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CLINICAL PRACTICE GUIDELINES DOCUMENT

European Society for Vascular Surgery (ESVS) 2026 Clinical Practice Guidelines on the Management of Vascular Graft and Endograft Infections

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Objective: The European Society for Vascular Surgery (ESVS) has developed clinical practice guidelines for the care of patients with vascular graft or endograft infection (VGEI), in succession to the 2020 version, with the aim of assisting physicians and patients in selecting the best management strategy.

Methods: The guidelines are based on scientific evidence complemented with expert opinion. By summarising and evaluating the best available evidence, recommendations for the evaluation and management of patients with VGEI have been formulated. The recommendations are graded according to the ESVS grading system, where the strength (class) of each recommendation is graded from I to III, and the level of evidence from A to C.

Results: Eighty-one recommendations have been issued across the following main topics: definitions, diagnosis, multidisciplinary team management, antimicrobial therapy, management of intracavity or extracavity VGEI, and follow up. A chapter addresses the concept of shared decision making, with supporting information for patients. A final chapter addresses unresolved issues.

Conclusion: These ESVS clinical practice guidelines provide comprehensive, up to date advice to clinicians and patients on the management of VGEI.

Keywords: Blood vessels prosthesis, Guideline, Infections, Vascular grafting

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ABBREVIATIONS

ABF	Aortobronchial fistula
AEF	Aorto-enteric fistula
AGREE	Appraisal of Guidelines for Research and Evaluation
AOF	Aorto-oesophageal fistula
APF	Aortopulmonary fistula
ASA	American Society of Anesthesiologists
AUF	Arterio-ureteral fistula
CEA	Carotid endarterectomy
cfDNA	Cell free DNA
CI	Confidence interval
CLTI	Chronic limb threatening ischaemia
CoNS	Coagulase negative staphylococci
CRP	C reactive protein
CT	Computed tomography
CTA	Computed tomography angiography
DUS	Duplex ultrasound
EANM	European Association of Nuclear Medicine
EJVES	*European Journal of Vascular and Endovascular Surgery*
EndoVAC	Endovascular vacuum assisted closure
ePTFE	Expanded polytetrafluoroethylene
ESVS	European Society for Vascular Surgery
EuREC	European Registry of Endovascular Aortic Repair Complications
EVAR	Endovascular aortic repair
[^{18}F]FDG PET/CT	[^{18}F]-fluoro-2-fluoro-deoxyglucose positron emission tomography—computed tomography
GSC	Guidelines Steering Committee
GWC	Guidelines Writing Committee
HR	Hazard ratio
Ig	Immunoglobulin
INAA	Infected native aortic aneurysm
MAGIC	Management of Aortic Graft Infection Collaboration
MDR	Multidrug resistant
MDT	Multidisciplinary team
MRI	Magnetic resonance imaging
MRSA	Methicillin resistant *Staphylococcus aureus*
MSSA	Methicillin sensitive *Staphylococcus aureus*
NPWT	Negative pressure wound therapy
OPAT	Outpatient parenteral antimicrobial therapy
OR	Odds ratio
PCR	Polymerase chain reaction
PET	Polyethylene terephthalate
PTFE	Polytetrafluoroethylene
RCT	Randomised controlled trial
rRNA	Ribosomal ribonucleic acid
SAT	Supra-aortic trunk
SDM	Shared decision making
SNMMI	Society of Nuclear Medicine and Molecular Imaging
SPECT/CT	Single photon emission computed tomography/computed tomography
SUV_{max}	Maximum standardised uptake value
TBR	Target to background ratio
TEVAR	Thoracic endovascular aortic repair
ToE	Table of evidence
VGEI	Vascular graft or endograft infection
WBC	White blood cell
WBCS	White blood cell scintigraphy
XDR	Extensively drug resistant

WHAT IS NEW COMPARED WITH THE EUROPEAN SOCIETY FOR VASCULAR SURGERY (ESVS) 2020 CLINICAL PRACTICE GUIDELINES ON THE MANAGEMENT OF VASCULAR GRAFT AND ENDOGRAFT INFECTIONS?

The European Society for Vascular Surgery (ESVS) 2026 clinical practice guidelines on the management of vascular graft and endograft infections (VGEIs) represent a comprehensive and fully updated revision of the previous 2020 edition.[1] Every section has been carefully revised or rewritten to reflect the clinical advances in the field over the past 5 years.

Eighty-one recommendations have been issued; 31 graded as Class I, 24 Class IIa, 21 Class IIb, one Class IIIa, and four Class IIIb. Compared with the previous 2020 edition, 40 recommendations are new (18 Class I, nine Class IIa, nine Class IIb, and four Class IIIb), 18 recommendations have been re-graded or significantly rephrased (two Class I, eight Class IIa, and eight Class IIb), and 23 recommendations remain unchanged (11 Class I, seven Class IIa, four Class IIb, and one Class IIIa) (Table 1). Nevertheless, the overall strength of evidence remains weak, since all recommendations except two are Level C evidence.

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Table 1. New, changed, and unchanged recommendations included in the European Society for Vascular Surgery (ESVS) 2026 clinical practice guidelines on the management of vascular graft and endograft infections compared with the previous 2020 guidelines. Numbers correspond to the numbers of the recommendations in the guideline document.

New Class I recommendations

2. When vascular graft or endograft infection is suspected according to the Management of Aortic Graft Infection Collaboration (MAGIC) criteria, further clinical, radiological, and laboratory assessment is recommended.
7. Involvement of an infectious diseases specialist and or microbiologist is recommended in suspected vascular graft or endograft infection.
8. When vascular graft or endograft infection is suspected or diagnosed, obtaining at least three pairs of blood cultures, aerobic and anaerobic, total volume 60 mL, is recommended, preferably before initiation of antimicrobial therapy.
9. When vascular graft or endograft infection is suspected or diagnosed, cultures from urine, nasopharynx and sputum, and superficial wounds are recommended when focal infection symptoms are present, preferably before initiation of antimicrobial therapy.
11. When vascular graft or endograft infection is suspected or diagnosed, collection of at least three deep perigraft or endograft tissue samples is recommended for optimal microbiological diagnosis.
12. When vascular graft or endograft infection is suspected or diagnosed, dividing explanted graft or endograft material and deep perigraft or endograft tissue into multiple pieces using sterile instruments and placing them promptly in separate sterile containers for microbiological analysis is recommended.
21. Multidisciplinary team management of vascular graft or endograft infection is recommended.
25. Targeted antimicrobial therapy is recommended once definitive microbiological results are available in patients with vascular graft or endograft infection.
30. Empirical antimicrobial treatment for thoracic vascular graft or endograft infection covering *Staphylococcus aureus*, coagulase negative staphylococci, streptococci, and enterococci is recommended.
31. Empirical antimicrobial treatment for abdominal vascular graft or endograft infection covering *Staphylococcus aureus*, coagulase negative staphylococci, streptococci, enterococci, Gram negative bacilli, and anaerobes is recommended.
32. Covering *Candida* species as part of the empirical antimicrobial treatment for abdominal vascular graft or endograft infection with erosion or fistula is recommended.
33. Empirical antimicrobial treatment for extracavity vascular graft or endograft infection covering methicillin sensitive and resistant staphylococci, enterococci, and Enterobacterales is recommended.
58. Bowel repair combined with complete graft or endograft explantation and vascular reconstruction is recommended for curative treatment of patients with abdominal vascular graft or endograft infection presenting with secondary aorto-enteric fistula.
61. Involvement of a gastrointestinal surgeon or a urologist is recommended in all cases of bowel or urinary tract repair in abdominal vascular graft or endograft infection with secondary fistula.
76. Endovascular treatment is recommended as first line strategy in lower limb vascular graft or endograft infection with profuse haemorrhage.
78. For patients with lower limb vascular graft or endograft infection requiring complex muscle flap reconstruction, involvement of plastic surgical expertise is recommended.
80. If long term antimicrobial therapy for vascular graft or endograft infection is required, management and monitoring through an outpatient parenteral antimicrobial therapy programme is recommended.
81. Individualised long term follow up by a multidisciplinary team is recommended for patients with vascular graft or endograft infection, guided by clinical, imaging, and laboratory findings.

New Class IIa recommendations

10. When vascular graft or endograft infection is suspected or diagnosed, prolonged incubation of blood, deep perigraft or endograft tissue, and explanted graft or endograft cultures for up to 10 − 14 days should be considered.
15. Delaying initiation of antimicrobial therapy until diagnostic microbiological tissue samples have been obtained should be considered in selected patients with suspected or diagnosed vascular graft or endograft infection, to improve microbiological diagnostic yield.
18. Molecular testing of deep tissue and or explanted graft or endograft should be considered in suspected or diagnosed vascular graft or endograft infection to improve microbiological diagnostic yield, particularly in patients receiving antimicrobial therapy and or with negative cultures.
19. Serology (e.g., *Coxiella burnetii*, *Bartonella*, *Brucella*) and mycobacterial cultures should be considered in culture negative patients with suspected or diagnosed vascular graft or endograft infection when relevant risk factors are present.
26. A minimum post-operative antimicrobial therapy duration of 6 weeks should be considered after complete graft or endograft explantation in patients with vascular graft or endograft infection.
27. Long term or lifelong antimicrobial therapy should be considered after surgical or conservative treatment in patients with vascular graft or endograft infection, when residual infection is likely.
37. Physician made bovine pericardium tube or antimicrobial prosthetic grafts should be considered for *in situ* reconstruction of thoracic vascular graft or endograft infection.

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Table 1-*continued*

47.	Conservative treatment should be considered in patients with abdominal vascular graft or endograft infection deemed unsuitable for aortic clamping or surgical intervention.
56.	Cultures of the graft margins should be considered to guide the duration of antimicrobial treatment when partial graft explantation is performed in patients with abdominal vascular graft or endograft infection.
New Class IIb recommendations	
16.	Temporary withdrawal of ongoing antimicrobial therapy for at least forty-eight hours before microbiological sampling may be considered in selected patients with culture negative suspected or diagnosed vascular graft or endograft infection, to improve microbiological diagnostic yield.
20.	Histopathological analysis of vascular and perigraft or endograft tissue may be considered in order to rule out alternative pathologies (e.g., lymphoma, sarcoma, vasculitides, IgG_4 related disease).
28.	Cessation of long term antimicrobial therapy, under close monitoring, may be considered after surgical or conservative treatment in patients with vascular graft or endograft infection, when residual infection is assessed to be unlikely.
29.	An early switch from intravenous to oral antimicrobial therapy (10 − 14 days post-surgery) may be considered in clinically stable patients with vascular graft or endograft infection, provided oral options with adequate bioavailability are available.
40.	Extra-anatomic reconstruction for thoracic vascular graft or endograft infection may be considered in highly selected patients when *in situ* reconstruction is deemed not possible or to avoid reconstruction in a contaminated field.
69.	For selected patients with lower limb vascular graft or endograft infection, partial graft explantation may be considered when the infection is of limited extent.
70.	For selected patients with lower limb vascular graft or endograft infection without involvement of the anastomoses and limited infection extent, a graft preservation strategy may be considered.
72.	For selected patients with lower limb vascular graft or endograft infection, extra-anatomic graft routing may be considered.
74.	Vascular reconstruction using bovine pericardium may be considered in patients with lower limb vascular graft or endograft infection limited to the groin.
New class IIIb recommendations	
34.	Rifampicin bonded grafts are not recommended for treatment of vascular graft and endovascular infection owing to their relatively narrow antimicrobial spectrum and the theoretical risk of developing rifampicin resistance.
38.	Cryopreserved arterial allografts are not recommended for *in situ* reconstruction of thoracic vascular graft or endograft infection.
52.	Cryopreserved allografts are not recommended for *in situ* reconstruction in patients with abdominal vascular graft or endograft infection.
75.	Antimicrobial grafts are not recommended for vascular reconstruction in patients with lower limb vascular graft or endograft infection.
Changed Class I recommendations	
44.	Endovascular treatment is recommended as first line strategy in thoracic vascular graft or endograft infection with profuse haemorrhage.
60.	Urinary tract repair combined with explantation of the infected graft or endograft with vascular reconstruction is recommended in abdominal vascular graft or endograft infection with arterio-ureteral fistula.
Changed Class IIa recommendations	
5.	[^{18}F]-Fluoro-2-fluoro-deoxyglucose positron emission tomography—computed tomography or white blood cell scintigraphy combined with single photon emission computed tomography/computed tomography should be considered as an adjunctive diagnostic tool in vascular graft or endograft infection.
14.	Percutaneous imaging guided sampling (e.g., perigraft aspiration or drainage) should be considered before surgical treatment in selected patients with suspected vascular graft or endograft infection when the diagnosis remains uncertain after non-invasive workup and or when conservative treatment is considered.
35.	Conservative treatment with close monitoring should be considered as first line therapy in most patients with thoracic vascular graft or endograft infection without fistula.
36.	Total graft or endograft explantation and *in situ* repair should be considered in selected patients with thoracic vascular graft or endograft infection who are suitable for surgery or in cases where conservative treatment has failed.
39.	Coverage of the vascular reconstruction for thoracic vascular graft or endograft infection with autologous vascularised tissue (e.g., omentum or muscle flaps) should be considered.
50.	Autologous veins or physician made bovine pericardium tube should be considered as the preferred method for *in situ* reconstruction in patients with abdominal vascular graft or endograft infection.
63.	For patients with supra-aortic trunk vascular graft or endograft infection presenting with profuse haemorrhage, endovascular treatment as a bridge to definitive management should be considered.

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Table 1-*continued*

66. For patients with supra-aortic trunk vascular graft or endograft infection in whom complete removal of the infected material is not achieved, adjunctive procedures such as muscle flap coverage or negative pressure wound therapy should be considered.

Changed Class IIb recommendations

6. When available, white blood cell scintigraphy combined with single photon emission computed tomography/computed tomography may be considered preferred over [^{18}F]-fluoro-2-fluoro-deoxyglucose positron emission tomography–computed tomography within the first 4 weeks following index surgery.
41. Partial graft explantation of thoracic vascular graft or endograft infection may be considered in selected patients when complete reconstruction is deemed high risk.
45. A graft preserving strategy may be considered in selected patients with aortobronchial or aortopulmonary fistula treated with emergency thoracic endovascular aortic repair.
46. Explantation and *in situ* reconstruction with airways repair and coverage with viable tissue may be considered in patients suitable for surgery as definitive treatment for thoracic vascular graft or endograft infection with an airway fistula.
48. Percutaneous drainage of perigraft fluid collections, with or without irrigation, may be considered in patients with abdominal vascular graft or endograft infection to achieve infection source control, either as a palliative strategy or as a bridge to later definitive surgical treatment.
51. Antimicrobial grafts may be considered as alternative substitutes for *in situ* reconstruction in patients with abdominal vascular graft or endograft infection, especially in complex repair.
57. Graft preservation surgery may be considered in selected patients with abdominal vascular graft or endograft infection when surgical explantation is not feasible.
73. Cryopreserved allografts or biosynthetic collagen grafts may be considered for alternative conduits for vascular reconstruction in lower limb vascular graft or endograft infection.

Unchanged Class I recommendations

1. The Management of Aortic Graft Infection Collaboration (MAGIC) criteria are recommended for the diagnosis of vascular graft or endograft infection.
3. When vascular graft or endograft infection is suspected or diagnosed, microbiological confirmation of infection is recommended.
4. Multiphase computed tomography angiography is recommended as first line imaging modality in suspected vascular graft or endograft infection.
22. Patients with suspected or diagnosed intracavity vascular graft or endograft infection are recommended to be referred to a tertiary centre with multidisciplinary team management.
23. When a vascular graft or endograft is implanted, systemic antimicrobial prophylaxis is recommended.
49. Complete explantation of graft material and infected tissue followed by *in situ* reconstruction is recommended as the first line curative treatment in patients with abdominal vascular graft or endograft infection.
53. Coverage of all vascular reconstructions with autologous vascularised tissue, such as omentoplasty, is recommended in patients with abdominal vascular graft or endograft infection.
59. Endovascular treatment as a bridge is recommended for patients with abdominal vascular graft or endograft infection complicated by aorto-enteric or arterio-ureteral fistula with profuse haemorrhage.
65. For patients with supra-aortic trunk vascular graft or endograft infection, explantation of the infected material and debridement of infected tissue followed by reconstruction with autologous material is recommended as first line treatment.
68. For patients with lower limb vascular graft or endograft infection, explantation of the infected material is recommended as first line treatment, with revascularisation if indicated.
71. For patients with lower limb vascular graft or endograft infection, *in situ* reconstruction with autologous vein is recommended as first line treatment.

Unchanged Class IIa recommendations

24. Prophylactic antimicrobial therapy should be considered in patients with intracavity vascular graft or endograft undergoing procedures that carry a high risk of bacteraemia.
42. Conservative treatment of patients with aorto-oesophageal fistula complicating thoracic vascular graft or endograft infection should be considered a palliative approach.
43. Explantation and *in situ* reconstruction with oesophageal repair and coverage with viable tissue should be considered in patients suitable for surgery as definitive treatment for thoracic vascular graft or endograft infection with an aorto-oesophageal fistula without profuse haemorrhage.
62. Conservative treatment should be considered as a palliative option in selected patients with supra-aortic trunk vascular graft or endograft infection ineligible for graft or endograft explantation.
67. Conservative treatment should be considered a palliative option in selected patients with lower limb vascular graft or endograft infection ineligible for graft or endograft explantation.

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Table 1-*continued*
77. Muscle flaps should be considered for coverage of complex wounds and vascular reconstructions in patients with lower limb vascular graft or endograft infection to promote wound healing and facilitate graft protection.
79. Negative pressure wound therapy should be considered as an adjunct in the management of lower limb vascular graft or endograft infection to promote wound healing and facilitate graft protection or preservation.
Unchanged Class IIb recommendations
17. Sonication of explanted vascular graft or endograft in patients with suspected or diagnosed vascular graft or endograft infection may be considered in order to improve microbiological diagnostic yield.
54. Extra-anatomic reconstruction may be considered an alternative option in selected patients with abdominal vascular graft or endograft infection, particularly in the presence of aorto-enteric fistulation, advanced age, or frailty.
55. Partial graft explantation may be considered in selected patients with abdominal vascular graft or endograft infection when infection is regarded as limited or if the remaining part of the graft or endograft is well incorporated.
64. The EndoVAC technique may be considered for selected patients with supra-aortic trunk vascular graft infection and complex anatomy.
Unchanged Class IIIa recommendations
13. Routine cultures from clinically non-infected superficial wounds, sinus tracts, or negative pressure wound therapy foams are not indicated in suspected or diagnosed vascular graft or endograft infection.

Accordingly, robust prospective studies using a standard format for reporting data on VGEI are required to strengthen the evidence for VGEI management.

Structurally, these guidelines introduce several new chapters, expand existing content, and place greater emphasis on multidisciplinary team (MDT) management and patient centred care.

A lack of consensus in the literature regarding several definitions related to VGEI was recognised, and a new chapter clearly defines different notions, such as MDT management, intestinal erosion or fistula, treatment strategies, as well as treatment outcomes. Another major chapter consolidates VGEI diagnosis, with emphasis on microbiological cultures and sampling, but also microbiological diagnostic adjuncts such as sonication, molecular techniques, serology, and histopathology. This introduces the concept of MDT management of VGEI by dedicated teams in specialised tertiary centres. Following the microbiological diagnosis section, a new chapter is devoted to antimicrobial therapy, which includes ten new recommendations. All efforts should be made to identify all causative pathogens along with their resistance patterns, thus ensuring optimal and targeted antimicrobial therapy.

Compared with the 2020 version, the management of VGEI is less radical. The complete explantation strategy followed by *in situ* reconstruction, considered the gold standard for intra- and extracavity VGEI in the 2020 version, is more debated in the present version. Accordingly, conservative management is now considered the first line treatment in thoracic VGEI. The use of cryopreserved allografts for intracavity vascular reconstruction is also questionable at present owing to the long term deterioration and profuse haemorrhage risk in a hard to reach area in life threatening situations.

This document also emphasises comprehensive patient information and shared decision making (SDM) to ensure individualised care. It also underlines the importance of MDT care, promoting centralised treatment and structured team based decision making. A greater focus is placed on patient centred care, with an emphasis on incorporating, when possible, patient perspectives, SDM, and tailoring interventions to life expectancy, functional status, and autonomy level. Finally, the guidelines acknowledge the persistent evidence gaps in many areas of VGEI management and call for robust prospective trials and a registry using a standard format for reporting data on VGEI.

Key themes across the 2026 update include:

- multidisciplinary care: stronger emphasis on centralised treatment and team decision making
- diagnosis: expanded focus on microbiological cultures and diagnostic microbiological adjuncts
- antimicrobial therapy: need to identify the causative pathogens along with their resistance patterns to ensure optimal antimicrobial therapy
- reporting items: call for robust prospective trials and a registry using a standard format for reporting data on VGEI

1. METHODOLOGY

1.1. Purpose of the guidelines

The European Society for Vascular Surgery (ESVS) has developed clinical practice guidelines for the care of patients with vascular graft or endograft infection (VGEI), in succession to the 2020 version.[1] These guidelines have been developed by a multidisciplinary panel, with the aim of assisting physicians in selecting the best management strategy.

Potential users of these guidelines include any physician involved in the management of patients with VGEI, such as

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cardiac, thoracic, vascular, and general surgeons, infectious diseases specialists, radiologists, cardiologists, nurse specialists, and other healthcare professionals. Furthermore, these guidelines aim to serve as an important source of unbiased information for the patient and their relatives to optimise shared decision making (SDM).

Guidelines promote standards of care but are not a legal standard of care. They are a guiding principle, and care delivered depends on patient presentation, choice, comorbidities, and setting (techniques available, local expertise).

These guidelines are informed by the best available evidence, which is limited in many areas since VGEI is a rare condition, and supplemented by expert opinion where data are lacking. By summarising and evaluating the best available evidence, recommendations for the evaluation and management of patients have been formulated. The recommendations represent the general knowledge at the time of writing these guidelines, but knowledge in this field changes, and recommendations may become outdated. The ESVS aims to update the guidelines when important new insights in the evaluation and management of VGEI become available. The ESVS 2026 clinical practice guidelines on the management of VGEI are published in the *European Journal of Vascular and Endovascular Surgery* (EJVES), as an online open access publication, as well as being free to access via the ESVS website. They are also available on a dedicated ESVS guideline app (https://esvs.org/new-and-improved-guidelines-app/).

1.2. Compliance with AGREE II standards

The 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 2026 clinical practice guidelines on the management of VGEI; a checklist is available as supplementary material in Appendix A.[2,3]

1.3. Guidelines Writing Committee

Guidelines Writing Committee (GWC) members were selected by the GWC chairs and the ESVS Guideline Steering Committee (GSC) to represent clinicians involved in the management of patients with VGEI. The GWC comprised 11 surgeons, four infectious diseases specialists, and one radiologist, from Austria, Denmark, Finland, France, Italy, the Netherlands, New Zealand, Sweden, Switzerland, the UK, and the USA.

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 headquarters of the ESVS (info@esvs.org) and are available upon request. GWC members received no financial support from any pharmaceutical, device, or industry body to develop these guidelines.

The ESVS GSC was responsible for the endorsement process of this guideline. All experts involved in the GWC have approved the final document. The guideline document underwent a formal external expert review process and was reviewed and approved by the ESVS GSC and by the EJVES. This document has been reviewed by 15 reviewers, including nine members of the GSC and six external reviewers from 11 countries. All reviewers assessed all versions and finally approved the final version of this document.

1.4. Methodology

1.4.1. Strategy. The GWC held a series of online conferences in March, April, and May 2024, at which time topics and tasks were allocated, and on a regular basis thereafter. Following preparation of the first draft, GWC members participated in a face to face meeting in Amsterdam, the Netherlands, in November 2024 to review the first draft as well as the wording and grading of each recommendation. Unanimous agreement was obtained for all recommendations. After several online follow up meetings, the GWC was able to agree on a final set of recommendations in March 2025. From March 2025 to April 2026, the document underwent three external review rounds. The final version of these guidelines was submitted in May 2026.

1.4.2. Literature search and selection. All GWC members performed the literature search for this guideline systematically in PubMed (MEDLINE), Embase, and the Cochrane Library up to October 2024. Reference checking and hand search by the GWC members added other relevant literature, including selected articles published up to March 2025. Members of the GWC performed the literature selection based on information provided in the title and abstract of the retrieved studies.

Only English full text peer reviewed publications were included, following the Pyramid of Evidence principle. Multiple randomised controlled trials (RCTs) or meta-analyses of multiple RCTs were at the top, then single RCTs or large non-randomised studies (including meta-analyses of large non-RCTs), followed by meta-analyses of small non-RCTs, observational studies, case series, and large prospective audits. Expert opinion was at the bottom of the pyramid, while case reports and abstracts were excluded. The evidence used in each of the recommendations is detailed in the Table of Evidence (Appendix A).

1.4.3. Studies commissioned for the guidelines. Two reviews and one consensus document were commissioned for these guidelines: (1) a Delphi consensus on VGEI definitions,[4] (2) a systematic review on supra-aortic trunk (SAT) VGEI,[5] and (3) a systematic review on biosynthetic collagen prosthesis extracavity reconstructions for VGEI.[6]

1.4.4. Recommendations. The recommendations are graded according to the ESVS clinical practice guidelines recommendation grading system, where the strength (class) of each recommendation is graded from I to III

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Table 2. Class of recommendation by the European Society for Vascular Surgery (ESVS) evidence grading system.[7]

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 2) and the letters A to C mark the level of evidence (Table 3).[7]

1.4.5. Limitations. Literature on VGEI is scarce, and analysis of the existing literature is difficult due to the heterogeneity of the studies. Moreover, data, when available, originate from different areas in the world, including different ethnicities, and not differentiating between men and women. The development of evidence based guidelines when evidence is scarce is therefore a difficult task, highlighting the importance of a balanced guidelines panel. The GWC based the recommendations on best available scientific evidence, when available, but the practical experience and expertise of the experts in the field were considered since evidence in the VGEI field is scarce. To mitigate bias in expert driven recommendations, the GWC applied a structured Delphi process and consensus voting.

1.4.6. The patient's perspective. Addressing the concept of SDM in the VGEI field is necessary. This requires access to information regarding all available treatment options, together with a balanced discussion of risks, benefits, and potential consequences in a manner that the patient understands and that takes his or her preferences, needs, and values into account.

In order to improve accessibility and interpretability for patients, the plain English summaries for these guidelines were subjected to a lay review process. Information for patients was drafted by the GWC for key subchapters. This draft was read and amended by 28 nurses and 34 patients (with or without VGEI), from Austria, Denmark, Finland, France, Italy, the Netherlands, New Zealand, Sweden, Switzerland, the UK, and the USA, in order to provide dedicated and clear information to patients.

Table 3. The European Society for Vascular Surgery (ESVS) clinical practice guidelines levels of evidence grading system.[7]

Level of Evidence A	Data derived from multiple randomised trials or meta-analyses of randomised trials
Level of Evidence B	Data 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. DEFINITIONS

This document provides guidelines regarding VGEI. Infective native aortic aneurysms (INAAs), primary aortic fistulas, inflammatory aneurysms, and penetrating aortic ulcers are therefore not covered in this document. However, if a primary infected arterial disease was managed by a graft or endograft that became infected, the patient is moved to the VGEI group with regard to management and reporting.

At the outset of creating these guidelines, a lack of consensus in the literature regarding several definitions related to VGEI was recognised. These included definitions of multidisciplinary team (MDT) management, categorisation of early and late VGEI, definitions of different treatment strategies including different antimicrobial strategies, what defines an intestinal erosion *vs.* a fistula, as well as treatment outcomes. To standardise terminology and disease related definitions in the expert driven recommendations, a modified Delphi process was used, involving a large international group of 43 experts in the field of aortic and peripheral VGEI including vascular, cardiac, and thoracic surgeons, and infectious diseases and microbiology, (interventional) radiology, and nuclear medicine specialists. Experts repeatedly voted and commented anonymously on statements dealing with the abovementioned VGEI issues. Statements were adapted accordingly throughout the iterative Delphi process in four rounds. Consensus was defined as $\geq$75% agreement among the experts. Consensus was reached for all statements, except for one (regarding early and late VGEI).[4]

The statements will improve and facilitate standardised research, the lack of which VGEI knowledge suffers from, as well as standardising understanding of the disease and enhancing comparability between studies. The statements are all an integrated part of this guideline document.

2.1. Multidisciplinary team management

Intracavity VGEI involves arteries in the abdomen or thorax, while extracavity VGEI involves arteries outside these regions. When the diagnosis of intracavity VGEI is suspected or diagnosed, given the complexity of management and the poor prognosis associated with these cases,

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prompt consultation in a tertiary centre with a MDT specialised in aortic infections, such as VGEI, is recommended. MDT management requires expertise consisting of vascular, cardiac, and thoracic surgeons, infectious diseases and microbiology specialists, radiologists, nuclear medicine specialists, and anaesthetists. In case of aorto-enteric or ureteral fistulation, a gastrointestinal surgeon or a urologist is required.

The management of extracavity VGEI requires an MDT with vascular surgeons working in close collaboration with infectious diseases and microbiology specialists. If nuclear imaging is performed for diagnostic purposes, the involvement of a nuclear medicine specialist is required.

2.2. Early and late vascular graft or endograft infection

The distinction between early and late infection was originally intended to guide antimicrobial therapy, as certain pathogens were believed to be more prevalent in early *vs.* late VGEI, and to account for differences in biofilm maturity. However, the temporal cutoffs for classifying VGEI into early or late were determined arbitrarily and varied. A cutoff of 2 months following the index surgery was based on cohort distribution,[8] while a cutoff of 4 months was later adopted arbitrarily.[9] Accordingly, a rigid unsupported temporal cutoff failed to achieve consensus among the international expert panel and was therefore discarded. Whether future studies will support the re-introduction of a specific cutoff remains uncertain.

2.3. Erosion and fistula

Intracavity VGEI may be complicated by fistulation to other hollow organs. When this occurs, the prognosis worsens significantly and the treatment requirements change, which is why it is considered a separate disease entity. Erosion and fistula differ in the grade of severity of the loss of integrity between gut and aorta in the presence of an aortic graft or endograft, and the distinction between them has severe implications for the patient and treatment requirements.[4]

An aortic graft enteric partial erosion is defined as pathological or inflammatory adherence of the bowel to the aortic graft or endograft, but with an intact intestinal wall (Fig. 1A). Partial erosions do not cause bleeding.* Partial erosions are diagnosed during surgery for VGEI but may be suspected on imaging such as computed tomography angiography (CTA).

A primary aorto-enteric fistula (AEF) is defined as direct communication between the aortic lumen and the enteric lumen in the absence of graft material.

A secondary graft enteric fistula is defined as an aortic graft or endograft having completely eroded through the enteric wall into its lumen, which causes a VGEI (Fig. 1B). Bleeding is possible (e.g., occult oozing from the mucosa).*

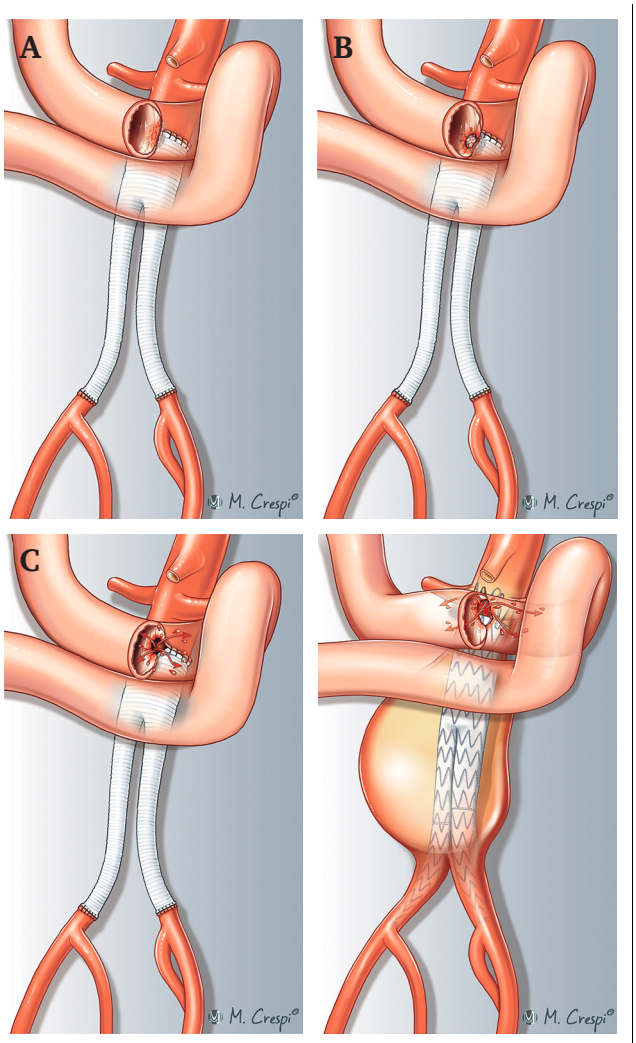

Figure 1. Illustrations of the terms erosion and fistula. (A) Aortic graft-enteric partial erosion is defined as intestine being adherent to the aortic graft/endograft, but with an intact intestinal wall. (B) Secondary graft-enteric fistula is defined as an aortic graft/endograft having completely eroded through the enteric wall into its lumen. (C) secondary aorto-enteric fistula is defined as an aortic graft (C) or endograft (D) with direct communication between the aortic lumen and the enteric lumen. In Figure 1D, this is depicted by a type I endoleak.

A secondary AEF is defined as an aortic graft or endograft with direct communication between the aortic lumen and the enteric lumen, which causes a VGEI (Fig. 1C). The risk of profuse bleeding is high (in case of suture line involvement after open surgical repair, and in the presence of endoleaks after endovascular aortic repair).*

It is difficult to clinically differentiate erosion from fistula. The management and outcome are more dependent on whether there is active bleeding or not as well as on the complexity of bowel repair. Moreover, in the case of fistula, multiple pathogens are common and make antimicrobial therapy more challenging. Accordingly, the treatment strategies in this guideline are divided between VGEI with or without fistula and with or without major bleeding.

* These statements are also applicable to erosions and fistulas engaging the oesophagus, and the respiratory- and urinary tracts.

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2.4. Treatment

Treatment of VGEI, whether intra- or extracavity, consists of antimicrobial treatment, often combined with surgery and or percutaneous drainage. Due to the lack of systematic and comprehensive differentiation between these approaches in the current literature, the following treatment strategies have been defined. These strategies can be adapted over time based on patient status and response to treatment.

2.4.1. Conservative treatment. Conservative treatment of VGEI is defined as antimicrobial treatment alone, with or without percutaneous drainage. Conservative treatment requires close patient monitoring in hospital, including clinical examination, laboratory testing, and imaging to ensure the patient responds well to treatment, as well as regular discussion at MDT meetings.

2.4.2. Surgical treatment. Surgical treatment of VGEI consists of debridement and either total graft explantation, partial graft explantation, or graft preservation.[4]

There is also the option of endovascular treatment for VGEI with profuse haemorrhage, e.g., from a suture line pseudoaneurysm or secondary fistula, as an emergency strategy to stop the bleeding, which might be lifesaving and gain time to decide on a further surgical treatment strategy, but does not treat the infection *per se*.

2.4.3. Curative approach. A curative approach has the intent to achieve VGEI cure (see below). Cure is considered as infection eradication with complete clinical recovery. All VGEI treatment strategies (conservative or different surgical approaches) can be applied individually or in combination to achieve this.

2.4.4. Palliative approach. A palliative approach does not aim to cure the disease or achieve disease in remission (see definitions below) but refers to symptom directed management. This approach involves supportive care for patients not eligible for surgery.

2.5. Antimicrobial therapy

Antimicrobial therapy of VGEI can be either *empirical* or *targeted*. Empirical treatment is defined as antimicrobial therapy directed against anticipated pathogen(s), e.g., if the causative microorganism(s) is not (yet) identified. Targeted treatment specifically targets the identified microorganism(s).[4]

Furthermore, antimicrobial therapy of VGEI can also be either *suppressive* or *curative*. Suppressive antimicrobial therapy is defined as antimicrobial therapy intended to suppress the infection, without being likely to eradicate the infection. Curative treatment is defined as antimicrobial therapy with the intention to eradicate the infection.[4]

2.6. Treatment outcomes

The existing literature focuses primarily on survival after treatment. In order to enhance and nuance the reporting of different treatment strategy outcomes, as opposed to solely reporting survival, and to make this reporting reflect the respective treatment strategies, the terms cure of VGEI, disease in remission, and treatment failure were introduced and defined.

2.6.1. Cure of vascular graft or endograft infection. Cure of VGEI, regardless of anatomic location, is defined as being achieved 1 year after cessation of antimicrobial therapy, without any signs of infection on follow up, including clinical evaluation, laboratory tests including white blood cell (WBC) count and C reactive protein (CRP), and imaging with CTA.

2.6.2. Disease in remission. VGEI disease in remission is defined as suppressive antimicrobial treatment for more than 1 year in a patient without symptoms and without deterioration on clinical evaluation, laboratory tests (WBC count and CRP), and imaging with CTA. This one year criterion is used to standardise reporting in clinical research, but not as a strict clinical threshold.

2.6.3. Treatment failure. VGEI treatment failure is defined as failure to achieve cure or remission, or as VGEI related death at any time. In clinical research, the outcome "treatment failure" is preferred over ambiguous terms such as "recurrent infection" or "persisting infection".

3. DIAGNOSIS

3.1. Incidence

VGEIs are usually multifactorial and result from the complex interplay of patient, surgical, and environmental factors. Reported incidences of VGEI by anatomic location will be developed in specific sections, although the true incidence is difficult to assess owing to under reporting and the lack of standardised diagnostic criteria.

3.2. Risk factors

Several factors, including patient related and pre-, intra-, or post-operative factors, have been described as risk factors for the development of VGEI and are listed in Table 4.[10–12]

3.3. Clinical presentation

The clinical presentation of VGEI is highly varied, ranging from mild symptoms to severe shock from sepsis and or haemorrhage. The duration of symptoms before presentation is also highly variable, and clinical manifestations depend on the anatomic location of the graft or endograft, pathogen virulence, and route of infection (Table 5).[13–20]

A detailed clinical history should be taken in patients with suspected VGEI, and systemic symptoms, e.g., fever, sweats, loss of appetite, unexplained anorexia or weight loss, lethargy, and malaise, should be thoroughly investigated.[14,15] Mild localising features can indicate significant pathology, especially if new, but focal localising symptoms are frequently absent. A history of infection (e.g., urinary tract infection, pneumonia, gastroenteritis, diverticulitis, dental caries, endocarditis) should be sought as this may indicate haematogenous seeding of the graft.[16] Although robust evidence is lacking, repeated instrumentation (e.g., intravascular line

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Table 4. Risk factors for vascular graft or endograft infection.

Patient related risk factors	Male sex* Obesity* Diabetes mellitus† Renal insufficiency† Congestive heart failure† Liver disease and or cirrhosis† Malignancy† Immunosuppressive conditions† Malnutrition† Lymphoproliferative disorders†
Pre-operative risk factors	Primary infected aortic pathology† Bacteraemia during hospitalisation† Infection in an adjacent or remote site† Emergency or redo procedure† Repeated instrumentations* Animal exposure†
Intra-operative risk factors	Groin incision* Breach in aseptic technique† Prolonged operating time* Haemorrhage* Concomitant gastrointestinal or genitourinary procedure† Inadequate antimicrobial prophylaxis*
Post-operative risk factors	Wound complications (wound dehiscence, skin necrosis)* Lymphocele, seroma, or haematoma*

* Data from the prospective Vascular Graft Infection Cohort Study.[10]
† Data from retrospective studies.[11,12]

insertion, urinary catheterisation, or procedures such as embolisation of a type II endoleak) might present a similar risk of causing haematogenous seeding or local inoculation. The first 6 — 8 weeks after graft or endograft placement is a particularly high risk period in this regard. Other more unusual exposure risks include dog or cat bites (*Capnocytophaga canimorsus*, pasteurellosis). Contact with parturient mammals or other animals (Q fever) are rarer causes of VGEI.[17]

VGEI typically has a progressive course, with additional clinical features, such as fistulation to adjacent organs, emerging over time.[9,18,19] Fistulation can form with the respiratory tract, leading to haemoptysis that may progress to oral exsanguination.[18,19] Fistula can also form with the intestinal tract, leading to sepsis, haemorrhage, or a combination of both, and should be considered in every patient with an aortic graft or endograft presenting with gastrointestinal bleeding. Involvement of the anastomosis or native vessel often leads to massive haemorrhage, typically preceded by smaller herald bleeds.[9,18]

3.4. Diagnostic criteria

The diagnosis of VGEI is a complex challenge and underscores the importance of an expert MDT approach. Until relatively recently, there was no widely accepted VGEI case definition, making meaningful evaluation of diagnostic and treatment strategies very difficult. In the last few years,

Table 5. Clinical signs and symptoms associated with vascular graft or endograft infection (VGEI).

Type of VGEI	VGEI specific clinical features	Non-specific clinical features	Laboratory findings
All types of VGEI[9,18]		Fever, sweats, chills	Elevated inflammatory markers (e.g., leukocytosis, CRP, ESR)
		Anorexia, weight loss	Anaemia
		Lethargy	Bloodstream infection
		Malaise	
Intracavity thoracic VGEI[18,19]	Aorto-oesophageal fistula	Chest or back pain	Bloodstream infection (mostly Gram positive pathogens, pathogens more like infective endocarditis)
	Aortobronchial fistula	Wound infection	Polymicrobial bloodstream infection in the case of fistula
	Aortopulmonary fistula	Emboli (including to viscera)	
		Stroke	
		Haemoptysis	
		Pseudoaneurysm	
Intracavity abdominal VGEI[9,18,20]	Aorto-enteric fistula	Abdominal or back pain	Bloodstream infection (including Gram negative pathogens and fungi)
	Aorto-ureteral fistula	Emboli (including to viscera)	Polymicrobial bloodstream infection in the case of fistula
		Haematemesis, melaena, haematochezia, haematuria	
		Pseudoaneurysm	
		Contiguous spondylodiscitis	
Extracavity VGEI[20]	Sinus tract to graft	Wound swelling and or warmth	Elevated inflammatory markers (e.g., leukocytosis, CRP, ESR)
	Graft exposure	Wound dehiscence	
		Lymphocele	
		Painful groin mass	
		Pseudoaneurysm	
		Limb ischaemia and or emboli	

VGEI = vascular graft or endograft infection; CRP = C reactive protein; ESR = erythrocyte sedimentation rate.

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however, a syndromic approach to VGEI diagnosis has been developed, which has been widely adopted for use in both intra- and extracavity VGEI.[15,21,22]

The Management of Aortic Graft Infection Collaboration (MAGIC) case definition was developed by an MDT of clinicians from several large vascular surgery centres by a modified Delphi process of expert consensus.[21] Features of VGEI from clinical, radiological, and laboratory categories are ranked as major or minor (Table 6). VGEI is suspected in the presence of one major criterion, or two minor criteria from different categories. VGEI is diagnosed when there is at least one major criterion and any other criterion from another category.

Using the MAGIC case definition, three diagnostic outcomes are possible. The patient may (1) not meet the suspected VGEI threshold, (2) meet only the lower suspected VGEI threshold, or (3) meet the more stringent diagnosed VGEI threshold. If suspected, exhaustive evaluation for the presence of further clinical, radiological, and laboratory criteria is recommended. In clinical practice, this most often includes imaging with CTA, [^{18}F]-fluoro-2-fluoro-deoxyglucose positron emission tomography—computed tomography ([^{18}F]FDG PET/CT), and imaging guided fluid aspiration. VGEI typically progresses and becomes more obvious over time, and the management of patients that meet the suspected but not the diagnosed threshold at their initial presentation can be challenging. These patients require a minimum of two year follow up, because up to half of suspected cases progress to diagnosed over this time. Importantly, some criteria can only be met following surgery, for example "organisms recovered from an intra-operative specimen" or "organisms recovered from an explanted graft", and it is possible that the diagnosis of VGEI may only be formally made following explantation surgery.[15]

The MAGIC criteria were validated in a prospective cohort study at a tertiary referral centre using consensus from the MDT as the reference measure.[22] Over all anatomic locations, a sensitivity of 93% and a specificity of 93% for the diagnosed threshold was reported. The sensitivity of the more easily reached suspected threshold was higher (99%), but with reduced specificity of 61%. Due to the very high sensitivity of the suspected threshold, VGEI can be considered to have been excluded in patients not meeting the suspected threshold, noting that this assumes that a thorough investigation has been undertaken. Sensitivity and specificity vary by anatomic location (Table 7).[22]

Recommendation 1			Unchanged
The Management of Aortic Graft Infection Collaboration (MAGIC) criteria are recommended for the diagnosis of vascular graft or endograft infection.			
Class	**Level**	**Reference**	**ToE**
I	C	Lyons *et al.* (2026)[15]	

Table 6. Vascular graft or endograft infection (VGEI) is suspected in a patient with any isolated major criterion, or minor criteria from two of the following three categories: clinical or surgical; radiological; or laboratory. VGEI is considered diagnosed in the presence of a single major criterion plus any other criterion (major or minor) from a different category. When laboratory investigations identify potential contaminating organisms (e.g., coagulase negative staphylococci, propionibacteria, corynebacteria, or other skin commensals), a minimum of (a) two intra-operative specimens, (b) two blood cultures, or (c) one intra-operative specimen plus one blood culture must be positive with an indistinguishable organism in each sample based on antibiograms or a recognised typing method, e.g., pulsed field gel electrophoresis. "Organisms recovered" includes identification by culture based techniques and, for example, 16S PCR.[21]

Criterion	Clinical or surgical	Radiological	Laboratory
Major	• Pus (confirmed by microscopy) around graft or in aneurysm sac at surgery • Open wound with exposed graft or communicating sinus • Fistula development, e.g., aorto-enteric or aortobronchial • Graft insertion in an infected site, e.g., fistula, infected native aneurysm, or infected pseudoaneurysm	• Perigraft fluid on computed tomography ≥3 months after insertion • Perigraft gas on computed tomography ≥7 weeks after insertion • Increase in perigraft gas volume demonstrated on serial imaging	• Organisms recovered from an explanted graft or endograft • Organisms recovered from an intra-operative specimen • Organisms recovered from a percutaneous, radiologically guided aspirate of perigraft or endograft fluid
Minor	• Localised clinical features of VGEI, e.g., erythema, warmth, swelling, purulent discharge, unexplained pain • Fever ≥38°C with VGEI as most likely cause	• Other, e.g., suspicious perigraft gas, fluid, or soft tissue inflammation; aneurysm expansion; pseudoaneurysm formation; focal bowel wall thickening; discitis or osteomyelitis • Suspicious metabolic activity on [^{18}F]-fluoro-2-fluoro-deoxyglucose positron emission tomography—computed tomography; radiolabelled leukocyte uptake	• Blood culture(s) positive and no apparent source except VGEI • Abnormally elevated inflammatory markers with VGEI as most likely cause, e.g., erythrocyte sedimentation rate, C reactive protein, white blood cell count

VGEI = vascular graft or endograft infection.

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Table 7. Accuracy of the Management of Aortic Graft Infection Collaboration (MAGIC) diagnosed threshold by graft location.[22]

Anatomic location	Sensitivity	Specificity
All graft sites, overall	93 (88–97)	93 (87–97)
Intracavity, abdominal ($n = 151$)	94 (84–99)	92 (85–96)
Intracavity, thoracic ($n = 56$)	86 (73–95)	100 (74–100)
Extracavity ($n = 43$)	100 (91–100)	67 (9–99)

Data are presented as % (95% confidence interval).

Recommendation 2 — New

When vascular graft or endograft infection is suspected according to the Management of Aortic Graft Infection Collaboration (MAGIC) criteria, further clinical, radiological, and laboratory assessment is recommended.

Class	Level	Reference	ToE
I	C	Lyons *et al.* (2026)[15]	

3.4.1. Clinical diagnostic criteria. Several clinical signs requiring subjective interpretation, such as unexplained fever or local erythema, were classified as minor criteria due to lower specificity (Table 6). For example, a fever $\geq 38^{\circ}C$ requires the clinician to exclude an alternative source, such as urinary tract or lower respiratory tract infection. This is also challenging when an endograft is infected by haematogenous seeding from a concomitant distant source, for example pneumonia or cholecystitis, and antimicrobial therapy is likely to target the same organism(s) in both locations.

Several clinical criteria for VGEI can be diagnosed intra-operatively by the surgeon. A fistula between a non-sterile site such as the respiratory or intestinal tract is incontrovertible evidence of VGEI. While discoloured or cloudy fluid around a graft may represent pus, microbiological examination to demonstrate neutrophils is needed for objectivity. Therefore, presence of leukocytes must be proven by direct microscopy or histology to be considered a major criterion. Lastly, deployment of a graft or endograft in an infected field (e.g., for an INAA) is a major criterion, even though the risk of VGEI is relatively low.[4,23,24]

3.4.2. Imaging diagnostic criteria. CTA remains the first choice imaging modality. Gas in the peri-aortic space or in the aneurysm sac is frequently seen on computed tomography (CT) during the 4 weeks following implantation for both open and endovascular repair. Gas that either accumulates over serial imaging or persists after 7 weeks indicates fistula or the presence of gas producing micro-organisms; and fluid that has not been resorbed by 3 months is also highly suspicious of infection, both considered as major criteria.[25,26] Rapid aneurysm enlargement on serial imaging can be a feature of infection, but requires clinical judgement and is therefore a minor criterion. [^{18}F]FDG PET/CT findings are minor criteria since interpretation has not been standardised.

3.4.3. Laboratory diagnostic criteria. The initial diagnostic workup should include basic laboratory tests for inflammatory markers and blood cultures. A normal CRP at presentation (without previous antibiotic administration) makes VGEI highly unlikely, but a normal WBC count is a much less discriminatory feature. Post-implantation syndrome, characterised by transitory fever associated with elevated WBC count and CRP, may occur after endograft insertion and can be observed in the absence of infection.[27] The cutoff after which elevated WBC count and CRP can no longer be attributed to post-implantation syndrome but are signs of infection is 8 weeks, since incorporation of the material must have taken place after 8 weeks.[25,26] Abnormally elevated inflammatory markers with VGEI as the most likely cause are minor criteria, such as positive blood cultures and no apparent source except VGEI.

Microorganisms recovered from an explanted graft or endograft or an intra-operative specimen, or from a peri-graft or endograft collection (e.g., percutaneous CT guided aspiration) are strong site specific evidence of VGEI and hence major criteria.[28]

Recommendation 3 — Unchanged

When vascular graft or endograft infection is suspected or diagnosed, microbiological confirmation of infection is recommended.

Class	Level	Reference	ToE
I	C	Colonna *et al.* (2025)[28]	

3.5. Imaging modalities

3.5.1. Duplex ultrasound. Duplex ultrasound (DUS) is a non-invasive, low cost, readily available imaging modality that is useful for the assessment of peripheral extracavity grafts but is less useful for intracavity grafts, which are difficult to assess due to overlying structures. DUS allows investigation for graft thrombosis, which may be a sign of VGEI. DUS can also assess for pseudoaneurysms and differentiate between haematoma or abscess formation. It may guide puncture for microbiological sampling and drainage of collections. However, a normal DUS does not rule out VGEI, and in cases where DUS is abnormal further imaging with CTA and or [^{18}F]FDG PET/CT is required to confirm the diagnosis and delineate the extent.[29]

3.5.2. Computed tomography angiography. CTA is the first line imaging modality for diagnosing VGEI as it is quick, widely available, and can direct image guided sampling for microbiological confirmation.

Core CTA protocols vary depending on institutional preferences but typically include high resolution images with multiplanar reconstructions acquired during pre-contrast, arterial, and delayed phases.[30] Electrocardiogram gating may be employed when assessing the ascending aorta to reduce motion artefact.[31] The pre-contrast study evaluates any hyperdense surgical material, outpouching of grafts

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(that can mimic pseudoaneurysms), and haematoma. CTA can evaluate graft anatomy, graft integrity, and involvement of adjacent structures. The delayed phase is useful to assess the presence of endoleaks and fistula.[30]

Typical CTA signs of VGEI include perigraft soft tissue stranding, fluid or collections, air, and perigraft soft tissue enhancement.[32–35] However, these signs can be normal in the immediate post-operative period. As per the MAGIC major criteria, perigraft air is considered abnormal beyond 7 weeks and perigraft fluid beyond 3 months, in addition to worsening signs on serial imaging.[25,26] Furthermore, perigraft air can be seen related to the use of bioabsorbable haemostatic agents that are used to control bleeding and designed to be absorbed over time.[36]

Complications of VGEI, such as development of pseudoaneurysms, abscesses, hydronephrosis, bowel erosions, fistula, and osteomyelitis, can also be detected on CTA. Direct but infrequent CT signs of fistula include visualisation of the fistula tract and contrast extravasation into the fistula with changing appearances of the extravasated contrast over the various CT acquisition phases.[37,38] Indirect signs are non-specific, including the presence of intraluminal air within the aorta or aneurysm sac, loss of the fat plane between the aorta and duodenum, and bowel wall thickening. In patients with an aortic graft presenting with upper gastrointestinal haemorrhage, often both endoscopy and CTA are performed to secure the diagnosis, but it may still be difficult to determine with certainty.[39]

In rare cases with direct infection extension to the vertebral bodies, CT can detect bony erosion but contrast magnetic resonance imaging (MRI) of the spine is more sensitive for assessment of early marrow based changes, delineating the extent of any associated osteomyelitis or discitis. [^{18}F]FDG PET/CT has also demonstrated good diagnostic performance for diagnosing vertebral osteomyelitis and can be considered complementary to MRI.[40,41]

The overall diagnostic accuracy of CTA for VGEI is moderate, with a pooled sensitivity of 67% and a pooled specificity of 63%.[42] In infections with low virulence, or VGEI occurring shortly after graft or endograft implantation, CTA has higher false negative rates and lower sensitivity rates, as it can be challenging to differentiate between normal post-operative inflammatory change and infection.[43] Accuracy increases in VGEI occurring later after graft or endograft implantation or when there are associated infection related complications. Serial imaging can improve diagnostic accuracy by detecting persistent or progressive findings.

In the presence of at least one major clinical or laboratory MAGIC criterion (high pre-test probability), a positive CTA can be sufficient for the diagnosis of VGEI, but a negative or equivocal CTA should be followed by nuclear medicine imaging.[29] In the presence of at least two minor clinical or laboratory MAGIC criteria (lower pre-test probability), a clearly positive CTA is considered sufficient without the need for additional nuclear medicine imaging.

Recommendation 4			Unchanged
Multiphase computed tomography angiography is recommended as first line imaging modality in suspected vascular graft or endograft infection.			
Class	**Level**	**Reference**	**ToE**
I	C	Reinders Folmer *et al.* (2018)[42]	

3.5.3. Nuclear medicine imaging

3.5.3.1. [^{18}F]-fluoro-2-fluoro-deoxyglucose positron emission tomography—computed tomography. [^{18}F]FDG PET/CT imaging is based on the uptake of radiolabelled glucose in inflammatory cells or tissue with enhanced glucose metabolism. [^{18}F]FDG PET/CT may provide accurate localisation of sites of infection, targets for sampling, and assessment of complications. However, there may be false positive findings, particularly in the early post-operative period owing to sterile post-surgical inflammation, haematomas, or lymphoceles and the use of surgical haemostatic agents during vascular graft placement.[44] In addition, diffuse FDG uptake can persist along vascular grafts for years post-surgery, especially with synthetic material, and atherosclerotic plaque can also show FDG uptake.[35,44,45] False negative findings have also been suggested to occur following prolonged antimicrobial exposure,[46] but others have reported no such association.[47,48]

The European Association of Nuclear Medicine (EANM) and the Society of Nuclear Medicine and Molecular Imaging (SNMMI) have published [^{18}F]FDG PET/CT procedure guidelines for infection workup.[49] Initial recommendations regarding a standard reporting format have been suggested. Interpretation of [^{18}F]FDG PET/CT for VGEI includes comparison with other available imaging modalities, assessment of pattern of tracer uptake (homogeneous diffuse *vs.* focal heterogeneous), intensity of tracer uptake (visual grading scores or semiquantitative scores), and associated morphological changes on the CT component of the PET/CT or CTA. Focal tracer uptake and the presence of ancillary findings on CT increases diagnostic accuracy for graft or endograft infection.[50,51]

Various visual grading scores have been proposed, some combining visual uptake scores and different tracer patterns,[32,33,52] and others also incorporating CT signs to improve the specificity of [^{18}F]FDG PET/CT for VGEI[53,54] (Fig. 2).

The use of maximum standardised uptake value (SUV_{max}) cutoffs to detect VGEI is not reliable owing to significant overlap between infected and non-infected devices, and in addition varies with reconstruction parameters and different PET/CT machines.[33,53–57]

Various target to background ratios (TBRs) have also been assessed; however, the choice of target graft value background reference tissue varies between studies, leading to heterogeneity in results and suggested threshold cutoffs.[33,54,56]

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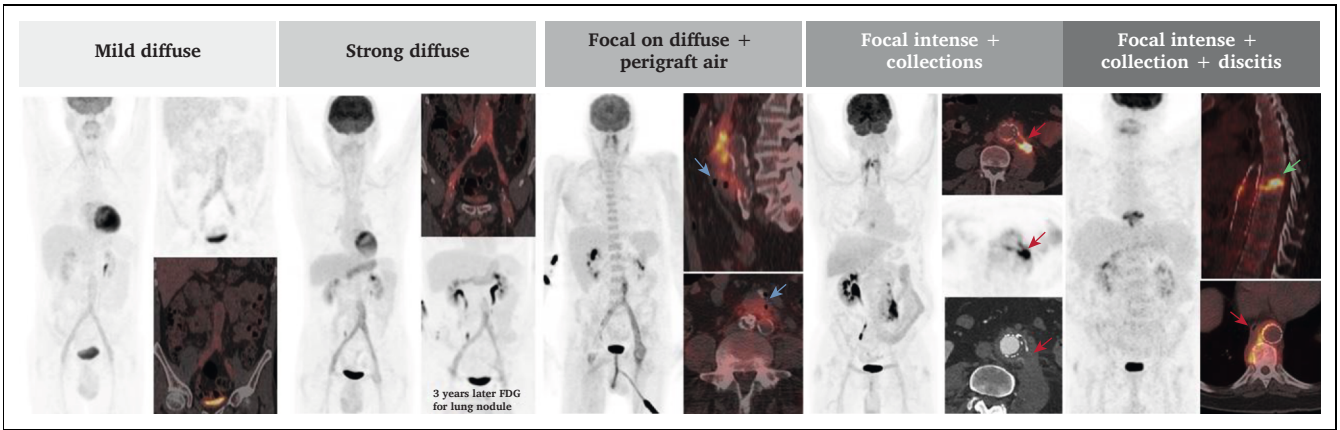

Figure 2. Examples of different uptake patterns on [^{18}F]-fluoro-2-fluoro-deoxyglucose positron emission tomography—computed tomography ([^{18}F]FDG PET/CT). The first two cases are examples of mild diffuse and strong diffuse without ancillary findings on computed tomography (CT) in patients without graft infection. The other cases are examples of confirmed graft infection with abnormal patterns of tracer uptake and ancillary findings on CT such as perigraft air (blue arrows), perigraft collections (red arrows), and graft infection associated discitis (green arrow).

Several meta-analyses have assessed the diagnostic accuracy of [^{18}F]FDG PET/CT for VGEI, each with slightly differing focus and some overlap in included studies (Table 8).[42,46,58–60]

An initial meta-analysis assessed the diagnostic accuracy of [^{18}F]FDG PET and [^{18}F]FDG PET/CT for VGEI using different imaging analysis methods and reported that focal uptake and SUV_{max} were the most reliable parameters but recognised that no clear SUV_{max} cutoff exists.[46] A second meta-analysis identified FDG uptake pattern as the most accurate assessment method.[59] The EANM evidence based guidelines conclude that focal uptake appears to be the most reliable method, but the combination of two or more parameters (visual grading score, pattern, SUV_{max}, TBR) alongside CT findings should be better explored.[29]

Similar results for [^{18}F]FDG PET/CT with high sensitivity and moderate specificity were reported in two more recent meta-analyses.[58,60] Combining contrast enhanced CT with [^{18}F]FDG PET/CT in one sitting leads to improved diagnostic accuracy over standalone imaging.[34]

There are limited data on the use of [^{18}F]FDG PET/CT in monitoring the response to antimicrobial therapy in VGEI. Husmann *et al.* reported that reduction in SUV_{max} and frequency of focal tracer uptake over time correlated with discontinuation of antimicrobial therapy, higher CRP correlated with higher SUV_{max}, and [^{18}F]FDG PET/CT imaging influenced decisions to escalate or continue antimicrobial treatment despite normalisation of CRP levels.[61] In the setting of diagnosed VGEI, where total graft or endograft explantation is not being considered, acquisition of nuclear medicine imaging as a baseline to aid in evaluation of treatment response over time appears reasonable. However, the clinical utility and impact of serial nuclear medicine imaging in follow up requires further investigation.

3.5.3.2. White blood cell scintigraphy. Radiolabelled autologous white blood cell scintigraphy (WBCS) detects infected sites by visualising the accumulation of radiolabelled WBCs over time. WBCS has high accuracy in differentiating infection, even low grade, from sterile inflammation but this relies on the application of well standardised acquisition protocols and interpretation criteria as proposed by the EANM guidelines.[62,63]

WBCs can be radiolabelled with either [^{99m}Tc] HMPAO or indium-111 [^{111}In]-oxine.[43] WBCS is a very specific method but has some limitations. The procedure is labour intensive and requires a laboratory specifically equipped to perform leukocyte labelling. In addition, the imaging needs to be performed at at least two different time points: early (thirty minutes to 1 hour) and delayed (2 — 4 hours) with the addition of late images (20 — 24 hours) in equivocal cases.[63] Since the tracer is eliminated via the intestinal tract and physiologically taken up in the bone marrow, combining WBCS with single photon emission computed tomography/computed tomography (SPECT/CT) imaging improves anatomic localisation and diagnostic accuracy.[42,63]

3.5.4. Imaging comparison studies. A meta-analysis comparing CTA, [^{18}F]FDG PET/CT, and WBCS reported that pooled sensitivities and specificities were 0.67 (95% confidence interval [CI] 0.57 — 0.75) and 0.63 (95% CI 0.48 — 0.86) for CTA, 0.95 (95% CI 0.87 — 0.99) and 0.80 (95% CI 0.69 — 0.89) for [^{18}F]FDG PET/CT, and 0.90 (95% CI 0.85 — 0.94) and 0.82 (95% CI 0.57 — 0.96) for WBCS.[42] The higher specificity and positive predictive value of WBCS over [^{18}F]FDG PET/CT have then been demonstrated in two other meta-analyses.[58,60] [^{18}F]FDG PET/CT therefore represents a suitable and more accessible alterative. Combining contrast enhanced CT with [^{18}F]FDG PET/CT in one sitting leads to improved diagnostic accuracy over standalone imaging by pairing the high sensitivity of [^{18}F]FDG PET/CT with the specificity of contrast CT.[34]

In a study comparing 99-metastable-technetium [^{99m}Tc] HMPAO WBCS SPECT/CT, [^{18}F]FDG PET/CT, and CTA in 26 patients with suspected abdominal VGEI of which 11 were

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Table 8. Meta-analysis of the diagnostic performance of [^{18}F] FDG PET/CT for vascular graft or endograft infection.

Author	Studies − *n* (patients − *n*)	Focus of analysis	Pooled sensitivity (95% CI) − %	Pooled specificity (95% CI) − %	Pooled positive LR (95% CI)	Pooled negative LR (95% CI)	Pooled DOR (95% CI)
Reinders Folmer, 2018[42]	5 (144)	PET/CT	95 (87–99)	80 (69–86)	NR	NR	38.0 (8.5–170.4)
Kim, 2019[58]	10 (286)	PET and PET/CT	96 (89–98)	74 (67–81)	3.7 (2.9–4.9)	0.06 (0.02–0.15)	63 (23–173)
Rojoa, 2019[46]	12 (NR)	6 (NR) Uptake intensity (VGS)	89 (73–96)	61 (48–74)	2.32 (1.27–4.22)	0.17 (0.06–0.53)	13.2 (3.1–56.6)
		7 (NR) Focal uptake	93 (83–97)	78 (53–92)	4.32 (1.71–10.90)	0.89 (0.03–0.25)	48.7 (9.6–246.7)
		6 (NR) SUV_{max}	98 (42–99)	80 (70–88)	4.98 (3.12–9.95)	0.03 (0–1.54)	176.7 (3–10 479)
Reinders Folmer, 2020[59]	13 (415)	6 (184) Uptake intensity (VGS)	90 (79–96)	56 (38–79)	NR	NR	10.7 (3.4–33.6)
		9 (317) Uptake pattern	94 (89–97)	81 (71–88)	NR	NR	52.4 (19.4–141.6)
		6 (160) SUV_{max}	95 (76–99)	77 (63–87)	NR	NR	37.1 (14.8–92.8)
Mahmoodi, 2022[60]	10 (320)	PET/CT	92 (88–95)	76 (70–81)	3.5 (2.5–5.3)	0.14 (0.09–0.23)	37.1 (14.8–92.8)

[^{18}F]FDG PET/CT = [^{18}F]-fluoro-2-fluoro-deoxyglucose positron emission tomography–computed tomography; CI = confidence interval; LR = likelihood ratio; DOR = diagnostic odds ratio; NR = not reported; VGS = visual grading score; SUV_{max} = maximum standardised uptake value.

confirmed as infected, [^{18}F]FDG PET/CT showed a high sensitivity and negative predictive value, but [^{99m}Tc] HMPAO WBCS SPECT/CT showed a higher specificity and positive predictive value.[35] Two false positive cases with [^{18}F]FDG PET/CT in the five cases performed within 4 months of surgery were reported.[35]

The 2022 EANM evidence based guidelines on imaging infection in vascular grafts and endografts suggest that following first line imaging with CTA, radiolabelled WBCS SPECT/CT, if available, is preferable in the early period after graft or endograft placement, while either radiolabelled WBCS SPECT/CT or [^{18}F]FDG PET/CT should be performed thereafter. In cases of discordant findings between CTA and nuclear medicine examinations, an additional nuclear medicine modality should be performed.[29,35]

Furthermore, the addition of whole body [^{18}F]FDG PET/CT or WBCS SPECT/CT may delineate the full extent of infection and reveal disseminated extra-aortic foci such as spondylodiscitis, osteomyelitis, or, more rarely, endocarditis. In previously reported VGEI cohorts, [^{18}F]FDG PET/CT has identified clinically relevant secondary infections and septic emboli in multiple cases, supporting its selective use beyond primary VGEI diagnosis, to map infection spread when surgical planning or atypical presentations warrant a comprehensive overview.[64,65]

In summary, the available evidence demonstrates that while CTA remains the essential first line modality for suspected VGEI, it lacks sufficient sensitivity and specificity to confirm or exclude infection. Functional imaging with [^{18}F]FDG PET/CT or WBCS SPECT/CT significantly enhances diagnostic accuracy, with the latter preferred in the early post-operative period, if available, owing to superior specificity. Additionally, nuclear medicine imaging can delineate the full extent of infection or detect extra-aortic foci when clinically indicated.

Figure 3 provides a summarised flowchart of the recommended diagnostic radiological workup in patients with VGEI.

Recommendation 5 — **Changed**

[^{18}F] -Fluoro-2-fluoro-deoxyglucose positron emission tomography–computed tomography or white blood cell scintigraphy combined with single photon emission computed tomography/computed tomography should be considered as an adjunctive diagnostic tool in vascular graft or endograft infection.

Class	Level	References	ToE
IIa	B	Reinders Folmer *et al.* (2018),[42] Rojoa *et al.* (2019),[46] Kim *et al.* (2019),[58] Reinders Folmer *et al.* (2020),[59] Mahmoodi *et al.* (2022)[60]	

Recommendation 6 — **Changed**

When available, white blood cell scintigraphy combined with single photon emission computed tomography/computed tomography may be considered preferred over [^{18}F] -fluoro-2-fluoro-deoxyglucose positron emission tomography–computed tomography within the first 4 weeks following index surgery.

Class	Level	Reference	ToE
IIb	C	Lauri *et al.* (2023)[35]	

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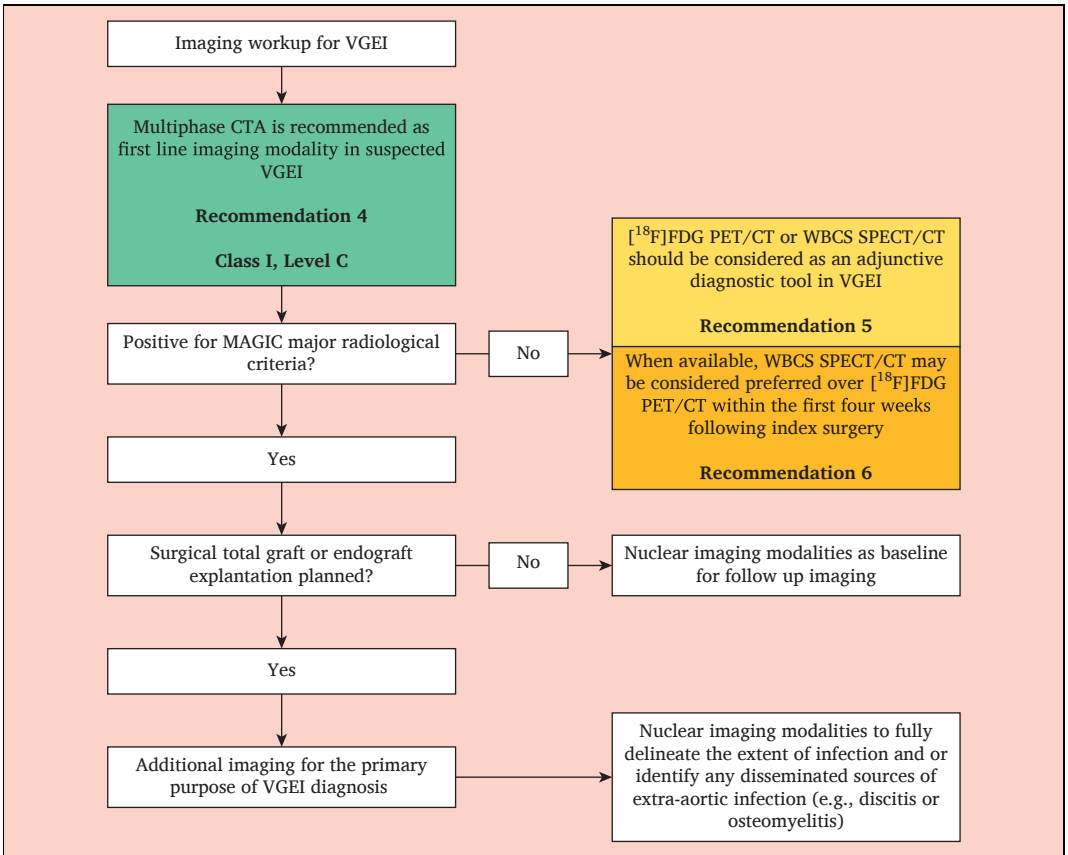

Figure 3. Flowchart summarising the recommendations for radiological diagnostic workup of patients with vascular graft or endograft infection (VGEI). CTA = computed tomography angiography; MAGIC = Management of Aortic Graft Infection Collaboration; [^{18}F]FDG PET/CT = [^{18}F]-fluoro-2-fluoro-deoxyglucose positron emission tomography–computed tomography; WBSC SPECT/CT = white blood cell scintigraphy combined with single photo emission computed tomography/computed tomography.

3.6. Microbiology

3.6.1. General considerations. A thorough microbiological workup for suspected VGEI serves two main critical purposes: firstly, to confirm the diagnosis of VGEI; and secondly, to identify all causative pathogens along with their resistance patterns to ensure optimal, targeted antimicrobial therapy. However, improper use of microbiological tests and sampling techniques can increase the risk of isolating contaminants (false positives), potentially resulting in misdiagnosis, unnecessary surgical interventions, or unwarranted exposure to additional antimicrobial therapy. Owing to the complexity of the microbiological diagnosis in patients with VGEI, early consultation with an infectious diseases specialist is required to ensure a thorough microbiological workup and the appropriate timing of antimicrobial initiation.

Recommendation 7		New
Involvement of an infectious diseases specialist and or microbiologist is recommended in suspected vascular graft or endograft infection.		
Class	**Level**	**Reference**
I	C	Consensus

Previous studies have demonstrated that a causative pathogen can be identified in approximately 60 – 90% of all VGEI cases.[18,66,67] These detection rates are influenced by several factors, including previous or ongoing antimicrobial therapy, the extent and quality of deep tissue or graft samples obtained for cultures, and the use of advanced microbiological diagnostic tools, such as sonication and microbial genomic identification via polymerase chain reason (PCR). Overall, two thirds of culture positive VGEIs are caused by Gram positive pathogens, with coagulase negative staphylococci (CoNS), *Staphylococcus aureus*, and enterococci being most common.[68,69] However, the spectrum of pathogens involved in VGEI has been shown to vary based on the anatomic location of the graft infection. For extracavity VGEI, Gram positive cocci, particularly *S. aureus* and CoNS, are identified as the primary causative pathogens in an average of 45% of cases.[70–72] In contrast, Gram negative bacteria, including the Enterobacterales order (e.g., *Escherichia coli*, *Klebsiella* spp., *Enterobacter* spp., and *Proteus* spp.) and anaerobes, are more common in intracavity abdominal VGEI.[66,72,73] Furthermore, recent findings have highlighted that intracavity abdominal VGEI with a concomitant secondary enteric fistula is frequently associated with polymicrobial (involving two or more

pathogens) and fungal infections, predominantly caused by *Candida* spp.[73–75] Atypical causative microorganisms such as *Bartonella* spp., *Brucella*, *Coxiella burnetii*, mycobacteria species, mycoplasma species, or *Pasteurella multocida* have also been reported.[76–79]

Additionally, the type of clinical presentation is probably associated with microbiological aetiology. A more fulminant presentation with relapsing high grade fever, rigors, and haemodynamic compromise is more likely to be caused by a highly virulent underlying pathogen (e.g., *S. aureus*), as opposed to a chronic, indolent, or grumbling clinical picture, more consistent with low virulence microorganisms requiring adherence to an implanted prosthetic surface (e.g., CoNS).

Biofilm formation on vascular devices represents a major complication in vascular surgery.[80] Biofilms are structured communities of microorganisms encased in a self produced extracellular polymeric substance that adhere to surfaces. When they form on vascular devices, they are associated with persistent infections, increased morbidity, and high treatment failure rates.[81] Cells from the mature biofilm can dislodge and spread to other areas, potentially causing systemic infections. The most common biofilm forming pathogens include *S. aureus*, *Staphylococcus epidermidis*, *Pseudomonas aeruginosa*, and *E. coli*.[80] Fungal pathogens, particularly *Candida* spp., are less common but can also form biofilms. It has been shown that a bacterial biofilm layer and associated bacterial cells could be detected under electron microscopy from explanted infected grafts or endografts as early as 1 month following VGEI onset.[82] However, while many VGEIs are associated with biofilm formation, particularly in chronic or late onset cases, acute infections, especially those occurring immediately after graft or endograft implantation, may not display mature biofilm structures.[82] Biofilm presence complicates treatment by limiting antimicrobial penetration and shielding microorganisms from host immune defences. This contributes to chronic infections, with biofilms persisting on grafts and often leading to recurrent infections despite systemic antimicrobial therapy. Emerging treatments therefore include biofilm disrupting agents, such as enzymes and chelating agents, and the use of antimicrobial coated or biofilm resistant graft materials.[81,83] Despite advances, biofilm associated VGEI still remains a serious clinical problem, and ongoing research into advanced materials and therapies offers hope for reducing biofilm formation and enhancing the durability and safety of vascular grafts.

3.6.2. Cultures and sampling

3.6.2.1. Indirect sampling. An initial microbiological investigation for a suspected VGEI should include at least indirect blood culture sampling.[4,14] In the presence of concurrent urinary tract or respiratory symptoms as well as superficial wound infections, wound, urine, and sputum cultures should also be obtained, in part to exclude other more common sites of infection. Negative blood cultures are common and several studies have shown that the microbiological yield of blood cultures in cohorts with a MAGIC suspected or diagnosed VGEI is around 25 – 40%.[9,72,84] In addition, some reports of intracavity VGEI have shown a low concordance of around 20% between pathogens identified in blood cultures and intra-operative tissue or graft cultures.[85]

Adequate blood sample volume is a well established predictor of positive blood culture yield. A 33% increase in blood culture yield for bloodstream infections has been shown when comparing standard volume (mean 8.7 mL) with low volume (mean 2.7 mL) blood cultures, with a 3% increase in yield for each additional millilitre sampled.[86] Identifying true, non-contaminant, bloodstream infections when going from two pairs (total volume 40 mL) to three pairs (total volume 60 mL) of blood culture sets increases sensitivity from 90% to 98%.[87–89]

Modern automated blood culture systems can typically detect most pathogens within 5 days of incubation. If cultures remain negative after 5 days, extending incubation up to 10 – 14 days is reasonable. The impact of prolonged blood culture incubation (> 5 days) in VGEI has not been studied but is routinely recommended, despite the risk of increasing false positives with prolonged incubation.[14,72] Studies on prosthetic valve infective endocarditis and prosthetic joint infections have shown improved detection of slow growing pathogens with extended incubation times (10 – 14 days), although this also increased contaminant yield.[90,91]

Recommendation 8 **New**

When vascular graft or endograft infection is suspected or diagnosed, obtaining at least three pairs of blood cultures, aerobic and anaerobic, total volume 60 mL, is recommended, preferably before initiation of antimicrobial therapy.

Class	Level	References	ToE
I	C	Mermel *et al.* (1993),[86] Li *et al.* (1994),[87] Lee *et al.* (2007),[88] Patel *et al.* (2011)[89]	

Recommendation 9 **New**

When vascular graft or endograft infection is suspected or diagnosed, cultures from urine, nasopharynx and sputum, and superficial wounds are recommended when focal infection symptoms are present, preferably before initiation of antimicrobial therapy.

Class	Level	Reference
I	C	Consensus

Recommendation 10 **New**

When vascular graft or endograft infection is suspected or diagnosed, prolonged incubation of blood, deep perigraft or endograft tissue, and explanted graft or endograft cultures for up to 10 – 14 days should be considered.

Class	Level	References	ToE
IIa	C	Schäfer *et al.* (2008),[90] Fihman *et al.* (2021)[91]	

3.6.2.2. Direct sampling. While not specifically studied in VGEI, prosthetic joint infection research has shown that an increased number of peri-operative deep tissue samples (at least three) increases the probability of identifying causative pathogens.[92] Extrapolation to VGEI would be that obtaining at least three direct deep specimens including perigraft or endograft tissue and graft or endograft sampling is necessary to increase the probability of identifying a causative pathogen.[14,21,69]

No study comparing specific tissue sampling techniques exists in the VGEI field, with the available literature also coming from orthopaedic surgery. It has been shown that placing sterile non-infected peri-operative fat tissue directly into a sterile container reduced the incidence of identified contaminants compared with placing them on the sterile back table for the duration of the elective orthopaedic procedure and that delays or suboptimal specimen handling have been associated with lower yields and higher false negatives.[93] In the VGEI context, extrapolated evidence suggests that reducing the time from acquisition of peri-operative tissue and graft or endograft samples to placement into sterile containers and subsequent incubation could diminish the contamination risk. Short term storage at room temperature may be acceptable when immediate processing is impossible, with refrigeration considered if delays are prolonged. Immersion in saline or excess fluid should be avoided as it can dilute organisms and reduce culture sensitivity.

Swab cultures from draining wounds or sinus tracts, as well as cultures from sampled negative pressure wound therapy (NPWT) foams, should be interpreted with caution as the results may reflect skin flora rather than the infecting pathogen, thus leading to misdiagnosis.[94,95]

Recommendation 11 — **New**

When vascular graft or endograft infection is suspected or diagnosed, collection of at least three deep perigraft or endograft tissue samples is recommended for optimal microbiological diagnosis.

Class	Level	Reference
I	C	Consensus

Recommendation 12 — **New**

When vascular graft or endograft infection is suspected or diagnosed, dividing explanted graft or endograft material and deep perigraft or endograft tissue into multiple pieces using sterile instruments and placing them promptly in separate sterile containers for microbiological analysis is recommended.

Class	Level	Reference
I	C	Consensus

Recommendation 13 — **Unchanged**

Routine cultures from clinically non-infected superficial wounds, sinus tracts, or negative pressure wound therapy foams are not indicated in suspected or diagnosed vascular graft or endograft infection.

Class	Level	Reference
IIIa	C	Consensus

Percutaneous punctures with direct fluid and tissue sampling, including direct aneurysm sac guided aspiration, play a possible role in diagnosing VGEI, particularly when the diagnosis is unclear, when less invasive methods have failed to identify a pathogen, or when surgical intervention is not appropriate.[67,96] Image guided needle biopsies or fine needle aspirations can target areas of suspected infection, such as perigraft fluid collections or abscesses, for microbiological and histopathological analysis. However, percutaneous punctures may introduce skin commensals such as *Cutibacterium* spp., and microbiological results should be interpreted carefully and contextually: isolation of the same microorganism from multiple deep samples increases the likelihood of causality *vs.* contamination.[67] Percutaneous punctures also carry procedure related risks such as organ damage, graft or endograft damage, or bleeding, and require MDT evaluation. In one report of 12 patients with intracavity VGEI who underwent percutaneous puncture and drainage, standard cultures of drain fluid were positive in nine patients, without any procedure related complications reported.[97] In another series of 31 aspirations in 27 patients with intracavity VGEI, a pathogen was identified in 25 aspirations, with moderate, non-permanent, and non-fatal procedure related complications reported in three patients.[67] In a retrospective cohort of 69 patients with VGEI (45 intracavity and 24 extracavity), cultures from percutaneous aspiration and or drainage were positive in 29 of 45 intracavity cases and 15 of 24 extracavity cases, with a low frequency of identified contaminants (six of 65) and complications (four of 65, including one fatal).[28]

Recommendation 14 — **Changed**

Percutaneous imaging guided sampling (e.g., perigraft aspiration or drainage) should be considered before surgical treatment in selected patients with suspected vascular graft or endograft infection when the diagnosis remains uncertain after non-invasive workup and or when conservative treatment is considered.

Class	Level	References	ToE
IIa	C	Colonna *et al.* (2025),[28] Ljungquist *et al.* (2021),[67] Kennedy *et al.* (2022)[97]	

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The majority of patients with VGEI receive antimicrobial therapy before specimen collection, often leading to a high rate of negative culture results. In selected haemodynamically stable patients without bloodstream infection or sepsis (including signs of organ dysfunction) and who are at low risk of immediate infective vascular complications, it is reasonable to consider delaying the initiation of antimicrobial therapy until surgical or percutaneous sampling has been completed.

In patients with suspected VGEI and early exposure to empirical antimicrobial therapy, the benefit of antimicrobial therapy withdrawal prior to direct tissue sampling in terms of microbiological yield is poorly understood. In a study of 331 patients with suspected prosthetic joint infections, withdrawal of antimicrobial therapy more than fourteen days before tissue sampling increased the yield of positive tissue cultures (76.9% *vs.* 41.2%).[98] A high degree of caution should be exercised when attempting to extrapolate these results to patients with VGEI to avoid harm: the risk of antimicrobial withdrawal, including early infections and vascular complications, needs to be individually assessed by an MDT in each suspected VGEI patient.

Recommendation 15 **New**

Delaying initiation of antimicrobial therapy until diagnostic microbiological tissue samples have been obtained should be considered in selected patients* with suspected or diagnosed vascular graft or endograft infection, to improve microbiological diagnostic yield.

Class	Level	Reference
IIa	C	Consensus

* Haemodynamically stable, without signs of sepsis, and at low risk of infection related complications.

Recommendation 16 **New**

Temporary withdrawal of ongoing antimicrobial therapy for at least forty-eight hours before microbiological sampling may be considered in selected patients* with culture negative suspected or diagnosed vascular graft or endograft infection, to improve microbiological diagnostic yield.

Class	Level	Reference
IIb	C	Consensus

* Haemodynamically stable, without signs of sepsis, and at low risk of infection related complications.

3.6.3. Microbiological diagnostic adjuncts

3.6.3.1. Sonication. The recovery in cultures of prosthetic samples may be improved by sonication, where ultrasonic waves disturb the surface biofilm on foreign material.[84,99,100] In this method, the explanted vascular graft or endograft is agitated in fluid, exposed to ultrasound to release adherent bacteria, and the resulting sonication fluid is cultured. Sonication can increase diagnostic yield.[84,99–101] A previous study showed that sonication of explanted vascular grafts or endografts improved the microbiological yield in nine of 57 samples.[100] Another prospective study compared sonication fluid inoculation into blood culture bottles with sonication fluid culture for diagnosing VGEI: the microbiological diagnosis rate was 64.9%, identical for both methods, increasing to 70.3% when both methods were combined. Sonication fluid culture identified a greater diversity of microorganisms, leading to more polymicrobial diagnoses, while both methods had the same rate of potential contaminant organisms.[101]

Although sonication can increase the diagnostic yield, its availability remains limited and the exact protocols may vary between institutions.

Recommendation 17 **Unchanged**

Sonication of explanted vascular graft or endograft in patients with suspected or diagnosed vascular graft or endograft infection may be considered in order to improve microbiological diagnostic yield.

Class	Level	References	ToE
IIb	C	Ulcar *et al.* (2018),[84] Puges *et al.* (2018),[99] Braams *et al.* (2023),[100] Puges *et al.* (2024)[101]	

3.6.3.2. Polymerase chain reaction and cell free DNA. Molecular diagnostic techniques, such as 16S rRNA PCR and 23S rRNA PCR, have enhanced pathogen detection in infective endocarditis and may offer similar benefits in VGEI. The combination of molecular techniques and conventional cultures of perigraft or endograft or of graft or endograft specimens improved bacterial identification in a small prospective cohort of patients with VGEI pre-treated with antibiotics.[102]

An emerging technology is metagenomic next generation sequencing of microbial cell free DNA (cfDNA) from tissue or plasma, which has gained attention for diagnosing infections such as infective endocarditis.[103,104] This method detects microbial cfDNA in plasma and is able to identify over 1 000 pathogens, including bacteria and fungi.[104] However, further studies are needed to confirm its utility in diagnosing VGEI. Molecular testing should be considered when other methods fail, but results need to be interpreted cautiously in polymicrobial infections, which are common in VGEI affecting the abdomen and groin, as the test may not reliably detect all pathogens.[14]

Recommendation 18 **New**

Molecular testing of deep tissue and or explanted graft or endograft should be considered in suspected or diagnosed vascular graft or endograft infection to improve microbiological diagnostic yield, particularly in patients receiving antimicrobial therapy and or with negative cultures.

Class	Level	Reference
IIa	C	Consensus

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3.6.3.3. Serology and mycobacterial cultures. In culture negative VGEI, serology can be a valuable diagnostic tool, especially when atypical and rare microorganisms are suspected (Table 9). It helps detect antibodies against specific pathogens when cultures fail to identify an organism. Serological tests can target rare organisms associated with VGEI, such as *C. burnetii*, *Brucella*, or *Bartonella* spp.[76–78] These pathogens are often implicated in chronic, low grade infections where cultures may be negative due to previous antimicrobial exposure. Although serology can support the diagnosis, it must be interpreted in the context of the clinical findings and imaging results, as false positives and cross reactivity may occur. Notably, *C. burnetii* infections can be polymicrobial, as they are occasionally complicated by primarily vasculodigestive fistulas.[79] Therefore, *C. burnetii* serology should not be limited to culture negative VGEIs in the presence of risk factors or fistula.

Mycobacterial VGEI is a rare condition, but *Mycobacterium chimaera*, part of the *Mycobacterium avium* complex, has been identified as a significant pathogen in cases of delayed onset, blood culture negative VGEI.[105] A large multicontinental outbreak of *M. chimaera* infections was linked to contaminated heater cooler devices used during cardiopulmonary bypass, with an estimated incidence of 156 – 282 cases per 100 000 population annually before preventive measures were implemented.[106]

Recommendation 19 **New**

Serology (e.g., *Coxiella burnetii*, *Bartonella*, *Brucella*) and mycobacterial cultures should be considered in culture negative patients with suspected or diagnosed vascular graft or endograft infection when relevant risk factors* are present.

Class	Level	Reference
IIa	C	Consensus

* **Mycobacteria:** cardiopulmonary bypass surgery using a 3T heater cooler unit (HCU) contaminated with *Mycobacterium chimaera*. History of Bacillus Calmette–Guérin immunotherapy for non-muscle invasive bladder cancer, leveraging the immune modulating properties of live attenuated *Mycobacterium bovis*. Secondary VGEI in cases of a *Mycobacterium tuberculosis* related mycotic aneurysm. ***Coxiella burnetii* (Q fever):** contact with infected animals or consumption of contaminated dairy products. ***Bartonella*:** exposure to lice or fleas (e.g., *Bartonella quintana*) or contact with infected cats (*Bartonella henselae*). ***Brucella*:** occupational exposure to livestock, consumption of unpasteurised dairy products, or contact with infected animal products.

3.6.3.4. Histopathology. Several structural and morphological changes in infected vascular grafts and endografts have been described, but their diagnostic value for VGEI remains unclear.[82,107] Histopathological analysis of peri-prosthetic tissue, aimed at identifying non-viable microbial structures or signs of acute inflammation, is routinely used in the diagnosis of prosthetic joint infections. Reported sensitivities and specificities are around 70 – 100% and 80 – 95%, respectively.[108–110] The role of histopathology in analysing

Table 9. Rare or unusual causes of vascular graft or endograft infection.

Pathogen (disease)	Exposure risk	Laboratory diagnosis
Bartonella spp. (*Bartonella henselae* ++, cat scratch disease; *Bartonella quintana*, trench fever)	Bites and scratches from kittens or feral cats: *B. henselae* Lice (homeless with fever): *B. quintana* Other animals: *Bartonella* spp. (*Bartonella alsatica*, rabbits…)	Serology (IgG >1:800), tissue culture, immunohistology using Warthin–Starry stain, 16S or 23S rRNA PCR
Brucella (brucellosis)	Zoonosis (cattle, goats, swine, dogs) Consumption of raw dairy Occupational risks (farmers, vets, laboratory and abattoir workers)	Serology, blood or tissue cultures (prolonged incubation), immunohistology, 16S or 23S rRNA PCR of tissue
Coxiella burnetii (Q fever)	Rearing livestock (cattle, sheep, goats) Domestic and wild birds, poultry Consumption of raw dairy, tick bites Sometimes rural exposure only	Serology (IgG phase I >1:800), tissue culture, immunohistology, 16S or 23S rRNA PCR of tissue
Mycobacteria spp.	Epidemiological risks (country of birth and ethnicity for tuberculosis) Chronic pulmonary disease, immunosuppression and BCG treatment (bladder cancer) for atypical mycobacteria	Acid fast staining of biopsy material and pus, mycobacteria blood cultures, mycobacteria specific PCR
Mycoplasma spp.	Sexual contact, per-operative contamination (*Mycoplasma hominis*) Respiratory tract infection (*Mycoplasma pneumoniae*)	Serology, 16S or 23S rRNA PCR
Pasteurella multocida (pasteurellosis)	Cat oral flora (less commonly canine) Pet bites	Standard culture

IgG = immunoglobulin G; rRNA = ribosomal ribonucleic acid; PCR = polymerase chain reaction; BCG = Bacillus Calmette–Guérin.

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perigraft tissue for VGEI is less established. An old study found acute inflammation (defined as presence of neutrophils, granulocytes, or necrosis) to be significantly more common in explanted infected vascular grafts or perigraft tissue compared with controls (75% *vs.* 14%).[111] The other potential utility of histopathology of aortic and perigraft tissue includes the diagnostic rule of rare pathologies occasionally mimicking VGEI on clinical presentation and radiological findings in selected cases, including lymphoma, soft tissue tumours, vasculitides, or immunoglobulin G4 (IgG_4) related disease.

Figure 4 provides a summarised flow chart of the recommended microbiological diagnostic workup in patients with VGEI.

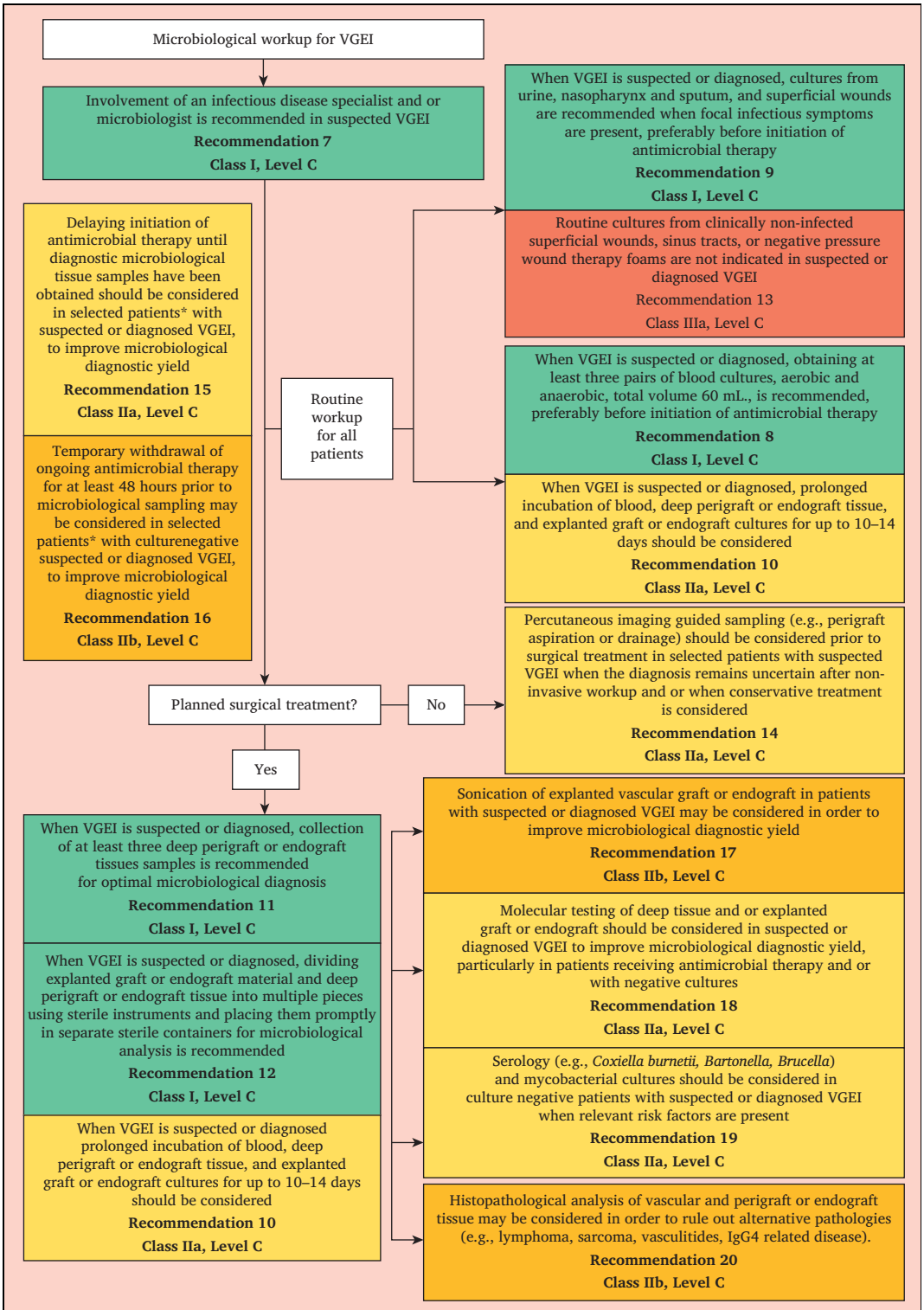

Figure 4. Flowchart summarising the recommendations for microbiological diagnostic workup of patients with vascular graft or endograft infection (VGEI). * Haemodynamically stable, without signs of sepsis, and at low risk of infection related complications.

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Recommendation 20		New
Histopathological analysis of vascular and perigraft or endograft tissue may be considered in order to rule out alternative pathologies (e.g., lymphoma, sarcoma, vasculitides, IgG_4 related disease).		
Class	**Level**	**Reference**
IIb	C	Consensus

4. MULTIDISCIPLINARY MANAGEMENT OF VASCULAR GRAFT OR ENDOGRAFT INFECTION

It is impossible to have a one size fits all approach to VGEI management.

In the absence of a life threatening emergency requiring immediate treatment, MDT expertise (refer to Chapter 2) is necessary in order to secure and obtain microbiological proof of infection, perform adequate imaging investigations, and plan optimal personalised management.

Recommendation 21		New
Multidisciplinary team management of vascular graft or endograft infection is recommended.		
Class	**Level**	**Reference**
I	C	Consensus

For active haemorrhage due to intracavity VGEI, endovascular treatment if often required as a first line procedure in order to stop the haemorrhage and stabilise the patient. MDT management in a tertiary centre is then mandatory, given the complexity of VGEI management and the poor prognosis associated with this pathology.

Recommendation 22		Unchanged
Patients with suspected or diagnosed intracavity vascular graft or endograft infection are recommended to be referred to a tertiary centre with multidisciplinary team management.		
Class	**Level**	**Reference**
I	C	Consensus

Important parameters must be considered by the MDT in order to decide on the best strategy to adopt: (1) clinical and functional status of the patient; (2) comorbidities of the patient; (3) location of the graft or endograft; (4) technical difficulties (e.g., complex fenestrated abdominal endograft explantation, presence of a fistula); (5) is the bacterial pathogen(s) known, which would enable a targeted antimicrobial therapy strategy, if not an empirical antimicrobial therapy strategy must be employed; and lastly (6) patient choice. The role of the MDT is to decide, with respect for the patient's views, preferences, and autonomy, between a curative or a palliative approach. The cornerstone of treatment is antimicrobial therapy, but the MDT should also decide between a conservative strategy (considering percutaneous drainage) or a surgical strategy (graft preserving strategy, partial or complete graft removal, and type of vascular reconstruction if needed).

Moreover, since VGEI has a dynamic course, individual treatment strategies might also change depending on the patient's clinical evolution and response to treatment. Hence, it is difficult to provide clear recommendations on the best treatment to provide in VGEI cases.

5. ANTIMICROBIAL THERAPY

5.1. Antimicrobial prophylaxis

Antimicrobial prophylaxis has been shown to reduce the risk of wound infection and VGEI in arterial reconstructions. Accordingly, in all patients undergoing open or endovascular arterial repair, peri-operative systemic antimicrobial prophylaxis is recommended.[4]

Recommendation 23			Unchanged
When a vascular graft or endograft is implanted, systemic antimicrobial prophylaxis is recommended.			
Class	**Level**	**References**	**ToE**
I	A	Stewart *et al.* (2007),[112] Cristino *et al.* (2025),[113] Correia *et al.* (2025)[114]	

Once implanted, the graft or endograft can become infected at any time after the index surgery, especially in the presence of bacteraemia. Analogous to prosthetic cardiac valves, antimicrobial prophylaxis should be considered for patients with a vascular graft or endograft undergoing procedures carrying a risk of bacteraemia. Such procedures are dental procedures with manipulation of the gingival or peri-apical region of teeth or perforation of the oral mucosa, including scaling and root canal procedures, gastrointestinal procedures with risk of bowel opening, or urogenital procedures.

Recommendation 24		Unchanged
Prophylactic antimicrobial therapy should be considered in patients with intracavity vascular graft or endograft undergoing procedures that carry a high risk of bacteraemia.		
Class	**Level**	**Reference**
IIa	C	Consensus

5.2. Timing of antimicrobial therapy

When VGEI is suspected in a clinically stable patient, microbiological sampling is recommended before antimicrobial therapy is initiated.[14,115] This includes obtaining three pairs of blood cultures and, in the presence of focal infection symptoms, relevant site specific cultures (see Chapter 2). If the clinical situation allows, delaying initiation of antimicrobial therapy until diagnostic intraoperative tissue samples are collected should be considered. This is particularly important in intracavity abdominal VGEIs, which are often polymicrobial and predominantly involve gut flora. Prolonged antimicrobial therapy before surgery should be avoided, as it may select for resistant

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bacterial strains and *Candida* species, complicating infection management.[116] In patients with culture negative VGEI, temporary discontinuation of ongoing antimicrobial therapy before microbiological sampling may be considered if the clinical situation allows, in order to improve the diagnostic yield. In haemodynamically unstable patients (sepsis or septic shock) or when awaiting urgent culture results, early initiation of broad spectrum empirical antimicrobial therapy remains critical (see Chapter 2).

Once definitive microbiological results are available, targeted antimicrobials can be administered, primarily guided by culture results and resistance patterns.

Recommendation 25 New

Targeted antimicrobial therapy is recommended once definitive microbiological results are available in patients with vascular graft or endograft infection.

Class	Level	Reference
I	C	Consensus

5.3. Duration of antimicrobial therapy

5.3.1. General approach. Various antimicrobial treatment durations have been reported for VGEI, but no comparative studies have been conducted to establish an optimal duration. Consequently, evidence based guidelines on treatment duration are lacking. In general, a minimum of 6 weeks of antimicrobial therapy (intravenous treatment with switch to oral treatment) is recommended for patients whose vascular graft or endograft has been completely removed and replaced, considering factors such as repair type, clinical progress, inflammatory parameters, and imaging findings.[14,69,115,117]

Recommendation 26 New

A minimum post-operative antimicrobial therapy duration of 6 weeks should be considered after complete graft or endograft explantation in patients with vascular graft or endograft infection.

Class	Level	Reference
IIa	C	Consensus

Shorter treatment durations may be considered after autologous venous reconstruction (since the absence of retained foreign material obviates a persistent biofilm nidus) or when no vascular reconstruction was required, provided all infected material has been fully resected and adequate source control is confirmed.[14,69,115,117] The criteria allowing the discontinuation of antimicrobial therapy have not yet been studied in the literature but should include the absence of clinical signs of infection, the normalisation of inflammation blood markers (WBC count, CRP), and the absence of radiological signs of infection (CTA and or nuclear imaging).

Treatment duration may need to be extended if inflammatory markers remain elevated at the end of the planned therapy or in the presence of undrained fluid collections.

5.3.2. Special situations. When partial explantation of the graft or endograft has been selected, prolonged antimicrobial therapy is often required due to the danger of occult retained infected material. A previous study reported outcomes of 169 patients with abdominal VGEI treated with a partial graft explantation approach *vs.* complete surgical explantation. Adjusted long term survival was comparable between the two approaches, particularly in patients without fistula. The only statistically significant difference was a higher re-infection rate (45% *vs.* 19%) in favour of complete surgical explantation. This suggests that a partial graft explantation approach may be a viable option for high risk surgical patients without fistula, but prolonged antimicrobial therapy is mandatory.[75]

Even conservative management may be a viable option for patients who are not candidates for surgery, although evidence supporting this strategy is limited, with small patient numbers, no control groups, and a focus on non-operative candidates.[19,96,118] However, for such patients, lifelong suppressive antimicrobial therapy is mostly the only option. Management and monitoring are then best coordinated through an outpatient parenteral antimicrobial therapy (OPAT) programme, with regular follow up to detect adverse effects and to assess treatment efficacy.[119] Close collaboration and MDT management is critical to ensure optimal patient outcomes.

The decision to shorten duration, extend therapy, or initiate long term suppression should be made on a case by case basis, after discussion in MDT meetings.

Recommendation 27 New

Long term or lifelong antimicrobial therapy should be considered after surgical or conservative treatment in patients with vascular graft or endograft infection, when residual infection is likely.

Class	Level	References	ToE
IIa	C	Kouijzer *et al.* (2022),[19] Ljungquist *et al.* (2023),[96] Van Hemelrijck *et al.* (2025)[118]	

Recommendation 28 New

Cessation of long term antimicrobial therapy, under close monitoring, may be considered after surgical or conservative treatment in patients with vascular graft or endograft infection, when residual infection is assessed to be unlikely.

Class	Level	Reference
IIb	C	Consensus

5.4. Switch to oral antimicrobial therapy

Data extrapolated from orthopaedic surgery and endocarditis management suggest that a transition from intravenous to oral antimicrobial therapy can be considered 10 — 14 days after surgery, provided there is evidence of sufficient bacterial load reduction, as indicated by a significant and rapid decline in serum inflammatory markers.[115] An

earlier switch may be considered depending on the patient's evolution and the microbial documentation including pathogen susceptibility, after MDT discussion. This switch is only appropriate if oral options with adequate bioavailability are available, the patient can tolerate the medication, and gastrointestinal drug absorption remains intact. Moreover, caution is warranted in patients with extensive bowel resections, as absorption may be impaired. In selected situations, such as extreme body weight, renal or hepatic failure, infections with multidrug resistant microorganisms, risk of significant drug—drug interactions, or shortages of specific antimicrobials, pharmacist assisted antimicrobial monitoring should be considered to support optimal dosing and treatment efficacy.

Recommendation 29 **New**

An early switch from intravenous to oral antimicrobial therapy (10 − 14 days post-surgery) may be considered in clinically stable patients with vascular graft or endograft infection, provided oral options with adequate bioavailability are available.

Class	Level	Reference
IIb	C	Consensus

5.5. Selection of empirical antimicrobial treatment

5.5.1. Intracavity thoracic vascular graft or endograft infection. Overall, research on thoracic VGEI is limited, with most studies involving fewer than 50 patients, highlighting the need for further investigation into treatment strategies and outcomes. Distinguishing thoracic VGEI from infective endocarditis can be challenging, particularly in patients with bacteraemia and non-diagnostic transoesophageal echocardiography findings. Table 10 summarises the causative microorganisms for intracavity thoracic VGEI, based on 11 studies involving 308 patients.[18,19,118,120–127] The likelihood of VGEI is higher in patients with thoracic aortic grafts with monomicrobial Gram positive bloodstream infections than with Gram negative bacteraemia, although the risk varies depending on the specific pathogen. Until more data are available on VGEI prevalence in patients with bloodstream infection, pathogens commonly associated with infective endocarditis should be considered high risk for thoracic VGEI, such as staphylococci, streptococci, and enterococci.

Given the wide range of potential pathogens involved, an empirical combination therapy targeting methicillin resistant staphylococci, enterococci, and streptococci is often initiated following infective endocarditis guidelines.[128] In cases of fistula to the respiratory tract, adding empirical coverage for Gram negative bacteria, anaerobes, and fungi is reasonable.[14,117] Once blood and tissue culture results are available, the antimicrobial regimen should be adjusted accordingly.[115,117]

5.5.1.1. Thoracic vascular graft or endograft infection due to Staphylococcus aureus and coagulase negative staphylococci. For methicillin sensitive *S. aureus* (MSSA) thoracic VGEI, therapy may be narrowed to flucloxacillin or cefazolin.[115] Daptomycin is a well established alternative to vancomycin in MSSA VGEI, particularly in patients at risk of nephrotoxicity. Rifampicin may be considered as an adjunct in staphylococcal infections owing to its activity against biofilm. Rifampicin should not be used for long term suppression owing to many drug—drug interactions and the risk of acquired resistance.

5.5.1.2. Thoracic vascular graft or endograft infection due to enterococci and streptococci. No studies specifically address the treatment of streptococcal or enterococcal thoracic VGEI. As a result, the following recommendations are based on extrapolations from endocarditis and a Delphi consensus.[115] For enterococcal VGEI, dual β lactam therapy with amoxicillin and ceftriaxone should be used as suggested for infective endocarditis. For streptococcal VGEI,

Table 10. Studies reporting on the causative microorganisms for intracavity thoracic vascular graft or endograft infection.

Author, year	Intracavity VGEI	*S. aureus*	CoNS	*Streptococcus* spp.	*Enterococcus* spp.	GNB	*P. aeruginosa*	Anaerobes	Yeasts	Culture negative	Polymicrobial
Van Hemelrijck, 2025[118]	66	12	7	14	3	2	1	NR	NR	14	NR
Kouijzer, 2022[19]	24	10	2	4	1	1	0	2	0	1	NR
Sandhu, 2021[120]	32	12	NR	NR	NR	6	NR	6	4	2	3
Schweizer, 2020[121]	28	7	3	4	2	1	1	5	0	5	NR
Mestres, 2019[122]	50	8	7	1	2	6	1	1	6	17	11
Machelart, 2017[123]	10	3	NR	2	1	NR	NR	NR	NR	3	1
Ramos, 2016[124]	31	4	7	4	3	1	1	2	1	2	NR
Erb, 2014[18]	24	4	8	0	5	5	1	7	1	NR	5
Takano, 2014[125]	8	5	1	1	1	0	0	0	0	0	NR
Shah, 2011[126]	15	5	0	1	1	0	1	4	0	2	NR
Coselli, 1999[127]	20	5	5	1	2	1	1	0	1	NR	3
Total	308	63 (20.0%)	40 (13.0%)	44 (14.3%)	21 (8.8%)	23 (7.5%)	7 (2.3%)	27 (8.8%)	13 (4.2%)	46 (14.9%)	23 (7.5%)

Data are presented as number of microorganisms. *S. aureus* = *Staphylococcus aureus*; CoNS = coagulase negative staphylococci; GNB = Gram negative bacilli; *P. aeruginosa* = *Pseudomonas aeruginosa*; NR = not reported.
Publications are ordered by the publication date, with the most recent publication listed first.

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penicillin is the preferred antibiotic. The use of aminoglycosides for severe streptococcal infections is currently debated due to concerns over their potential toxicity. The effectiveness of other antibiotics with antistreptococcal activity for treating VGEI has yet to be determined.

Recommendation 30			New
Empirical antimicrobial treatment for thoracic vascular graft or endograft infection covering *Staphylococcus aureus*, coagulase negative staphylococci, streptococci, and enterococci is recommended.			
Class	**Level**	**References**	**ToE**
I	C	Erb *et al.* (2014),[18] Koujizer *et al.* (2022),[19] Van Hemelrijck *et al.* (2025),[118] Sandhu *et al.* (2021),[120] Schweizer *et al.* (2020),[121] Mestres *et al.* (2019),[122] Machelart *et al.* (2017),[123] Ramos *et al.* (2016),[124] Takano *et al.* (2014),[125] Shah *et al.* (2011),[126] Coselli *et al.* (1999)[127]	

5.5.2. Intracavity abdominal vascular graft or endograft infection. Selecting an appropriate empirical antimicrobial regimen targeting the most common causative microorganisms is critical for the successful treatment of intracavity abdominal VGEI and has been shown to reduce the mortality rate in recent retrospective studies.[13,72] Table 11 summarises the causative microorganisms for intracavity abdominal VGEI, based on 14 studies involving 701 patients.[9,13,18,66,68,72,116,129–135] Polymicrobial infections accounted for 35.7% of cases. The most isolated pathogens included *S. aureus*, CoNS, streptococci, enterococci, Gram negative bacilli, anaerobes, and *Candida* species.

Two studies specifically addressed the prevalence of *Candida* species in patients with and without fistula to the bowel.[66,116] While the prevalence of *Candida* was significantly higher in patients with a fistula, approximately 10% of patients without a fistula also harboured *Candida* species.[66] Moreover, *Candida* infections were associated with a higher mortality rate compared with bacterial infections alone.[116,134] As such, covering *Candida* species in both clinical scenarios is required, but especially in the presence of a fistula to the bowel.

Due to variations in the incidence of AmpC producing microorganisms, non-fermenters, and resistance patterns among Gram positive and Gram negative bacteria across different regions, the specific choice of antibiotics should be guided by local epidemiological data. The currently available literature does not allow for specific recommendations: some studies reporting on Gram negative bacilli may include non-fermenters and or AmpC producing Gram negative bacteria, reports on enterococci often do not differentiate

Table 11. Studies reporting on the causative microorganisms for intracavity abdominal vascular graft or endograft infection (VGEI).

Author, year	Intracavity VGEI	*S. aureus*	CoNS	*Streptococcus* spp.	*Enterococcus* spp.	GNB	*P. aeruginosa*	Anaerobes	Yeasts	Polymicrobial
Monnier, 2024[116]	148	27	18	11	13	32	13	19	22	39
Stockschläder, 2024[13]	49	7	7	3	11	13	2	6	12	19
Khalid, 2023[135]	34	3	5	5	6	6	NR	2	4	12
Dominguez-Cainzos, 2023[131]	10	2	0	1	3	2	0	2	1	3
Sixt, 2022[72]	18	5	1	1	0	4	3	NR	0	NR
Dorpmans, 2022[134]	56	NR	NR	NR	NR	NR	NR	NR	10	NR
Caradu, 2022[133]	74	15	NR	NR	12	15	8	NR	13	20
Langenskiöld, 2021[132]	40	5	8	3	6	12	NR	10	0	NR
Gavali, 2021[66]	120	8	26	26	30	40	1	13	26	29
Gouveia E Melo, 2021[130]	16	0	5	2	4	14	NR	0	1	6
Dirven, 2015[129]	11	0	1	1	1	1	0	2	3	36
Erb, 2014[18]	27	6	3	0	11	16	1	10	10	18
Legout, 2012[9]	54	12	10	6	5	20	1	6	3	12
Saleem, 2010[68]	44	8	12	0	1	6	3	0	3	NR
Total*	701	98 (14.0%)	96 (13.7%)	59 (8.4%)	103 (14.7%)	181 (25.2%)	32 (4.6%)	70 (10.0%)	108 (15.4%)	194 (27.7%)

Data are presented as number of microorganisms. VGEI = vascular graft or endograft infection; *S. aureus* = *Staphylococcus aureus*; CoNS = coagulase negative staphylococci; GNB = Gram negative bacilli; *P. aeruginosa* = *Pseudomonas aeruginosa*; NR = not reported.
Publications are ordered by publication date, with the most recent publication listed first.
* Those studies not reporting on a specific microorganism were not included in the final calculation.

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between *Enterococcus faecalis* and *Enterococcus faecium*, and the distinction of anaerobes between Gram positive and Gram negative species is frequently omitted.

Recommendation 31 New

Empirical antimicrobial treatment for abdominal vascular graft or endograft infection covering *Staphylococcus aureus*, coagulase negative staphylococci, streptococci, enterococci, Gram negative bacilli, and anaerobes is recommended.

Class	Level	References	ToE
I	C	Legout *et al.* (2012),[9] Stockschläder *et al.* (2024),[13] Erb *et al.* (2014),[18] Gavali *et al.* (2021),[66] Saleem *et al.* (2010),[68] Sixt *et al.* (2022),[72] Monnier *et al.* (2024),[116] Dirven *et al.* (2015),[129] Gouveia E Melo *et al.* (2021),[130] Dominguez-Cainzos *et al.* (2023),[131] Langenskiöld *et al.* (2021),[132] Caradu *et al.* (2022),[133] Dorpmans *et al.* (2022),[134] Khalid *et al.* (2023)[135]	

Recommendation 32 New

Covering *Candida* species as part of the empirical antimicrobial treatment for abdominal vascular graft or endograft infection with erosion or fistula is recommended.

Class	Level	References	ToE
I	C	Gavali *et al.* (2021),[66] Monnier *et al.* (2024)[116]	

5.5.3. Extracavity vascular graft or endograft infection.

Table 12 summarises the causative microorganisms for extracavity VGEI, based on six studies involving 253 patients.[9,13,18,72,130,136] Polymicrobial infections accounted for 23.7% of cases. Staphylococci are the most common pathogens. *Staphylococcus aureus* predominated in some studies,[9,72] while others reported equal distributions between *S. aureus* and CoNS.[13,18,130] Other frequently isolated organisms include Enterobacterales, enterococci, non-fermenting Gram negative bacilli particularly *P. aeruginosa*, *Candida* spp., streptococci, and anaerobes.

Antimicrobial resistance rates should be assessed at each hospital to better understand local patterns and to optimise empirical antimicrobial therapy regimens. Notably, the prevalence of methicillin resistant staphylococci is a significant concern and must be addressed in empirical treatment strategies. Methicillin resistance is reported in 13.3 — 17.5% of patients with extracavity VGEI[9,72] and 34.5 — 44.0% of staphylococcal isolates,[9,72,130] with resistance distributed between methicillin resistant *S. aureus* (MRSA)[72] and methicillin resistant CoNS.[9,130] One study reported 53.8% of Gram negative bacilli as multidrug resistant (MDR), with 34.6% of these classified as extensively drug resistant (XDR).[130] Limited data are available on anaerobic or enterococcal resistance profiles.[13]

A broad spectrum β lactam (e.g., cefepime or piperacillin/tazobactam) combined with an agent active against methicillin resistant staphylococci (e.g., daptomycin, vancomycin, or linezolid) is required. Daptomycin has demonstrated safety in patients with VGEI, while the nephrotoxicity of vancomycin may complicate post-surgical management.[9] In patients with severe sepsis or septic shock, the addition of an aminoglycoside, such as amikacin, is required.

Table 12. Studies reporting on the causative microorganisms for extracavity vascular graft or endograft infection (VGEI).

Author, year	Extracavitary VGEI	*S. aureus*	CoNS	MR staphylococci	Streptococci	Enterococci	Enterobacterales	*P. aeruginosa*	MDR/ XDR GNB	Anaerobes	*Candida*	Polymicrobial	Culture negative
Stockschläder, 2024[13]	29	8	7	NR	4	9	12	5	NR	2	5	13	1
Sixt, 2022[72]	128	40	8	17	3	4	12	4	20	2	1	24	20
Gouveia E Melo, 2021[130]	36	14	15	20	1	11	20	6	5	1	7	18	NR
Erb, 2014[18]	10	4	5	NR		2	2	1	NR	1	0	4	0
Siracuse, 2013[136]	19	6	7	NR	1	2	1	1	NR	0	0	2	2
Legout, 2012[9]	31	17	3	NR	1	2	12	2	NR	2	1	8	1
Total	253	89 (35.2%)	45 (17.8%)	37 (14.6%)	10 (4.0%)	30 (11.9%)	59 (23.3%)	19 (7.5%)	25 (9.9%)	8 (3.2%)	14 (5.5%)	69 (27.3%)	24 (9.5%)

Data are presented as number of microorganisms. VGEI = vascular graft or endograft infection; *S. aureus* = *Staphylococcus aureus*; CoNS = coagulase negative staphylococci; MR = methicillin resistant; *P. aeruginosa* = *Pseudomonas aeruginosa*; MDR = multidrug resistant; XDR = extensively drug resistant GNB = Gram negative bacilli; NR = not reported.
Publications are ordered by publication date, with the most recent publication listed first.

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Recommendation 33			New
Empirical antimicrobial treatment for extracavity vascular graft or endograft infection covering methicillin sensitive and resistant staphylococci, enterococci, and Enterobacterales is recommended.			
Class	**Level**	**References**	**ToE**
I	C	Legout *et al.* (2012),[9] Stockschläder *et al.* (2024),[13] Erb *et al.* (2014),[18] Sixt *et al.* (2022),[72] Gouveia E Melo *et al.* (2021),[130] Siracuse *et al.* (2013)[136]	

5.6. Adjunctive therapy

5.6.1. Local antibiotic use. Local antibiotic use includes antibiotic beads, local antimicrobial irrigation, and antibiotic grafts.

There are insufficient data to assess the efficacy of local antibiotic beads in the setting of VGEI. Indeed, antimicrobial bead use has been analysed retrospectively in several small studies, especially in extracavity anatomic locations.[137–139] However, data on bead composition are highly heterogeneous, primarily involving polymethylmethacrylate, but sometimes also calcium sulphate, which may be bioabsorbable or not. The choice of antibiotics also varies significantly, with gentamicin and vancomycin being the most commonly used. Bacterial documentation is inconsistent across studies, and the antibiotic susceptibility profiles of the isolates are rarely detailed. Surgical management associated with the implantation of antimicrobial beads also varies considerably between studies, including graft preservation or removal, use in conjunction with flaps, and sometimes iterative debridement and bead replacement. These variations make it difficult to draw valid conclusions regarding the efficacy of antimicrobial beads, and further research is needed before recommending their use.

Antibiotic irrigation using agents such as rifampicin, gentamicin, or daptomycin, delivered through percutaneous drains or intra-operative lavage, has been experimented with, but available data remain too limited to support a recommendation.

There is ongoing concern regarding the use of rifampicin bonded grafts owing to their relatively narrow antimicrobial spectrum, mainly active against Gram positive cocci, and the documented risk of promoting rifampicin resistant strains, already demonstrated *in vitro*.[140] In addition, rifampicin rapidly elutes from graft material, often within hours to a few days, resulting in only short lived local antimicrobial activity. Once concentrations drop below inhibitory levels, the graft surface can be recolonised by bacteria. Rifampicin's low genetic barrier to resistance further compounds the problem, as subtherapeutic residual drug levels may select for rifampicin resistant staphylococci, potentially compromising future systemic treatment options. Evidence supporting its long term clinical benefit remains limited, with most data derived from *in vitro* studies, animal models, or small observational series; robust randomised trials are lacking. Moreover, rifampicin lacks coverage against important Gram negative pathogens such as *P. aeruginosa* and many Enterobacterales, which are common in VGEI. Given these limitations, the use of rifampicin bonded grafts is not recommended.[20,140]

Recommendation 34			New
Rifampicin bonded grafts are not recommended for treatment of vascular graft and endovascular infection owing to their relatively narrow antimicrobial spectrum and the theoretical risk of developing rifampicin resistance.			
Class	**Level**	**References**	**ToE**
IIIb	C	Bandyk *et al.* (2001),[20] Berard *et al.* (2019)[140]	

5.6.2. Bacteriophage therapy. Bacteriophages are naturally occurring viruses that selectively infect specific bacteria. Lytic bacteriophages induce bacterial cell lysis, releasing new virions that subsequently infect other bacterial cells in an exponential manner.[141] While the antibacterial properties of bacteriophages were discovered over 100 years ago, the rise of antibiotics hindered further development of phage therapy. Consequently, much of the knowledge regarding bacteriophage mechanisms, handling, clinical use, and limitations was lost. However, the growing concerns of antimicrobial resistance and the shift toward personalised medicine have prompted a renewed interest in this approach, beginning with respiratory tract infections and bone and joint infections, with cardiovascular and vascular surgery emerging as potential fields for bacteriophage therapy.[141] However, bacteriophage therapy remains experimental in the VGEI field and further evidence is required before it can be considered for routine clinical practice.

6. MANAGEMENT OF THORACIC VASCULAR GRAFT OR ENDOGRAFT INFECTION

6.1. General considerations

This chapter sets the stage for a discussion on the treatment of VGEI in the descending thoracic aorta beyond the left subclavian artery and briefly comments on the thoracoabdominal aorta. There is no distinction made between an infected graft or endograft, unless clearly stated. The literature on treatment of the thoracic aorta is particularly scarce and of very low quality, not only due to the absence of randomised trials and population based cohorts, but also due to non-homogeneous reporting mixing the thoracic and abdominal aortic regions and different managements. There is also a lack of studies reporting on conservative treatment. Hence, most of the following recommendations are based on low quality literature and are therefore expert opinions.

The true incidence of thoracic VGEI is unknown, but it is probably below 1 — 2% after endovascular repair and 1 — 3% after open repair.[12,142,143] Thoracic VGEI can be complicated

by fistula to the airways or oesophagus, or even a combination of both. The true incidence of fistula complicating VGEI is unknown and may be under reported. In the European Registry of Endovascular Aortic Repair Complications (EuREC) report, among 4 680 thoracic endovascular aortic repair (TEVAR) procedures, 15 were complicated by fistula to the airways and 11 by fistula to the oesophagus.[144] Since the management of thoracic VGEI depends on the presence or absence of fistulation, this chapter is divided into thoracic VGEI without and with fistula.

6.2. Thoracic vascular graft or endograft infection without fistula

6.2.1. Conservative treatment. For most patients, conservative treatment is often the most appropriate option in uncomplicated thoracic VGEI (absence of any fistulation and or haemothorax), given the poor outcomes of surgical explantation.[12,118] A conservative treatment strategy requires close in hospital monitoring, including clinical examination, laboratory testing as well as imaging to ensure the patient responds well to treatment. This needs to be discussed at regular MDT meetings. If the patient does not respond well to treatment, a change of treatment approach should be considered.

Conservative treatment might also be used as a bridging period to control the infection and optimise nutritional status for later MDT evaluation of whether the patient might then be a candidate for surgery.[19,133,145]

6.2.1.1. Percutaneous drainage. In the presence of perigraft fluid collections or abscesses, image guided percutaneous drainage can be performed in combination with systemic antimicrobial therapy. The drain is left in place until the collection is totally or sufficiently drained, but might also be combined with irrigation.[67] Not all perigraft or endograft collections are amenable to percutaneous drainage (e.g., proximity to vital structures), and the risks of percutaneous drainage (e.g., seeding, haemorrhage, graft puncture) should be discussed on an individual basis by the MDT.

6.2.1.2. Irrigation. Irrigation with saline or an antiseptic solution (e.g., diluted betadine irrigation) is commonly used in infected surgical fields. In thoracic VGEI, its use could reduce the bacterial burden, but the risk of tissue toxicity needs to be considered.

Recommendation 35		Changed
Conservative treatment with close monitoring should be considered as first line therapy in most patients with thoracic vascular graft or endograft infection without fistula.		
Class	**Level**	**Reference**
IIa	C	Consensus

6.2.2. *In situ* reconstruction. For diagnosed thoracic VGEI in selected patients who are physiologically able to cope with open aortic surgery, total graft explantation and *in situ* repair may be considered at presentation or when conservative treatment results in treatment failure. Explantation of the infected graft or endograft, aggressive debridement of the arterial bed, and arterial reconstruction with suturing in healthy non-infected tissue using infection resistant material constitutes the basis of this treatment modality.

When considering *in situ* repair, the following should be considered: cardiovascular and respiratory status of the patient; functional level of the patient; aortic and graft or endograft anatomy and extension; MDT position; and wishes of the patient.

Recommendation 36		Changed
Total graft or endograft explantation and *in situ* repair should be considered in selected patients with thoracic vascular graft or endograft infection who are suitable for surgery or in cases where conservative treatment has failed.		
Class	**Level**	**Reference**
IIa	C	Consensus

6.2.2.1. Technical aspects. The operative technique largely depends on the location of the graft or endograft. If the proximal extension of the graft or endograft is distal to the left subclavian artery, the procedure can be performed through a left thoracotomy with left lung exclusion and left heart bypass. Circulatory arrest is rarely needed except for an open proximal anastomosis when the infection approximates the supra-aortic branch vessels. If the VGEI extends to the level of the left subclavian artery and clamping between the left common carotid artery and the left subclavian artery is not possible, hypothermic circulatory arrest can be used to allow complete material excision. When exposure of the thoraco-abdominal aorta is required, a thoracophrenolaparotomy is the preferred approach.

Dissection of the proximal part of the graft or endograft can be difficult due to inflammation caused by the infection or previous interventions. Damage to the lungs, especially with re-operation procedures, is common and care should be taken to minimise lung injury as well as to avoid injury to the vagus and recurrent laryngeal nerves, phrenic nerve, thoracic duct, and oesophagus. Neuromonitoring with motor evoked potentials provides real time assessment of spinal cord integrity, and cerebrospinal fluid drainage can diminish the risk of spinal cord ischaemia.[146,147] Similar to primary non-infected procedures, selective visceral perfusion is typically performed by direct cannulation and perfusion of the coeliac artery and superior mesenteric artery using blood from the left heart bypass circuit.[148] With regard to renal perfusion, histidine—tryptophan—ketoglutarate solution at 4°C has demonstrated superior renoprotective effects.[149,150]

6.2.2.2. Graft materials. The literature on graft materials for thoracic VGEI is of very low quality. A thorough literature investigation was performed for both thoracic and abdominal VGEI, and the details are accounted for in the abdominal chapter and are only summarised here. With the new consensus for reporting items and outcomes definitions, the choice of graft material for thoracic VGEI

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reconstruction will hopefully be better supported by newly acquired research results.

Xenoprosthetic reconstruction using tubularised bovine pericardium has been reported for *in situ* reconstruction of thoracic VGEI, tailoring a custom made tube by sewing or stapling pericardial sheets. In a multicentre study, data for 168 patients (38 native aortic infection, 130 VGEI) who underwent *in situ* aortic reconstruction (from the aortic root to the iliac bifurcation) using bovine pericardium over a thirteen year period were analysed. The thirty day mortality rate was 15% and the estimated five year mortality rate was 50%. Estimated freedom from recurrent infection was 86% at 5 years. Estimated freedom from graft complications (anastomotic aneurysm, anastomotic stenosis, or graft occlusion) was 87% at 5 years, and graft degeneration was not observed.[151] Although reconstruction using tubularised bovine pericardium requires further study, physician made bovine pericardial grafts offer many advantages such as availability, easy handling, and avoidance of harvesting trauma and therefore provide an attractive option for thoracic aortic reconstruction in the setting of VGEI.

Aortic reconstruction using antimicrobial polyethylene terephthalate (PET) grafts has mostly been reported in abdominal VGEI, with satisfactory results. Similarly, these grafts could be used for thoracic VGEI reconstruction. However, future studies are required.

Cryopreserved arterial allografts have also been used, since arterial allografts have been shown to be resistant to bacterial colonisation. However, arterial allografts have a substantial risk of degeneration, rupture, and bleeding. A study reported the use of cryopreserved arterial allograft for thoracic or thoraco-abdominal vascular reconstruction in 35 patients (14 native aortic infection, 21 VGEI).[152] Fifteen patients (43%) died during the first month or before discharge (including one patient with proximal anastomotic disruption). During a median follow up of 32.3 months, three allograft related complications (15%) occurred in survivors.[152] This stability concern is of importance in the thoracic location, where massive uncontrolled bleeding is a life threatening risk. Accordingly, despite limited evidence, cryopreserved arterial allografts are not currently recommended as a conduit for thoracic VGEI owing to safety concerns. Understanding the long term effects of cryopreservation on vascular tissue integrity will optimise allograft production and durability.

Recommendation 37 — New

Physician made bovine pericardium tube or antimicrobial prosthetic grafts should be considered for *in situ* reconstruction of thoracic vascular graft or endograft infection.

Class	Level	Reference
IIa	C	Consensus

Recommendation 38 — New

Cryopreserved arterial allografts are not recommended for *in situ* reconstruction of thoracic vascular graft or endograft infection.

Class	Level	Reference
IIIb	C	Consensus

6.2.2.3. Graft coverage. Graft coverage with vascularised tissue has little scientific support but is believed to be of importance to prevent development or recurrence of fistulation, to fill space after debridement, and also to promote antibiotics reaching the outer surface of the graft. Coverage of the new graft to avoid direct contact with the lungs and oesophagus using the surrounding tissues is often not possible within the thoracic cavity, and alternative flap coverage should be considered. The type of flap depends on the type of reconstruction that has been performed. An intercostal flap has limited volume and is best prepared at the time of thoracotomy to avoid damage caused by the retractor. When a pericardial flap is used, the pericardial defect might need to be repaired with synthetic material. A *pectoralis major* flap can also be used. An omentum flap can be prepared and routed through the diaphragm via the aortic hiatus to cover the new graft. More extensive muscular flaps, such as *latissimus dorsi* or *serratus* muscles, have also been proposed but require the involvement of a plastic surgeon.[145,153–156]

Recommendation 39 — Changed

Coverage of the vascular reconstruction for thoracic vascular graft or endograft infection with autologous vascularised tissue (e.g., omentum or muscle flaps) should be considered.

Class	Level	Reference
IIa	C	Consensus

6.2.3. Extra-anatomic reconstruction. Extra-anatomic reconstruction for thoracic VGEI can be performed in rare cases, in patients fit enough for surgery but when *in situ* reconstruction is deemed not possible or to avoid reconstruction in a contaminated field. Extra-anatomic reconstruction outside the infected field and secondary aortic ligation with removal of the infected graft or endograft can be performed in one or two stages.

Recommendation 40 — New

Extra-anatomic reconstruction for thoracic vascular graft or endograft infection may be considered in highly selected patients when *in situ* reconstruction is deemed not possible or to avoid reconstruction in a contaminated field.

Class	Level	Reference
IIb	C	Consensus

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6.2.3.1. Technical aspects. To reconstitute distal perfusion, axillobifemoral or bilateral axillofemoral bypasses can be performed. However, it should be noted that retrograde blood flow to the visceral organs might be insufficient when the descending aorta is excluded.[145]

Another complex extra-anatomic reconstruction option, which requires the presence of a cardiothoracic surgeon, is the so called ventral aorta consisting of a retrosternally placed new graft, which originates from the ascending aorta, the distal anastomosis being to the supracoeliac abdominal aorta or more distally, to the infrarenal aorta or iliac arteries. If possible, this reconstruction is performed in two steps, the first step being the bypass through sternotomy and upper laparotomy, and the second step being removal of the infected graft or endograft through thoracotomy.[145,156]

6.2.3.2. Stump management. For extra-anatomic reconstruction, the aortic stump should be oversewn and covered with an omental or muscular flap in order to reinforce the stump and reduce the risk of blowout. Typically, the aorta is transected just distal to the left subclavian artery or at the level of the diaphragm.[145]

6.2.4. Partial or total graft explantation. Partial graft explantation has little scientific support in thoracic VGEI management but can be performed in cases when the entire graft or endograft is deemed too hazardous to explant, or impossible to explant due to dense adhesion to surrounding tissues.

Recommendation 41		Changed
Partial graft explantation of thoracic vascular graft or endograft infection may be considered in selected patients when complete reconstruction is deemed high risk.		
Class	**Level**	**Reference**
IIb	C	Consensus

6.3. Thoracic vascular graft or endograft infection with fistula to other organs

Thoracic VGEI can be complicated by fistula, either aorto-oesophageal fistula (AOF) or fistula to the airways, such as aortobronchial fistula (ABF) or aortopulmonary fistula (APF), or a combination of these. Presentation and management differ between AOF and ABF or APF, since patients with AOF present more frequently with hypovolaemic shock and systemic infection compared with patients with ABF or APF, and require more additional procedures.[12,157] Therefore, ABF and AOF are not comparable and are considered separately in the following subchapters.

6.3.1. Thoracic vascular graft or endograft infection with aorto-oesophageal fistula. In a review of 162 patients with AOF, secondary AOF due to VGEI was the most common cause (37.6%), followed by primary AOF due to aortic aneurysm, bone ingestion, and thoracic cancer.[157]

Conservative treatment of secondary AOF complicating a thoracic VGEI results in poor survival with an estimated one year survival close to 0%.[12,156,157] Accordingly, conservative treatment of patients with thoracic VGEI with an AOF is a palliative strategy.

Recommendation 42		Unchanged
Conservative treatment of patients with aorto-oesophageal fistula complicating thoracic vascular graft or endograft infection should be considered a palliative approach.		
Class	**Level**	**References** **ToE**
IIa	C	Gavali *et al.* (2026),[12] Kahlberg *et al.* (2019),[156] Takeno *et al.* (2020)[157]

When a curative approach is selected, the treatment depends on the clinical presentation, particularly whether or not the patient presents with profuse haemorrhage (e.g., haemodynamic instability and transfusion requirement).

6.3.1.1. Aorto-oesophageal fistula without profuse haemorrhage. Reviews of the literature showed significantly better survival for patients treated with repair of both the oesophagus and the descending thoracic aorta compared with treatment of only one of the two structures.[156–158] For this reason, in fit and haemodynamically stable patients with thoracic or thoraco-abdominal VGEI complicated by an AOF, explantation of the infected material followed by *in situ* revascularisation combined with repair of the oesophagus, by an upper gastrointestinal surgeon, and coverage with viable tissue is required. A pedicled omental flap or a pedicled intercostal muscle flap can be used to protect the repaired aorta from the resected and eventually repaired oeosophagus.[155,159]

A staged procedure can be adopted for oesophageal repair. Once the infected material is explanted and the descending aorta is repaired, the oesophagectomy can be done with a combined cervical oesophagostomy and a gastrostomy. Some weeks later, after optimised nutritional status, a second intervention can be planned to perform the oesophageal reconstruction with gastric or colonic pull up.[158]

Recommendation 43		Unchanged
Explantation and *in situ* reconstruction with oesophageal repair and coverage with viable tissue should be considered in patients suitable for surgery as definitive treatment for thoracic vascular graft or endograft infection with an aorto-oesophageal fistula without profuse haemorrhage.		
Class	**Level**	**References** **ToE**
IIa	C	Kahlberg *et al.* (2019),[156] Takeno *et al.* (2020),[157] Watanabe *et al.* (2020)[158]

6.3.1.2. Aorto-oesophageal fistula with profuse haemorrhage. For active bleeding from a previously repaired thoracic aorta, prompt sealing by endovascular repair has been proposed to rescue massive haemothorax or exteriorised bleeding in the form of haemoptysis or haematemesis (for ABF or APF and AOF, respectively). Use of an endograft without fixation elements (such as barbs or hooks) may simplify future explantation by avoiding direct penetration of the aortic wall. This bridge to definitive treatment strategy

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promptly restores haemodynamic stability and allows some delay during which to better identify patients suitable for complete explantation surgery and fistula repair *vs.* a conservative and most likely palliative treatment in this setting.[160] This delay, considering the risk of ongoing infection, facilitates optimisation of the patient by correcting malnutrition and major organ dysfunction.

Alternatively, oesophageal endoprostheses have been used to close the fistula from the oesophageal side and to prevent further contamination of the mediastinum. It can be performed as a standalone procedure or combined with an aortic endograft in case of bleeding.[161] In a report of the EuREC, survival at 1 year was only 17% with oesophageal stenting alone compared with 43% when oesophagectomy was performed.[162] Therefore, this technique should only be considered as a palliative option and be used in patients deemed to be unfit for further surgery or as a bridge to definitive therapy.[144,162] Of note, migration of the oesophageal endoprosthesis with the need for repositioning might occur.

Recommendation 44		Changed
Endovascular treatment is recommended as first line strategy in thoracic vascular graft or endograft infection with profuse haemorrhage.		
Class	**Level**	**Reference**
I	C	Consensus

6.3.2. Thoracic vascular graft or endograft infection with airways fistula

6.3.2.1. Endovascular treatment. A systematic review including 134 patients treated in an emergency setting with TEVAR for ABF or APF showed 93.2% technical success and a thirty day mortality rate of 5.9%. Aortic related death was 14.3% at 17.4 months, and ABF or APF recurrence was 11.1%.[163] Failure might, however, be underestimated due to limited follow up and censoring of early deaths, and the risk of recurrence of ABF or APF and infection of the newly inserted endograft is still present.[160]

Recommendation 45		Changed
A graft preserving strategy may be considered in selected patients with aortobronchial or aortopulmonary fistula treated with emergency thoracic endovascular aortic repair.		
Class	**Level**	**Reference**
IIb	C	Consensus

6.3.2.2. Open surgical treatment. Although some patients may be treated conservatively with endograft preservation, given the high risk of treatment failure without complete device explantation, endograft removal and *in situ* vascular repair should be considered in patients for whom surgery is being considered.[120,164,165] The EuREC registry showed that a radical surgical approach resulted in significantly better survival compared with any other treatment strategy, but operative mortality was high (up to 41%).[144]

The airways defect also needs to be repaired surgically. The defect can be closed primarily, but in most cases a bronchial resection and anastomosis or lung resection (mostly commonly wedge resection) is necessary. After bronchus repair, the endograft should be covered with a muscle or pleural flap.[163,166]

Recommendation 46		Changed
Explantation and *in situ* reconstruction with airways repair and coverage with viable tissue may be considered in patients suitable for surgery as definitive treatment for thoracic vascular graft or endograft infection with an airway fistula.		
Class	**Level**	**Reference**
IIb	C	Consensus

6.4. Thoraco-abdominal vascular graft or endograft infection

Dedicated literature regarding thoraco-abdominal VGEI (open thoraco-abdominal repair, and fenestrated, branched, or chimney endovascular aortic repair [EVAR]) is mostly limited to case reports.[167–169] A single centre study reported the outcomes of MDT management of complex aortic endograft infections (ten fenestrated EVAR, one chimney EVAR).[170] Seven patients were managed conservatively and four patients underwent open surgery for duodenal fistula or false aneurysm with septic embolism (graft explantation for three patients, duodenal fistula repair without graft explantation for one patient). Overall survival was 82% at 1 year. Conservatively treated patients demonstrated 86% infection remission with antimicrobial therapy, and one patient required emergency surgery for secondary duodenal fistulisation.

Another study reported the outcomes of 19 MAGIC diagnosed thoraco-abdominal VGEIs (four occurred in patients originally treated for primary infected aortic pathology), including six patients with a secondary fistula (two ABF, one AOF, two fistulae to the bowel, and one iliaco-ureteral fistula). In this series, 18 of 19 patients were deemed unfit for explantation after MDT review, reflecting the anatomic complexity and frailty in this cohort. Treatment was antimicrobial therapy alone in 11 cases, drainage in five cases, open surgical debridement in one case, drainage and surgical debridement in one case, and one failed explantation strategy initiated with extra-anatomic bypass. Among the 13 patients without secondary fistula, one year survival was 77%, and ten of 13 patients achieved remission or cure.[12] By contrast, outcomes in six patients with secondary fistula were poor (estimated one year survival 33%, 100% treatment failure).[12]

Pending future studies, proposing primarily conservative treatment may be considered in surgically unfit patients with uncomplicated VGEI, while surgical treatment may be considered in surgically fit patients with a bowel fistula or septic embolism or treatment failure after conservative treatment. Figure 5 provides a summarised algorithm of management of patients with thoracic VGEI.

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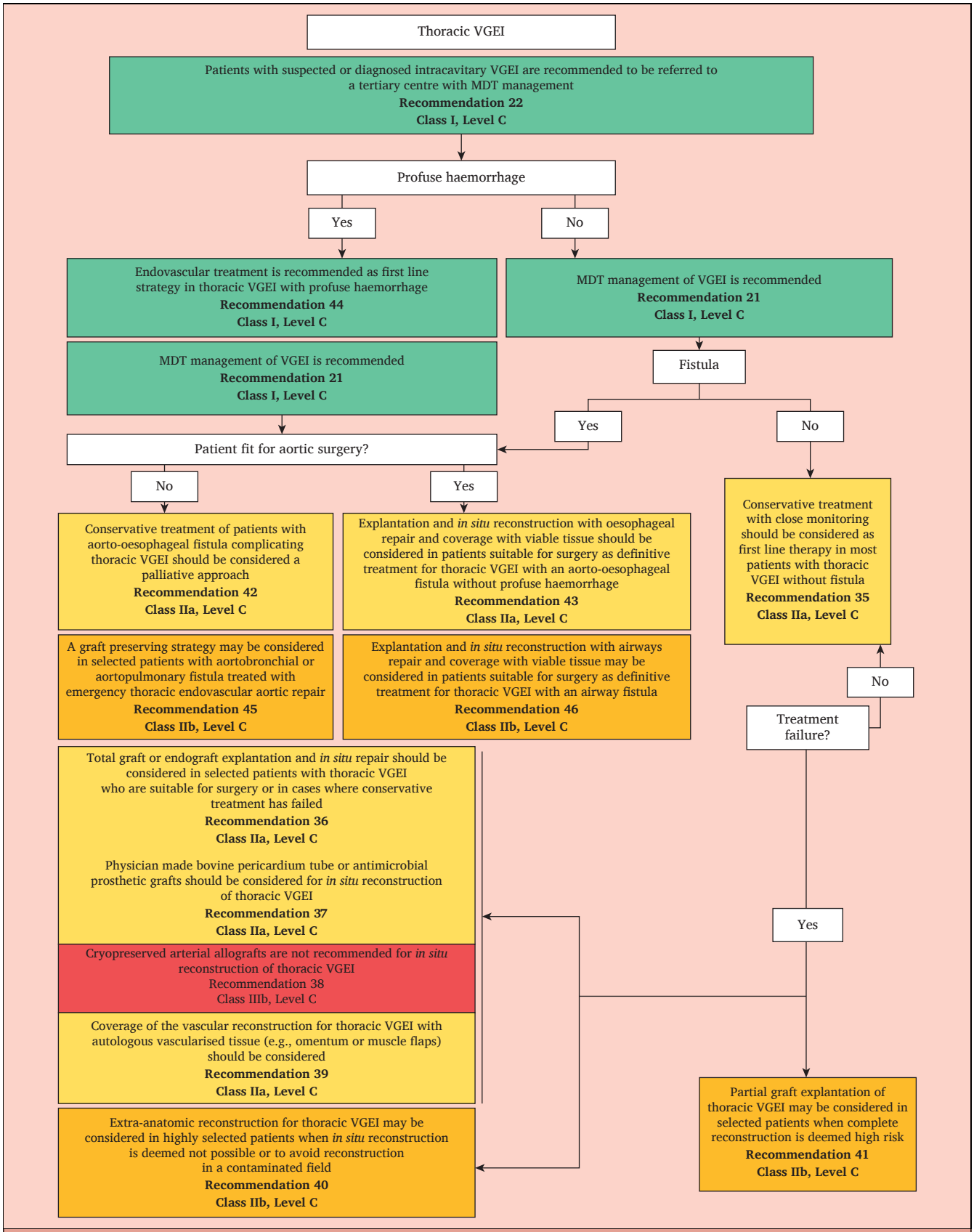

Figure 5. Algorithm for the management of thoracic vascular graft or endograft infection (VGEI). MDT = multidisciplinary team.

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7. MANAGEMENT OF ABDOMINAL VASCULAR GRAFT OR ENDOGRAFT INFECTION

7.1. General considerations

In this chapter, no distinction is made between an infected graft or endograft, unless clearly stated. The true incidence of abdominal VGEI is unknown because it has not been properly evaluated. It is estimated to be 0.2% after open surgery and 2.3% after EVAR.[12,171,172]

Abdominal VGEI can be complicated by fistulation to the bowel or urinary tract. In four population based studies, the rates of abdominal VGEI complicated by enteric erosion or fistula (AEF) were, respectively, 50%, 53%, 30%, and 53%.[66,75,173,174] The rate of abdominal VGEI complicated by fistulation to the urinary tract was 2%.[173] These rates may, however, be overestimated due to exclusion of conservatively managed patients in these studies.

The presence of fistula has severe implications on treatment demands, both medical and surgical, and consequently on survival, which is why this chapter is divided into abdominal VGEI without and with fistula.

7.2. Abdominal vascular graft or endograft infection without fistula

7.2.1. Conservative treatment. Conservative treatment may be proposed for patients deemed unsuitable for aortic clamping or surgical intervention. This conservative strategy, however, requires close in hospital patient monitoring, including clinical examination, laboratory testing as well as imaging. This needs to be discussed at regular MDT meetings.[12]

Conservative treatment might also be used as a bridging period to control the infection and optimise nutritional status for a later MDT to evaluate whether the patient might then be a candidate for surgery.

Recommendation 47		New
Conservative treatment should be considered in patients with abdominal vascular graft or endograft infection deemed unsuitable for aortic clamping or surgical intervention.		
Class	**Level**	**Reference**
IIa	C	Consensus

7.2.1.1. Percutaneous drainage. In the presence of perigraft fluid collections or abscesses, image guided percutaneous drainage can be performed in combination with systemic antimicrobial therapy. The drain is left in place until the collection is totally or sufficiently drained but might also be combined with irrigation. Not all perigraft and endograft collections are amenable to percutaneous drainage (e.g., proximity to vital structures), and risks of percutaneous drainage (e.g., seeding, haemorrhage, graft puncture) should be discussed on an individual basis by the MDT.

7.2.1.2. Irrigation. Irrigation with saline or an antiseptic solution (e.g., diluted betadine irrigation) is commonly used in infected surgical fields where it is thought to reduce the bacterial burden. Since irrigation does not constitute definitive infection control, it can be part of a conservative treatment strategy, as a palliative option, or performed in preparation for later curative treatment.

Recommendation 48		Changed
Percutaneous drainage of perigraft fluid collections, with or without irrigation, may be considered in patients with abdominal vascular graft or endograft infection to achieve infection source control, either as a palliative strategy or as a bridge to later definitive surgical treatment.		
Class	**Level**	**Reference**
IIb	C	Consensus

7.2.2. *In situ* reconstruction. *In situ* reconstruction involves complete excision of all graft material and infected tissue followed by anatomic reconstruction using autologous veins, xenografts, antimicrobial synthetic grafts (rifampicin bonded, silver coated or silver acetate, and triclosan loaded), or cryopreserved allografts.

Four meta-analyses demonstrated significantly better outcomes for *in situ* reconstruction over extra-anatomic reconstruction, with overall lower complication, re-infection, and mortality rates.[8] Accordingly, complete explantation of graft material and infected tissue followed by *in situ* reconstruction is the definitive treatment of choice for patients fit for aortic clamping.

Recommendation 49		Unchanged
Complete explantation of graft material and infected tissue followed by *in situ* reconstruction is recommended as the first line curative treatment in patients with abdominal vascular graft or endograft infection.		
Class	**Level**	**References** ToE
I	C	Batt *et al.* (2018),[175] O'Connor *et al.* (2006),[176] Post *et al.* (2019),[177] Shu *et al.* (2024)[178]

7.2.2.1. Technical aspects. For curative treatment, complete removal of infected material should be strived for. Reconstruction type and graft materials used for reconstruction are at the discretion of the MDT and chosen based on patient centred factors, the availability of substitutes, and hospital experience. After a careful analysis of the pre-operative CTA, the proximal clamping zone and organ protection strategies are defined. Securing the proximal aortic cross clamp zone is the first step of the laparotomy. Owing to anastomotic pseudoaneurysm, juxtarenal anastomosis, or the endografts' suprarenal hooks, suprarenal clamping is common. Alternatively, an intra-aortic balloon inserted through femoral access, brachial puncture, or through an endograft's iliac limb may be used.[179–181] All foreign material should be explanted; however, if removal of ingrown suprarenal fixation material

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poses a high risk to the orifices of visceral arteries, the bare metal barb anchored stents may be left in place in rare cases.[172,182] To facilitate extraction of the fixation material, a 20 mL syringe or disposable proctoscope may be used. Disconnecting the metal frame from the fabric and cutting the suprarenal stents free further eases the mobilisation of the barbed stents.[182,183] Wide debridement is recommended. Copious irrigation, local cleansing of adjacent tissues and previous tunnels or preserved graft is useful before new reconstruction.[172,184,185] If improper tunnelling contributed to the infection, a new bypass route should be created, preferably lateral to the previous tunnel, to avoid injury to deep veins or the ureter.

7.2.2.2. Graft materials. Most studies reporting surgical treatment of abdominal VGEI include patients with aortic VGEI and INAA. Combining two different entities prevents reliable conclusions about treatment efficacy. Furthermore, studies lack both consistent diagnostic criteria and standardised outcome measures for defining cure of treatment or treatment failure. The methodological limitations mean that the current literature cannot provide definitive guidance on the optimal reconstruction material for abdominal aortic VGEI.

7.2.2.3. Autologous veins. Several studies employed autologous femoral veins for *in situ* reconstruction of abdominal VGEI[132,172,184,186–194] (Table 13). A systematic review of 13 studies reported results on 388 patients with VGEI treated with graft explantation and reconstruction with autologous femoral veins. The thirty day mortality rate was 9.8% (range 0.0 — 28.6%), one year mortality rate was 18.4% (10.0 — 36.4%), and five year mortality rate (reported by eight studies) was 43.1% (25.0 — 59.1%). During the mean follow up of 48 months, the re-intervention rate was 21.7% (9.0 — 45.0%) and the re-infection rate was 5.6% (0.0 — 27.3%).[194]

The disadvantages of femoral veins include harvesting difficulties in emergency settings, prolonged operating time, and venous morbidity.[195] Vein harvest can result in the need for fasciotomy, but enhanced outcomes have been achieved through the implementation of lower limb pneumatic compression, low molecular weight heparin prophylaxis, and refined vein harvesting techniques.[184,189] The pooled mean amputation rate in a systematic review of 388 patients was 6.7%.[194]

Occasionally, technical challenges may arise when creating aorto-iliac grafts with two femoral veins, particularly when there is size mismatch due to large aortic diameter or small femoral vein calibre. Vein harvest can be associated with venous thrombosis in the early post-operative period, and chronic venous insufficiency thereafter.[184,189] However, venous morbidity occurred in only 11% of patients in the later period when the distal popliteal vein was preserved.[184]

Autologous vein ruptures have been reported in the immediate post-operative period secondary to technical issues, and afterwards due to re-infections.[184,188,192] Long term CTA studies demonstrate that autologous vein may dilate from the anastomotic sites or develop stenotic lesions; however, aneurysm formation has not been reported.[172,184,196]

A meta-analysis was conducted to determine the most appropriate conduit for aortic VGEI including autogenous veins, cryopreserved allografts, and synthetic grafts (standard PET, polytetrafluoroethylene [PTFE], or rifampicin or silver coated grafts).[175] Based on pooled estimates, the operative mortality rate was lowest for autogenous veins (10%), especially compared with cryopreserved grafts and silver coated grafts (22% and 16%, respectively). The pooled estimate for graft occlusion was lowest for autogenous veins (2%) compared with the others mentioned

Table 13. *In situ* reconstruction with autologous veins for abdominal vascular graft or endograft infection.

Author, year	Study type	*n*	FU — mo	30 day mortality — %	1 y/5 y mortality — %	Graft re-infection — %	Graft occlusion — %	Graft rupture — %
Järvinen, 2025[172] *	Retrospective	19	29	16	31/42	0	10	5
Girshfeld, 2024[194]	Retrospective	13	22	23	20/NR	8	NR	0
Glasgow, 2023[193]	Retrospective	18	26	11	23/NR	6	6	0
Kouijzer, 2022[192]	Retrospective	26	33	19	NR/46	11.5	NR	11.5
Filiberto, 2021[191]	Retrospective	49	14	5	NR/NR	8	NR	NR
Langenskiöld, 2021[132]	Prospective database	40	70	13	20/50	NA	3	8
Heinola, 2016[184]	Retrospective	55	32	9	18/41	4	3.6	5
Charlton-Ouw, 2014[190]	Retrospective	11	30	0	NR/46	27	NR	NR
Dorweiler, 2014[189]	Retrospective	55	45	9	NR/55	NR	NR	NR
Ali, 2009[188]	Prospective database	165	32	15	NR/NR	6	NR	6
Daenens, 2003[187] †	Retrospective	49	41	8	NR/40	0	4	0
Cardozo, 2002[186]	Retrospective	12	22	17	25/NR	0	0	0

FU = follow up; NR = not reported.

* Study included endovascular aortic repair infection patients. Outcome data for cases treated with autologous venous reconstructions were provided separately by the author upon request.

† Study excluded patients with enteral fistulas and erosions.

Table 14. *In situ* reconstruction with bovine pericardial patch for abdominal vascular graft or endograft infection.

Author, year	Study type	*n*	FU − mo	30 day mortality − %	Mortality − %	Re-infection rate − %	Graft complication − %
Grills, 2024[196] *	Meta-analysis	133	25	19	40	0	NA
Melloni, 2025[197]	Retrospective monocentric study	21	23	4.8	0	4.8	0
Weiss, 2024[197]	Prospective, multicentre study	130	26	17	42/56	7	9%

FU = follow up; NR = not reported.
* Study including vascular graft or endograft infection and infected native aortic aneurysm.

above (7% and 13%, respectively). There was no difference in pooled estimated re-infection rates comparing autologous veins (6%) with other grafts (9% and 11%, respectively).[175]

7.2.2.4. Xenografts. Several studies employed bovine pericardial patches for *in situ* reconstruction of abdominal VGEI[151,196,197] (Table 14). Bovine pericardial patches are an off the shelf solution and can be tailored (cut and sewn) to create physician made pericardial tube grafts (straight or bifurcated) for individualised *in situ* aortic repair. While laparotomy is performed, ideally a separate team uses a back table to construct the graft. This technique has been reported increasingly. Most series include thoracic, thoraco-abdominal, and abdominal reconstructions of mixed aetiology, such as INAA and VGEI.[196,197]

A meta-analysis including nine studies reported the results of 133 reconstructions using bovine pericardium grafts for INAA (33%) and VGEI (67%) in the thoracic (33%) and abdominal aorta (58%): thirty day mortality was 19% (95% CI 10.8 — 28.7%), late mortality was 19% (95% CI 7.8 — 32.8%), overall mortality was 40% (95% CI 29.8 — 50.9%), lower limb ischaemia was 6% (95% CI 0.5 — 13.8%), loss of graft patency leading to re-intervention was 1% (95% CI 0.0 — 7.7%), and the graft re-infection rate was 0% (95% CI 0.0 — 1.2%). The mean follow up of six studies was 25 months (1 — 65 months). Three studies reported median follow up ranging from 6 to 14 months.[196]

A large series was later published, reporting the outcomes of 130 abdominal reconstructions using bovine pericardium for VGEI at various aortic locations (63% involving the infrarenal aorta). The thirty day mortality rate was 17%. After a mean follow up of 26 months, re-infection occurred in 7%. The estimated survival at 1, 2, 3, and 5 years was 58%, 55%, 51%, and 44%, respectively. The combined results of 168 patients, including patients with INAA and aortic VGEI, resulted in an estimated freedom from graft complications of 91%, 89%, 87%, and 87%. Aneurysmal or stenotic graft degeneration, unrelated to an anastomosis, was not reported.[197]

There is limited experience of *in situ* ovine, mesh reinforced biosynthetic grafts (Omniflow II; LeMaitre Vascular) for abdominal aortic VGEI reconstructions, with a trend towards low re-infection rates. However, a considerable incidence of early occlusions has been reported.[198,199]

7.2.2.5. Cryopreserved allografts. Several studies employed cryopreserved allografts for *in situ* reconstruction of abdominal VGEI[200—211] (Table 15). The disadvantages of cryopreserved allografts are their limited availability, rate of graft rupture (up to 24%), and rate of aneurysmal degeneration (up to 20%).[200—211] Graft specific factors such as allograft age or cryopreservation technique were of no prognostic value in multivariable analysis for *in situ* reconstruction with cryopreserved allografts.[204] Despite some series showing improved graft durability, graft ruptures when they occur are often fatal.[203,207,209,210] Therefore, given the risk of fatal rupture and limited durability, cryopreserved allografts should be used with caution in abdominal aortic reconstructions and are generally not preferred when other options are available.

Data on cryopreserved veins for aorto-iliac reconstruction are scarce.[212,213] One study including 12 supra-inguinal graft infections treated with cryopreserved femoral veins reported one graft rupture due to *Candida* re-infection. During a median follow up of 15 months, there was one anastomotic dilatation but none of the grafts occluded.[212]

Lifelong surveillance is required for all the cryopreserved vessels owing to the risk of aneurysm development and anastomotic dilatations.[200,203,205,206,211]

7.2.2.6. Antimicrobial grafts. Rifampicin bonded grafts have also been used for *in situ* reconstruction of abdominal VGEI[20,211,214—220] (Table 16). Rifampicin bonded grafts have shown re-infection rates ranging from 0% to 12.8%. Experimentally there is weak activity against non-fermenter Gram negative bacilli such as *Pseudomonas* species or fungi.[140,221] Retrospective studies showed they might be less effective in infections caused by MRSA, Gram negative strains, and fungi.[20,216]

The antimicrobial efficiency of rifampicin bonded grafts is concentration and time dependent. Due to dilution, the protective effect is reduced after approximately 1 week.[20,221] The rifampicin bonded concentration currently used in the treatment solution is heterogeneous, making the comparison of outcomes difficult. Concentrations between 1 mg/mL and 60 mg/mL (soaked for 15 — 30 minutes) have been used in clinical and *in vitro* studies.[20,211,214—221] Despite the off the shelf availability and short preparation time in emergency settings and complex aortic reconstructions, rifampicin may not be ideal as monotherapy as it may lead to the emergence of antibiotic resistant strains, therefore the use of other substitutes should be preferred.[20,140]

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Table 15. *In situ* reconstruction with allografts for abdominal vascular graft or endograft infection.

Author, year	Study type	*n*	FU − mo	30 day mortality − %	1 y/5 y mortality − %	Graft re-infection − %	Graft occlusion − %	Graft rupture − %	Graft aneurysmal degeneration − %
Cryopreserved arterial allografts									
Tabiei, 2023[211]	Retrospective	69	20.5	7.2	NR/41	2.8	2.8	1.4	2.8
Couture, 2021[210]	Retrospective	200	49	11	24/44	12	8	6	4
Weiss, 2021[209]	Retrospective	33	96	6	12/38	9	15	3	0
Masabni, 2019[208]	Retrospective	16	19	6	25/NR	6	6	6	0
Ben Ahmed, 2018[207]	Retrospective	71	45	2.8	25/46	4	1.4	7	0
Lejay, 2017[206]	Retrospective	25	47	8	20/60	NR	NR	24	20
Minga Lowampa, 2016[205]	Retrospective	80	49	14	NR/51	5	1.5	12.5	10
Touma, 2014[204]	Retrospective	37	12	35	49/NR	5	NR	NR	NR
Harlander-Locke, 2014[203]	Retrospective	220	30	9	25/49	5	4	4	4
Garot, 2014[202]	Retrospective	22	12	50	50/NR	5	23	NR	NR
Lesèche, 2001[201]	Retrospective	23	35.4	17	33 (3 y)	0	NR	0	NR
Verhelst, 2000[200]	Retrospective	66	36	20	NR/NR	15	NR	18	10.6
Cryopreserved venous allografts									
Heinola, 2019[212]	Retrospective	12	15	8	17 (2 y)	8	0	8	0

FU = follow up; NR = not reported.

Cryopreserved arterial allografts and rifampicin bonded grafts were compared for *in situ* reconstruction for aorto-iliac VGEI. Overall survival and freedom from re-infection at 5 years were similar between groups, but freedom from graft related intervention at 5 years was statistically significantly lower in the cryopreserved arterial allografts group than in the rifampicin bonded grafts group: 66.2% *vs.* 92.5% (hazard ratio [HR] 3.2, 95% CI 1.2 − 8.2; $p = .02$).[211]

Silver has been appreciated for its wide antiseptic and antimicrobial effects. Silver coated polyester grafts are available in two different options: silver acetate, which dissolves within 2 − 4 weeks; and elemental silver, which remains for about 1 year.[222,223] The advantage of silver is its broad antimicrobial activity.

Use of silver grafts for *in situ* reconstruction of abdominal VGEI showed re-infection rates ranging from 0% to 20%, but there was no comparison between different silver coating and studies are heterogeneous[224−229] (Table 17). A systematic review including nine *in vitro* and five *in vivo* studies addressing the effect of silver in the prevention of aortic graft infection confirmed a positive antimicrobial effect in seven of nine *in vitro* studies and in two of five *in vivo* studies. *Candida* species were only tested *in vitro*, and a delayed efficacy was observed.[230] However, the overuse of silver in commercially available healthcare products is a growing potential concern due to the possible selection of tolerant or resistant bacteria, and potential silver resistance is therefore an important issue.

Triclosan (5-chloro-2-(2,4-dichlorophenoxy)phenol) is another antimicrobial agent. *In vitro* studies of triclosan showed better antimicrobial activity in a polyester graft coating combining silver and triclosan compared with silver alone or no coating after twenty-four hours.[231] This effect was confirmed after 7 days compared with silver alone, rifampicin soaked grafts, or no coating, which might be

Table 16. *In situ* reconstruction with rifampicin bonded grafts for abdominal vascular graft or endograft infection.

Author, year	Study type	*n*	FU − mo	Rifampicin dose − mg/mL	30 day mortality − %	Mortality − %	Re-infection rate − %	Graft occlusion − %
Tabiei, 2023[211]	Retrospective	80	21.5	2.4	7.5	41	12.8	1.25
Schaefers, 2019[220]	Retrospective	10	26	19	23.1	NR	0	NR
Batt, 2011[219]	Retrospective	8	41	NR	31.8	40	0	0
Oderich, 2011[218]	Retrospective	54	51	2.4	9	NR/41	4	8
Oderich, 2006[217]	Retrospective	52	41	2.4	8	16	11.5	8.8
Bandyk, 2001[20]	Retrospective	19	17	45−60	9.1	NR	8	0
Hayes, 1999[216]	Retrospective	11	12	45−60	18.2	36.4	0	18.2
Young, 1999[215]	Retrospective	25	36	1	8	24	4	14
Torsello, 1997[214]	Retrospective	11	33	60	18	18	9	NR

FU = follow up; NR = not reported.

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Table 17. *In situ* reconstruction with silver coated grafts for abdominal vascular graft or endograft infection.

Author, year	Study type	*n*	FU − mo	30 day mortality − %	Mortality − %	Re-infection rate − %	Graft occlusion − %
Zegelman, 2013[229]	Retrospective	10	18	10	NR	10	NR
Pupka, 2011[228]	Prospective	27	22.8	11	NR	16	0
Bisdas, 2011[227]	Retrospective	11	24	18	27	20	0
Batt, 2008[226]	Prospective	24	32.5	20.8	25	12.5	8.3
Mirzaie, 2007[225]	Prospective	11	30	0	0	0	0
Batt, 2003[224]	Prospective	24	17	16.6	16.6	0	0

FU = follow up; NR = not reported.

important regarding rifampicin resistance.[140] Triclosan coated grafts have been shown to offer a low adhesion profile in early stages of biofilm formation by *S. aureus* in another *in vitro* analysis.[232] Clinical results of the use of a silver—triclosan polyester graft in 54 *in situ* reconstructions of abdominal VGEI are promising, with an estimated freedom from re-infection of 97.8% at 1 year and 93.2% at 2 years.[233] Due to the off the shelf availability and short preparation time, the silver—triclosan polyester graft coating might serve as a good alternative to biological material especially in emergency settings and complex aortic reconstructions (e.g., including multiple grafts to branch vessels), but the literature is scarce and no valid conclusion can be drawn.

Recommendation 50 — **Changed**

Autologous veins or physician made bovine pericardium tube should be considered as the preferred method for *in situ* reconstruction in patients with abdominal vascular graft or endograft infection.

Class	Level	References	ToE
IIa	C	Langenskiöld *et al.* (2021),[132] Järvinen *et al.* (2025),[172] Heinola *et al.* (2016),[184] Cardozo *et al.* (2002),[186] Daenens *et al.* (2003),[187] Ali *et al.* (2009),[188] Dorweiler *et al.* (2014),[189] Charlton-Ouw *et al.* (2014),[190] Filiberto *et al.* (2021),[191] Kouijzer *et al.* (2022),[192] Glasgow *et al.* (2023),[193] Girshfeld *et al.* (2024),[194] Grills *et al.* (2024),[196] Weiss *et al.* (2024)[197]	

Recommendation 51 — **Changed**

Antimicrobial grafts may be considered as alternative substitutes for *in situ* reconstruction in patients with abdominal vascular graft or endograft infection, especially in complex repair.

Class	Level	Reference
IIb	C	Consensus

Recommendation 52 — **New**

Cryopreserved allografts are not recommended for *in situ* reconstruction in patients with abdominal vascular graft or endograft infection.

Class	Level	Reference
IIIb	C	Consensus

7.2.2.7. Graft coverage and adjunctive treatments. Graft coverage with vascularised tissue is believed to be important to prevent fistula development, to fill up dead space after debridement, to separate the graft from the bowel, and to promote antibiotics reaching the outer surface of the aortic graft. Therefore, it is advised to cover the new vascular reconstruction with a viable pedicled structure, usually with omentoplasty or retroperitoneal tissue. This is believed to be even more important in VGEI with AEF, to create an interposition between the bowel and the graft. If the groin is involved, the distal anastomosis might be covered with a muscle flap.[234,235]

Other adjunctive measures to consider when performing *in situ* reconstruction include reinforcing the proximal anastomosis with muscle or fascia, e.g., the *fascia lata*, or off the shelf bovine pericardium or peri-operative washout.[184,197,234] The use of open abdomen and negative pressure wound therapy (NPWT) in abdominal VGEI has also been described in a few studies, with low risk of treatment failure.[236]

Recommendation 53 — **Unchanged**

Coverage of all vascular reconstructions with autologous vascularised tissue, such as omentoplasty, is recommended in patients with abdominal vascular graft or endograft infection.

Class	Level	References	ToE
I	C	Rodrigues dos Santos *et al.* (2014),[234] Janko *et al.* (2022)[235]	

7.2.3. Extra-anatomic reconstruction

7.2.3.1. General considerations. Excision of the infected aortic vascular graft or endograft and extra-anatomic bypass grafting through a non-infected field can be

performed in the same procedure or as a staged approach, when the excision of the infected graft or endograft is performed a few days after extra-anatomic reconstruction. The disadvantages of extra-anatomic reconstruction include the high early mortality rate (up to 18%), high amputation rate (up to 11%), and high rate of recurrent infection (up to 18%).[66] Furthermore, long term patency rates for extra-anatomic bypass grafts in patients with abdominal VGEI have generally been low (43% primary patency at 3 years).[237]

7.2.3.2. Stump management. The risk of stump rupture is related to the mechanical consequences of a short stump and to the properties of the persistent infection after incomplete debridement. The growing volume of implanted EVARs, especially with suprarenal fixation, demands more complicated vascular diversions for stump management. With a short stump, transposition or bypasses to the renal and visceral arteries are likely to be necessary, adding further complexity and morbidity.[173,174] Moreover, revascularisation should be performed before explantation to avoid prolonged visceral ischaemia. Techniques to prevent stump rupture include double suture layers, and reinforcement with venous pledgets, bovine pericardium, pre-vertebral fascia, or a layer of *posterior rectus* fascia—peritoneum. Most authors recommend covering the stump with an omentoplasty or even pedicled *latissimus dorsi* muscle flap.[238,239]

7.2.3.3. Extra-anatomic vs. in situ reconstruction. Four meta-analyses demonstrated significantly better outcomes for *in situ* over extra-anatomic reconstruction with overall lower complication, re-infection, and mortality rates.[8] Pooled results of a recent network meta-analysis of 22 studies with 1 118 patients showed that complete graft removal with *in situ* repair had lower thirty day, 1 year, and 5 year mortality rates compared with graft removal with extra-anatomic repair (11.9% *vs.* 16.6%, 23.8% *vs.* 41.4 %, and 45.6% *vs.* 67.9%, respectively). Furthermore, the re-infection rate was higher among patients treated with extra-anatomic reconstruction compared with *in situ* reconstruction (22.4% *vs.* 8%, respectively).[178] Similar results were reported in a multicentre study of 241 patients, where re-infection free survival was significantly shorter among patients treated with extra-anatomic reconstruction compared with *in situ* reconstruction (HR 2.4, 95% CI 1.6 — 3.6; $p < .001$).[235] Conversely, two studies (126 and 213 patients, respectively) showed no significant differences in re-infection rates or early and late survival between patients treated with extra-anatomic *vs. in situ* reconstruction, but patients with AEF were over represented (more than 50%), which yields worse outcomes.[66,173] It has been highlighted that since early death is common in patients with AEF regardless of reconstruction type, their lifespan may be too short to gain the long term benefit of *in situ* reconstruction.[73] Therefore, careful MDT evaluation is necessary to identify patients with a potentially long life expectancy and favour *in situ* reconstruction.[234] Old and frail patients requiring re-reconstruction due to distal ischaemia serve as possible candidates for graft explantation and extra-anatomic reconstruction.

Recommendation 54			Unchanged
Extra-anatomic reconstruction may be considered an alternative option in selected patients with abdominal vascular graft or endograft infection, particularly in the presence of aorto-enteric fistulation, advanced age, or frailty.			
Class	**Level**	**References**	**ToE**
IIb	C	Gavali *et al.* (2021),[66] Janko *et al.* (2021),[73] Hosaka *et al.* (2023)[173]	

7.2.4. Partial or total graft explantation. Total explantation of the infected graft or endograft is the first line strategy to treat VGEI. However, total graft explantation is not always feasible in high risk patients, multiple and complex revascularisation cases, or emergency situations.

In retrospective studies, partial explantation was successful when infection was limited to the groin and supra-inguinal vascular grafts were incorporated.[201,225,240,241] Poor graft incorporation yielded a 47% re-infection rate in a small series of 15 patients, in whom bacterial growth above the resection line predicted re-infection with 71% accuracy.[241] Accordingly, cultures of the graft margins are required, as this may help guide the duration of antimicrobial treatment when partial graft explantation is performed.[14]

A network meta-analysis evaluated 1 118 patients reporting short and long term outcomes following complete graft explantation with *in situ* repair ($n = 852$), partial graft explantation with *in situ* repair ($n = 36$), complete graft explantation with extra-anatomic repair ($n = 228$), and partial graft explantation with extra-anatomic repair ($n = 2$). Both network meta-analysis results and pooled results of multi- and single arm cohorts indicated that partial graft explantation with *in situ* repair had the lowest thirty day and 1 year mortality rates (0% and 6.1%, respectively), compared with the second best option of complete graft explantation with *in situ* repair (11.9% and 23.8%, respectively). Nevertheless, only one study reported three year survival after partial explantation and *in situ* reconstruction (two of 17; 12%). Pooled re-infection rates after partial graft explantation with *in situ* repair were comparable with complete graft explantation with *in situ* repair (9.3% *vs.* 8.0%, respectively) and superior to extra-anatomic repair (22.4%).[178]

In a nationwide study, partial explantation of abdominal grafts was not associated with re-infection (odds ratio [OR] 1.21, 95% CI 0.56 — 2.64; $p = .063$) or a higher early (OR 0.81, 95% CI 0.42 — 1.59; $p = .54$) or late (OR 0.98, 95% CI 0.59 — 1.67; $p = .95$) mortality rate.[173] In contrast, partial explantation of abdominal endografts was associated with a higher mortality rate at ninety days (HR 3.4, 95% CI 1.3 — 8.9; $p = .01$) and at 3 years (HR 2.9, 95% CI 1.4 — 6.2; $p = .006$).[174]

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Recommendation 55			Unchanged
Partial graft explantation may be considered in selected patients with abdominal vascular graft or endograft infection when infection is regarded as limited or if the remaining part of the graft or endograft is well incorporated.			
Class	**Level**	**Reference**	**ToE**
IIb	C	Shu *et al.* (2024)[178]	

Recommendation 56		New
Cultures of the graft margins should be considered to guide the duration of antimicrobial treatment when partial graft explantation is performed in patients with abdominal vascular graft or endograft infection.		
Class	**Level**	**Reference**
IIa	C	Consensus

7.2.5. Graft preservation surgery. Open surgical debridement, saline irrigation, and retained graft local cleansing, in combination with graft omental wrapping, can help diminish the bacterial load and provide better perigraft antibiotic concentration owing to the presence of vascularised tissue.[75,242–244] However, series reporting graft preservation surgery are often mixed with conservatively treated patients, and conclusions are therefore hard to draw. Evidence regarding patient selection and the optimal treatment method for graft preservation is currently lacking.

Recommendation 57			Changed
Graft preservation surgery may be considered in selected patients with abdominal vascular graft or endograft infection when surgical explantation is not feasible.			
Class	**Level**	**References**	**ToE**
IIb	C	Gavali *et al.* (2023),[75] Shijo *et al.* (2021),[242] Maze *et al.* (2013),[243] Jamieson *et al.* (2012)[244]	

7.3. Abdominal vascular graft or endograft infection with fistula to other organs

7.3.1. Aorto-enteric fistula without profuse haemorrhage. A two centre retrospective study reporting outcomes of 74 frail patients with VGEI initially contra-indicated for explantation showed that VGEIs complicated by an AEF were associated with worse survival even without recurrent sepsis (HR 3.3, 95% CI 1.3 − 8.4; $p = .011$).[133]

For patients fit for aortic surgery, without active bleeding compromising haemodynamic stability, a bowel repair with excision of the infected vascular graft or endograft and *in situ* revascularisation is recommended for definitive treatment.[218,236,245] Compared with the retroperitoneal approach, a transperitoneal midline laparotomy provides superior exposure for secure bowel and vascular repair. Securing a supracoeliac clamping zone or using an aortic occlusion balloon should be considered as a first step before entering the area of the AEF. If possible, opening the enteral connection during dissection should be avoided. Furthermore, leaving the old graft patch attached to the bowel during intestinal dissection helps prevent spillage of enteral contents.[218] The type of bowel repair depends on the size and location of the defect.[218,236,245,246] A tension free duodenorrhaphy with direct suture of the duodenum can be performed if the bowel defect is small. For larger defects, a complex duodenal reconstruction with resection and anastomosis and re-routing decreases the risk of fistula and infection recurrence.[236]

In a review and pooled data analysis on secondary AEF, within the *in situ* repair group, impregnated synthetic grafts were associated with the worst overall and AEF related mortality rates, and vein grafts with the best.[247] However, the literature lacks comparative studies detailing subgroup outcomes of VGEI with AEF to recommend one type of graft over another in this specific setting.

For VGEI without bowel fistula, a pedicled omentoplasty is necessary to isolate the bowel repair from the new arterial reconstruction.

Recommendation 58			New
Bowel repair combined with complete graft or endograft explantation and vascular reconstruction is recommended for curative treatment of patients with abdominal vascular graft or endograft infection presenting with secondary aorto-enteric fistula.			
Class	**Level**	**References**	**ToE**
I	C	Oderich *et al.* (2011),[218] Mauriac *et al.* (2021),[236] Schoell *et al.* (2015)[245]	

7.3.2. Aorto-enteric fistula with profuse haemorrhage. In the last decade, several studies have reported the use of endografts to repair secondary AEF. In a retrospective multicentre study evaluating the outcome of patients after endovascular or open repair of secondary AEF, no in hospital death was reported in patients treated with endografts compared with an in hospital mortality of 35% in patients treated by open repair.[248] Late sepsis occurred significantly more often after endovascular repair than after open surgery (42% *vs.* 19% at 2 years).[248]

The use of a temporary stent graft as a bridging technique during active bleeding allows for patient stabilisation and offers the option of later extraction and *in situ* repair. In such cases, devices without suprarenal fixation are preferable to allow for easier subsequent explantation. For selected cases of non-septic bleeding, stent grafts can serve as the definitive vascular solution, followed by isolated laparotomy exclusively for enteric repair.[247]

7.3.3. Arterio-ureteral fistula. A secondary arterio-ureteral fistula (AUF) is defined as direct luminal communication between the ureteral lumen and the vascular graft or endograft (see Chapter 2). Clinical presentations include

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haematuria, recurrent urinary tract infections, or urinary outflow obstruction leading to hydronephrosis.[249] In acute haemorrhage cases, endograft insertion should be considered.[250]

A systematic review including 445 patients with primary and secondary AUF (26% underwent previous vascular surgery) reported a lower AUF related mortality rate for endovascular stent graft placement compared with open surgical repair (4% *vs.* 11%).[251] The AUF specific mortality rate before the year 2000 *vs.* after the year 2000 was 19% *vs.* 7%, coinciding with increasing use of endovascular stent grafts.[249,252]

For definitive treatment of the infection, open surgery with vascular graft or endograft explantation and AUF resection should be discussed in the MDT meeting including a urologist. Successful *in situ* repair with cryopreserved allografts, silver coated, or rifampicin bonded prosthetic grafts has been reported; however, the literature remains scarce.[249,250,253] Vascular graft or endograft removal with extra-anatomic femorofemoral crossover bypass and ligation of the iliac artery can also be considered in selected patients, particularly when *in situ* reconstruction is contraindicated or technically challenging.

Primary ureteric repair, ligation (with or without nephrectomy), re-implantation to a site away from the vessels, or urinary diversion with a nephrostomy may be necessary. Nephrectomy can be considered if irreversible damage to the renal collecting system has occurred.[252] Figure 6 provides a summarised algorithm of management of patients with abdominal VGEI.

Recommendation 59 — Unchanged

Endovascular treatment as a bridge is recommended for patients with abdominal vascular graft or endograft infection complicated by aorto-enteric or arterio-ureteral fistula with profuse haemorrhage.

Class	Level	Reference	ToE
I	C	Kamphorst *et al.* (2022)[252]	

Recommendation 60 — Changed

Urinary tract repair combined with explantation of the infected graft or endograft with vascular reconstruction is recommended in abdominal vascular graft or endograft infection with arterio-ureteral fistula.

Class	Level	Reference	ToE
I	C	Kamphorst *et al.* (2022)[252]	

Recommendation 61 — New

Involvement of a gastrointestinal surgeon or a urologist is recommended in all cases of bowel or urinary tract repair in abdominal vascular graft or endograft infection with secondary fistula.

Class	Level	Reference
I	C	Consensus

8. MANAGEMENT OF EXTRACAVITY VASCULAR GRAFT OR ENDOGRAFT INFECTION

8.1. Supra-aortic trunk vascular graft or endograft infection

Extracavity VGEIs include the supra-aortic trunks (SATs), upper limbs, and lower limbs. The precise VGEI incidence of the respective anatomic locations is unknown, but infection rates might be underestimated due to a lack of recognition and under reporting. Extracavity VGEIs include infections of prosthetic patches, vascular grafts, and endografts.

In a recent systematic review and meta-analysis including 149 patients with VGEI following carotid endarterectomy (CEA) with prosthetic patch closure, the occurrence rate was estimated at 0.7% (95% CI 0.4 — 0.9%).[5] In the same review, only 12 reported cases involved prosthetic SAT bypasses, which is too few to estimate an infection rate. The occurrence of SAT infection after endovascular treatment is even lower, with an estimated rate of less than 1 in 10 000 cases.[254] A recent systematic review of the literature identified only 21 patients with SAT stent or stent graft infections over the last four decades.[5]

As most of the available literature concerns carotid patch infections, the majority of the present recommendations are based on this evidence.[5,255] Where evidence specifically relates to other SAT infections (e.g., bypass grafts or stents), this will be explicitly stated.

8.1.1. Conservative treatment. Conservative treatment with antimicrobials alone has been documented in only four cases, three following patch infection after CEA and another involving a stent infection in the subclavian artery.[254,256–258] In three cases, the antimicrobial only strategy was successful, although the patient with the infected subclavian stent developed a false aneurysm that was left untreated.[254] In one case, a two stage procedure (stent insertion followed by carotid patch excision and debridement) became necessary after 1 year of follow up.[257] Thus, supporting a totally conservative management strategy is inadequate, as there is a risk of suture line rupture with patches or bypasses, and vascular wall necrosis with stent grafts, which could lead to life threatening complications such as uncontrolled major bleeding in the chest or tracheal compression in the neck.[254,257]

Recommendation 62 — Unchanged

Conservative treatment should be considered as a palliative option in selected patients with supra-aortic trunk vascular graft or endograft infection ineligible for graft or endograft explantation.

Class	Level	Reference
IIa	C	Consensus

8.1.2. Endovascular treatment. Endovascular treatment can also be an option, primarily in life threatening situations to control major VGEI bleeding.[259] Data regarding coil embolisation alone are insufficient to support its

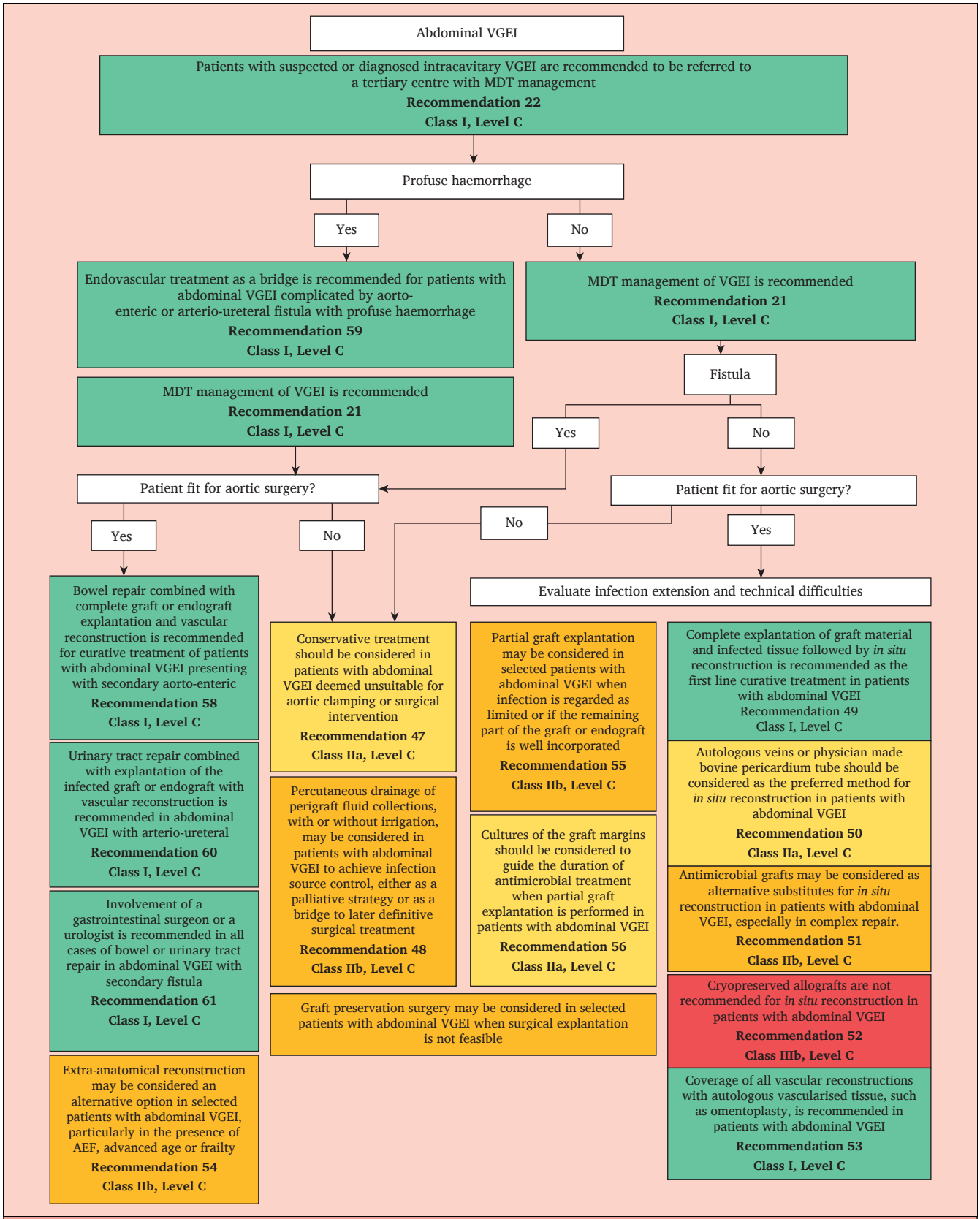

Figure 6. Algorithm for the management of abdominal vascular graft or endograft infection (VGEI).

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widespread use. It has been reported rarely, and information on stroke rates remains inadequate.[5,251,260]

The endovascular first strategy can be considered as a bridge to therapy in unstable patients.[257,259,261] This approach allows for controlled, semi-elective graft revision, including excision and reconstruction, providing temporary haemostasis and time to plan definitive management, thus avoiding emergency open surgery under unstable conditions. Such a strategy has also been described after carotid artery stenting or stent grafting.[260,262]

Recommendation 63		Changed
For patients with supra-aortic trunk vascular graft or endograft infection presenting with profuse haemorrhage, endovascular treatment as a bridge to definitive management should be considered.		
Class	**Level**	**Reference**
IIa	C	Consensus

The endovascular vacuum assisted closure (EndoVAC) technique is a hybrid approach that has been used in nine reported cases of infected carotid patches after endarterectomy[263,264] and in four cases of SAT bypass grafts.[263] The EndoVAC technique is a three step procedure: (1) relining the infected reconstruction with a covered stent; (2) surgical revision without clamping; and (3) using NPWT with the sponge applied directly on the vessel and the exposed stent graft. Continuous topical negative pressure is applied for the first twenty-four hours, followed by intermittent negative pressure at 125 mmHg to facilitate granulation and delayed secondary healing. This technique yielded promising results in a small, selected cohort: no re-infections were reported after EndoVAC treatment of carotid patch infections with a median follow up of 19 months, although re-interventions for stenosis or occlusion were observed (two of nine patients).

Recommendation 64		Unchanged
The EndoVAC technique* may be considered for selected patients with supra-aortic trunk vascular graft infection and complex anatomy.		
Class	**Level**	**Reference**
IIb	C	Consensus

* Endovascular relining with a covered stent followed by surgical revision and negative pressure wound therapy.

8.1.3. Surgical treatment. The first step in the surgical treatment of SAT VGEI is debridement of the surrounding infected tissue, as reported in nearly all cases in the literature.[5] Patients who did not undergo surgical debridement were typically considered to be at too high surgical risk due to severe comorbidities.[256,265] Debridement of infected tissues can be followed by explantation of the infected vascular materials, reconstruction, adjunctive procedures such as muscle flaps, and or long term antimicrobial administration, alone or in combination.

A surgical approach involving complete removal of infected foreign material is generally recommended in elective cases. In most instances, replacing the explanted graft, patch, or arterial patched or stented segment is necessary to prevent cerebral ischaemia or infarction. These are technically challenging procedures, with a reported post-operative mortality rate of 3.4% and stroke rate of 5.2% after surgery for carotid patch infections based on limited pooled data from small series.[5] Local complications are also frequent; in particular, the post-operative cranial nerve injury rate is 12.5% (higher than that observed after primary CEA), and approximatively one third of these injuries were reported as permanent.[5]

In emergency life threatening situations, primary ligation of the vessel has been proposed by some authors, particularly if the infected reconstruction is already thrombosed and the patient is asymptomatic neurologically, or if the cerebral infarction has already stabilised.[266]

Autologous material is typically preferred for reconstruction. Given the relatively short length and matching diameters of most of these reconstructions, autologous saphenous vein grafts can be used in the majority of planned procedures.[267,268] For patch infections, interposition grafts may be preferred over patch substitutions to avoid suturing in the same infected area of the artery, thereby reducing the risk of suture line rupture.[5] As an alternative to the saphenous vein, the use of jugular vein, superficial femoral vein, autologous arteries, cryopreserved allografts, or synthetic grafts has been proposed, although these options are employed less commonly (Figs 7 and 8).[5,211,269]

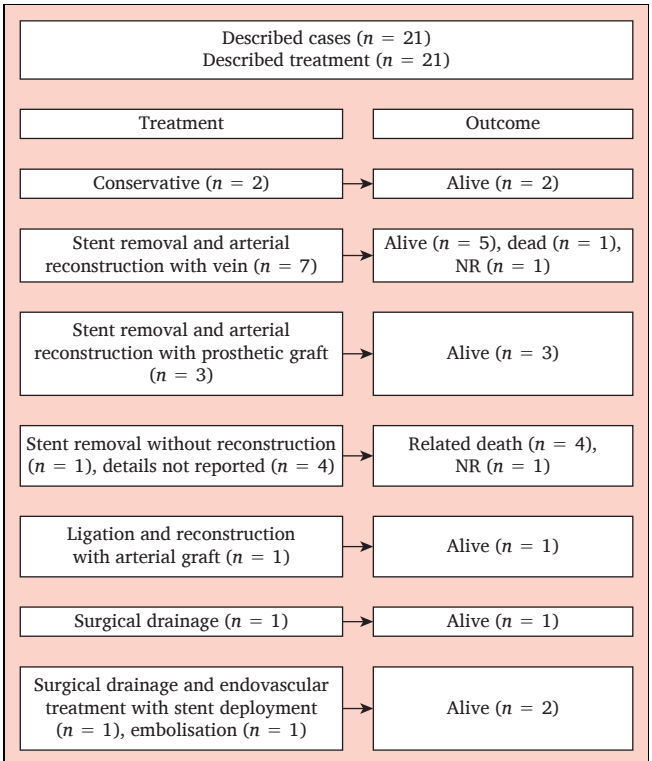

Figure 7. Management and outcomes of supra-aortic trunk infections after endovascular treatment. NR = not reported. From Perini *et al.* (2025).[5]

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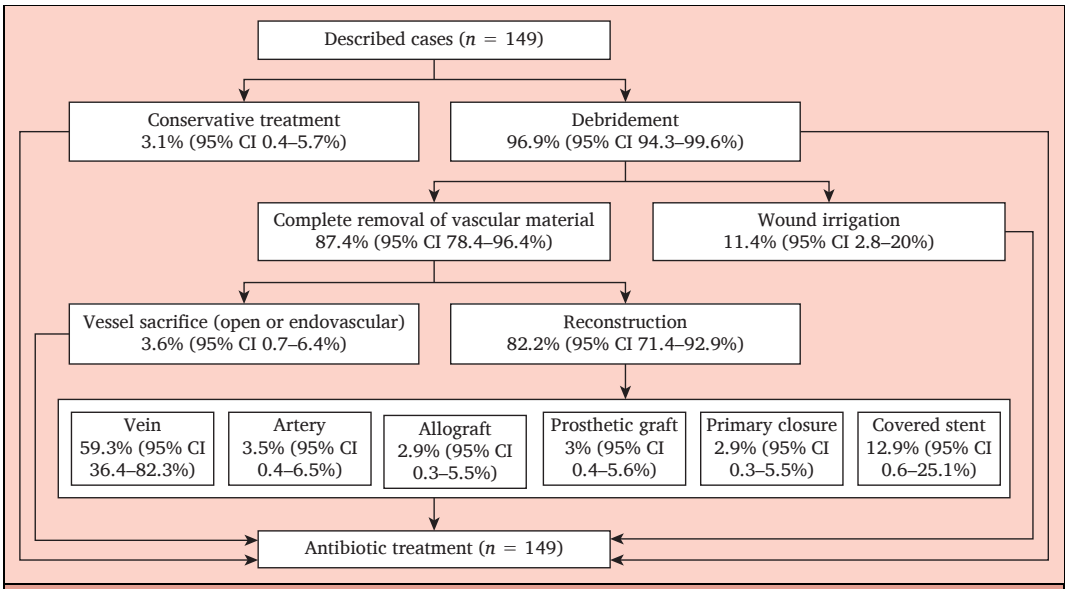

Figure 8. Management of supra-aortic trunk infections after open treatment. CI = confidence interval. From Perini *et al.* (2025).[5]

Total explantation is crucial as it ensures the removal of all infected material, even if it does not appear infected during surgical exploration. During follow up, the re-infection rate is reported to be low (3.4%) and re-interventions are infrequently necessary (3.4%) for re-stenosis, thrombosis, or false aneurysm formation.[5] Except for complex SAT bypasses, grafts and arterial segments with infected patches or stent grafts are generally short in the SAT, often making total explantation feasible. Achieving proximal arterial control is crucial, which may require an occlusion balloon or accessing an unscarred site via sternotomy or thoracotomy. This ensures a safer entry into the infected tissues and helps reduce peri-operative complications.[270]

The importance of obtaining proximal arterial control is heightened in cases with active bleeding caused by infection related arterial wall rupture.[270–272] Primary arterial control through the same cervical incision may be challenging, increasing the risk of significant blood loss and inadvertent injury to peripheral nerves.

Recommendation 65			**Unchanged**
For patients with supra-aortic trunk vascular graft or endograft infection, explantation of the infected material and debridement of infected tissue followed by reconstruction with autologous material is recommended as first line treatment.			
Class	**Level**	**References**	**ToE**
I	C	Perini *et al.* (2025),[5] Mann *et al.* (2012),[265] Fatima *et al.* (2019),[268] Small *et al.* (2021)[269]	

8.1.4. Adjunctive therapy. In addition to debridement of infected tissues and reconstruction, several adjunctive procedures can be employed in the management of SAT VGEI. Muscle flaps assist in healing and infection control by increasing local blood flow, thereby enhancing the delivery of antibiotic agents to the infected area, and have already demonstrated their effectiveness in groin infections.[273] Muscle flaps are particularly useful in cases where incomplete debridement and or material excision has occurred, or when a prosthetic graft has been used for reconstruction. Evidence for the use of muscle flaps to treat VGEI in the SAT is more limited (its use is reported in a pooled estimate of 5.2% after carotid patch infection), but it has provided encouraging results.[5] Muscle flaps have also been described in the management of carotid patch infections, both with patch preservation after local wound debridement and after patch removal with arterial reconstruction. Sternocleidomastoid, *pectoralis major*, and omohyoid muscle flaps have been reported in 18 cases of SAT VGEI. Of these, eight followed complete removal of the infected material and arterial reconstruction, while ten were combined with local wound debridement.[256,257,259,267,274–279] Among the ten patients treated with local wound debridement and muscle flap coverage, two complications were described during follow up: one false aneurysm required placement of a covered stent; and one carotid occlusion with hemispheric stroke approximately thirty days after muscle flap repair of an infected carotid patch.[256,276]

Another possible adjunctive treatment is NPWT, which has demonstrated its effectiveness in promoting drainage and encouraging granulation tissue formation in a small cohort of carotid patch infections and SAT bypasses.[263,264] Figure 9 provides a summarised algorithm of management of patients with SAT VGEI.

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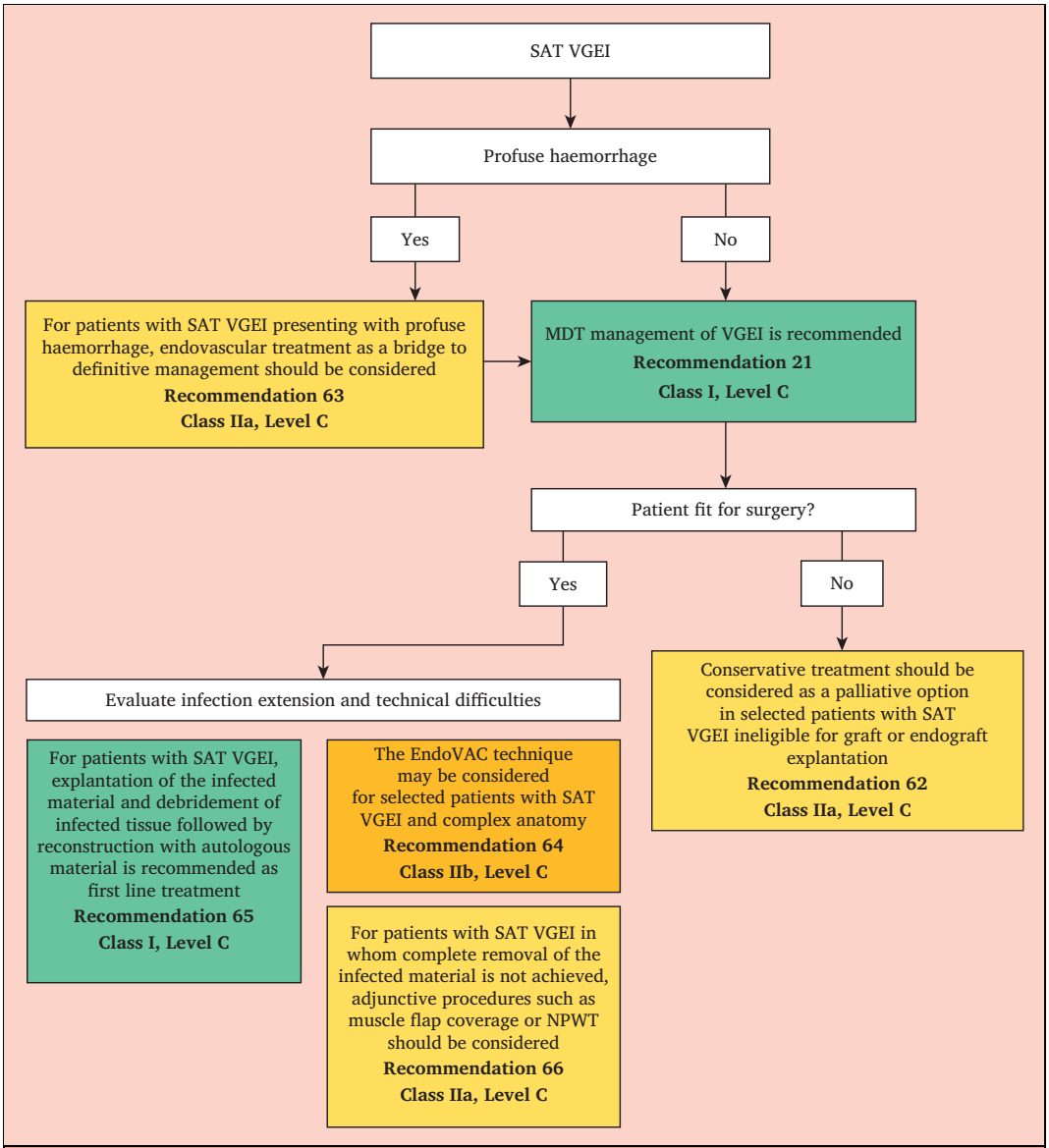

Figure 9. Algorithm for the management of supra-aortic trunk (SAT) vascular graft or endograft infection (VGEI). MDT = multidisciplinary team; NPWT = negative pressure wound therapy.

Recommendation 66			Changed
For patients with supra-aortic trunk vascular graft or endograft infection in whom complete removal of the infected material is not achieved, adjunctive procedures such as muscle flap coverage or negative pressure wound therapy should be considered.			
Class	**Level**	**References**	**ToE**
IIa	C	Perini *et al.* (2025),[5] Alawy *et al.* (2017),[256] Thorbjørnsen *et al.* (2016),[263] Shehab *et al.* (2024)[264]	

8.2. Upper limb vascular graft or endograft infection

Except for grafts used as vascular access for haemodialysis, upper limb VGEIs are rarely reported in the literature. It cannot be excluded that they are under reported; however, the low number of reported cases may also reflect the infrequent use of vascular grafts in the upper extremities for indications other than vascular access for haemodialysis. Only four reports (two case reports and two small case series, each including one patient with infection) were identified in the literature.[280–283] In the identified case reports, treatment typically involved explantation of the infected graft or endograft. Reconstruction was reported in one case,[282] although other surgical strategies may be considered depending on the location and clinical scenario.

8.2.1. Arteriovenous grafts for haemodialysis. Arteriovenous grafts are considered for haemodialysis access in patients without a suitable vein for creation of an arteriovenous fistula. The most commonly used synthetic graft is PTFE. A systematic review and meta-analysis evaluated 395 PTFE grafts for arteriovenous access. The pooled rate of infection was 95 per 1 000 patient years.[284]

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Staphylococcus aureus, particularly methicillin resistant strains, is the most frequently reported cause of arteriovenous graft infection, accounting for over 50% of cases in some cohorts.[284]

Infection is associated with the risk of sepsis and suture line disruption with life threatening bleeding. In general, extensive perigraft effusions require complete graft removal, while in some late infections unaffected well incorporated graft segments can be saved. Treatment requires antimicrobial therapy and graft explantation. Total graft explantation is the most effective way to eradicate infection. Subtotal explantation refers to removal of the graft leaving only a small stump on the arterial side to be closed. This approach avoids extensive dissection of the artery and the risk of nerve damage. If the infection is limited, localised to a segment of the graft, partial explantation can be performed. Infected graft outcomes are best following total graft explantation (1.6% recurrence rate) compared with subtotal explantation (19% recurrence rate) and partial explantation (29% recurrence rate).[285]

8.3. Lower limb vascular graft or endograft infection

The most common site for VGEI is the groin. Accordingly, graft infection rates of up to 4.2% in femorofemoral, 5.2% in femoropopliteal, and 5.9% in femorodistal bypasses have been reported, although these may include both wound and graft infections, and definitions vary between studies.[136] The incidence is, however, difficult to address since differentiation between deep wound infection and confirmed graft infection is unclear in many reports.

Regarding lower limb stent infection, the incidence is unknown.[286] Moreover, stent infection might be underestimated due to a lack of recognition and under reporting.

The literature on lower limb VGEI is particularly scarce and of very low quality. Hence, most of the following recommendations are based on low quality literature and therefore expert opinion.

8.3.1. Conservative treatment. Conservative treatment of lower limb VGEI (antimicrobial treatment, with or without percutaneous drainage/irrigation) is rarely an option owing to the risks of persistent infection, anastomotic disruption, and profuse haemorrhage.

Recommendation 67		Unchanged
Conservative treatment should be considered a palliative option in selected patients with lower limb vascular graft or endograft infection ineligible for graft or endograft explantation.		
Class	**Level**	**Reference**
IIa	C	Consensus

8.3.2. Debridement of infected tissues. Debridement of the arterial bed constitutes the basis of treatment and should be performed during any surgical intervention.

8.3.3. Graft preservation or graft explantation. If the initial indication for revascularisation was claudication or for a chronically occluded bypass, graft or endograft explantation without vascular reconstruction is allowed. Biological substitutes, such as autologous vein patch or bovine pericardial patch, should be used for closing arteriotomies.

If the initial indication for revascularisation was acute or chronic limb threatening ischaemia (CLTI) (Rutherford 4—6) and severe ischaemia of the leg is expected after graft or endograft removal, immediate revascularisation is required in most cases to avoid major amputation. Revascularisation is also needed when the arterial disease has progressed significantly, or removal of the infected graft or endograft is not possible without sacrificing crucial collaterals.[136]

Debridement with partial graft explantation may be considered if part of the graft is well incorporated and lacks obvious contamination. Debridement with graft preservation may be considered if the graft is patent, anastomoses are intact, and the whole graft is well incorporated.[287,288] However, risks of persistent infection, anastomotic disruption, and active bleeding need to be considered.

Recommendation 68		Unchanged
For patients with lower limb vascular graft or endograft infection, explantation of the infected material is recommended as first line treatment, with revascularisation if indicated.		
Class	**Level**	**Reference**
I	C	Consensus

Recommendation 69		New
For selected patients with lower limb vascular graft or endograft infection, partial graft explantation may be considered when the infection is of limited extent.		
Class	**Level**	**Reference**
IIb	C	Consensus

Recommendation 70		New
For selected patients with lower limb vascular graft or endograft infection without involvement of the anastomoses and limited infection extent, a graft preservation strategy may be considered.		
Class	**Level**	**Reference**
IIb	C	Consensus

8.3.4. Vascular reconstruction. *In situ* reconstruction using autologous vein is the preferred option, but extra-anatomic reconstruction can be an alternative to avoid routing in infected and or multi-operated areas. Extra-anatomic reconstructions mostly include obturator or lateral femoral bypasses, but lateral transiliac bypasses are also described (Fig. 10).

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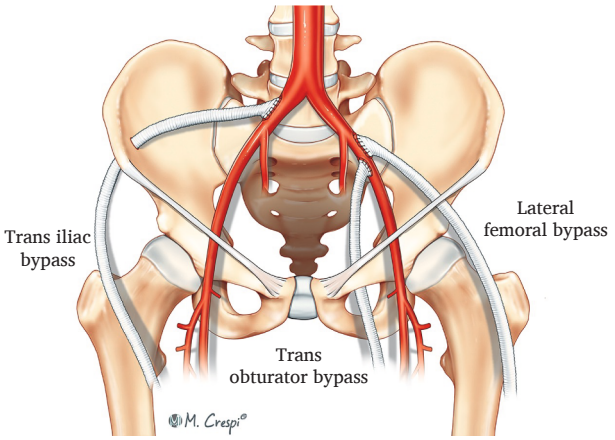

Figure 10. Extra-anatomic reconstructions used for groin infection.

Recommendation 71		**Unchanged**
For patients with lower limb vascular graft or endograft infection, *in situ* reconstruction with autologous vein is recommended as first line treatment.		
Class	**Level**	**Reference**
I	C	Consensus

Recommendation 72		**New**
For selected patients* with lower limb vascular graft or endograft infection, extra-anatomic graft routing may be considered.		
Class	**Level**	**References** **ToE**
IIb	C	Moawed *et al.* (2003),[289] Madden *et al.* (2019),[290] Enzmann *et al.* (2019),[291] Hoballah *et al.* (1996)[292]

* To avoid placement of the new graft into the infected area or to avoid heavily scarred anatomic zones from multiple previous operations.

The obturator bypass is the most used extra-anatomic bypass procedure. It avoids the femoral triangle by using a conduit from the aorto-iliac section to the superficial femoral, deep femoral, or popliteal artery, depending on the runoff circumstances. In the obturator bypass, the graft is tunnelled via the obturator foramen, dorsally to the hip joint, in a layer between the adductor magnus and longus muscles. In a prospective cohort including 100 patients (117 limbs) with groin infection undergoing an obturator bypass, the two year primary patency was 63% and the limb salvage rate was 97%.[289]

When the medial approach is not possible due to extended infection or after extensive approaches to the groin and pelvic region, lateral femoral bypass can be performed. During lateral femoral bypass, the graft is tunnelled lateral to the infected field, medial to the anterior superior iliac spine, and under the inguinal ligament through the psoas canal. It is considered a technically less demanding strategy than obturator bypass. In a retrospective cohort including 16 patients (19 limbs, mixed graft materials) with groin infection who underwent a lateral femoral bypass, two year primary patency was 83% and limb salvage was 94%.[290]

The transiliac bypass is an alternative technique. A small part of the iliacus muscle is separated from the iliac wing at its origin to prepare a path for the graft. The graft is then tunnelled through the upper part of the iliac wing, lateral to the infected field. In a retrospective cohort including eight patients (eight limbs) undergoing a transiliac bypass, two year primary patency was 37% and limb salvage was 100%.[291]

For a distal anastomosis to the popliteal and crural vessels, a lateral approach can be used. The graft is tunnelled laterally, far away from the infected tissues. In a cohort of 23 lateral bypasses (21 autologous veins and two expanded PTFE [ePTFE] grafts), the primary patency at 1 year was 61% and the secondary patency was 86%. One amputation was required due to poor runoff and graft thrombosis.[292]

8.3.5. Graft materials

8.3.5.1. Autologous vein. The great saphenous vein is the preferred substitute owing to infection resistance and its low graft related re-intervention rate (< 10% at 3 years) if available.[136,211,293] For short and large diameter interpositions, such as common femoral artery reconstructions, great saphenous vein panel grafts or spiral grafts are further possible alternatives.[294]

Arm veins have been used for revascularisation in the setting of CLTI with reported three year primary patency of 28.3% and secondary patency of 56.8%.[295] In a recent Vascular Quality Initiative analysis including 9 165 patients, arm vein bypasses were compared with synthetic and other biological reconstruction materials in patients with CLTI for femoropopliteal or femorotibial bypasses.[296] Arm veins yielded the highest amputation free and overall survival. Compared with veins, prosthetic grafts had a higher risk of re-infection (adjusted OR 2.89; $p = .045$), and other biological grafts had a higher risk of occlusion (adjusted OR 4.55; $p = .040$) and re-infection (adjusted OR 2.78; $p = .046$). However, the arm vein literature in the setting of lower limb VGEI is almost non-existent, and insufficient data exist to determine the optimal alternative vein graft for reconstruction if autologous great saphenous vein is not available.

8.3.5.2. Cryopreserved allografts. Cryopreserved arterial or venous allografts have been described as substitutes for revascularisation after infected graft removal for VGEI.[206,211,297–300]

Cryopreserved arterial allografts enjoy low re-infection rates of 0–9% in the peripheral setting and acceptable primary patency rates of 59–82% at 3 years.[206,211] Graft related re-interventions are more common (up to 25% at 3 years), including allograft rupture or allograft degeneration.[206] Nevertheless, as peripheral allograft pseudoaneurysms are more readily detected clinically and graft rupture is generally less catastrophic than in intracavity reconstructions, the use of cryopreserved arterial allografts may be acceptable for vascular reconstruction in lower limb VGEI.

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Cryopreserved veins have the advantage of a better size match in the crural vessels; furthermore, longer segments than cryopreserved arteries are available. In the setting of CLTI, patency remains limited (primary and secondary patency of 56% and 73% at 1 year, and 17% and 38% at 5 years), and neither immunosuppression nor anticoagulation provided better outcomes in a cohort of 92 patients.[298] In a retrospective study, 13 cryopreserved veins were used for peripheral VGEI and no re-infection was noted.[299]

8.3.5.3. Xenografts. Bovine pericardial patches can be used either to close the arterial defect after removal of infected material when revascularisation is unnecessary, or as self made bovine pericardial tube for short interpositions.[301,302]

In a recent study evaluating 21 reconstructions using a self made bovine pericardial tube for VGEI in 17 cases, two late re-infections appeared in drug users (therefore not attributable to material but rather to behavioural factors). All grafts were covered with *fascia lata.* Re-intervention free survival was 70% at 12 months.[302]

A recent meta-analysis evaluated outcomes of the Omniflow II biosynthetic graft for lower limb vascular reconstruction in the setting of VGEI. Among 116 patients, one and three year freedom from re-infection rates were 95% and 78%, one and three year primary patency rates were 79% and 68%, and one and three year secondary patency rates were 83% and 78%, respectively.[6]

8.3.5.4. Antimicrobial grafts. Off the shelf availability of antimicrobial grafts is the main advantage, and silver impregnated grafts or rifampicin soaked PET grafts have been used for revascularisation following the removal of infected material. However, most of the reports concern aorto-iliac reconstructions, and the literature is scarce on the peripheral level. In a study of 24 femoropopliteal bypass replacements with silver impregnated grafts, the re-infection rate was 19%, late patency 72%, and amputation rate 12%.[303] Rifampicin soaked grafts should be used with caution, since causative bacteria may not be susceptible to rifampicin (31% in a cohort of 48 patients).[304] Furthermore, rifampicin monotherapy may cause the emergence of antibiotic resistant strains, therefore a meticulous search for other possible reconstruction materials should be performed. Accordingly, antimicrobial grafts are not recommended as substitutes for revascularisation after infected graft removal for VGEI, since several other alternatives are available.

Recommendation 73 — **Changed**

Cryopreserved allografts or biosynthetic collagen grafts may be considered for alternative conduits for vascular reconstruction in lower limb vascular graft or endograft infection.

Class	Level	References	ToE
IIb	C	Aubertin *et al.* (2025),[6] Lejay *et al.* (2017),[206] Tabiei *et al.* (2023),[211] Furlough *et al.* (2019)[297]	

Recommendation 74 — **New**

Vascular reconstruction using bovine pericardium may be considered in patients with lower limb vascular graft or endograft infection limited to the groin.

Class	Level	References	ToE
IIb	C	McMillan *et al.* (2012),[301] Hostalrich *et al.* (2024)[302]	

Recommendation 75 — **New**

Antimicrobial grafts are not recommended for vascular reconstruction in patients with lower limb vascular graft or endograft infection.

Class	Level	Reference
IIIb	C	Consensus

8.3.6. Endovascular treatment. Endovascular treatment is an effective option to control profuse haemorrhage caused by an infected pseudoaneurysm or graft rupture.

Recommendation 76 — **New**

Endovascular treatment is recommended as first line strategy in lower limb vascular graft or endograft infection with profuse haemorrhage.

Class	Level	Reference
I	C	Consensus

Endovascular treatment can be used as a bridge to definitive surgical revision or as a part of hybrid approach such as the EndoVAC technique. Following covered stent placement to reline the infected graft or anastomosis, surgical debridement of infected tissue is performed with or without muscle flap, after which the wound is managed through secondary intention healing using NPWT and long term antimicrobial therapy. In a series of five groin infections, four patients were healed with the EndoVAC technique. The median duration of antimicrobial treatment was 2 months (range 1 — 9 months). However, in one case the deep femoral artery was embolised to achieve haemostasis in the acute phase and two grafts occluded during follow up.[263]

8.3.7. Graft coverage. Vascularised muscle flap wound coverage after debridement with or without vascular reconstruction is essential in filling dead space, increasing local tissue oxygenation, and increasing the transport of antimicrobial agents to the infected area.[273,305] Furthermore, the overlying muscle protects underlying tissues, the vascular reconstruction, and provides an ideal base for a later skin graft.

Sartorius, *rectus femoris*, and gracilis muscles are the most common flaps used in the setting of VGEI.[273,306] A sartorius muscle flap has historically been the favoured

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choice for rotational muscle flap coverage because of its proximity to the vessels. The harvest of the sartorius muscle is simple, quick, and associated with minimal lower extremity morbidity. However, concerns exist whether its blood supply (as the sartorius arterial blood supply originates from multiple segmental branches of the superficial and deep femoral arteries) and consequently muscle flap viability may be compromised in patients with CLTI, especially when previous vascular interventions could have altered its already tenuous blood supply.[307] An alternative to the sartorius muscle flap is the *rectus femoris* muscle. Since the flap is large, remote from the groin, and has a robust blood supply from the lateral femoral circumflex artery, it can provide a bulk to fill larger defects after aggressive debridement. However, it may be less appealing as its harvest can compromise knee extension.[308] The gracilis muscle flap can also be used. Although gracilis muscle harvest can be challenging, its viability is good. The blood supply to the gracilis muscle is derived from a single branch of the deep femoral artery, more likely to be spared from atherosclerosis. Moreover, the harvest site is distant from the infection site and in an area that is simple to close after harvest. The resulting functional deficit might also be less than after rectus femoris muscle flap.[309,310] However, the gracilis flap arc of rotation is limited unless microvascular techniques are used. Accordingly, if muscle flap harvesting is unfamiliar to the vascular surgeon, the involvement of a plastic surgeon is required.

A systematic review and meta-analysis evaluated the use of sartorius, *rectus femoris*, and gracilis muscle flaps in the setting of groin infection. Among 1 207 flaps, the sartorius muscle flap yielded the lowest rate of flap necrosis compared with *rectus femoris* and gracilis muscle flaps (3.4% *vs.* 7.5% *vs.* 5.6%, respectively), limb loss (6% *vs.* 7.4% *vs.* 17.2%, respectively), and thirty day mortality rate (5.3% *vs.* 10.0% *vs.* 10.5%, respectively).[273]

Recommendation 77 — **Unchanged**

Muscle flaps should be considered for coverage of complex wounds and vascular reconstructions in patients with lower limb vascular graft or endograft infection to promote wound healing and facilitate graft protection.

Class	Level	Reference
IIa	C	Consensus

Recommendation 78 — **New**

For patients with lower limb vascular graft or endograft infection requiring complex muscle flap reconstruction, involvement of plastic surgical expertise is recommended.

Class	Level	Reference
I	C	Consensus

8.3.8. Adjunctive therapy. To reduce the bacterial burden of the infected field, peri-operative irrigation should be an adjunct in every surgical procedure performed for VGEI. Continuous irrigation has also been proposed as part of a graft preservation strategy; however, no sufficient evidence exists to provide a recommendation.[311]

NPWT may be used as an adjunct after debridement, graft explantation or preservation, revascularisation, and muscle flap, to promote wound healing.[5] Usually, continuous 125 mmHg negative pressure allows faster granulation and healing. NPWT may be applied directly to the graft; however, all anastomoses should be covered with vascularised tissue to prevent rupture.[312,313] Using the abovementioned techniques, a graft preserving strategy was successful in over 80% of cases among 233 patients in two different series.[312,313] Synthetic grafts, however, were risk factors for failure (OR 6.1, 95% CI 2.6 — 14.2) and the re-infection rate remained 6.4% after wound healing.[313] Figure 11 provides a summarised algorithm of management of patients with lower limb VGEI.

Recommendation 79 — **Unchanged**

Negative pressure wound therapy should be considered as an adjunct in the management of lower limb vascular graft or endograft infection to promote wound healing and facilitate graft protection or preservation.

Class	Level	References	ToE
IIa	C	Monsen *et al.* (2014),[314] Cheng *et al.* (2014)[315]	

9. FOLLOW UP

The literature on VGEI follow up is particularly scarce. A Delphi consensus survey was performed in order to provide international expert recommendations on VGEI follow up.[115] It is important to note that follow up will vary depending on the treatment strategy (palliative or curative approach), bearing in mind that it may also vary over time. Each situation may therefore require a different approach and personalised follow up with MDT involvement.

Follow up of patients for whom a palliative strategy has been proposed may best serve to accommodate the patient's individual needs (manage symptoms, alleviate suffering, and address physical and psychological needs). Follow up of patients for whom a curative strategy has been proposed is required to ensure good patient compliance with treatment, to detect adverse effects of antimicrobial therapy, and for early detection of treatment failure. Extra observation and support should be given to nutritional status to optimise healing conditions.

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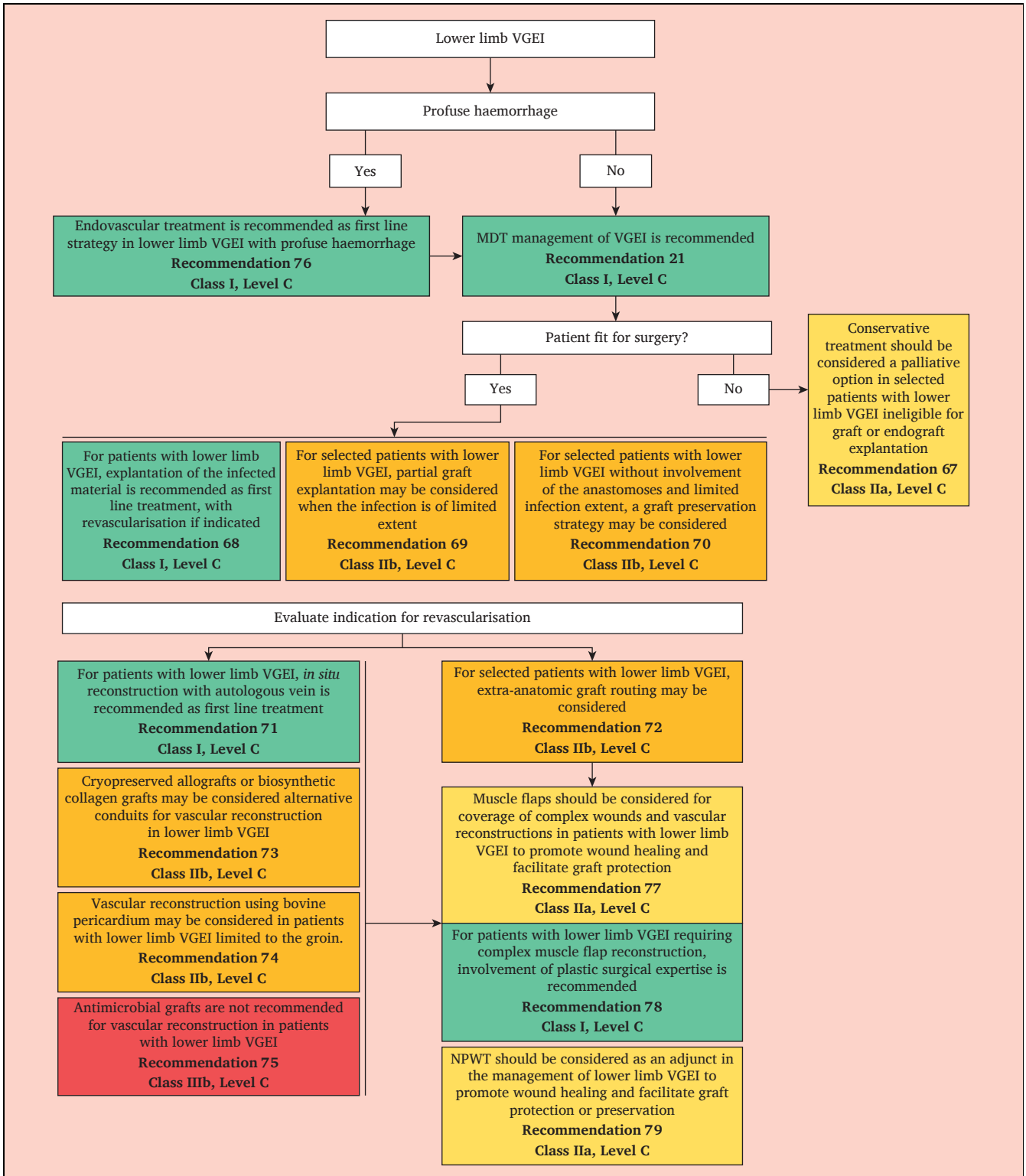

Figure 11. Algorithm for the management of lower limb vascular graft or endograft infection (VGEI). NPWT = negative pressure would therapy.

If long term or lifelong antimicrobial therapy is required, management and monitoring are best coordinated through an OPAT programme.[119] Before cessation of antimicrobial therapy, the following should be confirmed in an MDT meeting: absence of symptoms; normal WBC count and CRP; absence of infection signs on imaging, with confirmation sustained for at least 2 weeks post-antibiotic taper.[4]

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Recommendation 80		New
If long term antimicrobial therapy for vascular graft or endograft infection is required, management and monitoring through an outpatient parenteral antimicrobial therapy programme is recommended.		
Class	**Level**	**Reference**
I	C	Consensus

Since VGEI has a dynamic course, it is impossible to have a one size fits all follow up model.

Frequency of follow up is based on an individual patient tailored approach and depends on the treatment strategy, clinical evaluation, laboratory tests (WBC count and CRP), and imaging results. CTA is considered the preferred imaging modality for monitoring VGEI.[115] When CTA is insufficient to rule out treatment failure, the use of [^{18}F]FDG PET/CT can be discussed since it adds additional information to therapy control.[61] In addition to laboratory and clinical information, consecutive [^{18}F]FDG PET/CT examinations can therefore be a valuable source of information for treatment monitoring, often providing results in addition to clinical and laboratory parameters.

In summary, all patients treated for VGEI require a structured but flexible individual follow up plan to monitor for relapsing infection and potential complications.

Recommendation 81		New
Individualised long term follow up by a multidisciplinary team is recommended for patients with vascular graft or endograft infection, guided by clinical, imaging, and laboratory findings.		
Class	**Level**	**Reference**
I	C	Consensus

10. SHARED DECISION MAKING WITH SUPPORTING INFORMATION FOR PATIENTS

10.1. The concept of shared decision making

SDM is a process focusing on best quality patient centred healthcare that respects patients' views, preferences, and autonomy.[316–318] The concept of SDM is 50 years old but it gained a structured definition in the early 2000s. When more than one management option is available, the patient and healthcare professionals should jointly evaluate the available evidence and management options in order to undertake decisions together in formulating a care plan. In other disciplines, SDM has been proven to have important overall healthcare benefits including improving the patient–clinician relationship, increasing patient compliance, and reducing healthcare costs by avoiding unwanted treatments.[319]

In the setting of VGEI, certain consequences, such as persistent pain and suffering, prolonged hospital stay, inability to return to pre-operative functional capacity, and receipt of unwanted interventions, are missing from the discussion between surgeons and patients. One of the core arguments for SDM is that clinicians alone may be unable to fully appreciate how individual patients prioritise competing outcomes, for example, valuing independence over survival.[320]

10.2. Shared decision making in the vascular graft or endograft infection field

Although SDM is increasingly emphasised across medical disciplines, its application in the VGEI field has not been systematically studied. Given the complexity of treatment decisions, implementing SDM in VGEI is challenging. Moreover, SDM is not possible in urgent situations, such as VGEI with profuse haemorrhage. However, it is essential in elective VGEI situations.

In a systematic review, several barriers to patient participation in decision making have been identified, including time, continuity of care, interpersonal characteristics of the clinicians, trust, and characteristics of the patient. Specifically, characteristics of the patient, such as age, ethnicity, and education, affect the ability to participate in decision making, given attitudes and perceptions of patients and clinicians.[321] These barriers are important in the setting of VGEI, since most patients are old and frail, and they are facing major vascular surgery for which many alternatives are available, and clinicians must take care to ensure that the patient's values are reflected in the decisions that affect the patient the most.

Accordingly, the field of VGEI is an important area in which to implement SDM.

10.3. Implementing shared decision making in vascular graft or endograft infection

A systematic review summarised the relevant international scientific evidence on strategies aimed at facilitating or improving healthcare practitioners' adoption of SDM in elective surgery.[322] Several tools or models have therefore been developed and can be extrapolated to the VGEI field. These models and tools vary in form and purpose: some help identify the potential harms and benefits of a choice, other assist in ranking or rating values. Two approaches can be considered: the three talk model and decision aids.

10.3.1. Three talk model. The three talk model in SDM identifies and parses three separate parts of the physician–patient interaction, consisting of discussing the choices (team talk), discussing the options (option talk), and reaching a joint decision for the care plan (decision talk). This model emphasises that decisions should be influenced by exploring and respecting what matters most to the patient as an individual.

Accordingly, team talk places emphasis on the need to provide support to patients when they are made aware of choices, and to elicit their goals as a means of guiding the SDM process. Option talk refers to the task of comparing alternatives, using risk communication principles, and decision talk refers to the task of arriving at decisions that reflect the informed preferences of patients, guided by the experience and expertise of the MDT health professionals.[318]

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It is important to note that during the process patients may often be asked to repeat and explain the technical aspects of the options to check for understanding. Throughout the whole process, time is allotted for deliberation, a process whereby patients become aware of choice, can understand the different options, and have the time and support to consider what matters most to them.

10.3.2. Decision aids. Decision aids are tools that help physicians share information with patients. These can come in the form of pamphlets, anatomic models, personalised risk charts, scoring systems, videos, or other tools. Such decision aids have been developed in vascular surgery, but the literature is scarce in the VGEI setting.[323] Such tools could present relevant data in a variety of ways, from graphical displays to option grids, and could be merged into SDM conversations.[319]

A key prerequisite for effective SDM is a well informed patient, and this guideline contributes to that by providing balanced, evidence based recommendations together with relevant considerations, as well as detailed patient information.

10.4. Information for patients

This information has been developed by the European Society for Vascular Surgery (ESVS). The ESVS develops medical guidelines to help doctors and other healthcare professionals care for people with vascular conditions, including vascular graft or endograft infection (VGEI). The full medical guidelines are written for healthcare professionals. This section presents the same information in simple, non-technical language, so that patients, family members, and carers can understand it. The goal is to give clear and unbiased information to help you take part in shared decision making with your healthcare team. Details about how this information was developed, and how strong the scientific evidence is, are explained at the end of this section.

What is a VGEI? A VGEI is a serious complication that can happen after vascular surgery, when an artificial blood vessel (called a graft or endograft) that has been placed in the body becomes infected. VGEI can occur in different parts of the body:

- inside the chest or abdomen, affecting the main artery (the aorta)
- in the arms or legs (upper and lower limb arteries)
- in the neck (carotid arteries).

In rare cases, the infection can create an abnormal connection (called a fistula) between the graft or endograft and nearby organs, such as the lungs, bowel, or urinary tract.

How is VGEI diagnosed? Doctors use established diagnostic criteria (called the MAGIC criteria) to diagnose VGEI in a consistent and reliable way. These criteria are widely used in everyday medical practice. Diagnosis usually includes:

- a physical examination
- blood and other tests to look for infection
- imaging tests (such as computed tomography).

What happens if I am diagnosed with a VGEI? If you are diagnosed with a VGEI, your care will be managed by a multidisciplinary team. This team always include a vascular surgeon and an infectious diseases specialist, and may involve other experts as needed. The key question will be: what is the best treatment for you? All patients with VGEI receive antimicrobial treatment, usually after tests have identified the bacteria causing the infection.

Antimicrobial treatment may be:

- suppressive, meaning it controls the infection but does not fully eliminate it
- curative, meaning it aims to completely clear the infection.

Some patients may also need surgery. Surgical treatment can involve:

- cleaning infected tissues
- removing all or part of the infected graft or endograft
- keeping the graft in place if this is considered safer.

Not everyone benefits from surgery. For some patients, especially those who are older or have heart, lung, or renal disease, the risk of surgery may be higher than the potential benefits. Treatment decisions are made together with you, taking into account your overall health, preferences, and the expert opinions of the healthcare team.

Do I need to be followed up? Yes. If you have a VGEI, regular follow up is essential. Follow up care involves the same multidisciplinary team, at least including the vascular surgeon and infectious diseases specialist. How often and how long you will be followed up depends on your individual situation.

How was this information developed? This patient information is based on the official ESVS guidelines written for healthcare professionals. These guidelines were created by a group of experts who carefully reviewed all available medical research on VGEI.

For each treatment option, the committee assessed:

- how strong the scientific evidence is
- whether experts agree on the best approach.

Treatments are graded based on:

- quality of evidence (from high quality evidence to little or no evidence)
- strength of recommendation (from strong recommendations to treatments that are not advised).

This patient section was developed and reviewed together with patients to ensure it is clear, relevant, and helpful.

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Table 18. Summary of suggested reporting variables for vascular graft or endograft infection (VGEI).

	Variable
Patient characteristics	
Demographics	Age
	Sex
Cardiovascular risk factors	Hypertension
	Obesity
	Diabetes
	Smoking status
	Dyslipidaemia
Comorbidities	Cardiopulmonary disease
	Chronic obstructive pulmonary disease
	Renal disease
	Liver disease
	Peripheral vascular disease
	ASA physical status classification
Pharmacological therapy	Antithrombotic agents
	Lipid lowering agents
	Glycaemic control agents
	Antihypertensives
	Antimicrobial therapy
Vascular graft or endograft description	Graft or endograft type
	Graft or endograft location
	Indication for graft or endograft implantation
Predisposing risk factors for VGEI	Malignancy
	Immunosuppressive condition
	Malnutrition
	Lymphoproliferative disorders
	Primary infected aortic pathology
	Bacteraemia during hospitalisation
	Infection in an adjacent or remote site
	Emergency or redo procedure
	Repeated instrumentations
	Animal exposure
Clinical findings	Fever, sweats, chills
	Loss of appetite, anorexia, weight loss
	Pain
	Emboli (viscera or limb)
	Wound swelling, wound dehiscence
	Mass
	Haemorrhage (haematemesis, haematuria, melaena)
Imaging findings	*CTA*
	Change or increase of perigraft fluid, gas, or soft tissue
	Fat stranding
	Aneurysm expansion or pseudoaneurysm formation
	Focal bowel wall thickening
	Discitis or osteomyelitis
	Secondary fistula
	Nuclear medicine
	Uptake activity
Laboratory findings	WBC count, CRP
	Cultures results
	Microbiological results
	Molecular biology results
MAGIC criteria	Suspected or diagnosed VGEI
Antimicrobial therapy	Empirical or targeted
	Suppressive or curative
	Duration
	Adverse effects
Management	Emergency or elective
	Endovascular as a bridge
	Percutaneous drainage or irrigation
	Graft preservation strategy
	Partial graft explantation
	Total graft explantation
	Type of vascular reconstruction
	Graft material
	Fistula treatment
	Coverage
	Adjunctive therapy
Follow up	30 day death
	In hospital death
	Survival
	Infection free survival
	Graft related complications
	Graft patency
	Need for re-intervention
Outcome	Cure of disease
	Disease in remission
	Treatment failure

ASA = American Society of Anesthesiologists; VGEI = vascular graft or endograft infection; CTA = computed tomography angiography; WBC = white blood cell; CRP = C reactive protein; MAGIC = Management of Aortic Graft Infection Collaboration.

This section on information for patients has been put together and reviewed by patients and healthcare professionals.

11. REPORTING ITEMS ON VASCULAR GRAFT OR ENDOGRAFT INFECTION TO ENHANCE COMPARABILITY BETWEEN STUDIES

To date, the lack of common language with commonly included variables hampers the understanding of outcomes of treatment for a wide spectrum of vascular conditions, including VGEI. Moreover, the low incidence of VGEI and the heterogeneity of the current literature (e.g., mixed populations, different outcomes definitions) make it difficult to draw evidence based conclusions and provide recommendations. A standard format for reporting data on VGEI is therefore required to improve the comparison between different management approaches and outcomes (Table 18). Adherence to these reporting items standards will improve the clinical relevance and quality of future studies on patients with VGEI.

11.1. Patient characteristics

Relevant baseline information regarding patients must be given. This means use of the accounted terminology and definitions[4] as well as the MAGIC definition and diagnostic criteria.[21] INAAs, primary aortic fistulas, inflammatory aneurysms, and penetrating aortic ulcers should be excluded.

An exact definition of patient characteristics and comorbidities is required. The following data should be reported: age; sex; hypertension; obesity; diabetes;

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smoking status (current smoker, ex smoker, never smoked); dyslipidaemia; cardiopulmonary disease; chronic obstructive pulmonary disease; renal disease (estimated glomerular filtration rate); liver disease; and peripheral vascular disease. Pharmacological therapy should be reported and should include the following: antithrombotic agents; lipid lowering agents; glycaemic control agents; antihypertensives; and antimicrobial therapy. The American Society of Anesthesiologists (ASA) physical status classification is simple to use and is routinely recorded in anaesthetic charts. Contemporary evidence has reported that increasing ASA grade is strongly associated with an increased thirty day mortality rate after vascular surgery.[324]

A well documented vascular history of vascular graft or endograft implantation is recommended, including type of graft or endograft, indication for implantation, and precise level of graft or endograft location.

Risk factors predisposing to bacterial contamination and therefore VGEI should also be listed.[10]

11.2. Presentation

Clinical findings need to be reported. Levels of inflammatory markers such as WBC count and CRP, results from cultures, and molecular biology of microbiological specimens from relevant tissue, blood, peri-prosthetic samples from drainage, or explant surgery should be reported. Imaging findings should be recorded and adhere to proposed reporting standards: change or increase of perigraft fluid, gas, or soft tissue, fat stranding, aneurysm expansion, pseudoaneurysm formation, focal bowel wall thickening, discitis or osteomyelitis, and suspicious activity or uptake on [^{18}F]FDG PET/CT or WBCS.[4,15,325] The MAGIC criteria should be used.[15]

The presence of a secondary aortic fistula should be specified, and cohorts of aortic VGEI without fistula *vs.* aortic VGEI with fistula should preferably not be mixed, since the prognosis, urgency of treatment, and required treatment approaches might be vastly different.

11.3. Management

Patient management should be clearly distinguished between conservative and surgical treatment, with a further differentiation between curative and palliative approaches.

Antimicrobial therapy data should include type of antimicrobial therapy (empirical or targeted, suppressive or curative), pre-operative (if administered) and post-operative duration, as well as adverse reactions.[115]

The surgical procedure should be meticulously described. Parameters of interest are the following: use of acute bridging endovascular repair to treat bleeding complications; use of percutaneous drainage and irrigation; total or partial graft explantation; graft preservation; surgical debridement of perigraft tissue; total or partial sac resection; location of aortic clamping; *in situ* reconstruction or extra-anatomic bypass; graft material; fistula treatment; and use of graft covering technique (omentoplasty or other).

11.4. Follow up and outcomes

The surveillance protocol must include details about type and duration of antimicrobial therapy, follow up modalities (clinical, laboratory, and imaging data), duration, and intervals.

Outcomes data should include in hospital and thirty day death, survival, infection free survival, graft related complications, graft patency, and need for re-intervention. Long term results such as VGEI cure, disease in remission, or treatment failure should be assessed.[4]

12. UNRESOLVED ISSUES AND FUTURE RESEARCH

12.1. Early vs. late infection

The classification of VGEI into early and late phases, based on a time cutoff following the index surgery, was determined arbitrarily. This distinction was intended to guide antimicrobial therapy, as certain pathogens were believed to be more prevalent in early *vs.* late VGEI, and to account for differences in biofilm maturity. However, the time window that a vascular graft can be debrided and retained without the need for suppressive antimicrobial therapy still needs to be defined.

12.2. Diagnosis and follow up

The MAGIC criteria require further prospective validation, particularly for their use in extracavity VGEI.[22] The clinical outcomes of the suspected category remain unclear. Several MAGIC criteria rely on clinical judgement; in particular the role of inflammatory markers in VGEI diagnosis and the interpretation of [^{18}F]FDG PET/CT require further study.

Considerable uncertainly arises in the diagnosis and follow up of VGEI because no imaging modality can currently specifically identify infection from sterile inflammation, or even accurately differentiate bacterial from fungal infections in clinical use.[326]

Glucose uptake may also indicate inflammation or malignancy. New tracers such as ^{18}F-fluorodeoxysorbitol are promising in distinguishing inflammation and infection. On the other hand, the development of imaging tools that could directly and specifically localise and target microbial pathogens is highly desirable, as this would improve infection diagnosis and treatment.

Antigranulocyte scintigraphy with SPECT/CT has been developed in order to improve the accuracy of infection diagnosis, especially in implant associated infections, but needs further research to define its utility in VGEI follow up.

12.3. Choice of the best material for vascular reconstruction

While autologous material remains the preferred choice for vascular reconstruction in VGEI, the literature is lacking

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robust comparative data on alternative materials. Alternatives play a crucial role when autologous material is unavailable or when its use would significantly prolong surgical duration and increase procedural risk. This is particularly relevant in complex aortic reconstructions, where the superficial femoral vein could be used but may not always be a feasible option.

Currently, the most frequently considered alternatives include pericardial and carotid grafts, Omniflow II, silver coated grafts, cryopreserved allografts, and conventional prosthetic grafts. While the potential complications of these materials are known, comparative studies assessing their long term efficacy, durability, and resistance to infection remain insufficient. Further research is needed to establish clear recommendations on material selection based on infection severity, anatomic site, and patient specific factors.

Additionally, the development of biocompatible prosthetic grafts with proven infection resistance represents a key area for future innovation. Advances in biomaterials and surface modifications, as well as tissue engineered vascular grafts, could significantly enhance treatment strategies, ultimately improving both early and long term outcomes in patients with VGEI.

12.4. Microbiological diagnosis

The rate of undocumented VGEI remains unacceptably high in recently published studies, ranging from 10% to 30%. This high rate could negatively impact patient outcomes. While sonication has not demonstrated a sufficiently significant improvement in microbiological diagnosis, novel techniques should be evaluated to enhance the performance of conventional microbiological methods, such as tissue grinding.

Molecular biology approaches have also shown limited performance according to the current literature. However, promising new strategies, such as metagenomic sequencing, may offer substantial improvements in the coming years.

12.5. Antimicrobial therapy

12.5.1. Duration of antimicrobial therapy. The guidelines for antimicrobial therapy duration in VGEI are based on low quality evidence. Prospective comparative studies are needed to evaluate different treatment durations, such as 6 weeks *vs.* 3 months, as previously conducted in prosthetic joint infections.[327] Various clinical scenarios should be assessed, as they may influence patient outcomes: complete graft removal *vs.* partial graft removal, surgical debridement of early onset VGEI bacterial *vs.* fungal infections.

The issue of the duration of high dose initial antimicrobial therapy before transitioning to lower dose suppressive antimicrobial therapy remains unresolved.

Finally, the duration of suppressive antimicrobial therapy is also significantly understudied in the current literature. Key questions remain. Should suppressive therapy be continued lifelong? Could it be discontinued under specific conditions, such as prompt debridement of an early VGEI, partial removal of an infected graft or endograft, or early antimicrobial therapy following haematogenous contamination of the graft or endograft through bacteraemia or fungaemia? Additionally, nuclear medicine imaging could potentially assist in making such decisions.

12.5.2. Targeted antimicrobial therapy. Rifampicin has been associated with improved outcomes in VGEI treatment in several retrospective observational studies, particularly for staphylococcal infections. However, prospective studies are necessary to confirm these preliminary findings. Other antimicrobial agents used for targeted treatment are not well defined in the literature and should also be evaluated through future prospective studies.

For fungal VGEI, the optimal duration of initial antifungal therapy, often using echinocandins owing to their superior biofilm activity, and the appropriate timing for switching to triazoles for targeted therapy remains unresolved.

12.5.3. Antimicrobial dosage. No studies have been conducted to evaluate the impact of empirical and targeted antimicrobial therapy dosages on VGEI treatment. Infectious diseases physicians often rely on dosing regimens established for bone and joint infections or endocarditis, without clear evidence on vascular wall or biofilm penetration of various antimicrobial agents. Pharmacokinetic studies are needed to advance knowledge on this crucial topic.

12.5.4. Phage therapy. Further research is required to define the indications and optimal application of bacteriophage therapy. Standardising phage delivery is essential to better assess treatment outcomes and determine whether phages should be administered locally, intravenously, or both.

Additionally, treatment protocols should be clarified regarding the number of phages used, their dosage, duration of administration, and local delivery methods, which should be as minimally invasive as possible.

Currently, available literature suggests that bacteriophage therapy is primarily used as a salvage treatment within a conservative approach to VGEI management. This is mainly due to the logistical challenges associated with obtaining bacteriophages for therapeutic use. However, bacteriophage therapy may also hold promise as a component of curative treatment.

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APPENDIX A. SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ejvs.2026.05.035.

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