Eur J Vasc Endovasc Surg (2025) 70, 153—218

CLINICAL PRACTICE GUIDELINE DOCUMENT

Editor's Choice – European Society for Vascular Surgery (ESVS) 2025 Clinical Practice Guidelines on the Management of Diseases of the Mesenteric and Renal Arteries and Veins☆

Mark J. Koelemay, (Chair) [*], Robert H. Geelkerken, (Co-chair), Jussi Kärkkäinen, (Co-chair), Nicola Leone, (Co-chair), George A. Antoniou, Jorg L. de Bruin, Alexander Gombert, Anders Gottsäter, Elena Iborra, Sonia Ronchey, Konstantinos Spanos, Jos C. van den Berg, Sabine Wipper, Frederico Bastos Gonçalves, Martin Björck, Raphael Coscas, Sandro Lepidi, Timothy A. Resch, Jean-Baptiste Ricco, Riikka Tulamo, Anders Wanhainen, Olivier Corcos, Thomas S. Huber, Alexander Oberhuber, Annika Reintam Blaser, Matti Tolonen [†]

Objective: The European Society for Vascular Surgery (ESVS) has developed clinical practice guidelines for the care of patients with diseases of the mesenteric and renal arteries and veins, in succession to the first 2017 guidelines, with the aim of assisting physicians and patients in selecting the best management strategy.

Methods: These guidelines are based on scientific evidence and expert opinion. By summarising and evaluating the best available evidence, recommendations for the diagnosis and treatment of patients have been formulated. The recommendations are graded according to the new ESVS clinical practice guidelines class of recommendation grading system, where the strength (class) of each recommendation is graded from I to III, and the letter A to C marks the level of evidence.

Results: A total of 102 recommendations have been issued on the management of chronic arterial mesenteric ischaemia, median arcuate ligament syndrome, acute arterial mesenteric ischaemia, non-occlusive mesenteric ischaemia, venous mesenteric thrombosis and ischaemia, occlusive disease of the renal arteries and veins, visceral artery aneurysms, and spontaneous isolated dissection of the visceral arteries.

Conclusion: These 2025 ESVS clinical practice guidelines provide comprehensive and up to date advice to physicians and patients on the management of diseases of the mesenteric and renal arteries and veins.

Available online 11 June 2025

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[†] **Affiliations:** Mark J. Koelemay (Amsterdam, the Netherlands), Robert H. Geelkerken (Enschede, the Netherlands), Jussi Kärkkäinen (Turku, Finland), Nicola Leone (Modena, Italy), Elena Iborra (Barcelona, Spain), George Antoniou (Manchester, UK), Jorg L. de Bruin (Rotterdam, the Netherlands), Alexander Gombert (Aachen, Germany), Anders Gottsäter (Lund, Sweden), Sonia Ronchey (Rome, Italy), Konstantinos Spanos (Larissa, Greece), Jos C. van den Berg (Lugano, Switzerland), Sabine Wipper (Innsbruck, Austria), Frederico Bastos Gonçalves (Lisbon, Portugal), Martin Björck (Uppsala, Sweden), Raphael Coscas (Boulogne-Billancourt, France), Sandro Lepidi (Trieste, Italy), Timothy A. Resch (Copenhagen, Denmark), Jean-Baptiste Ricco (Poitiers, France), Riikka Tulamo (Helsinki, Finland), Anders Wanhainen (Uppsala, Sweden), Thomas S. Huber (Gainesville, FL, USA), Annika Reintam Blaser (Tartu, Estonia), Olivier Corcos (Paris, France), Alexander Oberhuber (Münster, Germany), Matti Tolonen (Helsinki, Finland).

☆ For a full list of the authors' affiliations, please refer to Appendix B.

* Corresponding author. Department of Surgery, Amsterdam UMC, location AMC, Meibergdreef 9, 1105 AZ, Amsterdam, the Netherlands.

E-mail address: m.j.koelemaij@amsterdamumc.nl (Mark J. Koelemay).

https://doi.org/10.1016/j.ejvs.2025.06.010

ABBREVIATIONS

ACEI	Angiotensin converting enzyme inhibitor
ACS	Abdominal compartment syndrome
AF	Atrial fibrillation
AGREE	Appraisal of Guidelines for Research and Evaluation
AI	Artificial intelligence
AMI	Acute mesenteric ischaemia
AMVT	Acute mesenteric venous thrombosis
ARB	Angiotensin receptor blocker
BP	Blood pressure
CA	Coeliac artery
CAA	Coeliac artery aneurysm
CEUS	Contrast enhanced ultrasound
CI	Confidence interval
CMI	Chronic mesenteric ischaemia
CMVT	Chronic mesenteric venous thrombosis
CRP	C reactive protein
CT	Computed tomography
CTA	Computed tomography angiography
DAPT	Dual antiplatelet therapy
DOAC	Direct oral anticoagulant
DSA	Digital subtraction angiography
DUS	Duplex ultrasound
EDUS	Endoscopic duplex ultrasound
EDV	End diastolic velocity
eGFR	Estimated glomerular filtration rate
EJVES	European Journal of Vascular and Endovascular Surgery
ELISA	Enzyme linked immunosorbent assay
ePTFE	Expanded polytetrafluoroethylene
ESVS	European Society for Vascular Surgery
FMD	Fibromuscular dysplasia
GSC	Guideline Steering Committee
GWC	Guideline Writing Committee
HAA	Hepatic artery aneurysm
HR	Hazard ratio
IFABP	Intestinal fatty acid binding protein
IAH	Intra-abdominal hypertension
IAP	Intra-abdominal pressure
ICGFI	Indocyanine green fluorescent imaging
IMA	Inferior mesenteric artery
IMAD	Isolated mesenteric artery dissection
ISR	In stent re-stenosis
IVUS	Intravascular ultrasound
JAK2 (V617F)	Janus kinase 2 (gain of function substitute of valine to phenylalanine at position 617)
LDL-C	Low density lipoprotein cholesterol
LoE	Level of evidence
LRV	Left renal vein
MALS	Median arcuate ligament syndrome
MAOD	Mesenteric artery occlusive disease
MRA	Magnetic resonance angiography
MRI	Magnetic resonance imaging
MVT	Mesenteric venous thrombosis
NCS	Nutcracker syndrome
NOMI	Non-occlusive mesenteric ischaemia
NPV	Negative predictive value
OMT	Optimal medical therapy
OR	Odds ratio
PDAA	Pancreaticoduodenal artery aneurysm
PN	Parenteral nutrition
PSV	Peak systolic velocity
PTRA	Percutaneous transluminal renal angioplasty
PTRAS	Percutaneous transluminal renal artery angioplasty and stenting
PV	Portal vein
RAA	Renal artery aneurysm
RAAS	Renin angiotensin aldosterone system
RAD	Renal artery dissection
RAS	Renal artery stenosis
RCT	Randomised controlled trial
ROC	Receiver operating characteristic
ROMS	Retrograde open mesenteric stenting
RR	Relative risk or risk ratio
SAA	Splenic artery aneurysm
SAPT	Single antiplatelet therapy
SARS-CoV-2	Severe acute respiratory syndrome coronavirus 2
SBS	Short bowel syndrome
SMA	Superior mesenteric artery
SMAA	Superior mesenteric artery aneurysm
SMV	Superior mesenteric vein
SV	Splenic vein
SVS	Society for Vascular Surgery
ToE	Table of Evidence
VAA	Visceral artery aneurysm
VKA	Vitamin K antagonist
VLS	Visible light spectroscopy
VMI	Venous mesenteric ischaemia
VTE	Venous thromboembolic event

STUDY ACRONYMS

AMESI	Acute MESenteric Ischaemia
ASTRAL	Angioplasty and Stenting for Renal Artery Lesions
CARoSO	Coeliac Artery Release or Sham Operation
CORAL	Cardiovascular Outcomes in Renal Atherosclerotic Lesions
EMMA	Essai Multicentrique Medicaments *vs.* Angioplastie
HERCULES	The Safety and Effectiveness Study of the Herculink Elite Renal Stent to Treat Renal Artery Stenosis
ORIAMI	ORal Antibiotics in Acute Mesenteric Ischemia

WHAT IS NEW IN THE 2025 GUIDELINES?

Each section of the 2017 European Society for Vascular Surgery (ESVS) clinical practice guidelines on the management of diseases of the mesenteric arteries and veins has been updated and revised, and recommendations are phrased and graded according to the new ESVS system. The new 2025 ESVS clinical practice guidelines now also cover diseases of the renal arteries and veins. The 2017 ESVS clinical practice guidelines comprised 64 recommendations, of which 13 have been deleted or merged with a new recommendation. The current guidelines comprise 102 recommendations, of which 60 are new, 21 have been regraded or significantly rephrased with a changed meaning to some extent, and 21 have not been changed. Diseases of the mesenteric and renal vessels are uncommon, which hampers the collection and synthesis of high quality data, with medical and endovascular treatment of hypertension as the exceptions. As a consequence, two recommendations are supported by Level A evidence, 12 by Level B evidence, and 88 by Level C evidence or consensus.

Chronic mesenteric ischaemia (CMI) is characterised by postprandial pain, weight loss, and occlusive disease of the mesenteric arteries. The diagnosis is often delayed because of overlap with other abdominal causes for these symptoms. Unfortunately, the definitive diagnosis can only be established after revascularisation. Most recommendations for diagnosis and management of CMI are essentially the same as in the previous guidelines. The primary investigation is duplex ultrasound (DUS), followed by computed tomography angiography (CTA) to exclude other abdominal pathologies and plan revascularisation. There are still no reliable functional tests to refine the selection of patients for revascularisation, a treatment that is recommended in patients with multivessel occlusive disease. The superior mesenteric artery (SMA) remains the primary target for revascularisation, preferably by an endovascular procedure. An upgraded recommendation suggests that covered stents should be preferred over bare metal stents in the SMA. There is no evidence to suggest superiority of single over dual antiplatelet therapy (DAPT) following endovascular revascularisation, but a short course of DAPT should be considered.

Median arcuate ligament syndrome (MALS) remains a controversial diagnosis, yet accumulated evidence suggests that 70 — 80% of patients experience symptom relief after coeliac artery (CA) release, and this has resulted in several new recommendations. In a new Class I recommendation, investigation of patients suspected of MALS is recommended to be done at specialised centres. In these patients, DUS is recommended to be done during inspiration and expiration and, after exclusion of all other abdominal pathologies, patients with MALS may be considered for video assisted retroperitoneal endoscopic or laparoscopic CA release.

In Chapter 5, arterial acute mesenteric ischaemia (AMI) is addressed, a condition that is associated with a 50% mortality risk. The diagnosis of AMI is difficult and a clinical suspicion is important. The Guideline Writing Committee (GWC) issued a Class IIIb recommendation against the measurement of a single biomarker, including D dimer and lactate, to confirm or rule out the diagnosis, which is different from the previous guideline that recommended to use D dimer to rule out AMI. Prompt CTA in at least the arterial and venous phase is indicated, and a new Class I recommendation prompts the clinician to mention the suspicion of AMI in the referral for CTA, since this is associated with better diagnosis and outcomes. Patients with AMI are recommended to be treated in centres with 24/7 multidisciplinary services and experience in both open and endovascular revascularisation. Endovascular revascularisation should now be considered for both thrombotic and embolic occlusions followed by bowel resection, if applicable. Assessment of bowel viability remains difficult, but might be aided by quantitative indocyanine green fluorescent imaging, which is currently used in only 7% of practices, yet is endorsed in a new Class IIb recommendation.

The mortality rate of non-occlusive mesenteric ischaemia (NOMI) is approximately 70%. The diagnosis is hard to establish, and in Chapter 6 the Class IIIb recommendation against single biomarker measurement to confirm or rule out NOMI is repeated. Angiography as the historical reference standard has been replaced by CTA, and radiological characteristics suggestive of NOMI are detailed in the chapter. Timely recognition remains essential, and decompression laparotomy may be life saving in patients with abdominal compartment syndrome. A new Class IIb recommendation is issued to consider administration of vasodilators such as papaverine or prostaglandin in the SMA in patients with NOMI.

Venous mesenteric thrombosis is the least common cause of mesenteric ischaemia and is relevant for surgeons perhaps only in case of an acute abdomen. A thorough evaluation of permanent and transient risk factors for venous thrombosis is recommended, and unfractionated heparin is recommended as initial treatment until symptoms subside or the patient deteriorates. Since 2017, more evidence on the effectiveness of intravenous thrombolysis has accumulated and a new Class IIb recommendation that this intervention may be considered in deteriorating patients is now issued. The duration and form of anticoagulation after the incident thrombosis is discussed in more detail in Chapter 7.

The GWC was commissioned to integrate the chapter on renal arterial and venous occlusive disease in the current guidelines, and this is the new Chapter 8. Medical treatment of hypertension is the standard of care, also in patients with a severe renal artery stenosis. The GWC recommends against percutaneous renal artery angioplasty and stenting (PTRAS) in patients with an atherosclerotic renal artery stenosis and controlled hypertension and stable renal function. PTRAS should be considered in patients with resistant hypertension despite three or more drugs and a $>70\%$ renal artery stenosis due to atherosclerosis or fibromuscular dysplasia. Other relative indications for PTRAS are flash pulmonary oedema and severe decline in renal function with preserved kidney viability. Nutcracker

syndrome is also new in these guidelines and is recommended to be treated conservatively in patients with mild symptoms and by various invasive options in patients with severe symptoms.

In Chapter 9 on visceral artery aneurysm (VAA), recommendations for surveillance and thresholds for elective treatment are given for aneurysmal disease in each individual vascular territory, as opposed to the general recommendations in the 2017 ESVS guidelines. A limiting factor for evidence based recommendations is the lack of robust studies on the natural history and of follow up after endovascular or surgical interventions for VAA. An important difference from the previous guideline is that the recommended threshold for elective endovascular repair as first line therapy is now set at 3.0 cm for all VAAs, except aneurysms of the pancreaticoduodenal arcade, which seem to rupture at lower diameters and should therefore be considered for treatment at a diameter of 15 mm. It is as yet uncertain whether prophylactic treatment of CA stenosis can modulate the course of pancreaticoduodenal artery aneurysms. Recommendations on management of renal artery aneurysms are new in this chapter and are similar to recommendations for the mesenteric arteries.

Chapter 10 on spontaneous isolated dissections of the mesenteric arteries has been updated with the latest evidence, which has not led to significant differences in the recommendations. Conservative management remains the cornerstone for both symptomatic and asymptomatic isolated dissections, unless complications such as rupture (0.4%) or bowel ischaemia (2.1%) occur. Despite a lack of compelling evidence on its benefits, there is a Class IIa consensus recommendation to consider antiplatelet therapy for patients with this disorder. Spontaneous renal artery dissections are even less common and are also now covered in this chapter. The recommendations for management are similar to those for mesenteric artery dissections.

This guideline document concludes with Chapter 11 on unresolved issues and opportunities for research.

Table 1. New and updated recommendations included in the European Society for Vascular Surgery (ESVS) 2025 clinical practice guidelines on the management of diseases of the mesenteric and renal arteries and veins compared with the 2017 ESVS guidelines. Numbers correspond to the numbers of the recommendations in the current guideline document.

New Class I recommendations

7. It is recommended to assess the cardiovascular risk profile of patients with asymptomatic atherosclerotic mesenteric artery disease and to offer secondary prevention.
8. It is recommended to counsel patients with asymptomatic multivessel mesenteric artery occlusive disease regarding abdominal symptoms related to mesenteric ischaemia.
23. Computed tomography angiography is recommended as second level imaging after duplex ultrasound diagnosis of re-stenosis in patients after revascularisation for chronic mesenteric ischaemia.
27. Duplex ultrasound of the mesenteric arteries during inspiration and expiration is recommended as the first line examination in patients suspected of median arcuate ligament syndrome.
28. Multidisciplinary management at specialised centres is recommended for patients suspected of having median arcuate ligament syndrome.
32. Clinicians are recommended to mention the suspicion of acute mesenteric ischaemia in the referral for computed tomography angiography.
34. It is recommended to treat patients with acute mesenteric ischaemia in centres with 24/7 multidisciplinary services and experience in both open and endovascular mesenteric artery revascularisation.
57. Duplex ultrasound is recommended as the first line imaging investigation for patients with suspected renal artery stenosis.
59. Computed tomography angiography or magnetic resonance angiography are recommended over catheter angiography for the establishment of diagnosis and treatment planning for patients with suspected renal artery stenosis.
60. Pharmacological treatment of hypertension with the same blood pressure targets as in other hypertensive patients is recommended for patients with a renal artery stenosis with blood pressure $\geq$ 140/90 mmHg, provided these levels are without side effects: $<$ 130/80 mmHg in patients aged $<$ 65 years, $<$ 140/80 mmHg in patients aged 65 $-$ 79 years, and systolic blood pressure 140 $-$ 150 mmHg in patients $\geq$ 80 years of age.
61. Inhibitors of the renin–angiotensin system (angiotensin converting enzyme inhibitors [ACEIs] and angiotensin II receptor blockers [ARBs]) are recommended as first line therapy for patients with unilateral renal artery stenosis and hypertension. Calcium channel blockers and thiazide diuretics are recommended as first line additional therapies.
69. Conservative management is recommended for patients with an established diagnosis of nutcracker syndrome who have mild symptoms.
95. Conservative management, including blood pressure and pain control and bowel rest, is recommended as first line strategy for patients with asymptomatic or uncomplicated symptomatic isolated dissection of the superior mesenteric or coeliac arteries.
100. Conservative management with blood pressure control and antiplatelet therapy is recommended as first line strategy for patients with asymptomatic or uncomplicated symptomatic renal artery dissection.
101. Endovascular revascularisation is recommended for patients with symptomatic renal artery dissection and hypertension not responding to medical management.

Continued

Table 1-*continued*

New Class IIa recommendations

22. Duplex ultrasound of the mesenteric arteries should be considered as the first line examination for patients with recurrent symptoms after revascularisation for chronic mesenteric ischaemia.
24. Endovascular revascularisation as first line treatment should be considered for patients with a symptomatic re-stenosis after endovascular or open revascularisation for chronic mesenteric ischaemia.
26. Median arcuate ligament syndrome should be considered in highly selected patients with otherwise unexplained chronic abdominal symptoms and coeliac artery stenosis or occlusion due to external compression.
43. Non-occlusive mesenteric ischaemia should be suspected in patients in shock if computed tomography angiography with contrast enhancement in arterial and venous phases with ≤ 1 mm slices (and optional non-contrast CT run) shows signs of lack of bowel enhancement, small bowel thickening, intestinal pneumatosis, or gas in the portal vein, in the absence of a significant stenosis of the superior mesenteric artery.
54. Extended anticoagulation beyond six months with a vitamin K antagonist should be considered for patients with acute mesenteric vein thrombosis and transient risk factors for venous thrombosis.
64. Percutaneous transluminal renal artery angioplasty with bailout stenting should be considered for patients with resistant hypertension despite taking three or more antihypertensive drugs and a renal artery stenosis $>70\%$ due to fibromuscular dysplasia.
65. Percutaneous transluminal renal artery angioplasty with stenting should be considered for patients with flash pulmonary oedema due to acute fluid retention caused by acute kidney failure and atherosclerotic bilateral renal artery stenosis $>50\%$.
66. Percutaneous transluminal renal artery angioplasty with stenting should be considered for selected patients with resistant hypertension despite taking three or more antihypertensive drugs and an atherosclerotic renal artery stenosis $>70\%$.
68. Surgical reconstruction of the renal artery should be considered for patients with renal artery stenosis in whom revascularisation is clinically indicated when endovascular treatment is not possible or has failed.
70. Open surgical treatment with left renal vein transposition should be considered for patients with an established diagnosis of nutcracker syndrome who experience severe symptoms.
74. Patients with a mycotic visceral artery aneurysm should be treated urgently by open surgical means and antibiotics.
79. Individualised surveillance with duplex ultrasound or computed tomography angiography in case of inadequate sonographic image quality should be considered for patients after endovascular or open visceral artery aneurysm repair.
80. Endovascular or open surgical treatment should be considered for patients with an asymptomatic splenic artery aneurysm with a diameter ≥ 30 mm.
81. Surveillance with individualised imaging follow up should be considered for patients with an asymptomatic splenic artery aneurysm with a diameter <30 mm.
82. Endovascular or open surgical treatment should be considered for pregnant patients with an asymptomatic splenic artery aneurysm, regardless of aneurysm size.
84. Endovascular or open surgical treatment should be considered for patients with an asymptomatic hepatic artery aneurysm with a diameter ≥ 30 mm.
85. Surveillance with individualised imaging follow up should be considered for patients with an asymptomatic hepatic artery aneurysm with a diameter <30 mm.
86. Endovascular or open surgical treatment should be considered for patients with an asymptomatic coeliac artery aneurysm with a diameter ≥ 30 mm.
87. Surveillance with individualised imaging follow up should be considered for patients with an asymptomatic coeliac artery aneurysm with a diameter <30 mm.
88. Endovascular or open surgical treatment should be considered for patients with an asymptomatic pancreaticoduodenal artery aneurysm with a diameter ≥ 15 mm.
89. Endovascular or open surgical treatment should be considered for patients with an asymptomatic superior mesenteric artery aneurysm with a diameter ≥ 30 mm.
90. Surveillance with individualised imaging follow up should be considered for patients with an asymptomatic superior mesenteric artery aneurysm with a diameter <30 mm.
91. Endovascular or open surgical treatment should be considered for patients with a mycotic or symptomatic superior mesenteric artery aneurysm irrespective of the aneurysm diameter.
92. Endovascular or open surgical treatment should be considered for patients with an asymptomatic renal artery aneurysm with a diameter ≥ 30 mm.
93. Annual surveillance with imaging should be considered for patients with an asymptomatic renal artery aneurysm with a diameter <30 mm.
94. Endovascular or open treatment should be considered for pregnant patients with a renal artery aneurysm, regardless of aneurysm size.
102. Annual imaging follow up with duplex ultrasound to detect aneurysm formation or progressive stenosis should be considered for patients with asymptomatic and symptomatic renal artery dissection who were managed conservatively.

Continued

Table 1-*continued*

New Class IIb recommendations

12. Revascularisation may be considered in patients suspected of chronic mesenteric ischaemia due to isolated coeliac artery disease.
17. Bare metal stents may be considered for lesions longer than 25 mm and or those required to land beyond any significant side branches in patients with chronic mesenteric ischaemia requiring mesenteric artery stenting.
25. Endovascular revascularisation may be considered for selected patients with an asymptomatic > 70% re-stenosis after endovascular or open revascularisation for chronic mesenteric ischaemia.
29. Surgical intervention may be considered for selected patients with median arcuate ligament syndrome.
30. Laparoscopic or video assisted endoscopic retroperitoneal coeliac artery release may be considered the preferred treatment option for patients with median arcuate ligament syndrome undergoing surgical intervention.
39. Quantitative indocyanine green fluorescent imaging may be considered as an aid in the assessment of bowel viability in patients undergoing laparotomy or laparoscopy for acute mesenteric ischaemia.
45. Intra-arterial administration of vasodilators, such as papaverine or prostaglandin, in the superior mesenteric artery under protective heparinisation may be considered for patients with non-occlusive mesenteric ischaemia.
51. Endovascular venous thrombolysis and mechanical thrombectomy may be considered for patients with acute venous mesenteric ischaemia who deteriorate during anticoagulant therapy.
53. Anticoagulation for three to six months with a direct oral anticoagulant as an alternative to a vitamin K antagonist or low molecular weight heparin may be considered for all patients with acute mesenteric vein thrombosis.
55. Extended anticoagulation beyond six months with a direct oral anticoagulant as an alternative to a vitamin K antagonist may be considered for all patients with acute mesenteric vein thrombosis and transient risk factors for venous thrombosis.
58. Contrast enhanced ultrasound may be considered for patients with suspected renal artery stenosis and inconclusive duplex ultrasound, or with contraindications to computed tomographic or magnetic resonance angiography.
62. Treatment with angiotensin converting enzyme inhibitors and angiotensin II receptor blockers may be considered for patients with bilateral severe renal artery stenosis or with a renal artery stenosis in a single functioning kidney, if without side effects and with regular follow up of renal function and blood pressure.
67. Percutaneous transluminal renal artery angioplasty with stenting may be considered for patients with an atherosclerotic renal artery stenosis > 70% and severe decline in renal function and with preserved kidney viability.
71. Endovascular stenting may be considered for selected patients with an established diagnosis of nutcracker syndrome who experience severe symptoms.
75. Endovascular treatment and antibiotics may be considered as a non-curative treatment option for patients with a mycotic visceral artery aneurysm who are unfit for surgery.
83. Endovascular or open surgical treatment may be considered for female patients of childbearing age with an asymptomatic splenic artery aneurysm, regardless of aneurysm size.

New Class IIIa recommendations

63. Percutaneous transluminal renal artery angioplasty with or without stenting is not indicated in patients with any degree of renal artery stenosis and controlled hypertension and renal function.
97. Primary endovascular or open revascularisation is not indicated in patients with asymptomatic or uncomplicated symptomatic isolated dissection of the superior mesenteric or coeliac arteries.

Updated Class I recommendations

1. Chronic mesenteric ischaemia is recommended to be considered present in patients with chronic abdominal symptoms and > 50% stenosis of the superior mesenteric artery and > 50% in another mesenteric artery. *(class upgraded from IIb to I)*
10. Revascularisation of at least the superior mesenteric artery is recommended for patients with multivessel occlusive mesenteric artery disease and chronic mesenteric ischaemia *(rephrased and LoE downgraded from B to C)*.
14. An endovascular first approach as preferred treatment strategy is recommended for patients with chronic mesenteric ischaemia and suitable anatomy who need revascularisation *(rephrased)*.
41. Treatment with antibiotics is recommended for patients with acute mesenteric ischaemia *(rephrased and LoE downgraded from B to C)*.
44. Urgent decompression laparotomy is recommended for patients with suspected non-occlusive mesenteric ischaemia and concurrent abdominal compartment syndrome *(rephrased and LoE downgraded from B to C)*.
49. Contrast enhanced computed tomography scanning with imaging in the arterial and portal phases (and optional non-contrast CT run) is recommended for patients suspected of mesenteric vein thrombosis *(rephrased and LoE downgraded from B to C)*.
77. Endovascular rather than open repair is recommended for patients with a visceral artery aneurysm who are anatomically suitable *(rephrased and class upgraded from IIa to I)*.
98. Endovascular revascularisation is recommended for patients with symptomatic isolated dissection of the superior mesenteric or coeliac arteries not responding to medical management and with a suspicion of bowel ischaemia *(rephrased and class upgraded from IIa to I)*.

Continued

Table 1-*continued*

Updated Class IIa recommendations

11. Revascularisation should be considered in patients with chronic mesenteric ischaemia due to isolated superior mesenteric artery disease *(class upgraded from IIb to IIa, LoE downgraded from B to C)*.
16. Covered stents should be considered for lesions shorter than 25 mm in patients with chronic mesenteric ischaemia requiring mesenteric artery stenting, if no significant side branches are covered *(class upgraded from IIb to IIa, LoE upgraded from C to B)*.
19. Open revascularisation in patients with chronic mesenteric ischaemia should be considered in fit patients, after failed endovascular intervention, or when endovascular intervention is not feasible or is contraindicated *(LoE downgraded from B to C)*.
21. Follow up with clinical and imaging assessment should be considered for patients in the first year after endovascular or open revascularisation for chronic mesenteric ischaemia *(class upgraded from IIb to IIa)*.
35. Revascularisation first before bowel resection should be considered in patients with acute mesenteric ischaemia *(LoE downgraded from B to C)*.
40. Resection without primary reconstruction and second look laparotomy for definitive treatment should be considered in patients undergoing acute mesenteric revascularisation who need bowel resection *(rephrased, consensus)*.
96. Antiplatelet therapy should be considered for patients with asymptomatic or symptomatic isolated dissection of the superior mesenteric or coeliac arteries who are managed conservatively *(rephrased)*.

Updated Class IIb recommendations

2. Chronic mesenteric ischaemia may be considered to be present in patients with chronic abdominal symptoms and $>70\%$ isolated superior mesenteric artery stenosis *(rephrased and class downgraded from IIa to IIb)*.
20. Retrograde open mesenteric artery stenting may be considered in patients with chronic mesenteric ischaemia needing superior mesenteric artery revascularisation when an antegrade percutaneous approach is not feasible or successful *(class downgraded from IIa to IIb)*.
38. Retrograde open mesenteric artery stenting may be considered in patients with acute mesenteric ischaemia needing superior mesenteric artery revascularisation when percutaneous stenting is not possible *(rephrased and class downgraded from IIa to IIb)*.
76. Annual surveillance for the first three years and individualised thereafter, preferably with duplex ultrasound and otherwise computed tomography angiography, may be considered for patients with an asymptomatic visceral artery aneurysm with a diameter <30 mm and for pancreaticoduodenal artery aneurysms with a diameter <15 mm *(rephrased)*.

Updated Class IIIb recommendations

31. A single measurement of a biomarker such as lactate or D dimer to confirm or rule out the diagnosis in patients with a clinical suspicion of acute mesenteric ischaemia is not recommended *(rephrased and class changed to IIIb)*.
42. The single measurement of a biomarker such as lactate or D dimer to confirm or rule out the diagnosis in patients with a clinical suspicion of non-occlusive mesenteric ischaemia is not recommended *(rephrased and class changed to IIIb)*.

Unchanged Class I recommendations

3. It is recommended that patients with chronic mesenteric ischaemia are investigated and treated at specialised centres that can offer multidisciplinary assessment as well as both open and endovascular revascularisation.
4. Duplex ultrasound of the mesenteric arteries after a minimum of four hours fasting is recommended as the first line examination in patients suspected of chronic mesenteric ischaemia.
5. Computed tomography angiography is recommended as the preferred imaging for diagnosis, treatment planning, and to exclude other intra-abdominal pathology in patients suspected of chronic mesenteric ischaemia.
9. Revascularisation is recommended in patients with multivessel occlusive mesenteric artery disease and chronic mesenteric ischaemia.
15. Routine mesenteric artery stenting as opposed to plain balloon angioplasty is recommended in patients with chronic mesenteric ischaemia requiring endovascular treatment.
33. Urgent computed tomography angiography with contrast enhancement in arterial and venous phases with ≤ 1 mm slices (and optional non-contrast CT run) is recommended in patients suspected of acute mesenteric ischaemia, regardless of renal function.
46. Investigation for the presence of an intra-abdominal malignancy, inflammatory disease, myeloproliferative neoplasm, cytomegalovirus and SARS-CoV-2 infection, and chronic liver disease is recommended for patients with mesenteric venous thrombosis.
50. Anticoagulation with unfractionated or low molecular weight heparin as first line therapy is recommended for all patients with acute mesenteric vein thrombosis.
52. Anticoagulation for three to six months with a vitamin K antagonist or low molecular weight heparin is recommended for all patients with acute mesenteric vein thrombosis.
56. Indefinite anticoagulation is recommended for patients with idiopathic acute mesenteric vein thrombosis and for patients with permanent risk factors for venous thrombosis.
72. Computed tomography angiography is recommended for diagnosis, anatomical characterisation, and procedure planning for patients with the suspicion of a visceral artery aneurysm.
73. Urgent repair is recommended for patients with a symptomatic visceral artery aneurysm irrespective of size and location.
78. Arterial reconstruction over occlusion when technically possible is recommended for patients with a visceral artery aneurysm if the patient is not at high risk for surgery.

Continued

Table 1-*continued*

Unchanged Class IIa recommendations
36. Completion control with intra-operative angiography, duplex ultrasound, or Doppler ultrasound should be considered in patients undergoing open surgical mesenteric artery revascularisation.
37. Endovascular revascularisation should be considered as first line therapy in patients with acute mesenteric ischaemia due to thrombotic or embolic superior mesenteric artery occlusion.
47. Patients with recurrent mesenteric venous thrombosis and recurrent foetal loss should be investigated for antiphospholipid antibody syndrome.
99. Annual imaging follow up with duplex ultrasound to detect aneurysm formation or progressive stenosis should be considered for patients with isolated dissection of the superior mesenteric or coeliac arteries who were managed conservatively or with endovascular stenting.
Unchanged Class IIb recommendations
6. Contrast enhanced magnetic resonance angiography may be considered as an alternative to computed tomography angiography for diagnosis, treatment planning, and to exclude other intra-abdominal pathology in patients suspected of chronic mesenteric ischaemia.
18. A short course (minimum of one month) of dual antiplatelet therapy (aspirin and clopidogrel) may be considered in order to reduce the risk of stent thrombosis for patients who underwent endovascular revascularisation for atherosclerotic mesenteric artery disease.
48. Testing for thrombophilia may be considered in selected patients with acute venous mesenteric thrombosis who will discontinue anticoagulation treatment after three to six months.
Unchanged Class IIIb recommendations
13. It is not recommended to improve nutritional status and delay urgent revascularisation in patients with severe chronic mesenteric ischaemia (severe weight loss, diarrhoea, constant pain).

ESVS = European Society for Vascular Surgery; CT = computed tomography; LoE = level of evidence; SARS-CoV-2 = severe acute respiratory syndrome coronavirus 2.

1. METHODOLOGY

1.1. Purpose of the guidelines

In 2017, the European Society for Vascular Surgery (ESVS) published its first clinical practice guidelines for the care of patients with diseases of the mesenteric arteries and veins.[1] The current document is an update of the 2017 ESVS clinical practice guidelines and has been established by members of the Guideline Writing Committee (GWC), who are ESVS members or non-members with specific expertise in the field, based on scientific evidence and expert opinion. By summarising and evaluating the best available evidence, recommendations for the evaluation and treatment of patients have been formulated.

The recommendations represent the general knowledge at the time of writing the guidelines, but they can become outdated as technology and knowledge in this field may change. Although guidelines have the purpose of promoting a standard of care according to specialists in the field, under no circumstance should these guidelines be seen as the legal standard of care in all patients. As the word "guideline" implies, the document is a "guiding principle", but the care given to a single patient is always dependent on the individual patient (symptoms, comorbidities, age, patient preference, etc.), treatment setting (techniques available), and other factors.

The ESVS guidelines are published freely available or open access in the *European Journal of Vascular and Endovascular Surgery* (EJVES), and are available via the ESVS website and through the ESVS guidelines app.

1.2. Compliance with AGREE II standards

Appraisal of Guidelines for Research and Evaluation (AGREE) II reporting standards for assessing the quality and reporting of practice guidelines were adopted during preparation of the 2025 guidelines, and the AGREE II checklist is available as supplementary material in Appendix A.[2]

1.3. Guideline Writing Committee

Members of the GWC were selected by the GWC chair and the ESVS Guideline Steering Committee (GSC) to represent physicians involved in the management of patients with diseases of the mesenteric and renal arteries and veins. Members of the GWC have provided disclosure statements of all relationships that might be perceived as real or potential sources of conflicts of interest. These disclosure forms are kept on file at the ESVS headquarters. The GWC members did not receive financial support from any pharmaceutical, device, or surgical company associated with developing the current guidelines. The ESVS GSC was responsible for the endorsement process of this guideline. All experts involved in the GWC have approved the final document. All versions of the guideline were reviewed internally by the GWC, by members of the ESVS GSC, and by independent external reviewers, and were approved by the EJVES.

1.4. Methodology

1.4.1. Strategy. The GWC was convened on 12 September 2022 at online meetings during which the tasks of creating the guideline were distributed among the committee members. The GWC met online every month and in person on 19 — 20 June 2023 in Amsterdam (the Netherlands) to review the wording and grading of the recommendations. All recommendations were agreed upon by the GWC members after discussion. Between March 2024 and March 2025 the guidelines underwent three rounds of internal and external review, until the final document could be submitted for publication in May 2025.

1.4.2. Literature search and study selection. Members of the GWC performed a systematic literature search for these guidelines in MEDLINE (through PubMed), Embase, and the Cochrane Library to identify relevant studies in English and German published between 2015 and December 2023. Reference checking and handsearching by the GWC members added other relevant papers. After the first round of document review, additional literature searches were performed in May 2024 and January 2025. Papers were selected based on information provided in the title and abstract. Relevant articles published after the search date or in another language were included, but only if they were of paramount importance to these guidelines. Selection and assessment of the literature to support the recommendations followed the pyramid of evidence. Multiple randomised controlled trials (RCTs) or meta-analyses of RCTs were at the top, then single RCTs or large non-randomised studies (including meta-analyses of observational studies), small non-randomised interventional studies, observational studies, case series, and large prospective audits. Expert opinion was at the bottom of the pyramid. Single case reports, animal studies, and *in vitro* studies were excluded. The evidence used to support the recommendations is summarised in the Table of Evidence (ToE) (Appendix A).

1.4.3. Recommendations. The recommendations in these guidelines are graded according to the new ESVS clinical practice guidelines class of recommendation grading system.[3] The strength (class) of each recommendation is graded from I to III (Table 2), and the letters A — C mark the level of evidence (Table 3).

1.4.4. The patient's perspective. One of the aims of these guidelines is to optimise shared decision making. This requires access to unbiased evidence based information regarding all available treatment options, and a balanced discussion of risks, benefits, and potential consequences in a manner the patient understands, and that takes the preferences, needs, and values of the patient into account.[4,5] No patients were involved in the development of the current guidelines, which is obviously an omission, but would also be difficult for the wide range of rare diseases discussed in this document.

Table 2. European Society for Vascular Surgery (ESVS) clinical practice guidelines class of recommendation grading system.

Class	Definition	Wording
I	Evidence and or general agreement that a given treatment or procedure is beneficial, useful, effective	is or are recommended
II	Conflicting evidence and or divergence of opinion about the usefulness or effectiveness of the given treatment or procedure	
IIa	Weight of evidence or opinion is in favour of usefulness or effectiveness	should be considered
IIb	Usefulness or effectiveness is less well established by evidence or opinion	may be considered
III	Evidence or general agreement that a given treatment or procedure is not useful or effective, and in some cases may be harmful	
IIIa	The given treatment or procedure is not necessarily useful or effective	is or are not indicated
IIIb	The given treatment or procedure may be dangerous or harmful to patients	is or are not recommended

Table 3. European Society for Vascular Surgery (ESVS) clinical practice guidelines levels of evidence grading system.

Level of Evidence A	Data derived from multiple randomised trials or meta-analyses of randomised trials
Level of Evidence B	Data derived from a single randomised trial, high quality* non-randomised studies or meta-analysis of such studies
Level of Evidence C	Consensus opinion of experts, data from low quality† studies, or meta-analysis of such studies

* Large prospective, population based, observational, or registry studies.
† Small retrospective studies or case series.

2. TERMINOLOGY, DEFINITIONS, ANATOMY, AND PHYSIOLOGY

2.1. Terminology and definitions

Similar to the 2017 ESVS clinical practice guideline, well established terms were chosen over "anatomically more correct" terms. "Mesenteric" and not "splanchnic" is used to indicate the coeliac artery (CA), superior mesenteric artery (SMA), and inferior mesenteric artery (IMA), and ischaemia in that region. In the chapter on aneurysms, the mesenteric and renal arteries are taken together as the "visceral arteries".

Mesenteric artery occlusive disease (MAOD) can be divided according to four characteristics: (i) presence of symptoms; (ii) clinical presentation (acute, chronic, and acute on chronic ischaemia); (iii) vessel involvement (the identification and number of involved arteries or veins); and (iv) the cause of the stenosis (atherosclerosis, thrombosis, external compression, or congenital). The presence of symptoms caused by atherosclerotic disease of the mesenteric arteries presenting with chronic stenosis or occlusion is defined as chronic mesenteric ischaemia (CMI). Median arcuate ligament syndrome (MALS) is a symptomatic MAOD caused by external compression of the CA. Congenital malformations (such as midaortic syndrome or gut malrotation) and ischaemia due to bowel strangulation are not covered by these guidelines.

2.2. Anatomy and physiology

The mesenteric arteries include the three ventral branches of the abdominal aorta, supplying blood flow to the viscera. The anatomy of the mesenteric arteries shows great variability. The CA is the most proximal mesenteric artery, followed distally by the SMA and IMA. The CA originates from the aorta just below the diaphragm, often with an up to 2 cm parallel course with the aorta. The arterial blood supply of the bowel is characterised by extensive collateralisation, which varies considerably and requires individual assessment. The CA and SMA are connected by several pancreaticoduodenal arteries

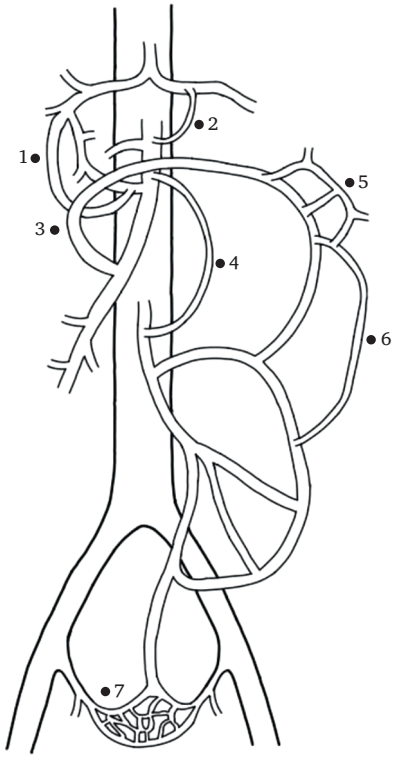

Figure 1. Diagram of the collateral arcades between the main digestive arterial trunks: the pancreaticoduodenal arcades described by Rio Branco (1) and Bühler (2) between the superior mesenteric artery and the coeliac trunk; the Riolan (3), Villemin (4), and Drummond (5) arcades, and marginal artery (6) between the inferior and superior mesenteric arteries (Griffith). The mesenteric artery ends with the superior rectal arteries, which are anastomosed to the internal iliac arteries via the middle rectal arteries (Sudeck) (7). Reproduced with permission of Dr Giovanni Francesco Baresi and Dr Lorenzo Leonardi (Modena, Italy).

described by Rio Branco and Bühler (Fig. 1) The SMA and IMA are anastomosed by the Riolan and Villemin arcades at the central mesenteric level, while the marginal arcade of Drummond is peripheral, close to the intestine. The macroscopic anastomoses between the three major vessels create a significant tolerance for central obstructions of the vessels: each one has the ability to supply the entire viscera via these anastomoses. The Griffith's point refers to the site of watershed anastomosis between the Riolan and the marginal artery of Drummond and is mostly situated in the region of the splenic flexure, and also located two thirds of the way along the transverse colon. The Sudeck point describes the junction in the rectosigmoid region, where arterial blood supply changes from the most distal branches of the IMA (superior rectal artery) to the branches of the internal iliac artery (inferior rectal artery), which is also an opportunity for collateral inflow to the mesenteric vascular bed. In case of reduced perfusion of the bowel, these two points are most prone to colonic ischaemia.

On a microscopic level, a capillary network in the submucosal layer provides blood supply to the villi and microvilli of the intestine, which is the most metabolically active layer. This network also includes anastomoses at the base of the villi, which allow re-direction of a compromised blood flow away from the mucosa while continuing to perfuse the muscularis and serosa, leading to ischaemic necrosis of the mucosa but preserving the integrity of the bowel basement layer, which may in fact be life saving.[6]

The viscera receive 10 — 20% of the cardiac output in a resting state and 35% in a postprandial state starting 10 — 30 minutes after a meal and continuing for up to three hours to meet the increased metabolic demand.[7,8] At the start of the meal the CA flow increases returning to baseline within an hour. The SMA flow increases after the meal, peaks in the first hour, and returns to baseline after two to three hours.[9] The arterial perfusion is regulated by various intrinsic and extrinsic factors with overlapping controls and restrictions, such as the autonomic nervous system, haemodynamic conditions, local metabolites, and hormones.[6] The abundant venous drainage of the viscera is via the inferior and superior mesenteric and splenic veins, culminating in the portal vein.

2.3. Pathologies

MAOD is predominantly caused by atherosclerosis affecting the ostia of the mesenteric arteries.[7,8] Compression of the CA by the median arcuate ligament is frequently observed and is most often asymptomatic. The less frequent causes of mesenteric vessel disease, such as non-occlusive mesenteric ischaemia (NOMI), venous mesenteric thrombosis, aneurysm, and isolated arterial dissection, are covered in other chapters of these guidelines. Other rare causes such as vasculitis (Takayasu's disease, Cogan's syndrome, Behçet's disease), trauma, and vascular malformations are not included in the present guidelines.

3. CHRONIC ARTERIAL MESENTERIC ISCHAEMIA

3.1. Epidemiology

The contemporary incidence and prevalence of CMI in populations is largely unknown. MAOD is mostly asymptomatic and its prevalence increases with age. A study in a Dutch referral centre estimated an annual CMI incidence of 9.2 (95% confidence interval [CI] 6.2 — 13.7) per 100 000 inhabitants.[10] Atherosclerotic CA and or SMA stenosis accounted for 7.3 (95% CI 4.6 — 11.3) per 100 000 inhabitants and MALS for 1.3 (95% CI 0.5 — 3.6) per 100 000 inhabitants. In the 358 patients included in that study, a definitive diagnosis of CMI was found in 12/75 patients (16%) with a single vessel stenosis, in 93/115 patients (81%) with stenosis of more than one vessel, and in 20/81 patients (25%) with CA compression.

Contemporary natural history studies are also scarce. In a retrospective study of 77 patients with a median follow up of 39 months, none of 24 patients with isolated SMA stenosis developed acute mesenteric ischaemia (AMI) or CMI.[11] Of the 53 patients with SMA stenosis plus CA and or IMA stenosis, five had AMI (most often triggered by another disease) and three had CMI during follow up.[11] The mortality rate of the entire cohort was 52% after a median of 23 months. The majority died from cancer and cardiovascular disease, and 5.2% from AMI. This study shows that the benefit of pre-

emptive intervention in patients with asymptomatic mesenteric artery disease is very uncertain.

3.2. Symptoms and signs

Symptomatic chronic MAOD is characterised by postprandial abdominal pain and, when severe, food aversion and weight loss in combination with insufficient bowel mucosal perfusion. The typical pain is midabdominal or epigastric and can be sharp or dull, usually beginning 20 — 30 minutes after eating and lasting one to two hours. The pain leads patients to restrict food intake, which may result in significant weight loss if not diagnosed and treated in a timely manner. Patients presenting with normal weight, vague abdominal discomfort, and multivessel atherosclerotic disease should be investigated for an alternative diagnosis. Considering the increasing global prevalence of obesity, weight loss over time is more important than weight at the time of presentation in order to establish the diagnosis (Table 4). The diagnosis of CMI is often delayed since patients may undergo extensive investigations for possible malignancy or are classified as having functional abdominal disorders. Atypical symptoms such as constant abdominal discomfort, nausea, vomiting, diarrhoea, or constipation may be present and may indicate severe CMI. CMI can also present as prolonged abdominal pain in the fasting state, which may be regarded as vascular abdominal rest pain. This condition is feared because it may turn into acute on chronic mesenteric ischaemia with a high risk of bowel gangrene. Earlier recognition and treatment of CMI can decrease the incidence of this often lethal complication.[12]

Clinical history and physical examination may reveal atherosclerosis in other locations but cannot be used to diagnose or exclude MAOD. Laboratory findings are not accurate enough to confirm or exclude CMI but may include anaemia, leucopenia, electrolyte abnormalities, and hypoalbuminaemia secondary to chronic malnutrition.

3.3. Diagnosis

The collateral pathways can compensate arterial supply when one or two of the mesenteric arteries are significantly stenotic or occluded, which prevents gastrointestinal ischaemia in single vessel disease. Although a stenosis in a single mesenteric artery is found in up to 18% of the general population, the diagnosis of mesenteric ischaemia is rare.[13,14] Symptoms of mesenteric ischaemia are usually not present until at least two of the three mesenteric arteries are significantly stenotic or occluded. Chronic single vessel atherosclerotic occlusion, however, can cause symptoms, especially if the SMA is involved and in the absence of a sufficient collateral network.[10] Terlouw *et al.* suggested that a >70% stenosis seems appropriate in patients with single vessel disease, considering that isolated 50 — 70% stenosis was associated with a 0 — 10% probability of CMI.[10] Moreover, different studies classifying stenosis grade as <50%, 50 — 70%, and >70% demonstrated that having a 50 — 70% stenosis of multiple mesenteric arteries was associated with a higher risk of CMI.[15,16] Female sex and age >65 years were also associated with a higher prevalence of CMI.

The presence of a mesenteric artery stenosis does not necessarily equate to actual ischaemia, since symptoms of CMI may overlap with other disorders affecting the pancreas, gallbladder, stomach, and duodenum, as well as functional disorders. This emphasises the need for a functional test that could actually prove ischaemia in the upper gastrointestinal tract and distinguish CMI from other conditions, specifically for single vessel disease. The number of reliable diagnostic tests, however, is limited. The highest diagnostic accuracy for CMI is achieved by combining clinical and radiological findings. A cohort of 436 patients suspected of CMI was used to develop a prediction model based on the abovementioned items.[15] This model includes computed tomography angiography (CTA) or magnetic resonance angiography (MRA) of the mesenteric arteries and allows for a low, intermediate, and high probability of CMI up to 21%, 46%, and >79%, respectively. The model was refined and validated in a prospective cohort study of 666 patients suspected of CMI in two referral centres,[17] with model scores ranging from 0 — 28 (Table 4). Its good discriminatory value makes the model a useful tool for clinical practice.

The associated probabilities of CMI (absolute CMI risk — %) according to the updated prediction model (Table 4) are as follows: total score 0—5 points, 19%; total score 6—18 points, 45%; and total score ≥19 points, 92%.

The definitive CMI diagnosis includes typical clinical symptoms, with weight loss being the most relevant, and radiological confirmation of chronic MAOD. A score ≥19 on the prediction chart corroborates the diagnosis as well as endoscopic findings of mucosal ischaemia.[17] The latter, and the relevance of a multidisciplinary assessment, are discussed in the following sections.

Table 4. Prediction model for chronic mesenteric ischaemia.[17]

Predictor	Scoring points
Weight loss (>5% of weight in kg)	
No	0
Yes	5
Cardiovascular disease	
No	0
Yes	2
Coeliac artery	
50—70% stenosis, vascular disease	4
50—70% stenosis, MALS	4
>70% stenosis, vascular disease	11
>70% stenosis, MALS	9
Superior mesenteric artery	
50—70% stenosis	4
>70% stenosis	10

MALS = median arcuate ligament syndrome.

Recommendation 1			Changed
Chronic mesenteric ischaemia is recommended to be considered present in patients with chronic abdominal symptoms and >50% stenosis of the superior mesenteric artery and >50% in another mesenteric artery.			
Class	**Level**	**References**	**ToE**
I	C	Terlouw *et al.* (2020),[10] Omran *et al.* (2022)[16]	

Recommendation 2			Changed
Chronic mesenteric ischaemia may be considered to be present in patients with chronic abdominal symptoms and >70% isolated superior mesenteric artery stenosis.			
Class	**Level**	**References**	**ToE**
IIb	C	Terlouw *et al.* (2020),[10] Omran *et al.* (2022)[16]	

3.4. Service standards

CMI is a rare condition that requires a multidisciplinary team approach. Diagnosis, selecting patients for revascularisation, and being able to offer different treatment options requires a team of dedicated and experienced specialists including, for instance, gastroenterologists, gastrointestinal surgeons, vascular surgeons, interventional radiologists, and dietary consultants. Members of the team may differ at each institution, prioritising the inclusion of physicians able to handle the entire spectrum of radiological, clinical, and surgical needs of this complex pathology.

Recommendation 3		Unchanged
It is recommended that patients with chronic mesenteric ischaemia are investigated and treated at specialised centres that can offer multidisciplinary assessment as well as both open and endovascular revascularisation.		
Class	**Level**	**Reference**
I	C	Consensus

3.5. Diagnostic imaging

3.5.1. Abdominal Xray. Plain abdominal Xray has no role in the diagnosis of CMI. Vascular calcification may indicate atherosclerotic disease, which can be a clue to the diagnosis, but a normal Xray does not exclude CMI.

3.5.2. Duplex ultrasound. Duplex ultrasound (DUS) is often used as the first imaging study to investigate the presence of MAOD.[1,18] The investigation may be technically challenging and requires a skilled operator. DUS has the benefit of dynamic assessment of flow through arterial segments. Peak systolic velocity (PSV) and end diastolic velocity (EDV) are used to quantify stenoses of the CA and SMA.[19] Most DUS validation studies with angiography as reference standard were done in patients after at least four hours of fasting,[20,21,23] and their results are displayed in Table 5.[20–23] The interpretation of flow velocities in the mesenteric arteries (e.g., SMA) should take into account both the presence of stenoses in the remaining mesenteric vessels (e.g., CA and IMA) and also the respiratory cycle, since the PSV and EDV in the CA and SMA are significantly higher during expiration than during inspiration.[23,24] However, the diagnostic accuracy of DUS expressed as area under the receiver operating characteristic (ROC) curve was similar for examinations during inspiration and expiration.[24]

Since the criteria for significant stenosis vary between these studies, ideally each individual centre should evaluate its own thresholds against a reference standard of CTA or digital subtraction angiography (DSA). A general cutoff value is hard to give because, as opposed to the carotid and peripheral arteries, there are just a few old validation studies. DUS findings should always be interpreted in conjunction with clinical symptoms and later confirmed with CTA to plan interventions.[25,26] The presence of a stent in the CA or SMA can generate artefacts and hinders quantification of the stenosis, and additional CTA may be required.[27,28]

Endoscopic DUS (EDUS) can obtain detailed images beyond the innermost lining of the digestive tract by combining endoscopy with DUS that is obtained with a

Table 5. Duplex criteria for mesenteric artery stenosis.

First author, publication year (patients – *n*)	SMA PSV ≥50% stenosis – cm/s	SMA PSV ≥70% stenosis – cm/s	CA PSV ≥50% stenosis – cm/s	CA PSV ≥70% stenosis – cm/s	SMA EDV ≥50% stenosis – cm/s	SMA EDV ≥70% stenosis – cm/s	CA EDV ≥50% stenosis – cm/s	CA EDV ≥70% stenosis – cm/s
Moneta, 1993[20] (*n* = 100)	–	275 (sens. 92%, spec. 96%)	–	200 (sens. 87%, spec. 80%)	–	–	–	–
Zwolak 1998[21] (*n* = 46)	300 (sens. 61%, spec. 100%)	–	200 (sens. 93%, spec. 94%)	–	45 (sens. 90%, spec. 91%)	–	55 (sens. 93%, spec. 100%)	–
AbuRahma 2012[22] (*n* = 150)	295 (sens. 87%, spec. 89%)	400 (sens. 72%, spec. 93%)	240 (sens. 87%, spec. 83%)	320 (sens. 80%, spec. 89%)	45 (sens. 79%, spec. 79%)	70 (sens. 65%, spec. 95%)	40 (sens. 84%, spec. 48%)	100 (sens. 58%, spec. 91%)
van Petersen 2013[23] (*n* = 324)	Expiration: ≥220 (sens. 84%, spec. 76%) Inspiration: ≥277 (sens. 68%, spec. 93%)	Expiration: ≥268 (sens. 75%, spec. 86%) Inspiration: ≥205 (sens. 78%, spec. 84%)	Expiration: ≥268 (sens. 66%, spec. 80%) Inspiration: ≥243 (sens. 68%, spec. 71%)	Expiration: ≥280 (sens. 66%, spec. 77%) Inspiration: ≥272 (sens. 72%, spec. 77%)	Expiration: ≥62 (sens. 75%, spec. 94%) Inspiration: ≥52 (sens. 76%, spec. 93%)	Expiration: ≥101 (sens. 74%, spec. 96%) Inspiration: ≥52 (sens. 78%, spec. 93%)	Expiration: ≥64 (sens. 78%, spec. 65%) Inspiration: ≥83 (sens. 53%, spec. 81%)	Expiration: ≥57 (sens. 83%, spec. 56%) Inspiration: ≥84 (sens. 66%, spec. 81%)

SMA = superior mesenteric artery; PSV = peak systolic velocity; CA = coeliac artery; EDV = end diastolic velocity; sens. = sensitivity; spec. = specificity.

curved linear array ultrasonic videoscope. The endoscope is placed along the lesser curvature of the stomach and a longitudinal view of the aorta, the origin of the CA, and the SMA can be assessed. In a validation study of 50 patients with CTA as reference standard, the sensitivity of EDUS to detect a $>70\%$ stenosis was 91% for the CA and 100% for the SMA, and for transabdominal DUS it was 81% for the CA and 92% for the SMA.[29] Whether there is a role in clinical practice for the more invasive EDUS has yet to be proven.

Recommendation 4			Unchanged
Duplex ultrasound of the mesenteric arteries after a minimum of four hours fasting is recommended as the first line examination in patients suspected of chronic mesenteric ischaemia.			
Class	**Level**	**References**	**ToE**
I	B	Moneta *et al.* (1993),[20] Zwolak *et al.* (1998),[21] AbuRahma *et al.* (2012),[22] van Petersen *et al.* (2013)[23]	

3.5.3. Computed tomography angiography. CTA is currently the imaging method of choice in patients with CMI. With 3D reformatting of arterial phase 0.75 mm axial slices that yields isotropic imaging, CTA can provide excellent reconstructions of the mesenteric arteries, and the sensitivity and specificity approaches 100% for the diagnosis of MAOD.[19,30] CTA also allows visualisation of the abdominal organs and helps to exclude other causes of chronic abdominal pain.

3D imaging allows static evaluation of the collateral circulation that develops in CMI. The CTA findings of localised narrowing, mostly only occurring in the expiratory phase, with post-stenotic dilatation and the absence of atherosclerotic plaques can support the diagnosis of MALS.[19] This always needs to be confirmed in the context of clinical symptoms and signs.[31]

3.5.4. Magnetic resonance angiography. Contrast enhanced MRA can also be used to assess the mesenteric arteries in patients suspected of CMI. The advantages of MRA are lack of radiation exposure and the possibility of flow measurements. MRA also allows assessment of the perivessel tissue and dynamic studies, e.g., during inspiration and expiration.[19] Most data on MRA come from small and old studies, which were not always performed in the target population. In a study of 26 patients, interobserver agreement for grading stenosis with contrast enhanced MRA was excellent for the CA ($\kappa = 0.90$) and SMA ($\kappa = 0.92$), and poor for the IMA ($\kappa = 0.48$).[32] In a series of 14 patients, the sensitivity and specificity of contrast enhanced MRA were both 100% for grading disease in the CA and SMA and 82% for the IMA compared with conventional angiography and surgery.[33] Others have also noted that IMA stenoses may be overestimated with MRA and that CTA shows these vessels better.[30] In a study of 52 patients, CTA had the highest accuracy to detect significant CA stenosis ($n = 12$) or SMA stenosis ($n = 2$) compared with DUS and MRA, although patients had DSA during endovascular abdominal aortic aneurysm repair, and not for CMI.[34]

In two pilot studies, patients with CMI had a significantly lower SMA and portal vein blood flow increase measured with MRA after a meal challenge compared with patients without CMI. This suggests that 4D flow magnetic resonance imaging (MRI) may be the future alternative to DUS and CTA because it can obtain anatomic and flow data from a single examination. Unfortunately, the studies were small and their results need to be confirmed in larger series.[35,36]

Recommendation 5			Unchanged
Computed tomography angiography is recommended as the preferred imaging for diagnosis, treatment planning, and to exclude other intra-abdominal pathology in patients suspected of chronic mesenteric ischaemia.			
Class	**Level**	**References**	**ToE**
I	C	van Dijk *et al.* (2017),[19] Hagspiel *et al.* (2015)[30]	

Recommendation 6			Unchanged
Contrast enhanced magnetic resonance angiography may be considered as an alternative to computed tomography angiography for diagnosis, treatment planning, and to exclude other intra-abdominal pathology in patients suspected of chronic mesenteric ischaemia.			
Class	**Level**	**References**	**ToE**
IIb	C	Hagspiel *et al.* (2015),[30] Carlos *et al.* (2001),[32] Meany *et al.* (1997)[33]	

3.5.5. Angiography. DSA is the historical reference standard for the diagnosis of MAOD. DSA should include selective injections into the CA, SMA, and IMA in a lateral projection. DSA provides high quality imaging of the mesenteric vasculature, demonstrates the dynamics of any collateral circulation, and allows endovascular procedures to be performed in the same session. Some centres use pressure gradient measurements across lesions to better define the clinical significance of mesenteric artery stenosis. Severe SMA stenosis has been defined as a mean arterial pressure gradient across the lesion of 20 mmHg[37] or a pressure gradient ratio after vasodilation of ≤ 0.8 achieved by intra-arterial injection of nitroglycerin 300 µg distal to the stenosis.[38] The pressure gradient can be measured using the pull back of a 4 F catheter or a 0.014" pressure wire landed beyond the lesion and a 5 or 6 F sheath with the tip positioned in the aorta. Both the absolute difference and the ratio of mesenteric artery pressure distal to the stenosis and aortic pressure should be evaluated. No recent studies have evaluated the role of angiography in the workup of CMI.

3.6. Proof of ischaemia (functional evaluation)

Proof of actual CMI can be obtained with three types of investigations, which currently have not demonstrated sufficient diagnostic accuracy and external validity in the workup of patients with CMI: (i) assessment of tissue or mucosal cell ischaemia during endoscopy; (ii) measurement of gastrointestinal blood flow; and (iii) measurement of ischaemia specific biomarkers.

Oesophagogastroduodenoscopy and colonoscopy are important examinations in the workup of patients with abdominal symptoms, especially to exclude malignancies. Gastric ulcer, ischaemic gastritis, duodenitis, and colitis may suggest the presence of CMI. On the other hand, a negative upper gastrointestinal endoscopy does not rule out CMI.[39] In a prospective study comparing 50 patients with and 26 without upper gastrointestinal ischaemia, no differences in histopathological changes were found in gastroduodenal biopsies.[40] Thus, endoscopic examinations are not included in the workup for CMI considering that none of the aforementioned findings are specific for CMI.

Visible light spectroscopy (VLS) can assess mucosal perfusion by measuring the intramucosal haemoglobin saturation. With saturation cutoff levels for ischaemia in the antrum (63%), duodenal bulb (62%), and descending duodenum (58%), VLS had a sensitivity of 90% and a specificity of 60% to detect CMI.[39] In a study of 32 patients with proven CMI and 38 controls, mean oxygen saturation measured in the stomach and duodenum with VLS was $67 \pm 9\%$ and $81 \pm 4\%$ ($p < .001$), respectively.[41] In a prospective study, mucosal oxygen saturation measured with VLS was lower in 23 patients with CMI compared with 37 patients without CMI. After enteral tube feeding, saturation increased in the descending duodenum; however, postprandial VLS did not add to the discriminative ability for the diagnosis of CMI.[42]

Serological markers for mesenteric ischaemia have rarely been studied in the context of CMI. Intestinal fatty acid binding protein (IFABP) is a marker of intestinal integrity and a potential biomarker for mesenteric ischaemia. In a pilot study in 24 patients with CMI, ischaemia was associated with IFABP increase after meals.[43] However, in another study IFABP levels were normal in patients with CMI and MALS.[44] In this study, plasma α-glutathione S-transferase levels were elevated compared with controls and had good discriminatory power at a threshold of 4 ng/mL. Levels decrease after revascularisation in patients with CMI and MALS.[44]

3.7. Treatment

3.7.1. Secondary prevention. Since MAOD is a manifestation of generalised atherosclerosis, it can be assumed that patients with asymptomatic MAOD are at increased risk of cardiovascular events. There are only a few natural history studies of patients with asymptomatic MAOD, and such patients have not been studied separately in trials on secondary prevention of cardiovascular events in patients with atherosclerosis. Yet it seems reasonable to offer patients with asymptomatic MAOD medical secondary prevention similar to patients with asymptomatic atherosclerotic disease.

Recommendation 7		New
It is recommended to assess the cardiovascular risk profile of patients with asymptomatic atherosclerotic mesenteric artery disease and to offer secondary prevention.		
Class	**Level**	**Reference**
I	C	Consensus

3.7.2. Asymptomatic mesenteric artery occlusive disease. Prophylactic revascularisation in patients with asymptomatic MAOD is controversial, and rarely performed, because the long term risk of becoming symptomatic is low, even in patients with multivessel disease whose prognosis is determined by events in other vascular territories, other comorbidities, and age.[11,46,47] Prophylactic revascularisation might be considered in highly selected cases with multivessel disease, balancing life expectancy along with the best cardiovascular risk management.[11,16] There is no evidence to suggest a benefit of follow up of patients with asymptomatic MAOD, who must however be counselled to attend a doctor when symptoms of mesenteric ischaemia develop.

Recommendation 8		New
It is recommended to counsel patients with asymptomatic multivessel mesenteric artery occlusive disease regarding abdominal symptoms related to mesenteric ischaemia.		
Class	**Level**	**Reference**
I	C	Consensus

3.7.3. Symptomatic mesenteric artery occlusive disease. Revascularisation is indicated in patients with MAOD with symptoms of CMI. Treatment goals are symptom relief, improved quality of life, return to normal weight, and prevention of AMI. There is no role for conservative treatment with chronic parenteral nutrition. There is no evidence of benefit for either enteral or parenteral feeding of patients with CMI before revascularisation, despite malnutrition and weight loss. Feeding in end stage CMI may provoke clinically significant complications such as transmural bowel infarction, but this has not been proven. Multivessel MAOD carries an AMI risk of up to 25% within one to six years, as demonstrated in 15 patients with three vessel disease.[45] In patients with single vessel disease, the probability of progression to CMI and AMI is very low.[11,46,47] Similarly, a prospective, single centre Dutch study demonstrated a higher probability of the diagnosis of CMI in patients with multivessel disease.[10] However, the prevalence of single vessel disease among those with a confirmed CMI diagnosis ranges between 12% and 40%. Up to 40% of atherosclerotic CMI is caused by a severe single vessel $>$ 70% SMA stenosis and 5.6% by a single vessel CA stenosis.[10] In a systematic review, the clinical success of mesenteric artery revascularisation, defined as improvement or disappearance of symptoms, was 87% (95% CI 80 – 92%).[47] Revascularisation has a higher clinical success rate in patients with multivessel disease (95.4%) compared with those with single vessel disease (69%).[10,50] Thus, revascularisation is recommended in patients with CMI and multivessel MAOD and should be considered for patients with single vessel disease.

Historically, the SMA is the primary target for revascularisation, and the CA or IMA are the second options.[48,49] Currently there is no compelling evidence comparing single and multivessel endovascular revascularisation in patients with symptomatic MAOD. Thus, the SMA remains

the main target in such patients, leaving the decision for multiple vessel revascularisation on a case by case basis.

Recommendation 9			Unchanged
Revascularisation is recommended in patients with multivessel occlusive mesenteric artery disease and chronic mesenteric ischaemia.			
Class	**Level**	**References**	**ToE**
I	B	Terlouw *et al.* (2020),[10] Omran *et al.* (2022),[16] Nana *et al.* (2023)[47]	

Recommendation 10			Changed
Revascularisation of at least the superior mesenteric artery is recommended for patients with multivessel occlusive mesenteric artery disease and chronic mesenteric ischaemia.			
Class	**Level**	**References**	**ToE**
I	C	Flis *et al.* (2016),[48] Wagenhäuser *et al.* (2017),[49] Malgor *et al.* (2010),[61] Ahanchi *et al.* (2013)[62]	

Recommendation 11			Changed
Revascularisation should be considered in patients with chronic mesenteric ischaemia due to isolated superior mesenteric artery disease.			
Class	**Level**	**References**	**ToE**
IIa	C	Terlouw *et al.* (2020),[10] van Dijk *et al.* (2018),[50] ter Steege *et al.* (2012)[51]	

Recommendation 12			New
Revascularisation may be considered in patients suspected of chronic mesenteric ischaemia due to isolated coeliac artery disease.			
Class	**Level**	**References**	**ToE**
IIb	C	Terlouw *et al.* (2020),[10] Omran *et al.* (2022),[16] Flis *et al.* (2016),[48] Wagenhäuser *et al.* (2017),[49] van Dijk *et al.* (2018)[50]	

Recommendation 13			Unchanged
It is not recommended to improve nutritional status and delay urgent revascularisation in patients with severe chronic mesenteric ischaemia (severe weight loss, diarrhoea, constant pain).			
Class	**Level**	**Reference**	
IIIb	C	Consensus	

3.8. Treatment strategies

Endovascular revascularisation and stenting have become the primary treatment modalities, reserving open surgical bypass for patients who are not suitable candidates or who fail endovascular therapy. Compared with open surgical bypass, endovascular revascularisation is associated with decreased morbidity and mortality rates, length of stay, and convalescence time.[52] A systematic review including 100 observational studies (18 726 patients) demonstrated that open surgery was associated with an increased risk of in hospital complications compared with endovascular revascularisation (relative risk [RR] 2.19, 95% CI 1.84 — 2.60), but not with 30 day mortality (RR 1.57, 95% CI 0.84 — 2.93).[52] Open surgery was associated with a lower risk of three year symptom recurrence (RR 0.47, 95% CI 0.34 — 0.66) and a similar three year survival (RR 0.96, 95% CI 0.86 — 1.07). A multicentre study including National Inpatient Sample patients treated for CMI showed that patients in the endovascular group, despite a higher comorbidity index, had a lower in hospital mortality rate (2.4% *vs.* 8.7%) and shorter admission, which incurred a cost saving of more than US$25 000 compared with patients in the open approach group.[53] Endovascular revascularisation demonstrated five year cost effectiveness over open revascularisation, providing higher quality adjusted life years.[54]

The long term results of endovascular revascularisation are scarcely reported. In a single centre series of 141 patients (86 CAs and 99 SMAs), the overall primary patency, primary assisted patency, and secondary patency rates after bare metal stenting were, respectively, 77.0%, 90.3%, and 98.3% at 12 months and 45.0%, 69.8%, and 93.6% at 60 months.[55] In a Danish national cohort of 245 patients with CMI treated between 2011 and 2015, 71% of patients had symptom relief and 7.3% had a re-intervention, which mainly occurred during the first year.[56] A recent meta-analysis estimated the re-intervention and symptom recurrence rates to be 26.0% (95% CI 17 — 37%) and 25.0% (95% CI 21 — 31%), respectively, after a mean follow up of 28 months.[47] Re-interventions were associated with a low mortality rate (3%), high risk of complications (27%), and excellent symptom improvement (92%).[57,58]

Concluding on the results of open and endovascular approaches for CMI, the latest published meta-analyses suffer from biases such as outdated publications and comparative studies including patients treated during the first decade of endovascular therapies. Still, endovascular mesenteric artery revascularisation carries a low morbidity and mortality rate, with good symptom relief, and thus should be considered as the first approach in patients with CMI. Irrespective of the revascularisation technique, patients with CMI should be counselled about the risk of symptom recurrence.

Recommendation 14			Changed
An endovascular first approach as preferred treatment strategy is recommended for patients with chronic mesenteric ischaemia and suitable anatomy who need revascularisation.			
Class	**Level**	**References**	**ToE**
I	B	Nana *et al.* (2023),[47] Alahdab *et al.* (2018),[52] Erben *et al.* (2018),[53] Bulut *et al.* (2017),[55] Altintas *et al.* (2021)[56]	

3.9. Endovascular revascularisation

The SMA is the primary target for revascularisation when possible. Revascularisation of the CA or IMA may be considered when the SMA is chronically occluded and not suitable for recanalisation. The characteristics of the SMA that affect treatment success include vessel diameter, extent of stenosis or occlusion, presence of tandem lesions, degree of calcification, and extent of collateralisation. Angioplasty and stenting are most effective for relatively short, focal SMA stenoses or occlusions with minimal to moderate calcification or thrombus. Plain balloon angioplasty has been largely replaced by primary stenting because of elastic recoil and re-stenosis, which limits its use for lesions at the ostium.[47] Although there are no prospective comparisons between angioplasty alone and primary stenting, in most centres routine mesenteric stenting is performed. In a systematic review of 1 224 endovascular procedures, only 90 (7.4%) were done by angioplasty alone.[47] The type of stent used is determined by the location of the lesion, the degree of calcification, and the tortuosity of the vessel. Balloon expandable stents are often used for ostial lesions, and self expanding stents for lesions distal to the ostium.[58] The meta-analysis of 1 224 patients with CMI who had endovascular treatment showed a technical success rate of 95% (95% CI 93 — 97%), an immediate symptom relief rate of 87% (95% CI 80 — 92%), and an early mortality rate of 2% (95% CI 1 — 4%).[47]

The indication for two vessel stenting (both CA and SMA) remains controversial, while most reports suggest that single vessel stenting may be sufficient. Two retrospective studies showed a non-significant trend towards lower risk of recurrence with two vessel stenting.[59,60] Another study, however, reported similar re-intervention rates at three years in patients treated with SMA stents (36%) compared with two vessel stenting (33%).[61] Two vessel mesenteric interventions may have a role in selected patients with severe gastric ischaemia who do not have a good collateral network between the CA and SMA, although a second intervention adds cost and potential risk of complications.

CA revascularisation may be considered in higher risk patients who have a failed SMA recanalisation or in those where an SMA intervention is felt to have a low chance of success, e.g., due to excessive calcification or long segment occlusion. Contraindications to endovascular treatment are external compression (MALS without surgical release of the median arcuate ligament) and small diameter distal vessels of either the CA or SMA.[63]

Evidence for the effectiveness of angioplasty and stenting of the IMA is limited to three case series comprising 17 patients with successful results.[64–67] Sizing of IMA stents can be an issue, since the post-stenotic dilatation that is often seen does not represent the true vessel diameter. Oversizing the stent too much with respect to the original ostial diameter increases the risk of rupture.

Recommendation 15			Unchanged
Routine mesenteric artery stenting as opposed to plain balloon angioplasty is recommended in patients with chronic mesenteric ischaemia requiring endovascular treatment.			
Class	**Level**	**Reference**	**ToE**
I	C	Nana *et al.* (2023)[47]	

3.9.1. Covered stents or bare metal stents. The benefit of using covered stents over bare metal stents to treat the SMA has been a matter of debate, with conflicting findings in two retrospective studies. In a study of 225 patients, covered stents were associated with a lower re-stenosis rate, a lower clinical symptom recurrence rate, and fewer re-interventions compared with bare metal stents (for all endpoints, some 8% *vs.* 50%).[67] On the other hand, in a series of 150 patients the three year primary patency of bare metal stents in the CA was 66% and in the SMA was 69%.[68] A Dutch RCT compared the outcomes of covered stents ($n = 47$) with bare metal stents ($n = 47$) in the CA or SMA in patients with atherosclerotic CMI.[69] Eligible patients had a target lesion length shorter than 25 mm, and those requiring coverage of a relevant side branch to adequately treat the lesion were excluded, as well as patients with previous stenting of the target vessel. In this RCT, the 24 month primary patency rate of covered stents was 80.8% *vs.* 49.1% for bare metal stents ($p < .001$). This study also reported a lower re-intervention rate, lower stent fracture rate, and similar safety profile, with an equally high technical success rate for covered stents.[69] It needs to be emphasised that the primary patency of bare metal stents in this study was lower than in previous studies and that extrapolation to other bare metal stent designs is not possible on a one to one basis. Still, covered stents should be the preferred stent for mesenteric artery stenting in CMI in short lesions that do not require coverage of any relevant side branches.

Recommendation 16			Changed
Covered stents should be considered for lesions shorter than 25 mm in patients with chronic mesenteric ischaemia requiring mesenteric artery stenting, if no significant side branches are covered.			
Class	**Level**	**References**	**ToE**
IIa	B	Oderich *et al.* (2013),[67] Terlouw *et al.* (2024)[69]	

Recommendation 17			New
Bare metal stents may be considered for lesions longer than 25 mm and or those required to land beyond any significant side branches in patients with chronic mesenteric ischaemia requiring mesenteric artery stenting.			
Class	**Level**	**Reference**	
IIb	C	Consensus	

3.10. Antithrombotic therapy

The ESVS 2023 clinical practice guidelines on antithrombotic therapy for vascular diseases recommend secondary prevention with single antiplatelet therapy (SAPT) for asymptomatic and symptomatic patients with atherosclerotic MAOD.[70] A consensus recommendation was issued regarding short term (one to six months) dual antiplatelet therapy (DAPT) following mesenteric revascularisation given an acceptable bleeding risk. This recommendation is an extrapolation of experience from peripheral and coronary artery disease because there were no data specific to patients with CMI. However, in an update of the literature, there was very low evidence to suggest that any of aspirin, clopidogrel, or DAPT was associated with a lower risk of distal embolisation or stent thrombosis.[71] The current literature does not support the use of DAPT or direct oral anticoagulant (DOAC), and no preference for aspirin or clopidogrel can be postulated.[71] In an umbrella review and meta-analysis of patients with peripheral arterial disease, DAPT following endovascular intervention conferred no clinical benefit.[72] Meta-regression comparing SAPT and DAPT after endovascular treatment of CMI showed similar outcomes, irrespective of the type of antithrombotic treatment (either SAPT or DAPT), in terms of technical success (98% *vs.* 94%), immediate symptom relief (82% *vs.* 91%), early death (3% *vs.* 2%), and morbidity (16% *vs.* 8%).[47]

Recommendation 18			Unchanged
A short course (minimum of one month) of dual antiplatelet therapy (aspirin and clopidogrel) may be considered to reduce the risk of stent thrombosis for patients who underwent endovascular revascularisation for atherosclerotic mesenteric artery disease.			
Class	**Level**	**Reference**	
IIb	C	Consensus	

3.11. Open surgery

Open surgical bypass is mainly used in patients who have MAOD that is unfavourable for endovascular treatment and or after failed endovascular intervention, and or who have recurrent in stent stenosis or occlusion. The early mortality rate following mesenteric bypass surgery has been reported as $< 4\%$.[73,74] However, in a meta-analysis the crude early mortality rate was estimated to be 5.5% (95% CI 4.8 — 6.3%), which according to the authors may be an underestimate due to publication bias.[52] Peri-operative mortality in the USA decreased from 12% to 5% during the first decade of the present century.[75] Mortality declined from 21% to 9% in patients treated with open surgery in the USA between 2000 and 2012.[12] During the same period, the volume of open surgery dropped significantly, and revascularisation shifted towards endovascular treatment, especially in comorbid patients.[53] The current mortality rates probably refer to highly selected fit patients, although from the literature it is not clear how fit is defined.

The SMA is the first target for revascularisation. A retrospective study of 27 patients demonstrated good results of retrograde iliac—SMA bypass even in cases with CA obstruction.[48] In a retrospective study of 100 patients, isolated open SMA revascularisation had higher primary and secondary patency rates compared with either isolated CA or combined CA and SMA revascularisation.[49] These results might be related to the greater invasiveness and challenges of open CA repair or to the increased competitive flow in two vessel revascularisation. Most small older retrospective cohort studies reported improved clinical outcome, free of symptoms, after open two vessel revascularisation.

Recommendation 19			Changed
Open revascularisation in patients with chronic mesenteric ischaemia should be considered in fit patients, after failed endovascular intervention, or when endovascular intervention is not feasible or is contraindicated.			
Class	**Level**	**References**	**ToE**
IIa	C	Flis *et al.* (2016),[48] Oderich *et al.* (2009),[73] Lejay *et al.* (2015),[74] Blauw *et al.* (2017)[79]	

3.11.1. Pre-operative evaluation prior to open surgery. Pre-operative evaluation should assess surgical risk, nutritional status, and anatomical factors that affect the choice of reconstruction. A comprehensive evaluation of cardiac, pulmonary, and renal function is needed because these procedures are usually required in patients with multiple comorbidities. Routine cardiac catheterisation is unnecessary, and cardiac evaluation and peri-operative management is guided by the recommendations of the European Society of Cardiology for patients undergoing major non-cardiac surgery.[76] There is no evidence to suggest that pre-operative oral or parenteral nutrition can improve outcomes of open mesenteric revascularisation or is potentially harmful in end stage CMI. However, it is relevant to assess nutritional status considering that a small retrospective cohort study found a 30 day mortality of 0% (0/16) in non-malnourished patients with CMI as opposed to 26% (10/38) in malnourished patients with CMI.[77]

3.11.2. Open surgical techniques. Recommendations on how to perform open surgical mesenteric artery repair rely on small cohort studies with substantial biases and expert opinion. Surgical planning involves selection of the approach (transperitoneal *vs.* retroperitoneal), conduit (vein *vs.* prosthetic), graft configuration (antegrade *vs.* retrograde), source of inflow (aortic *vs.* iliac), and number of vessels to be revascularised (single *vs.* multiple). The type of open reconstruction should be tailored to the anatomy and the patient's clinical risk assessment.[78,79]

3.11.2.1. Antegrade bypass. The distal thoracic or supraceliac aorta is selected as inflow source if it is spared from severe atherosclerotic disease. This graft configuration may offer a potential haemodynamic advantage by avoiding the potential risk of graft kinking that can occur with retrograde grafts.[80] Antegrade bypass can be performed using lateral or partially occluding aortic clamping, which maintains a minimum perfusion. For a heavily calcified aorta hindering

clamping, a hybrid approach combining a covered stent and prosthetic graft is an alternative.[81]

3.11.2.2. Retrograde bypass. A retrograde bypass with take off from the infrarenal aorta, a previous aortic graft, or the iliac arteries may be preferred if the supracoeliac aorta is diseased or in patients with limited physiological reserve. Most retrograde reconstructions deal with a single vessel, typically the SMA, but reconstruction of the CA or common hepatic artery can also be achieved by tunnelling the graft retroperitoneally or via the transverse mesocolon. Anastomosis at iliac artery level has the advantage of avoiding aortic cross clamping. As the graft assumes a C shaped configuration, it is important to avoid graft elongation, angulation, or kinking specifically when the bowel is repositioned in the abdominal cavity. It is important to cover a prosthetic graft to avoid contact with the intestines. Reports suggest that retrograde grafts perform as well as antegrade grafts.[48,81] A modified retrograde bypass technique to the SMA consisting of an autologous femoral or greater saphenous vein graft directly tunnelled under the small bowel avoids the C loop and prevents graft kinking.[82] A different technique has been described with a looped graft tunnelled behind the left renal pedicle with end to side anastomosis to the aorta or iliac artery.[83]

3.11.2.3. Endarterectomy. Transaortic endarterectomy is rarely used but can be considered in patients with bacterial contamination or perforated bowel, previous abdominal irradiation, extensive abdominal wall hernias, or other hostile conditions. Direct mesenteric endarterectomy, with or without stenting of proximal disease, is an alternative in some cases of coral reef aorta at the level of visceral artery ostia.

3.11.2.4. Superior mesenteric artery transposition. SMA transposition can be used in cases with a short proximal lesion and a healthy infrarenal aorta.

3.11.2.5. Retrograde open mesenteric stenting. Retrograde open mesenteric stenting (ROMS) combines open surgical exposure of the SMA and retrograde introduction of the stent in the SMA via direct puncture. ROMS is mostly described in the setting of AMI but may also be applied in patients with CMI when percutaneous stenting via the aorta is not feasible or successful. A detailed description and appraisal of ROMS is presented in Section 5.5.5.

Recommendation 20			Changed
Retrograde open mesenteric artery stenting may be considered in patients with chronic mesenteric ischaemia needing superior mesenteric artery revascularisation when an antegrade percutaneous approach is not feasible or successful.			
Class	**Level**	**References**	**ToE**
IIb	C	Sénémaud *et al.* (2021),[85] Crillo-Penn *et al.* (2023)[87]	

3.12. Follow up

Most studies on revascularisation of patients with CMI report 30 day and in hospital technical success, morbidity, and mortality rates. Unfortunately, longer follow up is often less well documented and incomplete and is focused on patency and re-interventions, which hampers assessment of long term outcomes, in particular clinical success. Some studies report outcomes five years after treatment, but there is little information beyond this time.[55]

It is unknown whether routine imaging follow up after mesenteric revascularisation is beneficial because uncertainty exists as to the best management of an asymptomatic re-stenosis. The 2021 Society for Vascular Surgery (SVS) guidelines recommend biannual follow up for the first two years after revascularisation and annually thereafter as good clinical practice, in the absence of scientific evidence.[88] Most patients with recurrent symptoms present during the first year after revascularisation.[25,89] Clinical follow up alone may be considered, with referral of recurrent symptom patients for DUS. Several studies have shown that the PSV is higher in stented than in native mesenteric arteries.[27,90] Various small studies have assessed DUS for the detection of $\geq$ 70% SMA in stent re-stenosis (ISR) and found optimal PSV cutoff values between 300 cm/s and 400 cm/s.[27,91–93] PSV thresholds for (clinically) significant ISR in the CA ranged between 289 cm/s[27] and 440 cm/s.[28] Interestingly, there is not always a direct correlation between ISR detected by DUS and symptom recurrence. In one study, an in stent PSV of 341 cm/s had only 80% sensitivity and 52% specificity to detect symptomatic SMA ISR requiring re-intervention.[28] This finding was corroborated in a study using a PSV threshold of 350 cm/s for SMA ISR, which had no predictive value at all for symptom recurrence.[89] In this study, CTA correlated better with DSA than DUS and therefore the authors recommended the use of multimodal imaging to detect ISR. In a retrospective study, 65/259 patients (25%) treated for CMI had recurrent symptoms or weight loss within one year, more often after endovascular (29%) than open revascularisation (13%).[25] In this study, re-interventions were done in patients with recurrent symptoms and severe re-stenosis on DUS. Of note, DSA could not confirm DUS findings in 24% of the patients in this study, which underscores the relevance of CTA as second level imaging if re-intervention is considered.

Although the ISR grade requiring re-intervention is not yet defined, it seems appropriate to re-intervene in patients with a symptomatic re-stenosis given that all other possible diagnoses have been excluded. Re-intervention is preferably done with an endovascular first approach.[57,94–96] In a systematic review of outcomes after endovascular revascularisation for CMI, symptom recurrence occurred in 25% (95% CI 21 — 31%) of patients after a mean follow up of 28 months.[47] The re-intervention rate was 26% (95% CI 17 — 37%), and was by endovascular means in 278 of 316 cases and by open conversion in 38 cases.

Several studies reported acceptable technical success of endovascular re-intervention after both primary endovascular and open revascularisation.[57,94,95] Morbidity and perioperative mortality rates were acceptable, and were lower with an endovascular compared with an open approach.[94,95]

Taken together, there is a lack of good follow up studies, which hinders specific recommendations about modalities and timing, leading the GWC to issue a recommendation that

clinical and radiological assessment within the first year after revascularisation is mandatory, also because recurrent symptoms may at first not be recognised by the patient. CTA is required to confirm a re-stenosis found with DUS, and a symptomatic re-stenosis should be treated with an endovascular intervention. Treatment of asymptomatic severe re-stenosis may be considered when the primary intervention was done for severe CMI if it can be treated with a low risk endovascular intervention, after discussion with the patient.

Recommendation 21 — Changed

Follow up with clinical and imaging assessment should be considered for patients in the first year after endovascular or open revascularisation for chronic mesenteric ischaemia.

Class	Level	References	ToE
IIa	C	Andraska *et al.* (2022),[25] Barnes *et al.* (2020),[28] Tallarita *et al.* (2011),[57] Sorour *et al.* (2024),[89] Green *et al.* (2021)[93]	

Recommendation 22 — New

Duplex ultrasound of the mesenteric arteries should be considered as first line examination for patients with recurrent symptoms after revascularisation for chronic mesenteric ischaemia.

Class	Level	References	ToE
IIa	C	Soult *et al.* (2016),[27] Barnes *et al.* (2020),[28] AbuRahma *et al.* (2012),[91] Pamulapati *et al.* (2021),[92] Green *et al.* (2021)[93]	

Recommendation 23 — New

Computed tomography angiography is recommended as second level imaging after duplex ultrasound diagnosis of re-stenosis in patients after revascularisation for chronic mesenteric ischaemia.

Class	Level	References	ToE
I	C	Andraska *et al.* (2022),[25] Barnes *et al.* (2020),[28] Sorour *et al.* (2024),[89] Green *et al.* (2021)[93]	

Recommendation 24 — New

Endovascular revascularisation as first line treatment should be considered for patients with a symptomatic re-stenosis after endovascular or open revascularisation for chronic mesenteric ischaemia.

Class	Level	References	ToE
IIa	C	Nana *et al.* (2023),[47] Tallarita *et al.* (2011),[57] Kanamori *et al.* (2014),[94] Zhou *et al.* (2019)[96]	

Recommendation 25 — New

Endovascular revascularisation may be considered for selected patients with an asymptomatic >70% re-stenosis after endovascular or open revascularisation for chronic mesenteric ischaemia.

Class	Level	Reference
IIb	C	Consensus

4. MEDIAN ARCUATE LIGAMENT SYNDROME

4.1. Diagnosis

The second most common cause of single vessel MAOD is external compression of the CA by the median arcuate ligament that crosses over the aorta at the level of the first lumbar vertebral body, above the CA origin (Fig. 2).[10] The muscular or fibrous diaphragmatic band compresses the CA, most pronounced during expiration, in patients who have a relatively low insertion of the diaphragm and or a relatively high CA origin. This configuration is frequently asymptomatic but may cause postprandial abdominal pain, which is known as MALS.

MALS is a controversial diagnosis that can only be established by exclusion of all other possible causes for abdominal pain. MALS must be agreed upon by a multidisciplinary team and shares most of the aforementioned recommendations in

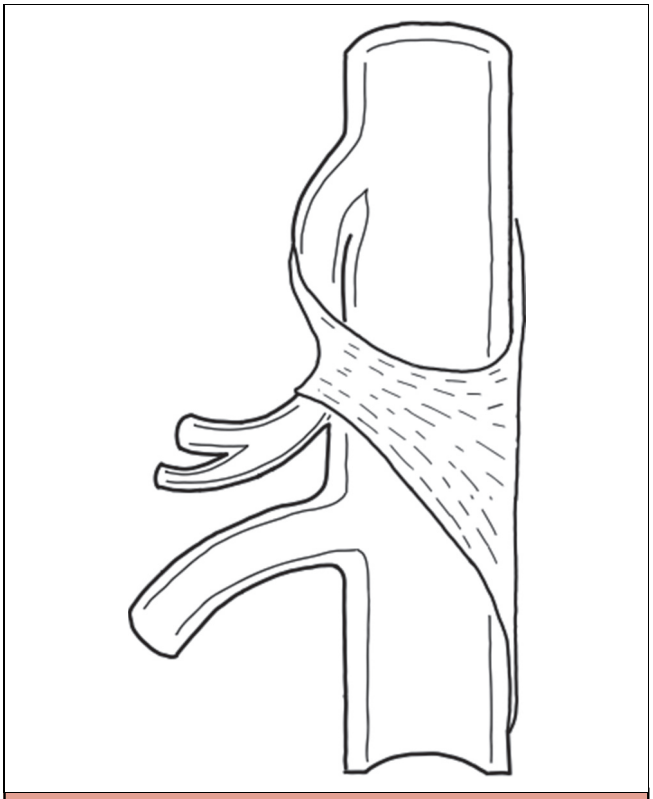

Figure 2. Mesenteric artery occlusive disease caused by external compression of the coeliac artery by the arcuate ligament. Reproduced with permission of Dr Giovanni Francesco Baresi and Dr Lorenzo Leonardi (Modena, Italy).

Chapter 3 regarding the diagnosis of CMI, with DUS, CTA, and angiography being the most relevant. Thus, only patients with otherwise unexplained chronic abdominal symptoms and a CA stenosis or occlusion by external compression may be considered as having MALS. Typical patients are 40 years old at intervention and 73% are female.[97,98] Although it is a common observation that the diagnosis of MALS is delayed, there is a widely shared experience that most of these patients had their first symptoms as teenagers, which suggests that CA compression exists during the teenage growth spurt. This observation is important since timely recognition and treatment is associated with better outcomes.[98] Inclusion of a psychologist or psychiatrist within the multidisciplinary team seems relevant considering the 25% prevalence of mental disorders in patients with MALS, and their association with worse quality of life outcomes.[99] In a currently recruiting RCT, patients with MALS are being allocated 1:1 to video assisted retroperitoneal endoscopic median arcuate ligament release or sham operation (CARoSO study; ClinicalTrials.gov ID NCT05468580).[100] Eligible patients must fulfil a multidisciplinary consensus diagnosis of MALS according to the following criteria: (1) postprandial pain associated with at least one of the following; dietary modification, unexplained weight loss, unexplained diarrhoea; (2) external compression of the CA causing a stenosis of $\geq$ 70% demonstrated by two imaging techniques (DUS, MRA, CTA, or DSA), which includes at least an inspiration and expiration CTA with 1 mm sections; and (3) abdominal ultrasound and oesophagogastroduodenoscopy with no abnormalities.

The GWC recommends use of the same criteria for establishing the diagnosis in current clinical practice.

Recommendation 26 New

Median arcuate ligament syndrome should be considered in highly selected* patients with otherwise unexplained chronic abdominal symptoms and coeliac artery stenosis or occlusion due to external compression.

Class	Level	References	ToE
IIa	C	Metz *et al.* (2022),[97] Jimenez *et al.* (2012)[101]	

* Postprandial pain associated with at least one of the following; dietary modification, unexplained weight loss, unexplained diarrhoea; external compression of the CA causing a $\geq$ 70% stenosis demonstrated by two imaging techniques (DUS, MRA, CTA, or DSA), which includes at least an inspiration and expiration CTA with 1 mm sections; and abdominal ultrasound and oesophagogastroduodenoscopy with no abnormalities.

Recommendation 27 New

Duplex ultrasound of the mesenteric arteries during inspiration and expiration is recommended as the first line examination in patients suspected of median arcuate ligament syndrome.

Class	Level	Reference	ToE
I	C	van Petersen *et al.* (2013)[23]	

Recommendation 28 New

Multidisciplinary management at specialised centres is recommended for patients suspected of having median arcuate ligament syndrome.

Class	Level	Reference
I	C	Consensus

4.2. Invasive treatment

The pathophysiology of MALS is most likely related to ischaemia. The arcuate ligament can be released by means of an open approach, laparoscopic access, as well as by a video assisted retroperitoneal endoscopic approach.[102] In two prospective cohort studies, normalisation of VLS or tonometry after CA release was associated with a long term benefit in 80 — 92% of cases.[103,104] However, patients with MALS with mesenteric collateral arterial circulation (at least one collateral clearly visible on non-selective DSA) were less likely to benefit from CA release than patients without an extensive collateral circulation.[105] In a review of 20 case series comprising 400 patients with MALS, immediate symptom improvement was reported in 85% of subjects after open or laparoscopic CA release, and symptoms recurred in 19 (6.8%) and seven (5.7%) patients after open and laparoscopic surgery, respectively.[101] A systematic review included 38 studies comprising 880 adult patients and six studies on 195 paediatric patients with MALS.[97] Durable symptom relief following open, laparoscopic, or video assisted retroperitoneal endoscopic CA release was $>$ 70% both in adults and children. In the few studies that measured quality of life, improvement was reported after surgery. The quality of the evidence was graded as low because of the heterogeneity of study populations and outcome assessments.

In an international, multicentre, retrospective observational study including 516 patients with MALS, freedom from treatment failure at three year follow up was 62% after laparoscopic CA release and 44% after open release.[98] An interesting observation in this study was the association between absence of symptom relief after coeliac plexus blockade and treatment failure (hazard ratio [HR] 2.18, 95% CI 1.00 — 4.72) in the 71 patients who had such blockade pre-operatively.[98] In a case series of 29 patients with MALS, 19 benefitted from laparoscopic CA release.[31] Since surgical success was not associated with pre-operative DUS or CTA vascular stenosis, the authors postulated a neurogenic rather than vascular cause for pain in patients with MALS.[31] Also, in a series of 28 patients who had pre-operative symptom relief after coeliac plexus blockade and underwent open CA release for MALS, 27 (96%) had immediate symptom relief after surgery.[106] However, mean follow up was only three months and longer term results are awaited. The definitive value of coeliac plexus blockade to improve patient selection for intervention has to be established.

There is no evidence to support primary CA revascularisation in patients with MALS. It seems appropriate to maintain a release first approach owing to the potential

complications arising from stenting a compressed CA. Revascularisation of the CA may be considered in selected patients with persistent symptoms and CA stenosis after arcuate ligament release. Stents in the CA have been noted to be at higher risk of fracture and disconnection in the setting of branched endovascular aneurysm repair.[107] Angulation, respiratory movements, and dynamic forces may be predisposing factors for CA stent complications.

Reporting standards, outcome definitions, and consensus descriptions of the intervention(s) should be the next steps to improve the quality of research and care for patients with MALS. The CARoSO study will hopefully give an answer to the questions regarding surgery for MALS.[100]

Recommendation 29			New
Surgical intervention may be considered for selected patients with median arcuate ligament syndrome.			
Class	**Level**	**References**	**ToE**
IIb	C	Metz *et al.* (2022),[97] DeCarlo *et al.* (2023)[98]	

Recommendation 30			New
Laparoscopic or video assisted endoscopic retroperitoneal coeliac artery release may be considered the preferred treatment option for patients with median arcuate ligament syndrome undergoing surgical intervention.			
Class	**Level**	**References**	**ToE**
IIb	C	Metz *et al.* (2022),[97] DeCarlo *et al.* (2023),[98] Jimenez *et al.* (2012),[101] van Petersen *et al.* (2009)[102]	

5. ACUTE ARTERIAL MESENTERIC ISCHAEMIA

5.1. Introduction

Acute thromboembolic occlusion of the mesenteric arteries most commonly affects the SMA.[108] Symptomatic acute occlusions of the CA and or its branches or the IMA are rare and very seldom lead to intestinal infarction owing to the extensive collateral network from a patent SMA. The exception is if there is a chronic SMA occlusion, in which case an acute occlusion of the CA or IMA may lead to AMI. Moreover, AMI may develop on chronic stenosis of the mesenteric arteries, in which case both the CA and SMA are affected by atherosclerotic disease. This condition is referred to as acute on chronic mesenteric ischaemia.[109,110] AMI may also develop due to mesenteric hypoperfusion caused by acute aortic dissection or isolated SMA dissection. The treatment of AMI in complicated acute aortic dissection aims to restore blood flow to the true lumen in the proximal aorta and, if necessary, adjunctive procedures to restore SMA patency. Treatment of complicated aortic dissection is not covered in these guidelines but will be covered in the updated ESVS descending aorta guidelines. Isolated mesenteric artery dissection is discussed in Chapter 10.

5.2. Epidemiology

AMI comprises a group of disorders with incidence rates that vary according to their aetiology (arterial, non-occlusive, venous). In a meta-analysis of five studies reporting incidence rates in the general population for AMI including all aetiologies, the estimated pooled incidence was 6.2 (95% CI 1.9 — 12.9) per 100 000 persons per year, and varied from 0.7 to 14.1/100 000/year between studies.[111] In this meta-analysis, 38 studies reported the proportions of different AMI aetiologies in their cohorts, the most common being arterial occlusion in approximately two thirds of all AMI cases. Around 50% of the arterial occlusions were embolic and 50% thrombotic.[111] A nationwide, hospital registry based study between 2016 and 2020 from Estonia reported an incidence of 8.7/100 000/year for all aetiology AMI in the total population and 60.2/100 000/year for those over 70 years of age.[112] A Finnish study of 470 patients with AMI diagnosed alive (71%) or *post mortem* (29%) between 2006 and 2015 showed an incidence of 3.1/100 000/year specifically for arterial occlusive AMI.[113] The median age of the patients was 79 and 81 years, and 57 — 62% were female in these two studies. While embolism used to be the most common cause of SMA occlusion, there is a shift towards atherosclerotic or thrombotic arterial occlusion in more recent studies.[110,113—115]

In a worldwide, prospective observational study in 32 acute care hospitals, AMI accounted for 0.01 — 0.18% of all hospital admissions during a ten month period.[115] In this study, the underlying aetiology for AMI was arterial occlusive in 55.3%, venous in 17.5%, and non-occlusive (NOMI) in 13.2%. The median age of the patients was 71 years, which is in line with the mean age of patients with occlusive AMI of 70.7 years (95% CI 66.8 — 74.6 years) in a systematic review.[116] The in hospital AMI mortality rate was 49.4%.[115] Many publications include only treated or revascularised patients, who tend to be younger than those who are not treated and not included in the reports.[117] Thus, the true mean age of patients with occlusive AMI may be higher than 70 years. A Finnish study showed a significant increase in the probability of occlusive AMI as the cause of acute abdominal pain in emergency room patients aged $\geq$ 75 years. In that age group, AMI was a more common cause of acute abdomen than appendicitis or ruptured abdominal aortic aneurysm.[114] In two Finnish studies from different regions, the arterial occlusive AMI incidence rates in patients aged $\geq$ 70 years or $\geq$ 75 years were 26.7 and 37.5/100 000/year, respectively.[113,117] In patients aged under 50 years, the likelihood of arterial occlusive AMI as a cause of acute abdomen is very low.[113,114]

5.3. Diagnosis

The diagnosis of AMI is based on (1) clinical presentation, (2) vascular stenosis or occlusion on contrast enhanced computed tomography (CT) (preferably arterial phase), and (3) signs of intestinal injury on the contrast enhanced CT (preferably venous phase) (Fig. 3).[118] In case of early ischaemia, before irreversible bowel injury develops, the

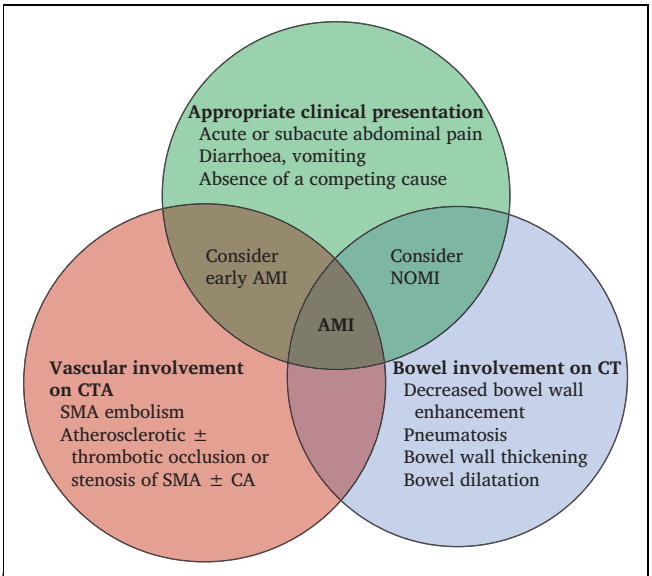

Figure 3. Basis for the diagnosis of acute mesenteric ischaemia (AMI). NOMI = non-occlusive mesenteric ischaemia; CTA = computed tomography angiography; SMA = superior mesenteric artery; CA = coeliac artery; CT = computed tomography.

diagnosis is ultimately confirmed by resolution of symptoms after revascularisation. Other options to assess early bowel ischaemia include laparotomy or laparoscopy accompanied by fluorescence angiography.[119] For late ischaemia, the diagnosis is confirmed by finding ischaemic bowel during laparotomy or at autopsy. Accurate triage is the key to a timely diagnosis of AMI. Time to CT, diagnosis, and operation were all shorter in patients with AMI initially referred to the surgical emergency room as opposed to non-surgical, which also led to shorter hospital stay, fewer bowel resections, and a lower 90 day mortality rate (50% *vs.* 75%).[120] In a worldwide survey on the management of AMI by a multinational study group, two thirds of the respondents indicated that AMI is rarely missed but that the diagnosis is often delayed.[121]

The common clinical symptoms of AMI include acute abdominal pain (60 — 100%), diffuse abdominal tenderness (54 — 90%), abdominal distension (18 — 54%), peritoneal signs (13 — 65%), nausea and or vomiting (39 — 93%), and diarrhoea and or rectal bleeding (12 — 48%).[122]

5.3.1. Clinical presentation: embolism. Awareness of AMI among emergency room physicians and radiologists as a potential cause of acute abdominal symptoms in certain patient groups is important. Since atrial fibrillation (AF) was present in 32.2% of patients with AMI, every patient with AF and acute abdominal pain should be suspected of having acute SMA embolism.[116] Cardiac emboli may also be associated with valvular disease, left atrium dilatation, myocardial infarction, and ventricular dilatation with mural thrombus. Concomitant stroke or leg ischaemia are also clues to a cardiac source of embolism.

The typical clinical triad for acute embolic SMA occlusion is (i) severe abdominal pain with minimal findings on examination (disproportionate pain), (ii) bowel emptying (vomiting, diarrhoea), and (iii) the presence of a source of embolus, most often AF. The sudden onset of abdominal pain (phase 1, reversible ischaemia) may decrease in intensity (phase 2), followed by an increase in abdominal pain associated with clinical deterioration and progression towards generalised peritonitis (phase 3, irreversible ischaemia).

5.3.2. Clinical presentation: thrombosis. Acute thrombotic SMA occlusion is more difficult to diagnose than acute embolic SMA occlusion. Thrombosis occurs in areas of severe atherosclerotic narrowing, most often at the origins of the SMA and CA.[108] Occlusive atherosclerotic lesions in the SMA are clinically more important compared with those in the CA. Concomitant atherosclerotic manifestations such as coronary, cerebrovascular, or peripheral arterial disease are common.[116] Every patient with atherosclerosis and acute abdominal pain should be suspected of having SMA thrombosis. In a substantial proportion of these patients, progressive atherosclerosis at the SMA origin may have developed over many years, resulting in collateral circulation to the SMA, mainly from the CA and IMA.

Acute abdominal pain may develop when a previously patent SMA occludes suddenly due to acute thrombosis of an underlying atherosclerotic lesion. However, if the SMA was already chronically severely obstructed with sufficiently developed collaterals, acute thrombosis of the SMA may not essentially change the total blood flow to the mesentery. AMI may develop upon severely obstructed or chronically occluded mesenteric arteries, typically involving both the CA and SMA, without any objective evidence of an acute thrombotic event (such as a soft thrombotic clot superimposed on a calcified stenosis in the SMA). This is the classic presentation of acute on chronic mesenteric ischaemia.[123] Dehydration, anaemia, low cardiac output, and hypercoagulable state are major contributing factors. In retrospect, a high proportion of the often misunderstood and misdiagnosed patients with acute thrombotic SMA occlusion may have had long standing pre-existing symptoms of CMI, including postprandial abdominal pain (abdominal angina), fear of eating, diarrhoea, and weight loss.[109] Patients with symptomatic CMI who have abdominal pain even between meals (abdominal rest pain) should be treated urgently since transition from CMI to AMI can be sudden and unpredictable.

5.3.3. Laboratory markers. Much research has been done in the hope of finding an accurate, non-invasive, rapid, 24/7 available, and cost effective biomarker that would aid in the diagnosis of AMI. The diagnostic accuracy of traditional biomarkers such as lactate, C reactive protein (CRP), leucocytes, D dimer, phosphate, and creatine kinase, and new biomarkers for intestinal cell and mucosa injury such as IFABP, ischaemia modified albumin, α-glutathione S-transferase, and D lactate has been summarised in several systematic reviews.[124—126] Treskes *et al.* reported pooled sensitivity and specificity, respectively, for investigated biomarkers as follows: IFABP (human enzyme linked immunosorbent assay [ELISA] kit) 79.0% (95% CI 66.5 — 88.5%) and 91.3% (95% CI 87.0 — 94.6%); IFABP (sandwich ELISA kit) 75.0%

(95% CI 67.9 — 81.2%) and 79.2% (95% CI 76.2 — 82.0%); D lactate 71.7% (95% CI 58.6 — 82.5%) and 74.2% (95% CI 69.0 — 79.0%); α-glutathione S-transferase 67.8% (95% CI 54.2 — 79.5%) and 84.2% (95% CI 75.3 — 90.9%); and ischaemia modified albumin 94.7% (95% CI 74.0 — 99.9%) and 86.4% (95% CI 65.1 — 97.1%).[124] Reintam Blaser *et al.* found the best diagnostic accuracy for ischaemia modified albumin, with pooled sensitivity and specificity, respectively, of 94.7% (95% CI 70.6 — 99.3%) and 90.5% (95% CI 68.9 — 97.6%); interleukin-6 100% (95% CI 0 — 100%) and 82% (95% CI 31.7 — 97.9%); IFABP 73.6% (95% CI 56.6 — 85.6%) and 89.8% (95% CI 79.1 — 95.3%); and procalcitonin 79.1% (95% CI 65.6 — 88.2%) and 89.1% (95% CI 81.5 — 93.8%).[126] The corresponding sensitivity and specificity, respectively, was 88.5% (95% CI 70.5 — 96.1%) and 61.7% (27.3-87.3) for D lactate and 87.9% (95% CI 77.0 — 94.0%) and 69.2% (95% CI 60.0 — 82.3%) for D dimer.[126]

In the most recent systematic review, a total of 60 different biomarkers for detecting arterial occlusive AMI in humans were identified.[127] Fifteen biomarkers were evaluated in five or more studies, 21 biomarkers in two or more studies, and 24 in a single study. Given the wide heterogeneity between the studies due to the differences in study populations, normal and cutoff values, verification bias, small sample sizes, and high risk of bias, the authors refrained from meta-analysis. It was concluded that clinical management cannot be based on the use of a single biomarker to establish or rule out AMI.[127]

In the 2017 ESVS clinical practice guidelines, D dimer was recommended as a biomarker to exclude the diagnosis of AMI owing to its 100% sensitivity and negative predictive value (NPV).[1] However, the NPV of D dimer in other studies ranged between 87% and 96%, indicating that the test is not perfect for ruling out AMI, also because of the variety of cutoff values and assays used.[127] D dimer is suggested to aid in the diagnostic process of AMI alongside a clinical suspicion and CTA.[128] However, the added value of D dimer in clinical decision making in a patient with acute abdominal pain is low because D dimer will be elevated with almost every acute abdominal pathology. Although D dimer is elevated in AMI due to acute occlusion of a large mesenteric artery or transmural intestinal necrosis, it is unknown how D dimer behaves in the early stages of AMI and acute on chronic mesenteric ischaemia. Due to the absence of clinical applicability and the limited scientific substantiation, the GWC advises against using D dimer as a single biomarker to exclude arterial occlusive AMI.

Lactate is an end product of glycolysis under anaerobic conditions and exists in two isomers: L lactate (primary isomer produced in humans) and D lactate (produced by bacteria in the human colon). Lactate is often regarded as a marker for bowel ischaemia, but this is a misconception. There is common agreement that high lactate levels and metabolic acidosis indicate general deterioration, systemic hypoperfusion, and high risk of death, irrespective of the cause.[129,130]

In summary, the ultimate single biomarker or combination of biomarkers that can solve the diagnostic dilemma and reduce the time to diagnosis of AMI is yet to be found. Laboratory tests together with contrast enhanced CT are all part of the clinical evaluation of the patient. Common biomarkers such as leukocytes, CRP, and lactate may help to raise the suspicion of AMI and associated bowel necrosis. Thus, none of these findings should be neglected. A recent study including 117 patients with arterial AMI undergoing first line endovascular revascularisation showed 86% bowel resection free survival for those with CRP < 100 mg/L and normal bowel wall enhancement at initial CT.[131]

Recommendation 31			Changed
A single measurement of a biomarker such as lactate or D dimer to confirm or rule out the diagnosis in patients with a clinical suspicion of acute mesenteric ischaemia is not recommended.			
Class	**Level**	**References**	**ToE**
IIIb	C	Treskes *et al.* (2017),[124] Khan *et al.* (2019),[125] Reintam Blaser *et al.* (2023),[126] Blauw *et al.* (2024)[127]	

5.3.4. Computed tomography angiography. The diagnosis of acute SMA occlusion and severity of intestinal ischaemia are greatly facilitated by the 24/7 availability of high resolution CTA.[132] The radiologist has an important role in raising the suspicion of AMI. Mesenteric vessel patency should be reported routinely rather than sporadically in patients with acute abdominal pain. The whole length of the mesenteric arteries should be examined carefully, as an embolic clot may often land distally in the SMA. The often subtle signs of ischaemic intestinal injury should not be overlooked.[133,134]

5.3.4.1. Optimal imaging protocol. Intravenous contrast enhanced CT with a slice thickness of ≤ 1 mm, performed with contrast in both the arterial and portal venous phases (biphasic or split bolus protocol), is recommended as the first line imaging technique to diagnose mesenteric artery occlusion and intestinal pathologies.[133] The 2017 ESVS clinical practice guidelines recommended triphasic CTA as the preferred imaging modality.[1] However, recent studies have questioned the necessity of the non-contrast phase for detection of bowel wall hyper attenuation and intramural haemorrhage.[133,135,136] Omitting the non-contrast CT is unlikely to result in a missed AMI diagnoses. The arterial phase with thin slices enables accurate detection of vascular stenosis, and the venous phase is required for the diagnosis of venous mesenteric thrombosis, assessment of bowel wall pathology and solid organ perfusion, and other causes of abdominal pain. Reconstructions of images in the sagittal, coronal, and transverse planes are helpful in assessing mesenteric artery patency. The optional non-contrast CT may help in the assessment of decreased bowel enhancement on contrast enhanced CT and offer some benefits in detecting alternative diagnoses.[133,135]

For patients with a baseline creatinine of < 1.5 mg/dL and an estimated glomerular filtration rate (eGFR) of ≥ 30 mL/min/1.73m^2, the risk of contrast induced acute kidney injury is extremely low.[137] Even patients with

impaired renal function or increased creatinine values should undergo emergency contrast enhanced CTA if there is a suspicion of mesenteric ischaemia in order to improve diagnostic accuracy and the chances of survival. Creatinine values are often temporary in patients with hypovolaemia due to vomiting and diarrhoea.

Dual energy CT could potentially improve visualisation of the vessels and bowel wall and assist in the detection of subtle bowel wall hypo-enhancement in both occlusive and non-occlusive mesenteric ischaemia. However, motion related artefacts due to peristalsis are common and more research is needed before any recommendation related to its use in AMI can be given.[133,138]

5.3.4.2. Vascular involvement. Embolic occlusion often appears as an oval shaped filling defect surrounded by contrast in a non-calcified arterial segment located in the middle and distal part of the main stem of the SMA. Synchronous emboli to other visceral, limb, or cerebral arteries is a common finding.[139] Increased awareness of the high likelihood of AF related and other cardio-embolic causes for acute abdominal pain may improve the diagnostic accuracy of CTA and triage of patients with acute embolic SMA occlusion.[116] To standardise reporting of SMA occlusion in AMI, it has been proposed that the SMA should be segmented into proximal (from the ostium to the inferior pancreaticoduodenal artery), middle (from the inferior pancreaticoduodenal artery to the ileocolic artery), and distal (downstream from the ileocolic artery) sections.[140] However, this proposal has not yet undergone clinical validation.

Thrombotic occlusion usually appears as a clot superimposed on a calcified occlusive lesion at the origin of the SMA. If there are no signs of acute SMA occlusion but the mesenteric arteries are severely calcified and obstructed, the likelihood of AMI in a patient with acute abdominal pain is still high. The radiologist should look for early signs of intestinal ischaemia on the CT.[110,141]

5.3.4.3. Bowel involvement. Decreased bowel wall enhancement, pneumatosis, and portomesenteric venous gas are considered signs of intestinal ischaemia and are suggestive, although not definitive signs, of bowel necrosis. In early AMI, early signs of intestinal injury are often present. These can be very subtle and difficult to detect, such as bowel wall thickening, mild luminal dilatation (as a sign of bowel paralysis), mesenteric oedema (fat stranding), and small amounts of peritoneal fluid (Table 6).[118,133]

It is important to distinguish between early and late AMI. In early AMI, bowel injury is limited and considered reversible. Patients with late AMI may have extensive bowel necrosis, organ failure, elevated serum lactate, and imaging features of intestinal necrosis. Therefore, radiologists should not only suggest the diagnosis of AMI but also recognise extensive bowel ischaemia that may require surgical resection. A systematic review and meta-analysis that aimed to identify CT findings associated with intestinal necrosis in AMI included 11 studies with 1 037 cases of AMI.[142] Bowel wall thinning had a diagnostic odds ratio (OR) of 13.1 (95% CI 3.71 — 46.3) for transmural intestinal necrosis. Diagnostic ORs for decreased or absent bowel wall enhancement, intestinal pneumatosis, and portomesenteric venous gas were between 5 and 6. However, none of these signs prove that the intestine is irreversibly injured.

5.3.4.4. Accuracy in diagnosing acute mesenteric ischaemia. If no clinical suspicion of AMI is raised in the information provided to the radiologist, the condition is highly likely to be underdiagnosed.[134,139] It is not unusual that a re-assessment of the imaging may detect overlooked radiological findings associated with AMI not reported at the initial reading, especially if the imaging protocol is not optimal.[143,144] Such diagnostic delay has a negative impact on the prognosis.

A systematic review of the diagnostic accuracy of CTA for detection of AMI that pooled data from eight studies including 213 patients with AMI and 651 without AMI reported a pooled sensitivity of 94% (95% CI 83 — 98%) and specificity of 97% (95% CI 93 — 99%).[145] However, these results should be interpreted with caution and the accuracy of the CT report by the emergency room radiologist should not be overestimated in AMI. A closer look at the individual

Table 6. Contrast enhanced computed tomography (CT) findings in acute mesenteric ischaemia (AMI).

Contrast enhanced CT findings	Specific for AMI	Specific for bowel necrosis
Vascular findings		
SMA emboli	++	−
SMA thrombosis	++	−
Mesenteric atherosclerosis (chronic occlusive multivessel disease)	−	−
Intestinal findings		
Bowel wall thickening	−	−
Bowel wall thinning (paper thin appearance)	+	++
Decreased or absent bowel wall enhancement	++	+
Increased bowel wall enhancement	−	−
Small bowel dilatation >25 mm	+	+
Pneumatosis or portomesenteric venous gas	++	+
Mesenteric fat stranding (oedema) and ascites	−	−
Involvement of other organs (solid organ perfusion defects)	−	−

CT = computed tomography; AMI = acute mesenteric ischaemia; SMA = superior mesenteric artery; ++ = highly specific finding; + = specific but not definitive finding; − = not a specific finding but is associated with AMI.[118,142,147]

studies in two other systematic reviews shows that the studies were performed exclusively in patients with a clinical suspicion of AMI prior to imaging and that the majority of the study patients had advanced bowel ischaemia.[122,132] In practice, however, AMI is rarely suspected prior to imaging, and without a clinical suspicion, CT of the abdomen is often performed in the venous phase alone. More importantly, AMI should be diagnosed early when the definitive signs of bowel ischaemia may still be absent.

5.3.5. Duplex ultrasound. Expertise in DUS of the visceral arteries may not be available around the clock. Furthermore, bowel paralysis associated with acute intestinal ischaemia may preclude accurate DUS. While proximal occlusive lesions of the visceral arteries can be identified, distal occlusions cannot. In a sample of patients with abdominal pain without a diagnosis after laboratory examinations, plain Xray, and abdominal ultrasound, the positive predictive value of DUS was only 50%.[146] Therefore, DUS is an inappropriate imaging method to assess acute occlusive lesions of the visceral arteries in the emergency setting.

5.3.6. Digital subtraction angiography. DSA can differentiate occlusive, embolic, and thrombotic from non-occlusive AMI, but it is seldom used for diagnostic purposes alone.

Recommendation 32			New
Clinicians are recommended to mention the suspicion of acute mesenteric ischaemia in the referral for computed tomography angiography.			
Class	**Level**	**References**	**ToE**
I	C	Lehtimäki *et al.* (2015),[134] Wadman *et al.* (2010),[139] Anglaret *et al.* (2021)[144]	

Recommendation 33			Unchanged
Urgent computed tomography angiography with contrast enhancement in arterial and venous phases with ≤1 mm slices (and optional non-contrast CT run) is recommended in patients suspected of acute mesenteric ischaemia, regardless of renal function.			
Class	**Level**	**References**	**ToE**
I	B	De Simone *et al.* (2018),[137] Zeng *et al.* (2023),[142] Yang *et al.* (2019)[145]	

5.4. Treatment of acute superior mesenteric artery occlusion

5.4.1. Current approaches. It is the vascular surgery paradigm that immediate revascularisation is necessary in most patients with acute SMA occlusion. However, there is significant variation between practices. A worldwide survey among specialists involved in the care for patients with AMI indicated that roughly half of the respondents rarely or never performed emergency revascularisation for AMI.[121] In contrast, 10 — 30% reported aiming to revascularise all patients with AMI. Some 45% of 231 patients with arterial AMI in the AMESI study underwent revascularisation.[115]

Optimal treatment may include both open and endovascular surgery, and patients are best managed in a vascular centre with a hybrid operating room, although logistical aspects need to be taken into consideration in this urgent situation. From pre-operative clinical and radiological evaluation, it should be determined whether there is the possibility that transmural bowel necrosis has already developed. The presence of gas in the intestinal wall or portomesenteric system on CTA is a sign of severe transmural ischaemia but does not mean irreversible bowel injury or a fatal outcome if treated in a timely manner. If bowel necrosis is suspected, the patient needs to be taken to the (hybrid) operating room for emergency laparotomy or laparoscopy. Otherwise, endovascular revascularisation without opening the abdomen may be considered first. A second aspect is to determine whether the arterial occlusion is embolic or thrombotic. This will guide the revascularisation approach.

Laparotomy or laparoscopy is indicated if there are signs of peritonitis or a high suspicion of intestinal infarction on CT, unless a palliative approach has been chosen. Laparotomy aims to assess the extent and severity of intestinal ischaemia, although this may require intra-operative fluorescence imaging or contrast angiography. Laparotomy, rather than laparoscopy, may be safer and quicker for evaluation of the visceral organs. Bowel viability may be impossible to evaluate at laparoscopy owing to extensive intestinal paralysis, and manipulation of dilated bowel with laparoscopic graspers may cause further bowel injury. In the AMESI study, some 5% of patients with arterial AMI were managed laparoscopically and 67% with laparotomy; 25% had bowel resection only.[115]

5.4.2. Acute mesenteric arterial revascularisation. There is rarely an indication for open surgical revascularisation of both the SMA and the CA in an emergency setting. SMA revascularisation appears to be most important, also for the endovascular approach. Even after successful endovascular revascularisation, patients may still require laparotomy when persisting signs of peritoneal irritation indicate the presence of non-viable bowel. This decision should not be delayed as necrotic bowel will lead to sepsis and more extensive intestinal necrosis unless resected in a timely manner.

Mesenteric artery revascularisation is preferably done before any bowel surgery so that the margins of viable bowel can be assessed after the blood flow has been restored (Fig. 4). If laparotomy has been performed due to an uncertain diagnosis of peritonitis in a hospital without a vascular surgery service, it may be preferable to resect clearly necrotic bowel sparingly without reconstruction, temporarily close the abdomen, and transport the patient to a vascular centre for revascularisation.

Patients with AMI should be treated in centres with 24/7 multidisciplinary services. The specialists involved in the diagnosis and treatment of patients with AMI include emergency room physicians, radiologists, vascular surgeons, gastrointestinal surgeons, specialised gastroenterologists, vascular interventional radiologists, intensivists, and anaesthetists.[150]

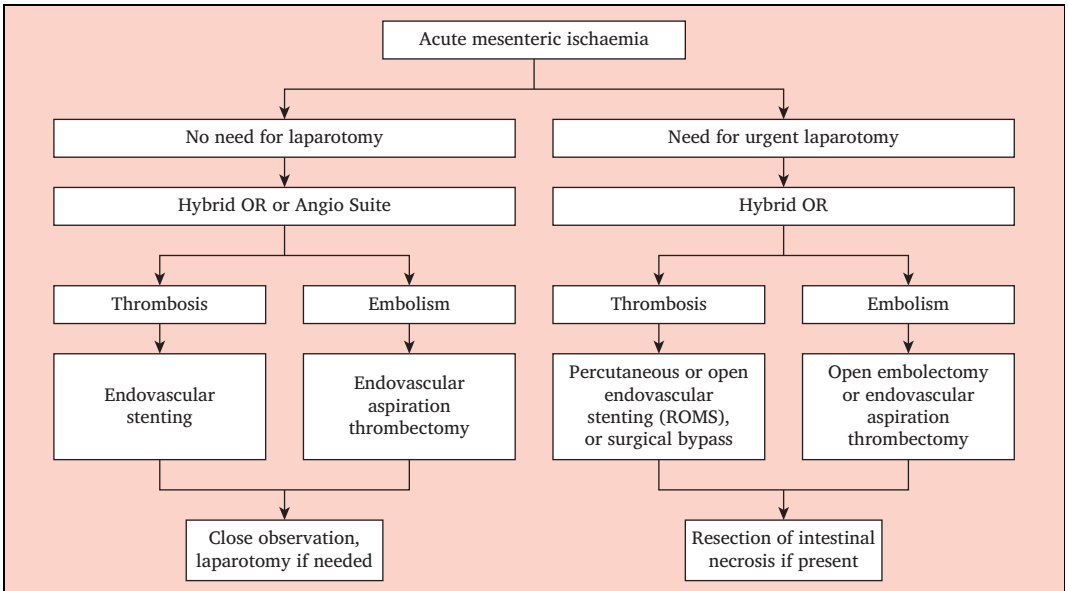

Figure 4. Flowchart for invasive treatment of acute mesenteric ischaemia. Adapted from.[150] OR = operating room; ROMS = retrograde open mesenteric stenting.

Recommendation 34 **New**

It is recommended to treat patients with acute mesenteric ischaemia in centres with 24/7 multidisciplinary services and experience in both open and endovascular mesenteric artery revascularisation.

Class	Level	References	ToE
I	C	Lemma *et al.* (2019),[120] Blauw *et al.* (2017),[148] Tolonen *et al.* (2021)[150]	

Recommendation 35 **Changed**

Revascularisation first before bowel resection should be considered in patients with acute mesenteric ischaemia.

Class	Level	References	ToE
IIa	C	Block *et al.* (2010),[149] Tolonen *et al.* (2021),[150] Arthurs *et al.* (2011),[152] Beaulieu *et al.* (2014)[153]	

The Swedish National Registry for Vascular Surgery (Swedvasc) noted a steady increase in endovascular revascularisations for AMI since 2004, a trend also seen in other European countries.[148–151] In the USA, endovascular and open revascularisations for AMI have been performed at equal rates since 2004.[12]

5.4.3. Open superior mesenteric artery embolectomy. Open SMA embolectomy may be done when laparotomy has been performed for suspected peritonitis. A transverse incision in the visceral peritoneum or transverse mesocolon in the mesenteric root just below the body of the pancreas is made to expose the SMA. Alternatively, the Treitz ligament may be mobilised to expose the SMA at the base. After arteriotomy, balloon embolectomy with a 3 or 4 F Fogarty catheter is performed with caution so as not to cause a dissection in the SMA. The result may be checked by some form of completion assessment, such as DUS, Doppler ultrasound, transit time measurement, or DSA of the SMA. If none of these modalities are available, pulse palpation distal in the mesentery can be performed. Of note, comparative data regarding completion assessment methods are lacking in the literature. The presence or absence of stenosis and dissection at the arteriotomy closure site, residual peripheral embolus in arterial branches, and venous return to the portal vein can only be assessed by DSA.

5.4.4. Open revascularisation for acute thrombotic superior mesenteric artery occlusion. Open revascularisation options include bypass distal to the occlusion, thromboendarterectomy with patch angioplasty, and division of the SMA distal to the occlusion and re-implantation into the infrarenal aorta. The pre-operative CTA is useful to determine the source of inflow artery and sites with extensive atherosclerotic lesions to avoid. A short synthetic bypass graft from the infrarenal aorta to the SMA seems to be straightforward but is seldom possible due to heavy infrarenal aortic calcification. The risk of kinking in such a short bypass is also a potential problem. In cases with co-existing extensive atherosclerotic lesions in the infrarenal aorta, the supracoeliac aorta or common iliac artery may be used as the inflow for the graft. Autologous reversed saphenous vein may be the preferred conduit in case of bacterial contamination. Vein grafts taking off from the infrarenal aorta or the common iliac artery are prone to kinking when the intestines are moved back into the abdomen after completion of the bypass. In order to prevent kinking, polyethylene terephthalate (e.g., Dacron) or reinforced expanded polytetrafluoroethylene (ePTFE) might be a preferred conduit material, especially in the emergency setting in a non-contaminated peritoneal cavity. It is important to tunnel the graft under the retroperitoneum or to cover the graft with an omental flap if possible in order to prevent contact between the graft and the intestines, which may result in a graft–enteric fistula.

Recommendation 36		Unchanged
Completion control with intra-operative angiography, duplex ultrasound, or Doppler ultrasound should be considered in patients undergoing open surgical mesenteric artery revascularisation.		
Class	**Level**	**Reference**
IIa	C	Consensus

5.5. Endovascular therapeutic options in acute mesenteric ischaemia

5.5.1. Access to the superior mesenteric artery. The SMA can be reached via the femoral and brachial routes. Brachial access may be preferable if there is a sharp downward angle between the aorta and the SMA. Steerable sheaths have reduced the need for a brachial approach that is associated with access related complications such as bleeding or occlusion at the puncture site, nerve injury, and stroke.[141,154] If an antegrade approach from the femoral or brachial artery fails, a retrograde approach through the exposed SMA after laparotomy can be attempted, unless open revascularisation is preferred.

5.5.2. Aspiration embolectomy of the superior mesenteric artery. In patients without peritonitis, endovascular aspiration embolectomy can be done by passing a catheter and a hydrophilic guidewire into the ileocolic branch of the SMA, which is then replaced by a stiff wire to achieve stability. Subsequently, an introducer with a removable hub is placed proximal to the embolus in the SMA. Inside this, a guiding catheter or a dedicated aspiration catheter is introduced into the clot, which is aspirated as the catheter is withdrawn. The hub of the introducer is removed to allow clearance of residual clots. Angiography is performed, usually followed by repeated aspirations. An alternative is to use an over the wire double lumen aspiration catheter, which may allow removal of smaller peripheral clots.

5.5.3. Local superior mesenteric artery thrombolysis. Local thrombolysis is an alternative in patients without peritonitis for cases of incomplete aspiration embolectomy or distal embolisation.[151,155] With the introducer placed in the proximal SMA, a multiple side hole catheter delivering thrombolytic agents over 10 cm, or an end hole catheter, is advanced to within the clot. Local thrombolysis is achieved by administration of recombinant tissue plasminogen activator at a rate of 0.5 — 1 mg/h (or other agents at different dosages, e.g., urokinase 120 000 IU/h), checking the patency with repeated angiography once or twice a day. Bleeding complications during local thrombolysis are infrequent and most often self limiting.[155] Small peripheral residual emboli can be treated conservatively with heparin anticoagulation as the marginal arteries in the mesentery may provide sufficient collateral circulation to the affected intestinal segment.[156] Endovascular rheolytic thrombectomy may be used as a supplementary technique to aspiration thrombectomy in cases where thrombolysis is contraindicated.[157]

5.5.4. Antegrade recanalisation and stenting of the superior mesenteric artery. Treatment of underlying stenotic or occlusive lesions is achieved during the same procedure after clot removal by aspiration or thrombolysis. Balloon expandable bare or covered stents are better than self expanding stents to maintain lumen diameter after stent deployment across hard calcified ostial lesions. Unfavourable artery angulation or a potential risk of arterial dissection at the distal end of the stent is treated by extension with a self expanding stent into the middle SMA. Results after stenting are checked by angiography.

5.5.5. Retrograde open recanalisation and stenting of the superior mesenteric artery. Laparotomy and exposure of the SMA may be performed for ROMS. This approach offers the opportunity to inspect the abdominal viscera, to have distal control of the SMA, and to avoid bypass surgery in the setting of necrotic bowel. The SMA is dissected below the pancreas, and any jejunal branches are controlled prior to catheter manipulations. Retrograde access is established with a guidewire and sheath after direct SMA puncture or puncture of one of its major branches. The stenosis or occlusion is treated by angioplasty and retrograde placement of a balloon expandable stent. It can sometimes be easier to snare the guidewire in the aorta and work from the groin or the arm rather than from the abdomen. The puncture site may be closed with interrupted sutures or a patch if necessary. Re-entry into the aorta in occlusion cases can be difficult with this technique, and there is a risk of causing an aortic dissection. The technical success rate of ROMS is around 90%.[85] In a case series of 37 patients who underwent ROMS for AMI ($n = 11$), acute on chronic ischaemia ($n = 18$), or CMI ($n = 8$), overall survival, freedom from re-intervention, primary patency, and primary assisted patency were 70.1%, 61.1%, 84.5%, and 92.4%, respectively, after one year.[85] These outcomes are similar to other published series and a systematic review of five case series.[86,87] Although the interpretation of the outcomes is difficult because most reports included patients both with CMI and AMI, ROMS appears to offer acceptable one year outcomes.

5.6. Outcomes after endovascular and open revascularisation for acute mesenteric ischaemia

Several non-randomised studies have compared endovascular *vs.* open revascularisation for arterial AMI. In a systematic review of 13 observational studies, the 30 day mortality rate was 15 — 39% after endovascular and 33 — 50% after open revascularisation.[158] Laparotomy rates ranged from 13 — 73% after endovascular revascularisation, and bowel resection rates from 14 — 40%.[158] The review included seven studies comparing endovascular and open revascularisation. The rate of bowel resection in the endovascular group ranged from 14 — 28% compared with 33 — 63% in the open cohort. In the same seven studies, the 30 day mortality rate ranged from 15 — 39% after endovascular revascularisation and from 33 — 50% after open revascularisation. In another meta-analysis, endovascular revascularisation was associated with a lower 30 day risk of death (RR 0.68, 95% CI 0.59 — 0.79) and

bowel resection (RR 0.64, 95% CI 0.45 — 0.91).[159] The latest meta-analysis did not confirm the lower 30 day mortality rate (OR 0.79, 95% CI 0.50 — 1.25) for endovascular revascularisation, whereas the need for bowel resection was again lower (OR 0.42, 95% CI 0.20 — 0.88).[160]

Although these non-randomised studies suggest a benefit of endovascular over open revascularisation, it should be considered that they are inherently flawed due to selection bias. The outcomes of AMI are highly dependent on whether all incoming patients with AMI received an attempt at revascularisation or whether only selected patients with early AMI were treated. In 20 studies reporting outcomes of endovascular or open revascularisation in AMI, the mean age of treated patients varied between studies from 50 years to 79 years. Moreover, only less than half of the studies reported revascularisation rates, which varied from 1% to 97%.[117] Revascularisation rate is the proportion of patients who underwent revascularisation among all patients treated with AMI. In future studies, researchers should always report the revascularisation rate to give a better understanding how patients were selected for revascularisation and how many were treated without a revascularisation attempt. Furthermore, the severity of the acute illness should be reported in future studies comparing open and endovascular revascularisation in AMI to be able to assess selection bias.

A nationwide analysis based on the US National Inpatient Sample showed a lower mortality rate for patients with AMI treated endovascularly compared with open revascularisation even though patients in the endovascular group had a higher comorbidity index. In addition, endovascular treatment was associated with cost savings, whereas patients in the open group had a higher risk of acute kidney injury and were often discharged to a nursing facility rather than home.[161] In patients with acute embolic SMA occlusion, there are no comparative studies to suggest that endovascular or open treatment is superior. Small, single centre studies have shown favourable results for endovascular mechanical thrombectomy in AMI.[141,162–165] The technical success rates for aspiration embolectomy ranged between 78% and 94%. In these studies, subsequent exploratory laparotomy was performed in 40 — 73% after endovascular embolectomy, bowel resection was performed in 12 — 41%, and the 30 day mortality rate ranged from 27 — 36%.[141,162–164]

5.6.1. Long term outcome after revascularisation for acute mesenteric ischaemia. Endovascular revascularisation may offer early survival benefit in AMI, but a comparison with open revascularisation showed equivalent five year survival rates of around 20%.[165] Re-admissions due to mesenteric ischaemia after surviving the initial episode of AMI are around 8%, and permanent institutionalisation after surviving AMI is also rare, especially in those treated endovascularly. In a small, single centre study with follow up ranging from eight to 13 years, those who survived the initial AMI episode typically died from other cardiovascular diseases, which highlights the importance of secondary prevention in these patients.[165]

Recommendation 37			Unchanged
Endovascular revascularisation should be considered as first line therapy in patients with acute mesenteric ischaemia due to thrombotic or embolic superior mesenteric artery occlusion.			
Class	**Level**	**References**	**ToE**
IIa	B	Kärkkäinen *et al.* (2015),[141] Murphy *et al.* (2019),[158] Salsano *et al.* (2018),[159] Shi *et al.* (2024),[160] Raupach *et al.* (2016),[162] Shi *et al.* (2022),[163] Li *et al.* (2022)[164]	

Recommendation 38			Changed
Retrograde open mesenteric artery stenting may be considered in patients with acute mesenteric ischaemia needing superior mesenteric artery revascularisation when percutaneous stenting is not possible.			
Class	**Level**	**References**	**ToE**
IIb	C	Blauw *et al.* (2014),[84] Sénémaud *et al.* (2021),[85] Hou *et al.* (2022),[86] Crillo-Penn *et al.* (2023)[87]	

5.7. Bowel management in acute mesenteric ischaemia

5.7.1. Clinical assessment of bowel viability. Intestinal ischaemia may be extensive with lesions in the jejunum, ileum, and colon. While the serosal surface of the intestine may have a normal appearance, mucosal ischaemic changes may be extensive. Severe intestinal ischaemia is characterised by patchy cyanosis, reddish black discolouration, decreased or absent peristalsis, and absent palpable pulsations in the mesentery. Clinical assessment at laparotomy remains the most frequently used method for assessment of bowel viability but is not infallible. Intra-operative Doppler ultrasound, intravenous injection of fluorescein for assessment of ultraviolet fluorescence pattern, and laser Doppler flowmetry have been evaluated in small studies but there is still no standardised technique to evaluate tissue perfusion status.

Although laparoscopy is minimally invasive, it is not ideal for safe assessment of ischaemia of the whole length of the intestine. Ischaemic bowel may be paralytic and distended, not allowing complete inspection. Laparoscopic manipulation of fragile bowel carries a risk of perforation. If a laparotomy has been performed, there are no grounds to perform a second look with laparoscopy. However, in patients undergoing successful endovascular revascularisation early in the course without intestinal CTA lesions, diagnostic laparoscopy may have a role in assessing bowel viability.[166]

5.7.2. Indocyanine green fluorescent imaging. Indocyanine green fluorescent imaging (ICGFI) is used in only 7% of centres for intra-operative quality control in visceral surgery to evaluate bowel viability.[121] However, several experimental and clinical reports have demonstrated the feasibility of ICGFI in mesenteric ischaemia for the evaluation of intestinal viability,

using various imaging systems and different ICG dosages. Up to now, visual interpretation of ICGFI has mainly been used.[167,168] The authors concluded that ICGFI is easy to use, takes little time, and can be used in an emergency situation.[167] ICGFI was found to be superior to visual assessment alone and may impact surgical management, especially regarding whether to perform a second look, resection, or even revascularisation. However, all investigators have only made a visual evaluation of ICGFI without objective quantitative interpretation.[167–170]

In a series of 52 patients with NOMI, ICGFI led to less aggressive bowel resection than based on clinical assessment in six patients and was false negative in two patients.[168] In a multicentre retrospective study of 93 patients with AMI due to mesenteric artery occlusion ($n = 42$) or bowel strangulation ($n = 41$), ICGFI led to no ($n = 4$ patients) or more conservative bowel resection ($n = 17$ patients), whereas it led to a more aggressive resection in six patients.[170]

Some authors have tried to establish quantitative assessment and concluded that ICGFI was a feasible, reliable, and valid technique for mesenteric blood flow assessment.[119,171–174] Real time ICGFI quantification using time to peak, slope of fluorescent intensity, and background subtracted peak fluorescent intensity may predict tissue injury, especially if visual assessment is difficult.[119,171,172] In an experimental setting even small differences in perfusion can be reliably determined by ICGFI. Evaluation of the maximum fluorescence intensity alone resulted in only a slightly better bowel evaluation compared with visual evaluation.[175]

Hyperintensity due to capillary leakage and the resulting pooling of ICG in the tissue may lead to misinterpretation. Already injured tissue can be estimated as optimally perfused by visual evaluation alone.[172,176] Haemodynamics, such as cardiac output, blood pressure, vascular resistance, volume load, and catecholamine therapy, have to be taken into consideration to avoid misinterpretation of ICGFI. More clinical studies are needed to determine the predictive value of ICGFI assessment.

Recommendation 39 — New

Quantitative indocyanine green fluorescent imaging may be considered as an aid in the assessment of bowel viability in patients undergoing laparotomy or laparoscopy for acute mesenteric ischaemia.

Class	Level	References	ToE
IIb	C	Vaassen *et al.* (2022),[119] Karampinis *et al.* (2018),[168] Liot *et al.* (2018),[169] Joosten *et al.* (2022)[170]	

5.7.3. Second look laparotomy. Surgical assessment of bowel viability may be necessary, sometimes repeatedly, depending upon the initial extent and severity of intestinal ischaemia prior to revascularisation, the expected effect of the revascularisation procedure, any bowel resection performed, and the physiological condition of the patient. The need to perform a second look laparotomy and bowel resection may indicate a more severe state of ischaemia and therefore be associated with a higher mortality rate. The decision to undertake a second look depends on the surgeon's interpretation at the initial laparotomy or if the patient's condition does not improve.

5.7.4. Damage control surgery. Bowel resection should be carried out according to the principles of damage control surgery. Only clearly transmurally non-viable segments should be resected, and doubtful ones should be spared and assessed at the second look. Bowel resections can be performed with staplers, delaying the creation of anastomoses or stomas until the second or third look laparotomy. The mesentery with all the important marginal collaterals should be preserved by keeping the resection line close to the bowel. Primary anastomosis should be avoided if the patient is haemodynamically unstable (requiring vasoactive drugs), the abdomen is severely contaminated, massive bowel resection or several bowel anastomoses are needed, the abdomen cannot be closed without tension, or the bowel cut end perfusion remains compromised.[123] The abdominal wall can be left unsutured when repeat laparotomy is planned. In this situation, skin only closure or temporary abdominal closure using a negative pressure wound therapy device may be applied.

Recommendation 40 — Changed

Resection without primary reconstruction and second look laparotomy for definitive treatment should be considered in patients undergoing acute mesenteric revascularisation who need bowel resection.

Class	Level	Reference
IIa	C	Consensus

5.8. Antibiotic therapy

It is well accepted that patients with bowel resection due to intestinal infarction and peritonitis should be treated with broad spectrum intravenous antibiotics.[121] Unfortunately, many observational studies on the management of patients with AMI did not specify the use of antibiotics, which precludes an assessment of its effectiveness in patients with AMI without peritonitis.[177] One observational study of 67 patients showed that administration of oral gentamicin and metronidazole was independently associated with a decreased risk of irreversible intestinal necrosis in AMI.[178] Antibiotics must be given according to local protocol. No specific recommendation can be made as to which type of antibiotics should be administered since different antibiotics had similar clinical outcome.[179]

Recommendation 41 — Changed

Treatment with antibiotics is recommended for patients with acute mesenteric ischaemia.

Class	Level	References	ToE
I	C	Consensus	

5.9. Feeding

Critically ill patients such as those after revascularisation with or without bowel resection for AMI are at risk of complications when enteral or parenteral nutrition (PN) is

given. Nutrition is necessary, but increasing the metabolic intake may lead to an increase in the demand for mesenteric blood circulation that cannot be met, and may exacerbate intestinal ischaemia. PN can also increase intestinal ischaemia by hepatic steal. There is wide clinical practice variation in enteral or parenteral feeding. In an international survey, haemodynamic stability was considered a prerequisite for starting PN for 42% of respondents, who would start PN between immediately and within 72 hours.[121] Enteral feeding would be resumed by 44% after evidence of bowel activity. The European Society for Clinical Nutrition and Metabolism guidelines recommend delaying enteral nutrition in patients with overt bowel ischaemia and to implement PN within three to seven days.[181]

Another complication, especially in malnourished patients, is refeeding syndrome. Although there is no uniform definition of refeeding syndrome, it is characterised by depletion of potassium, magnesium, and phosphorus, triggered by high insulin levels after nutrition is resumed. The clinical symptoms of refeeding syndrome are mostly weakness, encephalopathy, and cardiac failure. Monitoring potassium, magnesium, and phosphorus, as well as thiamine supplementation, are recommended to prevent refeeding syndrome in the consensus document of the American Society for Parenteral and Enteral Nutrition.[182]

5.9.1. Intestinal failure. The patient's prognosis is poor if the entire colon and terminal ileum are resected and the remaining bowel is $<$ 100 cm long. However, if the colon and terminal ileum can be preserved, some patients may cope with as little as 30 cm of small bowel. Transitory intestinal failure is common after major bowel resection, and PN should be started early to avoid the complications of severe malabsorption.[121] If long term intestinal failure, i.e., short bowel syndrome (SBS), develops, PN can be continued in the outpatient setting.[180] Intestinal failure does not necessarily mean the end of life for a patient surviving AMI and does not necessarily mean lifelong PN. Indeed, in an international observational study 25% of patients with SBS due to AMI were weaned from PN within two years.[183] In one study, 2% of all patients with AMI developed SBS and only 1% received PN after discharge. It was estimated that 5% of patients with AMI under 70 years of age could have been candidates for intestinal transplantation after resection of extensive bowel necrosis.[184]

6. NON-OCCLUSIVE MESENTERIC ISCHAEMIA

6.1. Background and definition

NOMI develops when the oxygen supply to the intestines is insufficient to meet its metabolic needs, e.g., caused by vasoconstriction in the non-occluded mesenteric vessels. The condition may be caused by low cardiac output owing to heart failure, hypovolaemic shock, medical therapy using vasoconstrictive medication, intra-abdominal hypertension (IAH), or abdominal compartment syndrome (ACS).

Although the concept of NOMI may seem straightforward, i.e., there is no occlusion of the mesenteric arteries, it is not easy to define. The main mechanism underlying NOMI is mesenteric vasoconstriction in response to reduced effective blood volume. NOMI is defined as "as a hypoperfusion syndrome that occurs when severe ischaemia of the intestines develops, despite the mesenteric arteries being patent. It is caused by either mesenteric vasoconstriction secondary to conditions such as heart failure, vasoconstrictive medication, and hypovolaemia or by increased intra-abdominal pressure".[1] However, NOMI may also develop in a patient with asymptomatic mesenteric atherosclerosis in shock. This combination may change a previously asymptomatic mesenteric artery stenosis into life threatening mesenteric ischaemia if hypotension, hypovolaemia, or ACS develops.

Table 7. Common clinical scenarios that may lead to non-occlusive mesenteric ischaemia (NOMI).

Cardiogenic shock needing inotropic and or vasoactive support or intra-aortic balloon pump
Post-operative period after cardiac surgery
Hypovolaemia during or after renal replacement therapy
Massive burn injury associated with hypovolaemia
Patients with mechanical causes such as intra-abdominal hypertension or abdominal compartment syndrome with or without trauma
Colonic ischaemia following ruptured abdominal aortic aneurysm repair (transient hypovolaemia or haemorrhagic shock)
Severe sepsis and shock
Toxic aetiologies such as cocaine abuse, digitalis, or vasoactive support

6.2. Epidemiology

NOMI is a rare and dangerous condition. There are few data on the incidence of NOMI in the literature, with a range of 0.9 — 2.0/100 000/year in studies from Finland and Sweden.[114,185,186] NOMI accounts for approximately 15% of all cases of AMI and carries an estimated mortality risk of 58.4% (95% CI 48.6 — 67.7%).[111] In the AMESI study, NOMI was found in 13.2% of all AMI cases and had in hospital and 90 day mortality rates of 72.7% and 74.5%, respectively.[115]

6.3. Diagnosis

In NOMI, promptness and accuracy of diagnosis are paramount to achieve decisive treatment, but this is difficult. While real life diagnostic management relies on a combination of physical examination, biomarkers, imaging, and endoscopy to detect NOMI, research studies only focus on a few elements at a time.[187]

In all the clinical scenarios listed above, the key to diagnosis is clinical suspicion and knowledge of which groups of patients are at risk of mesenteric ischaemia. Although rare, NOMI is a well known complication after cardiac surgery. In a prospective study of 865 cardiac surgery patients, the diagnosis was established through angiography in all patients with suspected NOMI and was confirmed in 78 (9%).[188] Renal insufficiency and age $>$ 70 years were pre-operative risk factors, but the odds ratios were higher for patients with intra-aortic balloon pump support or serum lactate $>$ 5 mmol/L.[188] In another study, 30 (0.3%) of 10 409 patients who underwent cardiac surgery died from NOMI.[189] Recent myocardial infarction,

intra-operative vasopressor use, and low mean intra-operative arterial pressure were associated with death due to NOMI. Old age, vasopressors, and high lactate levels were associated with NOMI in a cohort of 9 445 cardiac surgery patients, of whom 40 (0.4%) developed NOMI.[190]

Patients after ruptured abdominal aortic aneurysm repair are at risk of NOMI because of shock, IMA loss, IAH, and ACS. The consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome indicate that intra-abdominal pressure (IAP) >12 mmHg negatively affects bowel perfusion.[191] NOMI secondary to IAH or ACS can result in multi-organ failure, bowel gangrene, and death. The IAP should be measured routinely in patients with a risk of developing IAH and or ACS, followed by action according to a pre-defined protocol if IAH develops. Management of IAH and ACS is addressed in the 2024 ESVS clinical practice guidelines on the management of abdominal aorto-iliac artery aneurysms.[192]

As discussed in Section 5.3.3, there are no single biomarkers available to accurately confirm or rule out intestinal ischaemia, including lactate and D dimer. While no individual parameters provide sufficient diagnostic accuracy, a multimodal approach relying on artificial intelligence (AI) algorithms might increase the speed and accuracy of recognising NOMI. The combination of several biomarkers and imaging modalities and the study of their variation through time with the help of AI tools may, in the future, expedite the early diagnosis of NOMI and improve therapeutic management.[187]

Recommendation 42			Changed
The single measurement of a biomarker such as lactate or D dimer to confirm or rule out the diagnosis in patients with a clinical suspicion of non-occlusive mesenteric ischaemia is not recommended.			
Class	**Level**	**References**	**ToE**
IIIb	C	Treskes *et al.* (2017),[124] Khan *et al.* (2019),[125] Reintam Blaser *et al.* (2023),[126] Blauw *et al.* (2024)[127]	

6.4. Imaging

The historical reference standard for diagnosing NOMI is angiography.[193] However, in current clinical practice patients with shock will have CTA as part of the diagnostic workup and not angiography. NOMI will be suspected on other grounds, such as lack of bowel wall enhancement, small bowel wall thickening, intestinal pneumatosis, pneumoperitoneum, and gas in the portal vein in the absence of atherosclerotic mesenteric artery stenosis or thromboembolic occlusion. In a retrospective study reviewing contrast enhanced CT examinations in 84 patients with a confirmed diagnosis of NOMI, dichotomous signs of reperfusion and quantitative CT parameters were associated with poor outcome of patients with NOMI.[194] The diameters of the CA, SMA, and inferior vena cava were wider in surviving patients than in those who died. In particular, the combination of the inferior vena cava diameter and difference in Hounsfield Units of the small bowel wall (late phase compared with unenhanced CT) had the highest prognostic value.[194] In 55 patients with NOMI, the mean diameter of the SMA decreased from 7.32 mm to 5.39 mm.[195] However, constriction of the SMA is difficult to assess unless there is a previous CT scan for comparison. Also, in another retrospective study of 21 consecutive patients with DSA proven NOMI, there were no differences in mesenteric vessel diameters on CTA between survivors ($n=8$) and non-survivors ($n=11$).[196]

Recommendation 43			New
Non-occlusive mesenteric ischaemia should be suspected in patients in shock if computed tomography angiography with contrast enhancement in arterial and venous phases with ≤1 mm slices (and optional non-contrast CT run) shows signs of lack of bowel enhancement, small bowel thickening, intestinal pneumatosis, or gas in the portal vein, in the absence of a significant stenosis of the superior mesenteric artery.			
Class	**Level**	**References**	**ToE**
IIa	C	Bagnacci *et al.* (2022),[194] Pérez Garcia *et al.* (2018),[195] Miyazawa *et al.* (2020)[196]	

6.5. Treatment

The cornerstone of shock treatment is correcting the hypovolaemic state and organ hypoperfusion by any means, including volume replacement and vasoactive drugs. In a population based study on fatal NOMI, the autopsy rate was 87%.[185] In this study, 25/62 patients (40%) with non-occluded vessels had significant narrowing of the SMA that could have been treated by stenting. In a series of 121 patients who underwent laparotomy for NOMI, approximately 40% had a $>25\%$ SMA stenosis.[197] Among other factors such as use of catecholamines, the SMA calcium score was associated with death within 24 hours after CTA. These observations might have implications for potential treatment such as endovascular revascularisation of low grade SMA stenoses, although there are no clinical studies to support this.

In patients with IAH or ACS, a proactive approach to reduce IAP with medical treatment, followed by decompression laparotomy when indicated, may be life saving. This is particularly true if the patient has undergone resuscitation for shock, heart failure, sepsis, or major haemorrhage, since the burden of IAH or ACS is added to the hypoperfusion episode the patient has already experienced from the underlying condition. In patients with ACS, decompression laparotomy has been shown to effectively reduce IAP and to improve oxygenation and urinary output.[198] However, death after decompression laparotomy was still as high as 49.7%. The authors found a weak correlation between delay in surgery and death and thus suggest prompt operation.

Recommendation 44			Changed
Urgent decompression laparotomy is recommended for patients with suspected non-occlusive mesenteric ischaemia and concurrent abdominal compartment syndrome.			
Class	**Level**	**Reference**	**ToE**
I	C	Van Damme *et al.* (2018)[198]	

6.6. Catheter directed therapy

Patients with NOMI may benefit from intra-arterial administration of vasodilators such as papaverine, nitroglycerin, prostaglandin E1, or glucagon through a catheter in the SMA. A bolus dose of 80 mg of papaverine is given directly into the SMA followed by a continuous infusion of 30 — 60 mg of papaverine per hour for 24 — 72 hours. There are no comparative studies evaluating different vasodilators, dosages, or regimens, making it impossible to issue precise recommendations. A systematic review included 12 retrospective studies on 245 patients with NOMI receiving local vasodilator therapy, mainly with papaverine (six studies) and prostaglandin (four studies).[199] The diagnosis of NOMI was established by DSA in most patients, however not with uniform criteria. The overall mortality rate in these patients was 40.3% (95% CI 28.7 — 53%), which is lower than expected. In four comparative studies, intra-arterial administration of prostaglandin E1 (three studies) or tolazoline (one study) was associated with a lower mortality rate than standard care (OR 0.261, 95% CI 0.095 — 0.712). This finding was corroborated in another non-randomised retrospective comparative study of 66 patients with NOMI where intra-arterial papaverine was associated with a lower mortality rate (23/35; 66%) than standard treatment (30/31; 97%).[200] In a study of 42 patients with NOMI, intra-arterial prostaglandin E1 gave a better reduction in lactate levels than standard care. The overall 28 day mortality rate was 30/42 (71.4%).[201] Of note, lactate is not a valid biomarker specifically for NOMI, but rather for hypoperfusion. Mortality was 59% in patients with a lowering of lactate $>$ 2 mmol/L compared with 85% with $<$ 2 mmol/L lactate lowering, which was a statistically significant difference.[201] It must be recognised that there is a risk of sudden death due to generalised hypotension when vasodilators are administered into the mesenteric circulation, but vasodilation is used as last resort to potentially save life.

Because of the loss of mucosal integrity, most authors suggest antibiotic treatment, which is supported by animal experiments. In the absence of bleeding, anticoagulation may be considered to prevent (micro)thrombi in narrow arteries. However, no controlled studies comparing different pharmacological agents exist.

Recommendation 45			New
Intra-arterial administration of vasodilators, such as papaverine or prostaglandin, in the superior mesenteric artery under protective heparinisation may be considered for patients with non-occlusive mesenteric ischaemia.			
Class	**Level**	**References**	**ToE**
IIb	C	Stahl *et al.* (2020),[199] Winzer *et al.* (2020),[200] Rittgerodt *et al.* (2022)[201]	

7. MESENTERIC VENOUS THROMBOSIS

7.1. Introduction

Acute mesenteric venous thrombosis (AMVT) is the least common cause of AMI. AMVT can occur with obstruction of the superior mesenteric vein (SMV), inferior mesenteric vein, splenic vein (SV), and portal vein (PV). The common definition of mesenteric venous thrombosis (MVT) is thrombosis within the SMV *with* or *without* extension into the PV or SV. Concomitant involvement of more than one venous segment is frequent, most often the PV and SMV, followed by the SV and inferior mesenteric vein.[202—204] AMVT may lead to venous mesenteric ischaemia (VMI), which is defined as the acute onset of symptoms in the presence of AMVT, and this is the main focus of the current guideline. Budd-Chiari syndrome with or without liver cirrhosis, isolated PV obstruction, isolated outflow obstruction caused by hepatic veno-occlusive (sinusoidal obstruction) disease, or hepatic disorders associated with congestive heart failure or chemotherapy are not covered in this guideline. Since the 2017 ESVS clinical practice guidelines were issued, four relevant reviews on AMVT have been published.[111,205—207] However, the current level of evidence for diagnosis and treatment of AMVT is low because only retrospective case series with a high risk of bias have been published. In 2020, the American College of Gastroenterology issued clinical guidelines on disorders on the hepatic and mesenteric circulation in which MVT was addressed.[208]

7.2. Epidemiology

AMVT is a rare condition that accounts for 11.5% (95% CI 9 — 14%) of all cases of AMI and for 1 in 1 000 emergency department admissions.[111] In a population based study in Sweden including 402 patients with an autopsy rate of 87%, the estimated incidence of VMI was 2.7/100 000 person years.[209] In Finland the incidence of VMI was 0.5/100 000 person years.[114] The mean age of patients at presentation was 45 — 62 years, and the minority were female (34.3%, 95% CI 30.5 — 38.5%).[205] In a worldwide, multicentre, prospective observational study, the proportion of acute VMI was 0.004% (95% CI 0.002 — 0.007%) of all adult hospital admissions.[115]

7.3. Diagnosis

7.3.1. Risk factors. Most MVT can be considered as provoked due to the presence of permanent risk factors, such as solid cancer, liver cirrhosis, and myeloproliferative diseases, or transient risk factors such as abdominal inflammation or infection, recent surgery, hormonal therapy, and trauma. In the 20 — 30% of patients in whom the underlying aetiology cannot be identified, AMVT is considered unprovoked or idiopathic. A systematic review including 1 725 patients from observational studies found pooled prevalences of liver disease (27.8%), cirrhosis or portal hypertension (28.8%), pancreatitis (11.1%), and inflammatory bowel disease (10.0%) as risk factors in patients with MVT.[207]

A thorough investigation can identify one or more systemic prothrombotic factors in approximately 60 — 70% of patients with AMVT, and further local triggering factors in as many as 30 — 40% of cases.[202,204,210] However, currently available investigations fail to identify a causal factor in approximately 20 — 30% of patients.[202,211,212] In a systematic review of 14 studies, a higher prevalence of prothrombin *G20210A* gene mutation and antithrombin and protein S deficiency was found compared with the general population and patients with a single venous thromboembolic event (VTE).[213] AMVT is a

common first clinical manifestation in patients with newly diagnosed myeloproliferative diseases such as polycythemia vera or essential thrombocytosis. The *JAK2* mutation is diagnostic of myeloproliferative diseases and appears to be linked to the development of AMVT. A meta-analysis reported a *JAK2* mutation prevalence of 32.7% (95% CI 25.5 — 35.9%) in patients with AMVT compared with approximately 1% in patients with VTE in other sites, and found a strong association between *JAK2* mutation and MVT (OR 53.9, 95% CI 13.1 — 222.5).[214] Moreover, 52% (95% CI 38 — 67%) of patients with MVT and *JAK2* mutation were diagnosed with a myeloproliferative disease during follow up, with MVT being the first manifestation of the underlying condition.[214] Therefore, it is important to screen for *JAK2* V617F mutation in patients with MVT without evidence of other major systemic or local risk factors.

7.3.2. Laboratory testing. Like arterial mesenteric ischaemia, there are no specific and reliable biomarkers available to diagnose MVT. Cohort studies found a high prevalence of thrombophilia in patients with MVT including over representation of Factor V Leiden and prothrombin *G20210A* gene mutation, or deficiencies of antithrombin, protein C and protein S, and antiphospholipid antibodies including Lupus anticoagulants.[213] It is as yet unclear to what extent these factors have implications for prognosis and duration of therapy after MVT. However, with this high prevalence of prothrombotic factors, many experts tend to offer patients with permanent risk factors indefinite anticoagulation, despite the lack of evidence for such treatment. The American Society of Hematology guidelines recommend conditional testing for thrombophilia in patients with MVT who would discontinue anticoagulation after three to six months, with a very low certainty of evidence about effects.[215] In patients with MVT and another indication for anticoagulation for other reasons, there is no indication to test for thrombophilia.[215]

Patients with recurrent venous and or arterial thrombosis or with recurrent foetal loss may be considered for testing for antiphospholipid antibody syndrome, including Lupus anticoagulants, anticardiolipin antibodies, β2 glycoprotein, and deficiencies of antithrombin, protein C, and protein S.[216] Although rare, cytomegalovirus has been found to be associated with AMVT and might act synergistically in patients with a *G20210A* mutation.[217] A similar association was found between severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection and AMVT.[218,219]

Recommendation 46			Unchanged
Investigation for the presence of an intra-abdominal malignancy, inflammatory disease, myeloproliferative neoplasm, cytomegalovirus and SARS-CoV-2 infection, and chronic liver disease is recommended for patients with mesenteric venous thrombosis.			
Class	**Level**	**References**	**ToE**
I	B	Thatipelli *et al.* (2010),[204] Primignani (2010),[210] De Broucker *et al.* (2022),[217] El-Hady *et al.* (2023),[218] Elkrief *et al.* (2023)[219]	

Recommendation 47			Unchanged
Patients with recurrent mesenteric venous thrombosis and recurrent foetal loss should be investigated for antiphospholipid antibody syndrome.			
Class	**Level**	**References**	**ToE**
IIa	C	Thatipelli *et al.* (2010),[204] Primignani (2010),[210] Elkrief *et al.* (2023)[219]	

Recommendation 48			Unchanged
Testing for thrombophilia may be considered in selected patients with acute venous mesenteric thrombosis who will discontinue anticoagulation treatment after three to six months.			
Class	**Level**	**Reference**	**ToE**
IIb	C	Middeldorp *et al.* (2023)[215]	

7.3.3. Clinical manifestations. MVT may appear as two different entities, *acute* and *chronic.* Both represent successive stages of the same disease and share similar causes but require different management. Patients with acute onset of symptoms within four weeks of presentation are arbitrarily classified as having AMVT. The mean duration of symptoms is often reported to vary from six to 14 days.[220] Chronic MVT (CMVT) is used to describe patients with symptoms lasting longer than four weeks, but without bowel infarction, or where MVT was an incidental finding on abdominal imaging.

Acute presentation accounts for 20 — 74% of cases, depending on the vein segment involved, and is more common if thrombosis involves the mesenteric veins.[204] After an acute onset, symptoms persist for two to three days in > 75% of cases before the diagnosis is made.[220]

SMV involvement, in contrast to isolated PV thrombosis, is associated with symptoms in 92% of cases and carries a 33 — 45% risk of developing haemorrhagic bowel infarction if left untreated. Abdominal pain, anorexia, and diarrhoea are the most common symptoms. Non-specific abdominal pain is often present in the early phase, whereas localised abdominal tenderness develops later. Fever and peritoneal signs are suggestive of progression of ischaemia to bowel infarction. High fever, malaise, abdominal tenderness, and sepsis are features of pylephlebitis (infectious thrombophlebitis of the PV and its branches), which may be accompanied by liver abscesses, and is a rare complication of intra-abdominal infections caused by appendicitis, pancreatitis, and diverticulitis.[202,210,221]

CMVT may manifest as portal cavernoma, where the obstructed PV is replaced by a network of collateral veins. Complete occlusion of the PV, or of its two main branches, is virtually always associated with portal hypertension and the development of portosystemic collaterals. The classical presentation of cavernoma with ruptured oesophageal and or gastric varices or biliary symptoms related to portal cholangiopathy (jaundice, cholangitis, cholecystitis, or pancreatitis) is rare.

7.4. Imaging

In patients with unexplained, serious, and or longstanding abdominal complaints, a three phase CTA with 1 mm

maximum slice thickness is indicated. CTA of the abdomen, with intravenous contrast injection and imaging in the arterial and portal venous phase, is the most important and accurate diagnostic tool and is the imaging investigation of choice.[222] Such a CT is accurate to confirm or refute the diagnosis of arterial or venous mesenteric ischaemia.[223] The venous phase CT can accurately visualise both the extent of thrombosis within the portomesenteric venous system and secondary abnormal intestinal findings (Fig. 5).

The assessment of "non-vascular findings" includes intestinal signs such as bowel wall thickening, bowel dilatation, mesenteric fat stranding, pneumatosis intestinalis, and portal venous gas.

Duplex ultrasound alone is insufficient to evaluate the extent of portomesenteric thrombosis and secondary intestinal abnormalities, and needs to be complemented by CT.[205] Depending on local expertise, diagnostic laparoscopy or laparotomy to assess bowel viability is necessary in some patients, especially those with peritonitis. However, the distinction between irreversible and reversible intestinal ischaemia is notably difficult, especially in AMVT.

Recommendation 49			Changed
Contrast enhanced computed tomography scanning with imaging in the arterial and portal phases (and optional non-contrast CT run) is recommended for patients suspected of mesenteric vein thrombosis.			
Class	**Level**	**References**	**ToE**
I	C	Salim *et al.* (2018),[222] Henes *et al.* (2017)[223]	

7.5. Treatment

The management of AMVT has changed over the recent decades, and the in hospital mortality rate has decreased to around 10%.[115,205] Nevertheless, there is no agreement on the optimal treatment strategy owing to the rarity of the condition and the absence of solid scientific evidence. The goal of AMVT treatment is to prevent thrombus propagation and to promote mesenteric vein recanalisation with the aim of preventing bowel infarction, portal hypertension, and thrombosis recurrence. In CMVT, additional goals are the prevention and treatment of gastrointestinal bleeding and portal cholangiopathy.

Most patients with AMVT are treated successfully with therapeutic anticoagulation and supportive treatment.[206,224] The size and extent of venous thrombosis largely affect the outcome, clinical presentation, and probability of bowel infarction, which mostly requires involvement of the venous arcades and vasa recta, which in turn causes complete venous occlusion. Endovascular treatment and surgery are the next steps in deteriorating patients.

7.5.1. Anticoagulation. In the absence of major contraindications, systemic anticoagulation should be initiated soon after the diagnosis is made, with unfractionated heparin or low molecular weight heparin to reduce the risk of thrombosis propagation, VTE recurrence, and overall mortality.[205,206,225] In the acute phase, unfractionated heparin carries the advantage that its action can be reversed by protamine if laparotomy is necessary or bleeding complications occur. Early anticoagulation results in recanalisation in 40 — 80% of patients, and complete recanalisation is associated with less extensive thrombosis.[2,210,226,227]

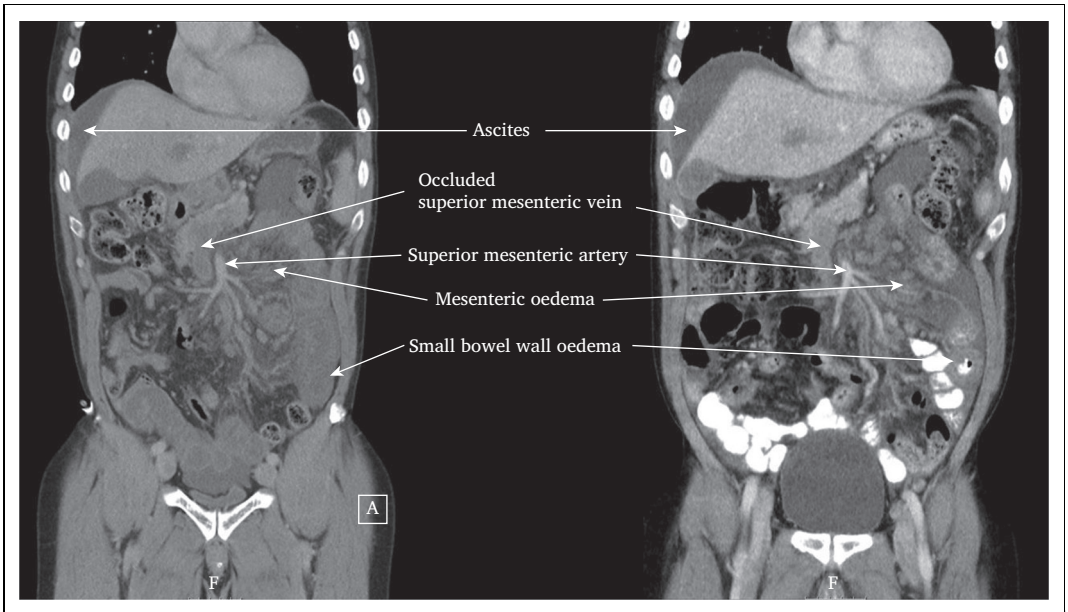

Figure 5. Schematic anatomy of mesenteric venous ischaemia. Computed tomography angiography of the abdomen showing extensive thrombosis of the superior mesenteric, splenic, extrahepatic, and intrahepatic portal venous system (on the left). Partial recanalisation (extrahepatic portal vein and a few branches of the superior mesenteric vein) using an endovascular procedure (on the right).

Recommendation 50			Unchanged
Anticoagulation with unfractionated or low molecular weight heparin as first line therapy is recommended for all patients with acute mesenteric vein thrombosis.			
Class	**Level**	**Reference**	**ToE**
I	C	Wang *et al.* (2022)[206]	

7.5.2. Supportive treatment. Pain control, fluid and electrolyte supplementation, and bowel rest should be initiated immediately. Nasogastric aspiration is used in case of ileus, abdominal distension, and intractable nausea and vomiting. Supportive treatment also includes blood transfusion for patients presenting with bleeding, and parenteral nutrition. The use of antibiotics has not been shown to be associated with a lower mortality rate or shorter hospital stay in two small retrospective studies, but is indicated if the patient suffers from bowel perforation, sepsis secondary to bacterial translocation, pylephlebitis, or septic thrombophlebitis.[228,229]

7.5.3. Endovascular treatment. Patients with persisting symptoms, worsening abdominal pain after initiation of anticoagulation, or developing signs of peritonitis may be considered for endovascular treatment, if necessary followed by diagnostic laparoscopy or exploratory laparotomy (Fig. 6). Recently developed endovascular procedures for the treatment of AMVT include transjugular intrahepatic portosystemic shunting (TIPS) with mechanical aspiration thrombectomy, direct thrombolysis or thrombolysis followed by angioplasty, percutaneous transhepatic mechanical thrombectomy, percutaneous transhepatic thrombolysis, and thrombolysis via the SMA. Rapid thrombus removal or dissolution can be achieved through these techniques.

Catheter directed thrombolysis can be administered by different approaches. An indirect approach with a catheter in the SMA is one possibility.[230] Thrombolysis by a direct venous approach has been used in more centres and may be more effective than via the SMA. Although technically challenging, both transjugular intrahepatic and percutaneous transhepatic approaches provide resolution of thrombus by direct access to mesenteric veins, after a mean of 40 hours.[231] In a systematic review of small case series (total 480 patients) and 159 cases of acute portal and MVT with heterogeneous presentation and lysis regimens (most often urokinase) in combination with anticoagulation, the pooled rate of complete recanalisation was 58% (95% CI 46 — 70%), with 18% (95% CI 7 — 32%) bleeding complications and 3% (95% CI 0 — 8%) requiring a bowel resection.[231] In another systematic review, the clinical effectiveness (death, complications, and symptom relief) was 89% with anticoagulation and 93% with endovascular treatment.[206] The level of evidence for the effectiveness of endovascular treatment of AMVT is low because of obvious selection bias and poor quality studies.[205,206]

More research is needed to assess the additional value of endovenous thrombolysis when anticoagulation fails in patients with extensive thrombosis of the mesenteric and or portal venous systems.

Recommendation 51			New
Endovascular venous thrombolysis and mechanical thrombectomy may be considered for patients with acute venous mesenteric ischaemia who deteriorate during anticoagulant therapy.			
Class	**Level**	**References**	**ToE**
IIb	C	Acosta *et al.* (2021),[205] Wang *et al.* (2022),[206] Gao *et al.* (2023)[231]	

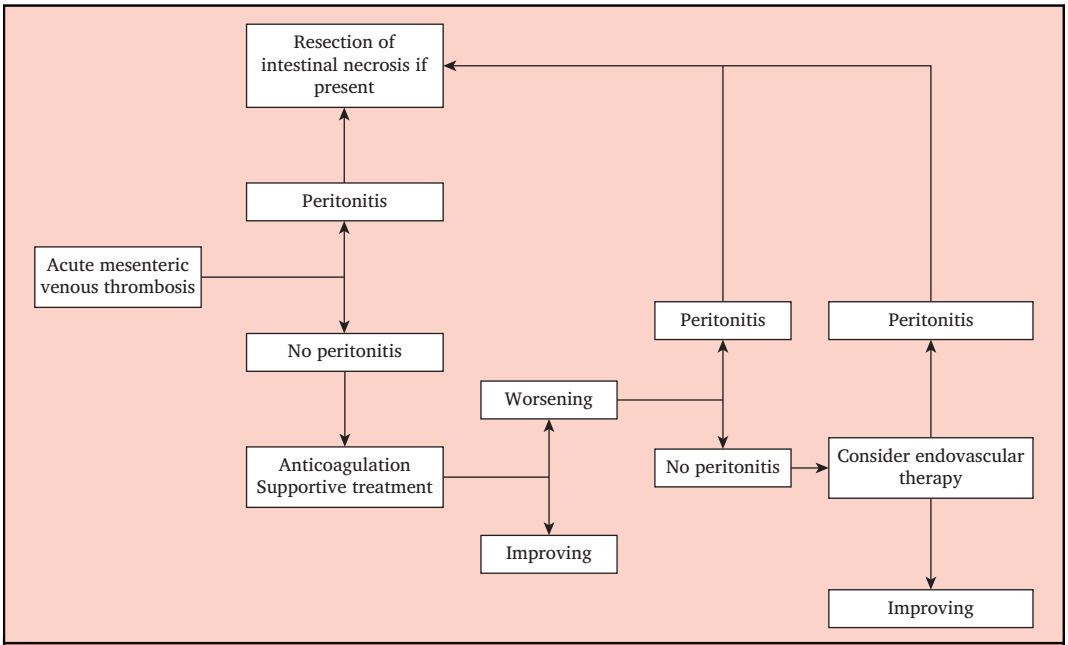

Figure 6. Suggested algorithm for the management of acute mesenteric venous thrombosis.[205]

7.5.4. Open surgery. Patients with persisting or worsening symptoms, organ failure, and those with perforation or signs of peritonitis require open surgical intervention. The aim of surgery is to remove irreversibly ischaemic bowel and preserve as much bowel as possible. The rate of bowel resection varies widely between studies and was 43.9% in a systematic review of 599 patients.[205] There are two difficulties in the surgical management of patients with AMVT: choosing when to perform laparotomy and deciding when a bowel segment is irreversibly injured. The recommendations in Chapter 5 regarding bowel resection, antibiotic treatment, and second look laparotomy are also valid for AMVT.

7.6. Long term treatment

After the acute phase of AMVT, anticoagulation should be maintained to prevent progression and recurrence of thrombosis, which is estimated to be 2.7% in one year.[215] Oral anticoagulation with a vitamin K antagonist (VKA) can usually be started two to three weeks after the onset of AMVT when the phase of acute ischaemic injury has passed. Historically, VKA is the drug of choice. Off label use of a DOAC after AMVT in small retrospective studies suggests that rivaroxaban and apixaban may be equally as effective as VKA or enoxaparin for this indication.[225,232] In a retrospective study of 102 patients with MVT with repeat CTA imaging, recanalisation was demonstrated after a median of six months in 71% of patients on VKA and 69% of patients on a DOAC.[225] Major bleeding rates were 14.3% and 9.1% for patients on VKA and DOAC, respectively. No significant differences in outcomes were found between patients on VKA or DOAC, except a higher gastrointestinal bleeding rate in patients using a DOAC. In a multicentre, prospective, single arm intervention study treating 96 patients with AMVT with rivaroxaban for three months, recurrent VTEs occurred within three months in two patients (2.1%, 95% CI 0.6 — 7.2%).[233] After three months, 11 patients stopped anticoagulation and 85 continued rivaroxaban. The cumulative VTE incidence after six months was 3.1% (95% CI 1.1 — 8.7%).[233]

Anticoagulation should be given for at least three to six months, or indefinitely in unprovoked MVT or if underlying persistent prothrombotic factors or thrombophilia are identified.[215] The optimal duration of anticoagulant treatment after AVMT is unknown, although there may be an argument to extend treatment beyond six months. In an individual patient data meta-analysis of observational studies of 1 635 patients with VMT with a mean anticoagulation use of 316 days, the recurrent VTE incidence rate was lower during anticoagulant treatment (3.4 per 100 patient years, 95% CI 3.2 — 3.6) than after treatment discontinuation (6.6 per 100 patient years, 95% CI 6.0 — 7.2).[212] VTE recurrence was in the mesenteric veins in 38.5% of cases. The major bleeding rate was 3.1 per 100 patient years (95% CI 2.9 — 3.3) in patients on treatment and 5.8 per 100 patient years (95% CI 5.3 — 6.4) after discontinuation. In this dataset, 31.9% of patients used low molecular weight heparin, 25.4% used a VKA, and only 1.7% used a DOAC.[212] Treatment effects were similar for provoked and unprovoked MVT. These results are in line with a meta-analysis showing a beneficial effect of anticoagulation on recanalisation (RR 2.39, 95% CI 1.66 — 3.44), major bleeding (RR 0.73, 95% CI 0.58 — 0.92), as well as overall mortality (RR 0.45, 95% CI 0.33 — 0.60) compared with discontinuation.[234] There was no difference in VTE recurrence (RR 0.91, 95% CI 0.44 — 1.87). In this study, 12.7% of patients used a DOAC.

In a multicentre, prospective registry of 604 patients with MVT, anticoagulation with parenteral treatment only or VKA was administered to 465 patients in the entire cohort (77%) for a mean duration of 13.9 months.[235] Two year follow up data showed that the incidence of thrombotic events was 5.6 per 100 patient years (95% CI 3.9 — 8.0) during anticoagulant treatment and 10.5 per 100 patient years (95% CI 6.8 — 16.3) after treatment discontinuation. The major bleeding rate was 3.9 per 100 patient years (95% CI 2.6 — 6.0) and 1.0 per 100 patient years (95% CI 0.3 — 4.2), respectively. The highest rates of both thrombotic events and major bleeding during the whole study period were observed in patients with cirrhosis (11.3 per 100 patient years and 10.0 per 100 patient years, respectively). The lowest rates were in patients with MVT secondary to transient risk factors.[235] A large cohort study of patients with isolated SV thrombosis showed the highest recurrence free survival at ten years (97%) and those with isolated MVT the lowest (60%).[204]

Extended anticoagulation must be considered during shared decision making, after carefully balancing (i) risk factors for bleeding (e.g., the presence of varices, low platelet count, and previous bleeding episodes), (ii) the risk of recurrence (e.g., persisting risk factors and previous venous thromboembolism), and (iii) the consequences of recurrence or progression (extensive bowel involvement and SBS). Patients with permanent risk factors for MVT or idiopathic MVT must be considered for indefinite treatment.[208,216]

Recommendation 52			**Unchanged**
Anticoagulation for three to six months with a vitamin K antagonist or low molecular weight heparin is recommended for all patients with acute mesenteric vein thrombosis.			
Class	**Level**	**References**	**ToE**
I	B	Candeloro *et al.* (2022),[212] Valeriani *et al.* (2021),[234] Ageno *et al.* (2015)[235]	

Recommendation 53			**New**
Anticoagulation for three to six months with a direct oral anticoagulant as an alternative to a vitamin K antagonist or low molecular weight heparin may be considered for all patients with acute mesenteric vein thrombosis.			
Class	**Level**	**References**	**ToE**
IIb	C	Salim *et al.* (2019),[225] Janczak *et al.* (2018),[232] Ageno *et al.* (2022)[233], Valeriani *et al.* (2021)[234]	

Recommendation 54			New
Extended anticoagulation beyond six months with a vitamin K antagonist should be considered for patients with acute mesenteric vein thrombosis and transient risk factors for venous thrombosis.			
Class	**Level**	**References**	**ToE**
IIa	C	Candeloro *et al.* (2022),[212] Valeriani *et al.* (2021),[234] Ageno *et al.* (2015)[235]	

Recommendation 55			New
Extended anticoagulation beyond six months with a direct oral anticoagulant as an alternative to a vitamin K antagonist may be considered for all patients with acute mesenteric vein thrombosis and transient risk factors for venous thrombosis.			
Class	**Level**	**Reference**	**ToE**
IIb	C	Valeriani *et al.* (2021)[234]	

Recommendation 56		Unchanged
Indefinite anticoagulation is recommended for patients with idiopathic acute mesenteric vein thrombosis and patients with permanent risk factors for venous thrombosis.		
Class	**Level**	**Reference**
I	C	Consensus

8. OCCLUSIVE RENAL ARTERY AND VENOUS DISEASES

8.1. Definition and epidemiology

Renal artery stenosis (RAS) refers to > 50% narrowing of at least one renal artery or its branches resulting in impaired blood flow to the kidney.[236–238] By consensus, a RAS > 70% is considered to be severe and 50 — 70% as moderate. As RAS is most commonly caused by atherosclerosis, its occurrence is related to the presence of common atherosclerotic risk factors.[236,237] The prevalence of RAS increases with age and has been estimated to be 6.8% in those older than 65 years (9.1% in men and 5.8% in women).[239] Approximately 10% of RAS cases are caused by fibromuscular dysplasia (FMD), which is "an idiopathic, segmental, non-atherosclerotic and non-inflammatory disease of the musculature of arterial walls, leading to stenosis of small and medium sized arteries."[240] FMD has a female preponderance, may have a genetic basis, and is associated with smoking and exposure to female hormones; however, its aetiology is largely unknown.[241] Whereas the prevalence of FMD in the general population is unknown, it varied between 2.3% and 6.6% in healthy live kidney donors[242] and was 5.8% in a retrospective assessment of the CORAL study participants.[243] The diagnosis is established with CTA or MRA and is characterised by focal or multifocal (string of beads) stenosis, most often in the middle part of the artery.

8.2. Clinical presentation

The most common clinical manifestations of RAS are arterial hypertension and ischaemic nephropathy, both related to activation of the renin—angiotensin—aldosterone system (RAAS).[237,244]

8.2.1. Arterial hypertension. Among patients with arterial hypertension, 5 — 15% are reported to have secondary hypertension.[245] Screening for secondary hypertension is suggested in younger patients, in acute worsening, severe, or treatment resistant hypertension, or when symptoms or biochemical characteristics suggest underlying renal or endocrine disease.[238] Renovascular disease as a cause of secondary hypertension is suggested in patients with atherosclerosis or its risk factors presenting with resistant hypertension, abdominal bruit, unexplained renal failure after treatment with angiotensin converting enzyme inhibitors (ACEIs) or angiotensin receptor blockers (ARBs), discrepancy in kidney size, or flash pulmonary oedema.[238,246] FMD may be suspected in younger female patients.

8.2.2. Ischaemic nephropathy. Atherosclerotic RAS is progressive.[247] Hypoperfusion or recurrent microembolism leads to significant loss of renal function, reported in 19 — 25% of patients,[243] or even renal artery occlusion or renal atrophy.[248,249] In a patient with unilateral RAS, the contralateral kidney has the ability to increase sodium excretion, preventing sodium retention and extracellular volume overload. In patients with severe bilateral RAS or with RAS in a single functioning kidney, on the other hand, renal failure is accelerated and fluid retention might cause acute flash pulmonary oedema.[250] This rare condition entails the clinical presentation of acute pulmonary oedema in patients with bilateral RAS, normal systolic cardiac function, and absence of coronary artery stenosis.[251] It is postulated that excessive RAAS activation in such patients leads to fluid retention and acute pulmonary oedema.

8.3. Diagnostic strategy for renal artery disease

8.3.1. Duplex ultrasound. DUS is the first line imaging investigation for the diagnosis of renal artery disease. DUS uses grey scale, colour, and spectral Doppler parameters, which provide anatomic and haemodynamic information to evaluate the renal artery and the kidney. Such methods allow identification and quantification of the degree of arterial stenosis and may help in therapeutic strategies by determining which patients might benefit from interventional treatment.

Direct and indirect DUS parameters have been described for the diagnosis and assessment of the degree of RAS.[252,253]

The direct criteria are (1) the PSV at the site of stenosis and (2) the ratio of the PSV at the site of renal artery stenosis to the PSV in the non-diseased aorta, also called renal aortic ratio.

The indirect criteria are (1) renal resistance index, which is usually measured in an interlobar artery and is obtained from the difference between the PSV and the EDV divided by the PSV and (2) waveform shape, flow acceleration, and delayed acceleration time (also known as *tardus parvus*), which is measured distal to the stenosis and is the delay in systolic rise from end diastole up to PSV.

Direct and indirect parameters can be used in combination to increase the diagnostic accuracy of DUS. Recent studies set a PSV cutoff for a significant (>50%) stenosis between 180 cm/s and 285 cm/s, with sensitivities and specificities ranging between 70% and 90% depending on the PSV cutoff value, i.e., selecting higher cutoff values results in lower sensitivity and higher specificity, and *vice versa*.[252,254,255] A renal—aortic PSV ratio >3.5 indicates a >60% stenosis, with varying sensitivities and specificities reported.[252,254] Furthermore, a side to side difference (reduction) in renal resistance index >0.05 has been reported as an indication of RAS.[252]

Ultrasound of the renal parenchyma may provide additional information in cases of suspected RAS with implications for therapeutic strategies. A normal kidney size and echogenicity of the renal parenchyma in the presence of RAS confirmed with spectral analyses (or other diagnostic methods) indicate that the renovascular disease may be at an early stage with a higher likelihood of a successful intervention such as stenting. In contrast, a small and or hypoechoic kidney is suggestive of a more advanced stage of renal artery disease for which intervention may not be effective.

In recent years, contrast enhanced ultrasound (CEUS) has been proposed to address the limitations of DUS and also offers additional diagnostic value with direct visualisation of the RAS. In a cohort study of 60 patients with 94 stenotic renal arteries, the sensitivity and specificity of CEUS were 95% and 84%, respectively, using DSA or CTA as reference standard.[256] In another study of 122 renal arteries with suspected RAS, the sensitivity and specificity of CEUS were 89% and 88%, respectively, with DSA as reference standard.[257] CEUS has also been used to assess the perfusion of the renal parenchyma and renal microcirculation, with a potential role in identifying individuals who might benefit from intervention for RAS.[258]

DUS is a widely accepted screening method in individuals with suspected renovascular hypertension because it is safe, non-invasive, and widely available, it has a low cost, and there is no need for nephrotoxic agents or radiation. Limitations include body habitus, overlying bowel gas, and osseous structures, which may make it difficult to visualise the renal artery, especially in the case of FMD where the stenosis is usually located in the middle third of the artery, as opposed to atherosclerotic stenosis that is located at the origin of the artery. Furthermore, DUS is limited by interobserver variation and the requirement for a certain level of expertise and should therefore be performed in vascular laboratories with experience in renovascular imaging (Table 8).

Limited bibliographic data have described intravascular ultrasound (IVUS) as an adjunctive diagnostic tool. It directly assesses atherosclerosis and remodelling of the arterial wall and has been proposed as a predictor of haemodynamically significant stenosis, based on minimum arterial lumen diameter measurements.[259,260]

Recommendation 57			New
Duplex ultrasound is recommended as the first line imaging investigation for patients with suspected renal artery stenosis.			
Class	**Level**	**References**	**ToE**
I	B	Schäberle *et al.* (2016),[252] AbuRahma *et al.* (2012),[254] Kawarada *et al.* (2006)[255]	

Table 8. Advantages of diagnostic imaging investigations in renal artery stenosis.

Advantage	DUS	CEUS	CTA	Gadolinium enhanced MRA	Non-enhanced MRA	Catheter angiography
Non-invasive	+++	+++	+++	+++	+++	−
Widely available	+++	−	+++	++	++	−
Low cost	+++	++	+	−	−	−
Low risk	+++	+	+	+	++	−
Accepted by patients	+++	++	+	+	+	−
Haemodynamic information	+++	−	−	−	−	+++
Functional assessment of blood flow and renal parenchyma	++	++	−	+++	+++	−
Information on mural calcification and thrombus	+	++	+++	−	−	−
Assessment of in stent stenosis	+	++	+++	−	−	+++
Assessment of middle and distal segment	+	+	+++	+++	+++	+++
Assessment of accessory renal arteries	−	−	+++	+++	+++	+++
Suitable for intervention planning	−	+	+++	+++	+++	+++
No risk of nephrotoxicity	+++	+++	−	−	+++	−
No radiation	+++	+++	−	+++	+++	−
Not limited by body habitus, bowel gas, or osseous structures	−	−	+++	+++	+++	++
Limited interobserver variation	−	+	+++	++	+++	+++
Limited performer experience dependent	−	+	+++	+++	+++	+++
Not limited by allergies	+++	++	−	+	+++	−
Not limited by implantable devices	+++	+++	+++	−	−	+++
Not limited by claustrophobia	+++	+++	+++	−	−	+++

DUS = duplex ultrasound; CEUS = contrast enhanced ultrasound; CTA = computed tomography angiography; MRA = magnetic resonance angiography; +++ = indicates the greatest advantage; − indicates no advantage.

Recommendation 58			New
Contrast enhanced ultrasound may be considered for patients with suspected renal artery stenosis and inconclusive duplex ultrasound, or with contraindications to computed tomographic or magnetic resonance angiography.			
Class	**Level**	**References**	**ToE**
IIb	C	Li *et al.* (2022),[256] Cui *et al.* (2020)[257]	

8.3.2. Computed tomographic and magnetic resonance angiography. CTA and MRA have evolved over the past few decades as the preferred non-invasive tests over DSA for the diagnosis and treatment planning of RAS. They should be used when interventional RAS treatment is considered in order to establish the diagnosis and to help plan the intervention. Reported sensitivities and specificities of CTA range from 64% to 100% and 62% to 98%, respectively, with DSA as the reference test.[261] CTA is widely available, has a high spatial and temporal resolution, allows multiplanar imaging of the renal arteries, provides information on vessel wall calcification and mural plaque, and is suited for the assessment of in stent stenosis. It is limited, however, by the need for radiation and may be contraindicated in individuals with chronic kidney disease owing to the risk of contrast induced kidney injury (Table 8).

Gadolinium enhanced MRA is an alternative imaging modality for the diagnosis and intervention planning of RAS, with the area under the ROC curve being 0.97, which is similar to that of CTA (0.99).[262] To evaluate diagnostic test accuracy, a meta-analysis of four studies reporting 486 individuals with suspected RAS found a summary sensitivity of 70% and specificity of 82% for gadolinium enhanced MRA and a summary sensitivity of 73% and specificity of 96% for CTA to detect a 50% RAS.[263] Recently, non-enhanced MRA has been found to have high sensitivity and specificity for significant RAS (respectively, 97% and 90% for 1.5 T and 96% and 91% for 3.0 T), with DSA as the reference standard, and has been proposed as an alternative to gadolinium enhanced MRA, thereby avoiding gadolinium related toxicity.[264,265] MRA has the additional advantage of providing a functional assessment of blood flow and organ function, which can help determine those patients who would benefit from revascularisation. On the other hand, MRA cannot be used in individuals with claustrophobia, implantable devices, or nephropathy, and is susceptible to artefacts from neighbouring metal or gas containing organs (Table 8).

Recommendation 59			New
Computed tomography angiography or magnetic resonance angiography are recommended over catheter angiography for the establishment of diagnosis and treatment planning for patients with suspected renal artery stenosis.			
Class	**Level**	**References**	**ToE**
I	B	Zhang *et al.* (2009),[261] Wang *et al.* (2021),[263] Guo *et al.* (2020)[264]	

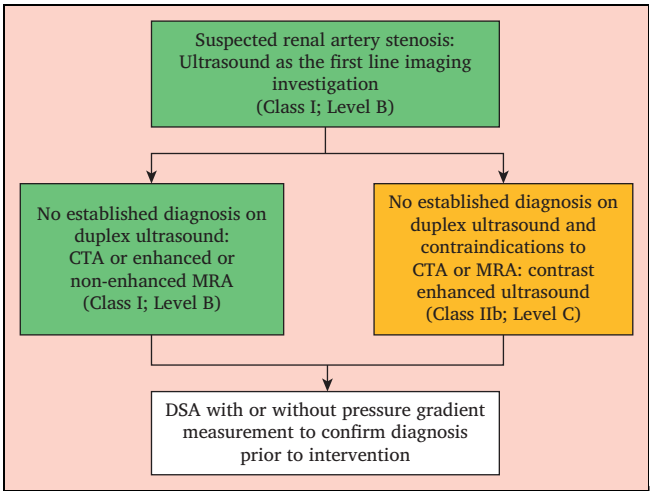

Figure 7. Diagnostic algorithm for renal artery stenosis. CTA = computed tomography angiography; MRA = magnetic resonance angiography; DSA = digital subtraction angiography.

8.3.3. Digital subtraction angiography. DSA has been considered the gold standard diagnostic investigation in individuals with suspected RAS. Catheter directed angiography also enables quantitative haemodynamic assessment through direct pressure measurement. A pressure gradient across the stenosis $>10\%$ of the mean arterial pressure has been proposed to signify a haemodynamically significant stenosis.[266] An expert consensus has determined a haemodynamically significant RAS to be present when the translesional mean pressure gradient is ≥ 10 mmHg or when the translesional peak systolic gradient is ≥ 20 mmHg.[267] DSA is an invasive diagnostic procedure that, apart from radiation exposure and contrast induced kidney injury, carries risks related to arterial access such as bleeding, arterial damage, arterial thrombosis, dissection, and pseudoaneurysm. With the advent of CT and magnetic resonance technologies, DSA is rarely used for diagnostic purposes and should rather be used as confirmatory imaging prior to intervention (Fig. 7).

8.4. Medical treatment in atherosclerotic renal artery stenosis

Risk assessment, lifestyle recommendations, and medical treatment of patients with atherosclerotic RAS should follow current guidelines from the European Society of Hypertension[238] and the European Society of Cardiology.[268]

Statin therapy is warranted in all patients with established atherosclerotic disease, including RAS.[268] A low density lipoprotein cholesterol (LDL-C) goal of <1.4 mmol/L (55 mg/dL) and an LDL-C reduction of $>50\%$ from baseline is recommended, and follow up is important to ensure that these targets are reached. According to the recent European Society of Cardiology/European Atherosclerosis Society guidelines, an LDL-C level <1.0 mmol/L (40 mg/dL) can be the target in patients with recurrent cardiovascular events (within two years).[268] Intensive lipid lowering has also been proven beneficial for renal function after renal artery stenting.[269] If LDL-C targets are not reached with maximum

tolerable statin doses, combination with other drugs such as ezetimibe (which has limited side effects in most, but not all, patients and is relatively inexpensive)[270] or proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors may be needed.[271,272] The latter are monoclonal antibodies that have limited side effects but have the drawback of high costs, parental administration, and at present there is very limited evidence on their costs effectiveness.

According to the ESVS 2023 clinical practice guidelines on antithrombotic therapy for vascular diseases, asymptomatic and symptomatic patients with RAS should have secondary prevention with single aspirin or clopidogrel antiplatelet therapy.[70] When RAS has caused hypertension through activation of the RAAS system, decreased glomerular filtration rate, and renal ischaemia, then antihypertensive treatment is warranted.[237] Blood pressure (BP) lowering reduces cardiovascular events and mortality rate, and current guidelines are also applicable for patients with hypertension caused by RAS.[238]

Pharmacological treatment is first choice and recommended at a BP $\geq$ 140/90 mmHg. As the optimal BP in patients with hypertension caused by RAS has not been established, target BP should be set at levels generally recommended for hypertensive patients: $<$ 130/80 mmHg in patients aged $<$ 65 years; $<$ 140/80 mmHg for patients aged 65 — 79 years; and systolic BP 140 — 150 mmHg in those $\geq$ 80 years of age, provided these levels are tolerated.[238] ACEI and ARB are first line therapies as they lower BP effectively and reduce morbidity and mortality in this group.[273—277] Most patients require two or more drug classes to achieve target BP, however, and calcium channel blockers and thiazide diuretics are recommended as first additions in such cases.[238] If the BP remains uncontrolled despite the combination of these three drug classes, spironolactone is a more effective addition than alpha or beta blockers in the general hypertensive population.[238] Pharmacological blockade of the RAAS system with ACEI or ARB might reduce glomerular capillary hydrostatic pressure and thereby cause a decrease in glomerular filtration rate, however, calling for caution and close follow up after institution of ACEI or ARB especially in patients with bilateral RAS or stenosis in a renal artery supplying a single functioning kidney, as renal failure might occur.[276—278] In the CORAL trial, only 46 — 62% of patients with atherosclerotic RAS used an ACEI or ARB, 37 — 58% used calcium channel blockers, and 64 — 75% used statins.[279] Medical management of patients with atherosclerotic RAS therefore needs to be improved.

8.4.1. Medical treatment in renal artery stenosis caused by fibromuscular dysplasia. In the first international consensus report on FMD, antiplatelet therapy with aspirin 75 — 100 mg daily in the absence of contraindications is recommended in FMD to prevent adverse cardiovascular events, since FMD may lead to stenosis, dissection, or aneurysm formation.[241] Statin or other lipid lowering treatment is not indicated for this indication. The recommendations regarding BP lowering are the same as in atherosclerotic RAS.[241]

Recommendation 60 — New

Pharmacological treatment of hypertension with the same blood pressure targets as in other hypertensive patients is recommended for patients with a renal artery stenosis with blood pressure ≥ 140/90 mmHg, provided these levels are without side effects: < 130/80 mmHg in patients aged < 65 years, < 140/80 mmHg in patients aged 65 — 79 years, and systolic blood pressure 140 — 150 mmHg in patients ≥ 80 years of age.

Class	Level	Reference	ToE
I	A	Mancia *et al.* (2023)[238]	

Recommendation 61 — New

Inhibitors of the renin—angiotensin system (angiotensin converting enzyme inhibitors [ACEIs] and angiotensin II receptor blockers [ARBs]) are recommended as first line therapy for patients with unilateral renal artery stenosis and hypertension. Calcium channel blockers and thiazide diuretics are recommended as first line additional therapies.

Class	Level	References	ToE
I	B	Mancia *et al.* (2023),[238] Losito *et al.* (2005),[273] Hackam *et al.* (2008),[274] Chianci *et al.* (2011),[275] Chrysochou *et al.* (2012),[276] Evans *et al.* (2014)[277]	

Recommendation 62 — New

Treatment with angiotensin converting enzyme inhibitors and angiotensin II receptor blockers may be considered for patients with bilateral severe renal artery stenosis or with a renal artery stenosis in a single functioning kidney, if without side effects and with regular follow up of renal function and blood pressure.

Class	Level	References	ToE
IIb	B	Chrysochou *et al.* (2012),[276] Evans *et al.* (2014)[277]	

8.5. Revascularisation for atherosclerotic renal artery stenosis

While many case series have reported a beneficial effect of percutaneous transluminal renal artery angioplasty and stenting (PTRAS), a less biased assessment of its effectiveness *vs.* control had to come from RCTs. A total of nine RCTs performed between 1985 and 2015 compared the outcomes of PTRAS and optimal medical therapy (OMT) *vs.* OMT alone in patients with atherosclerotic RAS. The two largest RCTs are summarised briefly. The ASTRAL RCT included 806 patients with uncontrolled or refractory hypertension or renal dysfunction, potentially attributable to substantial anatomical atherosclerotic stenosis in at least one of the renal arteries, and in whom there was clinical equipoise whether they could benefit from PTRAS.[280] Patients were randomly allocated to PTRAS plus OMT or

OMT alone. The primary endpoint was change in renal function; secondary endpoints were blood pressure, renal and cardiovascular events, and death. After a median follow up of 33.6 months, there were no significant differences in the primary and secondary endpoints between groups. ASTRAL has been criticised for the low technical success rate in the intervention group, with only 335/403 patients (83%) indeed receiving PTRAS, with a technical success rate of 95%.[281] The risk of procedural complications was as high as 9%. Another limitation was the 40% of patients with a 50 — 70% RAS, although a *post hoc* analysis in patients with a > 70% RAS did not reveal significant differences in clinical outcomes. The final analysis of ASTRAL after a median follow up of 56.4 months again showed no significant differences in endpoints.[282]

The CORAL RCT randomised 931 patients with an atherosclerotic RAS > 60% to PTRAS plus OMT *vs.* OMT alone.[278] The primary endpoint was a composite of major cardiovascular and renal events. After a median follow up of 43 months, the primary endpoint occurred in 35.1% in the intervention group and 35.8% in the OMT alone group. There were no differences in any of the secondary endpoints including progressive renal insufficiency (16.8% *vs.* 18.9%), permanent dialysis (3.5% *vs.* 1.7%), and mean number of antihypertensive drugs (3.3 *vs.* 3.5). Of note, the mean number of antihypertensive drugs at baseline was 2.1 in both groups. The total number of angiographic complications was 26/495 (5.3%). CORAL has been criticised for having included too many patients with a moderate RAS < 80% (33% even had a < 60% stenosis), too many diabetics, and unreliable pre-interventional imaging, thereby selecting patients who were unlikely to benefit from PTRAS because they had only moderate disease.

A 2021 systematic review and meta-analysis of the nine trials comparing PTRAS and OMT found no differences in the rates of renal and cardiac events, stroke, and cardiac and all cause mortality over a follow up of three to five years.[283] The only benefit of PTRAS was a reduction of the incidence of refractory hypertension within two years of follow up in a subset of three RCTs (OR 0.09, 95% CI 0.01 — 0.70). However, the difference in this subset was driven by the EMMA study, with refractory hypertension in 0/23 patients in the intervention group and 7/26 in the control group.[284] The latter were all patients who were treated for refractory hypertension within six months after randomisation. The EMMA study was small and biased, however, since the outcome assessment was not blinded.

Taken together, the RCTs comparing medical therapy alone or with PTRAS found that most patients with atherosclerotic RAS who have controlled hypertension and stable renal function on medical treatment do not benefit from PTRAS. The studies failed to define subgroups of patients who might benefit from PTRAS.

Recommendation 63 New

Percutaneous transluminal renal artery angioplasty with or without stenting is not indicated in patients with any degree of renal artery stenosis and controlled hypertension and renal function.

Class	Level	References	ToE
IIIa	A	Cooper *et al.* (CORAL study) (2014),[278] ASTRAL Investigators *et al.* (2009),[280] Chen *et al.* (2021)[283]	

8.6. Specific indications for renal artery revascularisation

8.6.1. Fibromuscular dysplasia. Hypertension in the context of FMD may be an indication for invasive treatment. There is an indication for percutaneous transluminal renal angioplasty (PTRA) when a pressure drop > 10% is measured between the renal artery and the aorta in patients with FMD and refractory hypertension.[241] This threshold is based on findings in patients with atherosclerotic RAS and has not been validated in patients with FMD. Renal artery stenting is not indicated in patients with FMD because of an increased risk of stent fracture and re-stenosis due to mobility of the renal arteries and the length of stents, which is different from patients with atherosclerotic RAS. Stents may be used as bailout in case of dissection or failed angioplasty. Lifelong antiplatelet therapy is mandatory after PTRA.

A 2010 systematic review summarised the outcomes of 1 616 patients with FMD treated with PTRA and 1 014 treated with surgical revascularisation for hypertension.[285] The pooled cure rates of hypertension, defined as BP < 140/90 mmHg without medication, were 36% for PTRA and 54% for surgery, with major complication rates of 6% and 15%, respectively. A 2022 systematic review and meta-analysis of 36 non-controlled studies comprising 1 191 patients with FMD found an overall hypertension cure rate by PTRA of 37% (95% CI 27 — 47%).[286] In the included studies, cure was defined as no antihypertensive drugs and either BP 140/90 mmHg, BP 165/90 mmHg, diastolic BP < 90 mmHg, or diastolic BP < 90 — 100 mmHg. All patients were treated with balloon angioplasty alone, except for 20 patients who had stent placement. When cure was defined as a BP of 140/90 mmHg without medication, the success rate of PTRA was 18.1% (95% CI 7.0 — 32.8%).[286] In a meta-regression analysis, older age and longer duration of hypertension were negatively associated with cure.[286] The pooled rate of major complications (death, transfusion, prolonged hospitalisation, or additional procedures) after PTRA was 4.6% (95% CI 2.5 — 7.2%).[286] Renal complications (renal failure, embolisation or infarction, and renal artery dissection, occlusion, thrombosis, perforation, or rupture) occurred in 7.0% (95% CI 3.3 — 14.8%).[286] Pooled improvement rates declined over time, being 83.5%, 73.8%, and 63.3% after one,

three, and five years, respectively.[286] Rates of freedom from re-stenosis followed a similar pattern, being 86.8%, 72.8%, and 70.9% after one, three, and five years, respectively.[286] PTRA had no effect on renal function.[286] In a recent study of 36 patients, IVUS found that patients with FMD and intima—media thickening had better BP lowering 48 hours after PTRA than patients with negative vessel remodelling.[287] The clinical implication of this finding is uncertain since long term follow up is missing.

Recommendation 64			New
Percutaneous transluminal renal artery angioplasty with bailout stenting should be considered for patients with resistant hypertension despite taking three or more antihypertensive drugs and a renal artery stenosis > 70% due to fibromuscular dysplasia.			
Class	**Level**	**Reference**	**ToE**
IIa	C	Tian *et al.* (2022)[286]	

8.6.2. Flash pulmonary oedema. In a systematic review of case reports and small case series, PTRAS of one renal artery prevented new episodes of flash pulmonary oedema by acute fluid retention due to kidney failure in 53/79 patients (67%).[288] PTRAS led to symptom relief in all 94 patients with congestive heart failure and renal insufficiency. BP and renal function improved when reported, but there were many missing data. The level of evidence for these findings is low due to the inherent biases of case reports and small case series without controls. Richie *et al.* compared the outcomes of medical treatment ($n = 25$) and PTRAS ($n = 12$) in patients with flash pulmonary oedema and atherosclerotic RAS.[289] PTRAS lowered the risk of death (HR 0.43, 95% CI 0.2 — 0.9), although patients managed medically had a mean age of 72 years compared with 62 years in the intervention group. In a follow up observational study by the same group, there was no difference in long term survival, hospitalisation for heart failure, or cardiovascular events in a comparison of 36 patients with medical management and 16 with PTRAS.[290]

Recommendation 65			New
Percutaneous transluminal renal artery angioplasty with stenting should be considered for patients with flash pulmonary oedema due to acute fluid retention caused by acute kidney failure and atherosclerotic bilateral renal artery stenosis > 50%.			
Class	**Level**	**Reference**	**ToE**
IIa	C	van den Berg *et al.* (2012)[288]	

8.6.3. Resistant hypertension. Although PTRAS is not indicated in patients with RAS and controlled hypertension, there may be a role in selected patients with uncontrolled hypertension. In the HERCULES study, 202 patients (71% requiring at least three antihypertensive drugs) with a RAS > 80% and a translesional pressure gradient > 10 mmHg on DSA underwent PTRAS. After three years there was a persistent reduction in mean systolic BP from 162 mmHg to 146 mmHg, however the mean number of antihypertensive drugs (3.4) was unchanged from baseline.[291] Renal function also remained unchanged. Most international scientific societies on the management of hypertension agree to some point that there is a place for PTRAS in selected cases, with recommendations based on consensus.[292—297]

Recommendation 66			New
Percutaneous transluminal renal artery angioplasty with stenting should be considered for selected patients with resistant hypertension despite taking three or more antihypertensive drugs and an atherosclerotic renal artery stenosis > 70%.			
Class	**Level**	**Reference**	**ToE**
IIa	C	Chrysant *et al.* (HERCULES) (2014)[291]	

8.6.4. Rapid deterioration of renal function. There are some case reports and cases series suggesting that PTRAS has benefits in patients with RAS and a rapid deterioration of renal function. In a retrospective study of 50 patients with hypertension or rapid decline in eGFR treated with PTRAS for RAS > 70% and a translesional gradient > 30 mmHg, the mean eGFR improved from 33.3 mL/min to 54 mL/min.[298] A prerequisite for PTRAS is kidney viability, indicated by a renal size > 8 cm, a distinct cortex > 0.5 cm, an albumin:creatinine ratio < 20 mg/mmol, and a renal resistance index < 0.8.

Recommendation 67			New
Percutaneous transluminal renal artery angioplasty with stenting may be considered for patients with an atherosclerotic renal artery stenosis > 70% and severe decline in renal function and with preserved kidney viability.			
Class	**Level**	**Reference**	**ToE**
IIb	C	Ramos *et al.* (2003)[298]	

8.7. Surgical instead of endovascular treatment?

The role of surgery has decreased due to the evolution of endovascular techniques and accumulated knowledge on indication for RAS revascularisation. However, when endovascular therapy is not feasible or has failed, a range of surgical solutions is available depending on the anatomy of the patient, including renal artery bypass with take off from the aorta, iliac, hepatic, or splenic artery, transposition of a renal artery to the aorta, endarterectomy and patch angioplasty, and autotransplantation after bench reconstruction. An analysis of studies published between 1975 and 2004 compared short term outcomes of surgical renal artery revascularisation and PTRAS.[299] Pre-interventional renal function was worse in surgical patients, who had a lower mean age than patients treated with PTRAS. Surgery conferred a greater improvement of hypertension control by 21% (95% CI 9 — 33%) and of renal function by 34% (95% CI 18 — 54%), as well as a greater creatinine reduction by 32 µmol/L (95% CI 7 — 57 µmol/L). The peri-procedural mortality rate was 3.1% (95% CI 1.8 — 4.4%) higher for all surgical *vs.* endovascular procedures, but was no longer statistically significant when

procedures with simultaneous aortic repair were excluded (0.18%, 95% CI −0.7 — 1.1%). A direct comparison of surgical and endovascular revascularisation is inherently biased because of variation in technical suitability as well as anatomic and patient factors. In one RCT, 22 patients were allocated to PTRA(S) and 27 patients to open surgery.[300] There were no significant differences in outcomes between groups, and both hypertension and renal function improved significantly after endovascular and surgical treatment.

Recommendation 68			New
Surgical reconstruction of the renal artery should be considered for patients with renal artery stenosis in whom revascularisation is clinically indicated when endovascular treatment is not possible or has failed.			
Class	**Level**	**Reference**	**ToE**
IIa	C	Abela *et al.* (2009)[299]	

8.8. Nutcracker syndrome

8.8.1. Introduction. Nutcracker syndrome (NCS) is an uncommon disorder due to extrinsic compression of the left renal vein (LRV) between the aorta and the SMA (anterior NCS) or between the aorta and the vertebral column (posterior NCS) that leads to distal dilatation of the LRV and an increase in renal vein pressure with venous hypertension. NCS must be distinguished from other causes of LRV compression such as lymphadenopathy, tumours, overarching testicular artery, intestinal malrotation, reduced retroperitoneal and mesenteric fat, pregnancy, and lordosis. Asymptomatic LRV compression is known as nutcracker phenomenon.

8.8.2. Clinical signs. NCS is most common in young and middle aged patients.[301,302] Symptoms and signs include microscopic (8.6 — 21.7%) and macroscopic haematuria (39.1 — 69.5%), abdominal flank, back, and pelvic pain (43.4 — 65.2%), proteinuria (4.3 — 26.1%), dyspareunia, dysmenorrhoea, orthostatic hypotension, and fatigue. Left sided varicocele in men and gonadal varices in women can develop due to renal and pelvic congestion in 8.7 — 21.7%.[303,304] Isolated left sided haematuria at cystoscopy supports the diagnosis.[303] The diagnosis of NCS is challenging and is generally established after excluding other more common pathological conditions such as urinary tract infection, nephritis, kidney stones, endometriosis, and autoimmune disease.

8.8.3. Diagnosis. DUS is the first line examination and has a sensitivity of 69 — 90% and a specificity of 89 — 100% to detect NCS.[305] DUS criteria include an aorta—SMA distance <10 mm that suggests the existence of LRV compression,[306] as well as a ratio >4 of the LRV diameter at the hilum and at the aortomesenteric angle.[304] A peak velocity ratio of LRV flow at the level of the aortomesenteric angle to the hilar portion of the LRV of 4.2 — 5 is considered to confirm the diagnosis of NCS.[307,308]

CTA and magnetic resonance venography can accurately demonstrate compression of the LRV, dilatation of the LRV proximal to the kidney, large tributaries of the LRV, gonadal vein distension, and pelvic congestion.[305,309] Other features are the beak sign and an SMA—aortic angle $<35°$.[303] Invasive assessment with venography combined with IVUS is sometimes required for a definitive diagnosis by demonstrating a >2 mmHg pressure gradient between the LRV and the vena cava, and to measure the LRV diameter at the level of the compression.[304,309]

8.8.4. Management. Most often observation is preferred in cases with mild symptoms, particularly in patients <18 years because during growth there is an increase in retroperitoneal fibrous and adipose tissue, which may release the obstruction at the level of the SMA origin and may also alter the position of the left kidney with consequently less tension on the renal vein. In two small case series, conservative management was successful in 50 — 68% of patients.[309,310] Invasive treatment may be considered for severe pain, repeated gross haematuria, and renal function damage. Open surgery is considered the standard of care for NCS and in most cases consists of LRV transposition 3 — 5 cm inferiorly on the vena cava, combined if needed with a greater saphenous vein patch or cuff to enlarge a narrow LRV. LRV transposition led to symptom relief in 90% of 47 cases, although ten patients needed angioplasty with or without stenting during follow up to maintain LRV patency.[309,311] The dilated ovarian or spermatic vein facilitates transposition of this vein into the inferior vena cava or left common iliac vein.[312] Robot assisted LRV transposition, nephropexy, renal autotransplantation, SMA transposition, and nephrectomy are alternative surgical options. Renal autotransplantation may also be used for cases of LRV re-stenosis after transposition.

Endovascular treatment by stenting of the LRV may offer a less invasive solution and has gained popularity due to the promising results in recent case series. However, endovascular treatment may not be preferable in younger patients. In a review of seven case series comprising 223 patients (70 female) who underwent LRV stenting, partial or complete symptom relief was reported in 76% (range 50 — 100%) of cases.[313] Haematuria resolved in 86% (range 60 — 100%) of cases. Follow up ranged between one month and 60 months during which nine patients had a re-intervention for in stent stenosis ($n=6$) or stent migration into the vena cava ($n=3$).[313] It is likely that the risk of migration is partially related to the stent diameter and length. Sizing of the stent is quite difficult because of the discrepancy between the normal renal vein (12 mm) and the LRV dilatation close to the renal hilum. The use of larger stents does not avoid migration, which has been reported for stents size from 10 mm to 20 mm. IVUS may be useful in choosing the most adequate stent diameter.

There is no consensus on post-operative anticoagulation after endovascular therapy: some authors suggest anticoagulation for two to 12 months and subsequent antiplatelet therapy, whereas others use DAPT for one to six months and subsequent SAPT.[302]

Another option is laparoscopic release of the complete LRV followed by reinforcement of the LRV by wrapping it with extravascular stent reinforcement. This technique has been

described in case series mainly from China. A review of four case series comprising 63 patients (nine female) reported symptom resolution in 83% (range 71 — 100%).[313] In a single centre case series of 76 patients who underwent laparoscopic extravascular stenting by a transperitoneal or retroperitoneal approach, complete symptom relief was achieved in 79%.[314] Currently there are no comparative studies available on surgery, endovascular, or laparoscopic treatments.

Recommendation 69			New
Conservative management is recommended for patients with an established diagnosis of nutcracker syndrome who have mild symptoms.			
Class	**Level**	**Reference**	
I	C	Consensus	

Recommendation 70			New
Open surgical treatment with left renal vein transposition should be considered for patients with an established diagnosis of nutcracker syndrome who experience severe symptoms.			
Class	**Level**	**References**	**ToE**
IIa	C	Velasquez *et al.* (2018),[303] Fuentes-Perez *et al.* (2023)[313]	

Recommendation 71			New
Endovascular stenting may be considered for selected patients with an established diagnosis of nutcracker syndrome who experience severe symptoms.			
Class	**Level**	**References**	**ToE**
IIb	C	Velasquez *et al.* (2018),[303] Fuentes-Perez *et al.* (2023)[313]	

9. VISCERAL ARTERY ANEURYSMS

9.1. Introduction

Visceral artery aneurysm (VAA) is a rare condition with an estimated prevalence of 0.01 — 0.2% in autopsy studies. The pathogenesis is poorly understood and is most likely related to atherosclerosis, abdominal trauma, infection, or inflammatory diseases such as panarteritis or polyarteritis nodosa. A minority may be associated with vascular Ehlers—Danlos syndrome, FMD, or Kawasaki disease. Increased blood flow may also contribute to the development of VAA.[315,316,340]

Most VAAs are incidental findings on abdominal CT or MRI. Since VAAs are detected more often due to widespread use of imaging, a careful and critical debate among physicians regarding indications for treatment is needed. Increased detection should not automatically lead to increased treatment rates, but rather to adjustment of clinically significant thresholds, to help physicians inform and treat patients.

While most VAAs remain stable over time, they may rupture and are challenging to diagnose and treat under emergency conditions.[317] Except for the splenic and renal arteries, the 2020 SVS clinical practice guidelines on the management of visceral aneurysms recommend early treatment of VAA regardless of the aneurysm diameter and the low level of evidence for treatment.[318] While assessing the risk of death in case of rupture, especially in patients such as pregnant females with splenic artery aneurysm, these recommendations are entirely understandable. However, the few follow up studies on VAA surveillance suggest that the risk of rupture is very low, and that the natural history of VAAs seems to be benign.[319,320] Accordingly, a reasonable cutoff for observational management between 20 — 30 mm has been suggested in the literature and in the 2020 SVS guidelines.[316,318,319]

Some aneurysm characteristics need to be considered, such as aneurysm size, growth, shape, and degree of calcification. Saccular aneurysms may be more prone to rupture than fusiform aneurysms of similar diameter owing to increased wall stress. Calcification may be interpreted as a sign of aneurysm chronicity.[322]

Treatment is aimed at preventing aneurysm enlargement and potential rupture by excluding the arterial circulation and maintaining necessary distal perfusion. Endovascular techniques are used more often than open surgery to treat VAA. However, an individualised approach considering aneurysm location and consequences for organ perfusion is always necessary.[323,324] Some studies showed similar rates of technical and clinical success as well as death during follow up for endovascular and open approaches.[325] However, peri-procedural morbidity is significantly higher after open surgery. Moreover, an analysis of 9 260 interventions in the USA showed that open treatment of VAA was associated with higher mortality (OR 1.70, 95% CI 1.03 — 2.80) and complication rates (OR 1.78, 95% CI 1.43 — 2.21) and longer hospital stay.[326] Endovascular treatment comprises different techniques such as coiling of the aneurysm, proximal and distal coiling of the artery, stent assisted coiling, balloon assisted coiling, stent graft, microvascular plugs, or flow diverting stents.[317] Depending on the location and aneurysm characteristics, open surgery techniques include bypass (venous or prosthetic), aneurysmectomy with end to end anastomosis, aneurysm resection with saphenous vein patch closure, or aneurysmorrhaphy and ligation.[327]

Treatment of symptomatic VAA should be based on clinical assessment, imaging findings, and informed consent. The level of evidence for this acute pathology, regardless of the vessel involved, is too low for robust recommendations. If a symptomatic aneurysm cannot be ruled out, urgent treatment is justified.[328] Mycotic VAAs are extremely rare and should be treated urgently because of the risk of rupture and sepsis. Patients typically present with abdominal pain, malaise, and fever. *Streptococcus* and *Staphylococcus* species are the most commonly isolated pathogens.[329] Open surgical correction and antibiotics are the preferred treatment for mycotic aneurysms.[329] If a patient is not fit for surgery, endovascular and or conservative treatment may be considered.

This chapter is focused on true aneurysms of the mesenteric and renal arteries. A separate section has been used for each vascular territory, which has several benefits, such as easier handling of the guideline for clinical practice and more explicit recommendations for the different VAAs with a better level of transparency and reliability regarding the

level of evidence. Pseudoaneurysms are not addressed in this guideline. The GWC could not identify any relevant data on medical treatment following surgery or follow up regimens. Accordingly, the recommendations focusing on these aspects are based on Level C evidence and consensus.

9.2. General recommendations

Recommendation 72	Unchanged	
Computed tomography angiography is recommended for diagnosis, anatomical characterisation, and procedure planning for patients with the suspicion of a visceral artery aneurysm.		
Class	**Level**	**Reference**
I	C	Consensus

Recommendation 73	Unchanged	
Urgent repair is recommended for patients with a symptomatic visceral artery aneurysm irrespective of size and location.		
Class	**Level**	**Reference**
I	C	Consensus

Recommendation 74	New	
Patients with a mycotic visceral artery aneurysm should be treated urgently by open surgical means and antibiotics.		
Class	**Level**	**Reference**
IIa	C	Consensus

Recommendation 75	New	
Endovascular treatment and antibiotics may be considered as a non-curative treatment option for patients with a mycotic visceral artery aneurysm who are unfit for surgery.		
Class	**Level**	**Reference**
IIb	C	Consensus

Recommendation 76	Changed	
Annual surveillance for the first three years and individualised thereafter, preferably with duplex ultrasound and otherwise computed tomography angiography, may be considered for patients with an asymptomatic visceral artery aneurysm with a diameter < 30 mm and for pancreaticoduodenal artery aneurysms with a diameter < 15 mm.		
Class	**Level**	**Reference**
IIb	C	Consensus

Recommendation 77	Changed		
Endovascular rather than open repair is recommended for patients with a visceral artery aneurysm who are anatomically suitable.			
Class	**Level**	**Reference**	**ToE**
I	C	Chin *et al.* (2017)[326]	

Recommendation 78	Unchanged	
Arterial reconstruction over occlusion when technically possible is recommended for patients with a visceral artery aneurysm if the patient is not at high risk for surgery.		
Class	**Level**	**References ToE**
I	C	Venturini *et al.* (2018),[323] Song *et al.* (2018)[324]

Recommendation 79	New	
Individualised surveillance with duplex ultrasound or computed tomography angiography in case of inadequate sonographic image quality should be considered for patients after endovascular or open visceral artery aneurysm repair.		
Class	**Level**	**Reference**
IIa	C	Consensus

9.3. Splenic artery aneurysm

Splenic artery aneurysms (SAAs) are considered the most common VAA, while the most updated literature reports a lower incidence compared with other entities.[315] SAAs are most often detected incidentally at a mean diameter of 16 — 17 mm.[316,330] CTA is the method of choice to assess SAAs, as certain limitations have been described for MRI and DUS, especially for small aneurysm diameters and because of shadowing from bowel gas in obese patients.[331]

The literature assessing the risk of SAA rupture is scarce. In a study of 38 patients with VAA, including 25 SAAs with a mean diameter of 20 mm, no interventions or ruptures occurred after a mean follow up of 31 months, although the dropout rate was 50%.[332] In another series of 41 patients with SAAs with an initial mean diameter of 19.9 mm who had serial CT scans, the growth rate was 1.08 mm/year after 42.5 months of follow up.[320] Two SAAs measuring 12 mm and 15 mm ruptured during follow up.

Endovascular and open surgical techniques are available to treat SAAs under elective and emergency conditions.[317] Endovascular treatment includes coil embolisation and exclusion with bare metal or covered stents. Open surgery includes ligation of the splenic artery combined with splenectomy in case of a proximal aneurysm or splenic artery reconstruction.[333,334] Recently, the feasibility of robot assisted ligation, resection, and end to end anastomosis or graft interposition has been reported in a literature review of 30 patients.[335] In a comprehensive review comparing the outcome of 511 open repairs, 385 endovascular repairs, and 425 conservatively managed SAAs, the mean SAA diameter in patients with an intervention was 30 — 31 mm, and was 21 mm in patients managed conservatively. No data on the natural history could be derived from this analysis, other than an intervention rate of 1.2% per year in patients with initial conservative management.[336] Mortality was significantly higher after open repair (5.1% *vs.* 0.6%), probably due to the higher proportion of ruptured SAAs in the open repair subgroup. Conversely, the re-intervention rate was higher after endovascular treatment than after open surgery (3.2% and 0.5%, respectively).

Recommendation 80			New
Endovascular or open surgical treatment should be considered for patients with an asymptomatic splenic artery aneurysm with a diameter ≥ 30 mm.			
Class	**Level**	**References**	**ToE**
IIa	C	Barrionuevo *et al.* (2020),[315] Battagini *et al.* (2021),[320] Saltzberg *et al.* (2005),[332] Lakin *et al.* (2011),[333] Hoogendoorn *et al.* (2014)[336]	

Recommendation 81			New
Surveillance with individualised imaging follow up should be considered for patients with an asymptomatic splenic artery aneurysm with a diameter < 30 mm.			
Class	**Level**	**References**	**ToE**
IIa	C	Barrionuevo *et al.* (2020),[315] Battagini *et al.* (2021),[320] Saltzberg *et al.* (2005),[332] Lakin *et al.* (2011),[333] Hoogendoorn *et al.* (2014)[336]	

9.3.1. Splenic artery aneurysm and pregnancy. SAAs in pregnant women require special attention. According to the *Maternal Deaths in Australia* reports, 2% of all 449 maternal deaths over a 21 year period were caused by SAA rupture.[337] A comprehensive review assessed nine cases of intact and 75 cases of ruptured SAAs in pregnant women published between 2000 and 2020.[338] The mean age at presentation was 31 years, and more than 50% presented only in the third trimester. For unruptured cases the mean SAA diameter was 28.2 ± 18.4 mm and for ruptured cases was 21.6 ± 12.3 mm. Patients with a ruptured SAA were treated by laparotomy in 81%, whereas intact aneurysms were excluded by embolisation or laparotomy. Maternal and foetal mortality rates in patients with a ruptured SAA were as high as 25% and 50%, respectively.

Recommendation 82			New
Endovascular or open surgical treatment should be considered for pregnant patients with an asymptomatic splenic artery aneurysm, regardless of aneurysm size.			
Class	**Level**	**Reference**	**ToE**
IIa	C	Aung *et al.* (2023)[338]	

Recommendation 83			New
Endovascular or open surgical treatment may be considered for female patients of childbearing age with an asymptomatic splenic artery aneurysm, regardless of aneurysm size.			
Class	**Level**	**Reference**	
IIb	C	Consensus	

9.4. Hepatic artery aneurysm

Hepatic artery aneurysm (HAA) is the second most common VAA, with a reported prevalence of 0.01% in autopsy studies.[339] In a single centre study at Mayo clinics, an HAA incidence of 0.002% per year was reported.[340] The aetiology is most often degenerative and atherosclerosis, while FMD, vasculitis, connective tissue disease, polyarteritis nodosa, and systemic lupus erythematosus have also been reported to be related to HAA.[339,341] HAAs are classified as extrahepatic or intrahepatic, with extrahepatic aneurysms being three times more common. HAA is commonly diagnosed in patients older than 60 years, 78% being extrahepatic, and 92% presenting as a single dilation, according to a single centre retrospective study.[340] This case series of 36 patients reported five ruptures of non-atherosclerotic HAAs. The mean diameter of symptomatic non-ruptured HAAs was 35 mm. Twenty two patients in this series with a mean HAA diameter of 23 mm (range 15 — 50 mm) who were managed conservatively did not experience rupture within a mean follow up of 68.4 months, although this was poorly documented. In general, surveillance with ultrasound, CTA, or MRI can be suggested for HAA with a diameter of 20 mm, since they tend to grow by 1 mm per year.[340,341] The risk of HAA rupture may be higher in females.[341] HAAs are traditionally treated by open surgical means preserving arterial flow, although endovascular techniques have evolved and include stent implantation and coil embolisation, depending on the anatomic configuration and the hepatic collateral circulation.[342] A Norwegian series of 57 patients with HAA over 20 years confirmed the trend towards endovascular repair.[343] There is currently no literature available to provide indications regarding either the type of treatment or thresholds for repair, hence we recommend the application of the general VAA recommendations.

Recommendation 84			New
Endovascular or open surgical treatment should be considered for patients with an asymptomatic hepatic artery aneurysm with a diameter ≥ 30 mm.			
Class	**Level**	**References**	**ToE**
IIa	C	Barrionuevo *et al.* (2020),[315] Erben *et al.* (2015),[339] Stark *et al.* (2022),[341] Melissano *et al.* (2018)[342]	

Recommendation 85			New
Surveillance with individualised imaging follow up should be considered for patients with an asymptomatic hepatic artery aneurysm with a diameter < 30 mm.			
Class	**Level**	**References**	**ToE**
IIa	C	Barrionuevo *et al.* (2020),[315] Erben *et al.* (2015),[339] Stark *et al.* (2022),[341] Melissano *et al.* (2018)[342]	

9.5. Coeliac artery, gastroduodenal and pancreaticoduodenal artery aneurysms

Literature assessing the prevalence, natural history, and outcome of interventions for coeliac artery aneurysm (CAA),

gastroduodenal artery aneurysm, and pancreaticoduodenal artery aneurysm (PDAA) is scarce. CAA rupture seems to be rare. In an observational study of various VAAs, only 3/73 (5%) CAAs with a mean diameter of 16 mm showed significant growth over a mean follow up of 44 months.[321] In a systematic review, only 27 interventions for (un)ruptured CAA were found, and four patients (14.8%) died.[315] In a series not included in the systematic review, nine patients had elective open repair with no deaths, and nine were managed conservatively, with one rupture after five years in a patient with a 25 mm CAA.[344] In another series of 19 patients with CAA (mean diameter 25 mm, range 10 — 52 mm), ten had endovascular repair with no complications after the intervention. Follow up data of the patients with expectant management were not given.[345]

The prevalence of PDAA is unknown, but it has been estimated that 1 — 2% of all VAAs are PDAAs. Although they are extremely rare, PDAAs are clinically significant because of a possible association with CA stenosis and the high risk of death in rupture cases. It has been postulated that in patients with a CA stenosis or occlusion, compensatory high flow in the communicating branches of the pancreaticoduodenal arcade may cause local hypertension and aneurysmal degeneration of these branches.[346] This is known as Sutton—Kadir syndrome. In a literature review of 125 patients treated for PDAA ($n = 105$), gastroduodenal artery aneurysm ($n = 10$), or both ($n = 10$) with concomitant CA occlusive disease and a mean aneurysm diameter of 21 mm, 48 patients (38%) had a rupture and six of them died (12.5%), as opposed to one (1.3%) elective patient.[347] In a retrospective study of 57 patients with a mean PDAA diameter of 29 mm and concomitant CA compression, 31 were treated by open median arcuate ligament release and exclusion of the PDAA (mean diameter 29 mm) and 26 had endovascular PDAA exclusion (mean diameter 27 mm) and CA stenting or laparoscopic median arcuate ligament release.[348] Mortality in both groups was 0%; however, endovascular treatment was associated with late aneurysm recanalisation (12%) and CA stent stenosis (40%). However, there are insufficient data to suggest that prophylactic treatment of CA stenosis can modulate the course of a PDAA, and this needs to be addressed in a prospective multicentre study. The same applies to CA revascularisation during surgery or intervention. Operative management has shifted from open surgery to endovascular and hybrid interventions over time. In a series of 59 patients with a PDAA, 22 patients were managed conservatively.[349] The mean aneurysm diameter at diagnosis was 14 mm (range 8 — 47 mm). Only eight of these patients had serial imaging, which detected stable aneurysms after a mean follow up exceeding five years and no ruptures reported. In the 19 patients presenting with rupture, the mean aneurysm diameter was 16.4 mm. Eight (42%) of these patients had significant CA disease. Although 16 patients could be managed by embolisation, the mortality rate was as high as 30%. The 18 electively treated patients had a PDAA with a mean diameter of 19.4 mm. Taken together, there is no association between PDAA diameter and rupture, and death after rupture was 30% as opposed to 5.6% for elective cases.

Recommendation 86 — New

Endovascular or open surgical treatment should be considered for patients with an asymptomatic coeliac artery aneurysm with a diameter ≥ 30 mm.

Class	Level	References	ToE
IIa	C	Barrionuevo *et al.* (2020),[315] Corey *et al.* (2016),[321] Mascia *et al.* (2022)[345]	

Recommendation 87 — New

Surveillance with individualised imaging follow up should be considered for patients with an asymptomatic coeliac artery aneurysm with a diameter < 30 mm.

Class	Level	References	ToE
IIa	C	Barrionuevo *et al.* (2020),[315] Corey *et al.* (2016),[321] Mascia *et al.* (2022)[345]	

Recommendation 88 — New

Endovascular or open surgical treatment should be considered for patients with an asymptomatic pancreaticoduodenal artery aneurysm with a diameter ≥ 15 mm.

Class	Level	References	ToE
IIa	C	Vandy *et al.* (2017),[347] Illuminati *et al.* (2021),[348] Stoecker *et al.* (2022)[349]	

9.6. Superior mesenteric artery aneurysm

The prevalence of true superior mesenteric artery aneurysms (SMAAs) is unknown, but these represent 3 — 6% of all VAAs.[313] While mycotic aetiology has been historically considered the most common cause, contemporary series have shown that a non-mycotic cause is the aetiologic factor in 92% of cases (atherosclerotic, collagen vascular disorder, cystic medial dysplasia, or polyarteritis nodosa).[350,351] In the light of the possible mycotic aetiology, patients with endocarditis and abdominal pain should undergo a CTA since it has been estimated that 2.5 — 10% of all episodes of endocarditis are associated with mycotic aneurysms. The distribution in both sexes appears to be equal, and some series suggest a higher incidence of rupture in males. There are virtually no data on the natural history of SMAA. One series of 13 patients reported no change in the initial diameter of 16.5 mm after a mean follow up of 57 months,[320] and similar results have been reported in another series of 11 patients.[321] Given their rarity, the natural history and risks associated with invasive therapy for SMAA are still poorly understood. The aggregated outcomes of several series suggest a mortality rate of < 10% following repair of intact aneurysms and < 40% for ruptured aneurysms, and a complication rate of < 20%.[351]

Patient comorbidities, aneurysm location, underlying aetiology, symptoms, and intestinal perfusion status are all factors influencing management. For mycotic aneurysms, the therapeutic objectives include avoiding rupture,

embolisation, and thrombosis while preserving visceral perfusion and managing the infective source. The SVS clinical practice guidelines recommend repair of all true SMAAs and pseudoaneurysms as soon as the diagnosis is made, regardless of size (Level of recommendation, Grade 1 [Strong]; Quality of evidence, A [High]).[318] The SVS recommendation was considering the potentially mycotic aetiology of SMAA. The GWC has not been able to replicate these recommendations. For an asymptomatic <30 mm SMAA, conservative management should be considered with imaging follow up with duplex ultrasound, CTA, or MRI.[328]

It is essential to evaluate intestinal perfusion and to take collateral visceral flow into account when deciding on interventional therapy. In patients with adequate collateral visceral circulation, the choice could be embolisation or ligation without revascularisation. In some cases, especially in emergencies, intra-operative assessment of bowel perfusion could be necessary, including visual inspection, Doppler ultrasound, ICGFI, and second look exploration, as discussed in Chapter 5.

Recommendation 89 New

Endovascular or open surgical treatment should be considered for patients with an asymptomatic superior mesenteric artery aneurysm with a diameter ≥ 30 mm.

Class	Level	References	ToE
IIa	C	Barrionuevo *et al.* (2020),[315] Shukla *et al.* (2015),[328] Stone *et al.* (2002),[350] Jacobs *et al.* (2021)[351]	

Recommendation 90 New

Surveillance with individualised imaging follow up should be considered for patients with an asymptomatic superior mesenteric artery aneurysm with a diameter < 30 mm.

Class	Level	References	ToE
IIa	C	Barrionuevo *et al.* (2020),[315] Shukla *et al.* (2015),[328] Stone *et al.* (2002),[350] Jacobs *et al.* (2021)[351]	

Recommendation 91 New

Endovascular or open surgical treatment should be considered for patients with a mycotic or symptomatic superior mesenteric artery aneurysm irrespective of the aneurysm diameter.

Class	Level	References	ToE
IIa	C	Barrionuevo *et al.* (2020),[315] Stone *et al.* (2002),[350] Jacobs *et al.* (2021)[351]	

9.7. Renal artery aneurysm

The prevalence of renal artery aneurysm (RAA) varies between 0.01% and 1%, but this information is biased because there is no dedicated cross sectional study.[352,353] In an autopsy study of 36 656 sudden deaths in a Swedish population, none was caused by ruptured RAA.[352] A mean annual growth of 0.86 ± 0.8 mm/year was found in a retrospective, multicentre study of 865 patients with RAA undergoing surveillance.[319] Growth did not appear to be associated with aneurysm morphology or calcification.

The diagnosis of RAA is most often incidental. However, RAA presentation might be acute with back or flank pain due to aneurysm enlargement, embolisation resulting in renal infarction, or aneurysm rupture. The diagnostic workup should be based on CTA, and imaging enhancement by post-processing reconstructions is recommended.[353,354] Non-contrast enhanced MRA may be considered when CTA is contraindicated.[355] Catheter based angiography is mainly used in interventional settings, however if implemented with 3D rotational application, may be useful to determine the morphological features of RAAs.

Contemporary RAA rupture rates are largely unknown. In two studies, growth rates were low and there were no ruptures in patients with a >20 mm diameter RAA who were managed conservatively.[352,353] A multicentre, retrospective study of patients with RAA reported a rupture rate of 0% in 547 patients (624 RAAs) over 55 months (mean 29 months) follow up, including 88 patients with a mean RAA diameter of 27 mm.[319] In a recent, single centre, retrospective study of 338 RAAs in 331 patients, no rupture was observed during 41 months follow up (281 patients). The median growth rate was 0.23 mm/year in the 31% of patients experiencing growth.[358] These studies suggest that a threshold for intervention for RAA of at least 30 mm is safe.

More than two thirds of patients with RAA have clinically relevant hypertension. The mechanism behind this is not completely understood considering that a stenosis cannot be demonstrated in all cases.[319] Nevertheless, clinical benefit has been demonstrated in more than half of those operated on for refractory hypertension.[319]

RAAs can be treated both by open surgery and endovascular techniques. The historical treatment for RAA included nephrectomy, and has more recently shifted towards *in situ* or *ex vivo* repair or autotransplantation, which mostly suits complex distal branch lesions.[359,360] Endovascular treatment encompasses coil embolisation, stent graft, and stent assisted coil detachment. These techniques have mainly been used to treat aneurysms involving the proximal segment of the renal artery. In a systematic review of 26 case series comprising 427 patients, endovascular RAA treatment was technically successful in 96%, with severe adverse events in 6.7% and no deaths.[361] Approximately 3% needed a secondary intervention due to aneurysm progression during follow up. A recent meta-analysis of observational studies comparing endovascular and open repair demonstrated no significant differences regarding short and long term mortality and re-intervention rates, incidence of end organ infarction, and respiratory complications as well as the length of hospital stay.[315] The largest retrospective study on RAA included 2 709 elective procedures from 2000 to 2011 (1 627 open and 1 082 endovascular repairs) from the US Nationwide Inpatient Sample.[362] A statistically significantly higher crude in hospital mortality rate was found for endovascular repair (1.8% *vs.*

0.9%; $p = .037$), but this was no longer statistically significant after multivariable adjustment for the presence of coronary artery disease, dysrhythmia, chronic renal failure, and peripheral vascular disease. A systematic review of case series (4 026 patients) showed no differences between endovascular, open, and autotransplantation repair of RAAs with mean diameters of around 20 mm regarding complications and mortality.[363] Length of stay was shorter after endovascular treatment, and nephrectomy occurred more often after autotransplantation.

Recommendation 92		New
Endovascular or open surgical treatment should be considered for patients with an asymptomatic renal artery aneurysm with a diameter ≥ 30 mm.		
Class	**Level**	**References** **ToE**
IIa	C	Klausner *et al.* (2015),[319] Wayne *et al.* (2014),[356] Brownstein *et al.* (2018),[357] Zhang *et al.* (2023),[358] Choksi *et al.* (2023)[363]

Recommendation 93		New
Annual surveillance with imaging should be considered for patients with an asymptomatic renal artery aneurysm with a diameter < 30 mm.		
Class	**Level**	**References** **ToE**
IIa	C	Klausner *et al.* (2015),[319] Wayne *et al.* (2014),[356] Brownstein *et al.* (2018),[357] Zhang *et al.* (2023)[358]

9.7.1. Renal artery aneurysm and pregnancy. Like SAA, pregnancy has been considered to be associated with an increased risk of RAA rupture, although pregnancy and a ruptured RAA at the same time is extremely rare. A recent literature review of 53 cases confirmed the high maternal and foetal mortality rates after RAA rupture.[364] A 5.1% maternal mortality rate has been estimated since the 1980s, with a significant decrease over the past decades. However, foetal mortality was 38% without a significant decrease over time.

Recommendation 94		New
Endovascular or open treatment should be considered for pregnant patients with a renal artery aneurysm, regardless of aneurysm size.		
Class	**Level**	**Reference** **ToE**
IIa	C	Augustin *et al.* (2019)[364]

10. ISOLATED DISSECTION OF THE VISCERAL ARTERIES

10.1. Introduction

This chapter concerns the diagnosis and management of spontaneous isolated dissection of the CA, SMA, and renal arteries. Dissections resulting from extension of a primary aortic dissection or trauma are different entities and are not considered here. Spontaneous isolated mesenteric artery dissection (IMAD) and isolated renal artery dissection are rare but increasingly diagnosed due to the wide availability of CTA. They can be encountered as an incidental finding on CTA in asymptomatic patients or in patients admitted with abdominal pain or suspected of mesenteric ischaemia. After publication of the 2017 ESVS clinical practice guidelines,

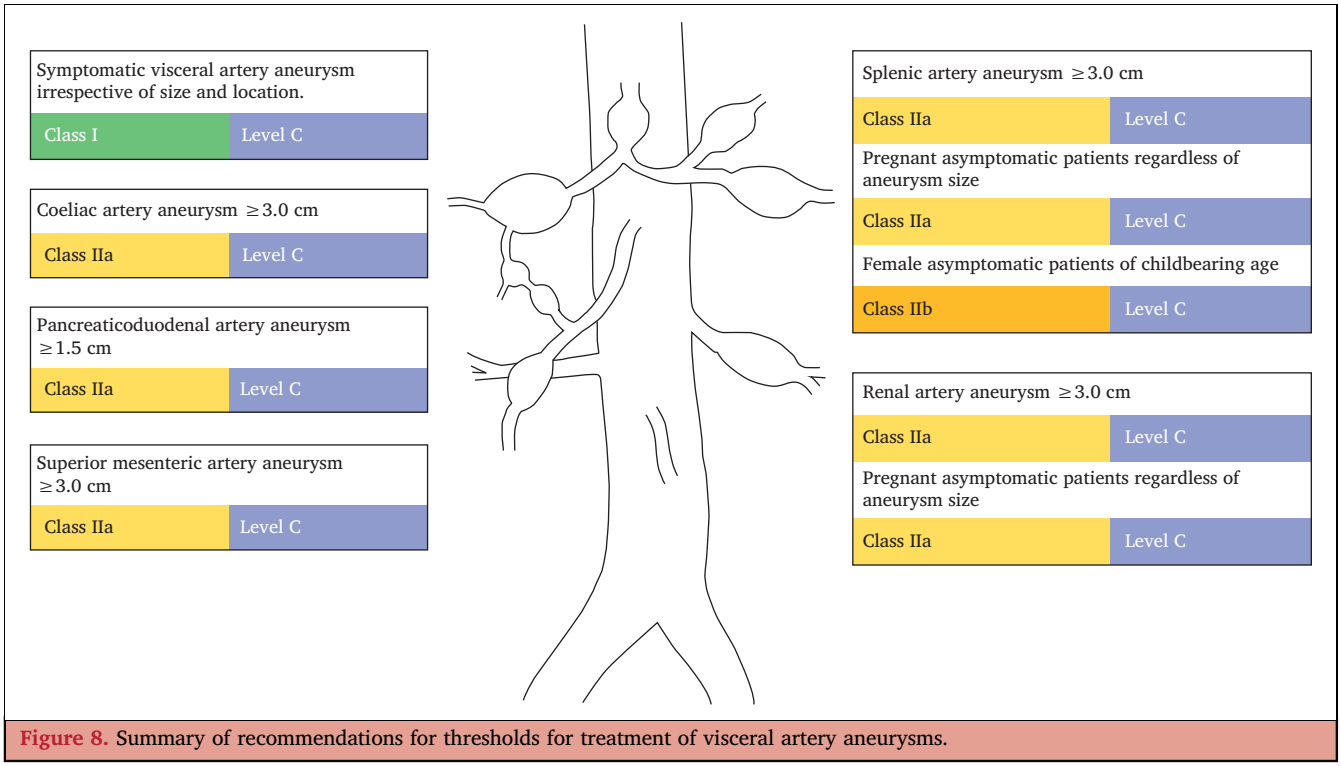

Figure 8. Summary of recommendations for thresholds for treatment of visceral artery aneurysms.

numerous systematic reviews of case series have been published, and these form the evidence base for this chapter.

10.2. Epidemiology

The prevalence and incidence of IMAD are unknown but seem to be higher in Asian countries such as China, South Korea, and Japan than in Caucasian populations, since most published studies are from that region. In a South Korean study among symptomatic patients admitted to the emergency department, the prevalence of IMAD was 0.96%, as opposed to 0.03% in a French study.[365,366] The aetiology of IMAD is as yet unknown. A systematic review of 97 case series (4 239 patients) found that patients with IMAD had a mean age of 54 years and most of them were male (88%, 95% CI 87 — 89%).[365] Regarding risk factors, 44% (95% CI 43 — 46%) had hypertension, 41% (95% CI 40 — 43%) were current or ex-smokers, and 7% (95% CI 6 — 8%) had diabetes. IMAD affected the SMA in 80.4% and the CA in 17.9%.[365] In another review of patients with IMAD, the dissection was located in the SMA in 60% (95% CI 50 — 71%) and the CA in 37% (95% CI 27 — 46%).[367] In a retrospective case series of 77 patients, 13% of IMAD seemed to be associated with segmental arterial mediolysis and FMD.[368] Segmental arterial mediolysis has a predilection for the mesenteric arteries and entails lysis of the outer media through formation of vacuoles in the cytoplasm of smooth muscle cells. Rupture of these vacuoles may damage the inner media, thus forming a possible entry site for dissection. Another hypothesis is damage to the SMA endothelium owing to high shear stress in patients with an aortomesenteric angle $> 70^{\circ}$.[366,369]

10.3. Diagnosis

Abdominal pain is the presenting symptom of IMAD in $>$ 90% of patients, and nausea, vomiting, and bowel distension may also be present. There are no specific laboratory tests to help in the diagnosis of IMAD, which can only be established by CTA. The combination of DUS and contrast enhanced DUS may be used as an alternative to CTA for IMAD surveillance.[370]

There are at least five classification systems for IMAD. The Sakamoto and Yun classifications are most often used in research studies to classify the extent of the dissection (Figs 9 and 10).[371,372]

10.4. Treatment

Patients with asymptomatic IMAD do not require intervention and can be treated medically to control hypertension, if applicable. Treatment of symptomatic patients with IMAD is aimed at prevention of bowel ischaemia (2.1%) or rupture of the artery (0.4%).[365] Although most patients present with acute abdominal pain, 82 — 85% fare well with conservative treatment.[365,373] Conservative management comprises food withdrawal, hydration, analgesia, and BP control in most studies, however no recommendation can be given with regard to optimal medical treatment. Close clinical observation is necessary, and when there is a suspicion of bowel ischaemia endovascular intervention is the treatment of choice. Anatomical considerations define the endovascular strategy, which will most often consist of stenting. The course of symptomatic IMAD is usually benign, with 16% of patients ultimately needing stenting of the affected CA or SMA, and in 2.3% open revascularisation may be necessary.[365] The risk of bowel resection is 2.1% in patients with IMAD of the SMA and is negligible in patients with a CA dissection.[365] Neither the Sakamoto nor the Yun classification can predict the clinical course, although in a French series all six patients with Yun type III SMA dissection needed revascularisation for bowel ischaemia.[374] IMAD carries an estimated mortality rate of 0.5%, which occurs most often in patients with bowel ischaemia or rupture. If endovascular treatment fails, surgical bypass is the next step, although other procedures such as intimectomy, thrombectomy, or patch angioplasty can also be performed, depending on the local anatomical situation.

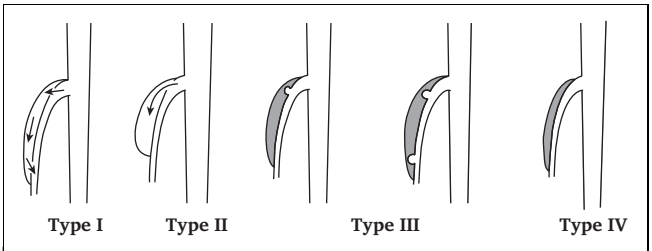

Figure 9. The Sakamoto classification describes computed tomography angiography findings as type I (entry and re-entry tear visible, patent false lumen), type II (only entry tear visible, patent false lumen), type III (thrombosis of false lumen and ulcer like projection), and type IV (occlusion of false lumen without ulcer like projection).[371]

The role of antithrombotic therapy in patients with IMAD is controversial. In a systematic review of observational studies on the benefits of antithrombotic therapy in patients with symptomatic SMA IMAD, 98/110 (89.1%) had an uneventful recovery after conservative treatment with antithrombotic therapy compared with 128/142 (90.1%) without (RR 0.96, 95% CI 0.87 — 1.05).[375] These findings corroborate the conclusions of a previous systematic review.[376] However, the 2023 ESVS clinical practice guidelines on antithrombotic therapy for vascular diseases have a consensus recommendation for antiplatelet therapy in patients with IMAD, which is followed in the current guideline.[70]

Recommendation 95 New

Conservative management, including blood pressure and pain control and bowel rest, is recommended as first line strategy for patients with asymptomatic or uncomplicated symptomatic isolated dissection of the superior mesenteric or coeliac arteries.

Class	Level	References	ToE
I	C	Acosta *et al.* (2021),[365] Shi *et al.* (2023)[367]	

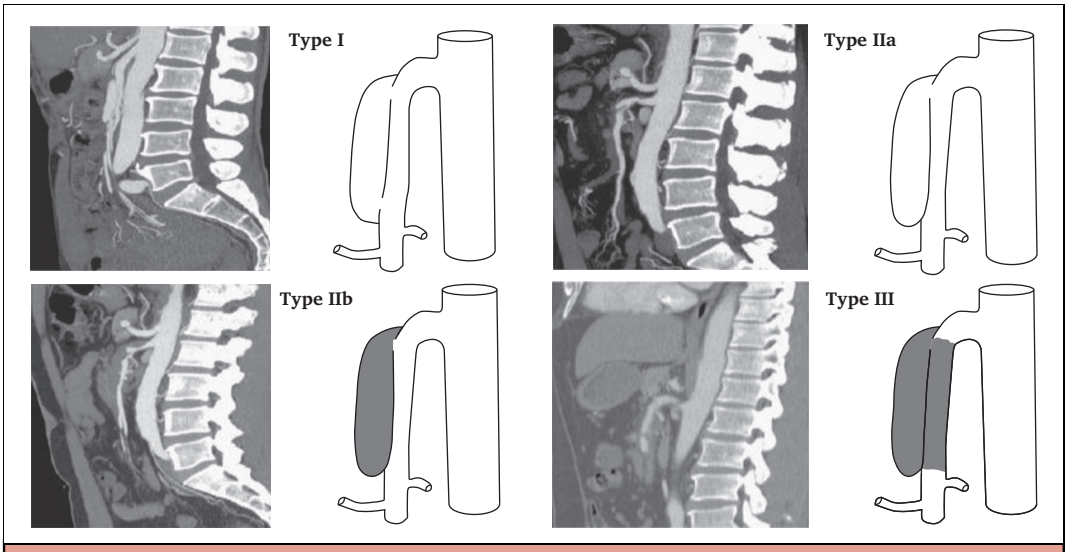

Figure 10. The Yun classification classifies the dissection of the visceral artery on computed tomography angiography as type I (entry and re-entry tear visible, patent false lumen), type IIa (only entry tear visible, patent false lumen), type IIb (thrombosis of false lumen, patent true lumen), and type III (occlusion of true and false lumen).[372]

Recommendation 96			Changed
Antiplatelet therapy should be considered for patients with asymptomatic or symptomatic isolated dissection of the superior mesenteric artery or coeliac arteries who are managed conservatively.			
Class	**Level**	**Reference**	
IIa	C	Consensus	

Recommendation 97			New
Primary endovascular or open revascularisation is not indicated in patients with asymptomatic or uncomplicated symptomatic isolated dissection of the superior mesenteric or coeliac arteries.			
Class	**Level**	**References**	**ToE**
IIIa	C	Acosta *et al.* (2021),[365] Shi *et al.* (2023)[367]	

Recommendation 98			Changed
Endovascular revascularisation is recommended for patients with symptomatic isolated dissection of the superior mesenteric or coeliac arteries not responding to medical management and with a suspicion of bowel ischaemia.			
Class	**Level**	**References**	**ToE**
I	C	Acosta *et al.* (2021),[365] Shi *et al.* (2023)[367]	

10.5. Follow up

Given the weakening of the dissected arterial wall, aneurysmal degeneration in the years following IMAD might be expected. DUS, CTA, or MRA are suitable modalities for surveillance. Patients with favourable remodelling and unchanged anatomy over time may be discharged from surveillance. Unfortunately, there are no studies with systematic and sufficiently long follow up to determine the natural course after IMAD. After conservative treatment, complete artery remodelling was seen in 43% (95% CI 43 — 47%) of 593 patients after a median follow up of 22 months with CTA.[365] Late interventions appear to rarely be necessary since in the same review there were only six interventions for occlusive disease and nine for post-dissection aneurysm in a total of 15 studies.[365] In a series of 190 patients with symptomatic IMAD, complete SMA remodelling occurred in 73/141 patients (51.8%) managed conservatively as opposed to 47/49 (96%) after endovascular treatment after a follow up of 37 — 42 months.[377] The probability of remodelling was lowest for Sakamoto type II compared with all other Sakamoto types (OR 0.34, 95% CI 0.13 — 0.91). Pain relief was reported in 88% after conservative and endovascular management. Endovascular stenting of SMA dissection has an 88% (95% CI 82 — 94%) chance of successful remodelling, however also a 12% (95% CI 4 — 23%) risk of stenosis after a mean follow up of 27 months and a 3% (95% CI 0 — 5%) risk of re-intervention.[378]

Recommendation 99			Unchanged
Annual imaging follow up with duplex ultrasound to detect aneurysm formation or progressive stenosis should be considered for patients with isolated dissection of the superior mesenteric or coeliac arteries who were managed conservatively or with endovascular stenting.			
Class	**Level**	**Reference**	**ToE**
IIa	C	Acosta *et al.* (2021)[365]	

10.6. Renal artery dissection

Spontaneous renal artery dissection (RAD) is an even more uncommon disorder than IMAD. RAD is associated with FMD, connective tissue disease, vasculitis, male sex, hypertension, and smoking and most often occurs in the fifth decade of life.[379,380] Complications of RAD are hypertension resistant to medical therapy, loss of renal function, aneurysm formation, or rupture. In a retrospective study of 73 symptomatic patients with RAD in Boston (USA), 74% were male, 40% had FMD, and 27% had hypertension.[380] The presenting symptom was flank pain in 96%. Sixty eight patients (93%) were managed medically with antihypertensive (52%), anticoagulant (55%), and antiplatelet therapy (46%). Six patients had a percutaneous transluminal angioplasty (two with stenting) during admission, and within 2.6 years one additional endovascular intervention was necessary.[380] No patients died due to dissection. Faucon *et al.* retrospectively reviewed 61 French patients with renal infarction and RAD due to FMD ($n = 16$), dissecting or aneurysmal multisite arterial disease ($n = 21$), and isolated RAD ($n = 24$).[379] Fifty two patients were managed with antihypertensive, anticoagulant, and antiplatelet therapy, whereas nine were treated with renal artery angioplasty and stenting because of uncontrollable hypertension or decline in renal function. During a median follow up of 51 months, 41 dissections remodelled, five developed a RAS, and 15 had mild aneurysmal dilatation. Patients with dissecting or aneurysmal multisite arterial disease were prone to develop new dissections of extrarenal arteries ($n = 8/21$). There were no long term complications of RAD in any of the groups. Although the clinical course of RAD appears to be benign, in a series of 23 patients in a single South Korean centre four patients (7%) died within a median follow up of 20 months due to rupture of a growing dissecting RAA caused by a significant true lumen stenosis.[381] Three of these patients had an endovascular procedure as initial treatment. Conservative management was successful in 14 patients (61%) in this series. A similar observation was made in small Chinese series of 13 patients with RAD, with one death due to aneurysm rupture and one after stent placement for ongoing symptoms.[382] There is no obvious explanation for the unfortunate clinical course in the studies from Asia, which may be different due to ethnic differences, but this is speculative.

In a comprehensive review of the case reports of 129 patients with a mean age of 43 years, 79% were male, 54% had hypertension, and 8.5% had FMD.[383] Pain (88%) and haematuria (15%) were presenting symptoms. In this dataset, 57% had conservative management with anticoagulation (45%) and antihypertensive drugs (69%), whereas 35% of patients had renal artery stenting because of deteriorating renal function or refractory hypertension. This high rate of interventions is probably a reflection of selection bias due to including case reports. Taking all studies together, it seems reasonable to follow a similar management strategy as in patients with IMAD.

Recommendation 100 — New

Conservative management with blood pressure control and antiplatelet therapy is recommended as first line strategy for patients with asymptomatic or uncomplicated symptomatic renal artery dissection.

Class	Level	References	ToE
I	C	Faucon *et al.* (2021),[379] Dicks *et al.* (2023),[380] Jeong *et al.* (2018),[381] He *et al.* (2025)[382]	

Recommendation 101 — New

Endovascular revascularisation is recommended for patients with symptomatic renal artery dissection and hypertension not responding to medical management.

Class	Level	References	ToE
I	C	Faucon *et al.* (2021),[379] Dicks *et al.* (2023),[380] Jeong *et al.* (2018),[381] He *et al.* (2025)[382]	

Recommendation 102 — New

Annual imaging follow up with duplex ultrasound to detect aneurysm formation or progressive stenosis should be considered for patients with asymptomatic and symptomatic renal artery dissection who were managed conservatively.

Class	Level	References	ToE
IIa	C	Faucon *et al.* (2021),[379] Dicks *et al.* (2023),[380] Jeong *et al.* (2018),[381] He *et al.* (2025)[382]	

11. UNRESOLVED ISSUES

Diseases of the mesenteric and renal arteries and veins are rare entities, which hampers the possibility of conducting clinical trials. Observational studies with high quality data may be a better way to increase knowledge and improve the evidence base for clinical practice. Compared with the previous version of these guidelines, it is obvious that more data are available but that the scientific standard of research has only improved moderately. In the current guidelines data from one RCT comparing outcomes of covered and bare metal stents for treatment of SMA stenosis[69] and important data from one large international observational study of management and outcomes of patients with AMI were available.[115] The only way to move forward is by international scientific collaboration in the form of high quality registries or RCTs and agreement on reporting standards. Below, some areas in which there is room for improvement are highlighted, which are more or less the same as in the 2017 ESVS clinical practice guidelines.[1]

11.1. Chronic mesenteric ischaemia

The selection of patients undergoing interventions for CMI must be improved, since approximately 15% of patients do

not benefit from revascularisation. Currently there is no accurate diagnostic test or combination of easy applicable functional tests that can prove the diagnosis of CMI. This is pivotal in patients with chronic abdominal symptoms fitting to CMI and especially in single vessel mesenteric disease.

Reporting standards, outcome definitions, and consensus descriptions of the intervention(s) should be the next steps aiming to improve the quality of research and to facilitate meta-analyses or meta-analyses of individual patient data.

Evidence is needed to determine whether it is sufficient to target one stenotic mesenteric artery for intervention or two for symptom relief in patients with CMI.

More research is needed on the effect of revascularisation on clinical outcomes such as pain, weight gain, and quality of life, instead of patency and re-intervention rates in the long run.

11.2. Median arcuate ligament syndrome

Reporting standards, outcome definitions, and consensus descriptions of the intervention(s) should be the next steps to improve the quality of research and care for patients with MALS. In the currently recruiting CARoSo study (NCT05468580), patients with MALS are randomly allocated to video assisted retroperitoneal endoscopic median arcuate ligament release or sham operation. Its outcomes will hopefully give an answer to questions regarding the benefit of surgery for MALS.

The definitive value of coeliac plexus blockade to improve patient selection for interventions for MALS has to be established.

11.3. Acute mesenteric ischaemia

Future observational studies should add more details on subtypes of AMI and severity of illness in each patient group in order to improve our understanding of the effectiveness of conservative and revascularisation strategies.

Oral antibiotics in patients with AMI were associated with improved intestinal preservation in an observational study.[178] The ORIAMI study (NCT06387147) has recently started and is an RCT comparing the relative efficacy of oral gentamicin, metronidazole, and placebo on intestinal preservation and survival in patients with AMI and a $>75\%$ stenosis in the SMA.

Data are needed to support whether revascularisation is indeed necessary for survival and preservation of bowel function in acute thromboembolic SMA occlusion. This is sometimes a controversy between general and colorectal surgeons who claim that most patients can be cured by bowel resection only, and vascular surgeons who claim the opposite.

The role of ICGFI guided assessment of bowel viability requires further study. The technique appears to be promising but is used in only 7% of centres.

There is uncertainty regarding the effectiveness of completion assessment after surgical embolectomy for AMI, and the modality that should be used.

11.4. Venous mesenteric thrombosis

More studies are required to assess the additional value of endovascular invasive treatments when anticoagulation fails in patients with extensive thrombosis of the mesenteric and or portal venous systems.

11.5. Visceral artery aneurysms

There is a lack of data on the natural history and outcomes after open and endovascular treatment of VAAs. Prospective registries such as the European Vascular Research Collaborative could be very useful to enable future studies assessing the natural course or outcome after open and or endovascular VAA repair. The indication for treatment of hepatic, coeliac, as well as gastroduodenal and pancreaticoduodenal artery aneurysms is not well established, and no clear recommendation for these particular entities was possible in these guidelines. Another open question is the effectiveness of regular follow up after interventions for VAA.

There are insufficient data to suggest that prophylactic treatment of CA stenosis can modulate the course of a PDAA, and this needs to be addressed in a prospective multicentre study.

11.6. Isolated dissections of the mesenteric arteries

The role of endovascular treatment as well as the duration and frequency of follow up examinations need to be defined in rigorous prospective cohort studies. The majority of outcome data are based on case series of Asian patients. Since the disease may behave differently in Asian and European populations, there is a need for multicentre collaboration in Europe, where the disease is less common.

APPENDIX A. SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ejvs.2025.06.010.

APPENDIX B. AUTHORS' AFFILIATIONS

Mark J.W. Koelemay (Chair), Department of Surgery, Amsterdam University Medical Center, Amsterdam, location AMC, the Netherlands; Robert Herman (Bob) Geelkerken (co-chair), Department of Vascular Surgery, Medisch Spectrum Twente, Enschede, the Netherlands, and Multi-Modality Medical Imaging Group, TechMed Centrum, University Twente, the Netherlands; Jussi M. Kärkkäinen (co-chair), Heart Center, Turku University Hospital, Turku, Finland; Nicola Leone (co-chair), Department of Vascular Surgery, Ospedale Civile di Baggiovara, Azienda Ospedaliero-Universitaria di Modena, University of Modena and Reggio Emilia, Modena, Italy; George A. Antoniou, Manchester Vascular Centre, Manchester University NHS Foundation Trust, Manchester, United Kingdom, Division of Cardiovascular Sciences, School of Medical Sciences,

Manchester Academic Health Science Centre, The University of Manchester, Manchester, United Kingdom; Jorg L de Bruin, Erasmus Medical Center, Department of Surgery, Rotterdam, The Netherlands; Alexander Gombert, Department of Vascular Surgery, University Hospital RWTH Aachen, Germany; Anders Gottsätter, Lund University, Department of Medicine, Skåne University Hospital, Malmö, Sweden; Elena Iborra, Bellvitge University Hospital, Barcelona, Spain Barcelona University, Barcelona, Spain, Bio-Heart-Idibell, Barcelona, Spain; Sonia Ronchey, ASL Roma 1 – San Filippo Neri Hospital, Rome, Italy; Konstantinos Spanos, Department of Vascular Surgery, School of Health Sciences, Faculty of Medicine, University Hospital of Larissa, University of Thessaly, Larissa, Greece; Jos C. van den Berg, Clinica Luganese Moncucco, Lugano, Switzerland and Universitätsinstitut für Diagnostische, Interventionelle und Pädiatrische Radiologie Inselspital, Universitätsspital Bern, Bern, Switzerland; Sabine H. Wipper, Department for Vascular Surgery, Medical University of Innsbruck; Frederico Bastos Gonçalves, NOVA Medical School|Faculdade de Ciências Médicas, NMS|FCM, Universidade Nova de Lisboa; Lisboa, Portugal and Hospital de Santa Marta, Unidade Local de Saúde São José , Centro Clínico Académico de Lisboa, Lisboa, Portugal and Hospital CUF Tejo, Lisboa, Portugal; Martin Björck, Department of Surgical Sciences, Uppsala University, Uppsala, Sweden and Institute of Clinical Medicine, University of Tartu, Tartu, Estonia; Raphaël Coscas, Ambroise Paré University Hospital, AP-HP, Boulogne-Billancourt and Versailles-Saint-Quentin Paris-Saclay Universities, Paris, France; Sandro Lepidi, Vascular and Endovascular Surgery, University of Trieste Hospital, School of Medicine, Trieste, Italy; Timothy A. Resch, Department of Vascular Surgery, Heart Center, Copenhagen University Hospital, Denmark and Faculty of Health Sciences, Copenhagen University, Denmark; Jean-Baptiste Ricco, University of Poitiers, Medical School, Poitiers, France; Riikka Tulamo, Helsinki University Hospital and University of Helsinki, Helsinki, Finland; Anders Wanhainen, Section of Vascular Surgery, Department of Surgical Sciences, Uppsala University, Uppsala, Sweden, and Department of Diagnostics and Intervention, Surgery, Umeå University, Umeå, Sweden; Olivier Corcos, Assistance-Publique/Hopitaux de Paris (APHP), Paris, France; Thomas S. Huber, University of Florida College of Medicine, Gainesville, Florida, USA; Alexander Oberhuber, Department of Vascular and Endovascular Surgery University Hospital Münster, Münster, Germany; Annika Reintam Blaser, University of Tartu, Tartu, Estonia, and Lucerne Cantonal Hospital, Lucerne, Switzerland); Matti Tolonen, HUS Helsinki University Hospital, Abdominal Center, Department of Abdominal Surgery, Helsinki, Finland.

Author contributions

ESVS Guideline Writing Committee: Mark J. Koelemay (Chair), Robert H. Geelkerken (Co-chair), Jussi Kärkkäinen (Co-chair), Nicola Leone (Co-chair), George Antoniou, Jorg L. de Bruin, Alexander Gombert, Anders Gottsäter, Elena Iborra, Sonia Ronchey, Konstantinos Spanos, Jos C. van den Berg, Sabine Wipper.

ESVS Guidelines Steering Committee: Frederico Bastos Gonçalves, Martin Björck, Raphael Coscas, Sandro Lepidi, Timothy A. Resch, Jean-Baptiste Ricco, Riikka Tulamo, Anders Wanhainen.

Document reviewers: Olivier Corcos, Thomas S. Huber, Alexander Oberhuber, Annika Reintam Blaser, Matti Tolonen.

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