Vascular Society of Southern Africa

Vaskulêre Vereniging van Suidelike Afrika

South African

Guidelines on the Management of Aneurysmal Disease

Developed October 2016

Preamble

VASSA has embarked upon a programme of Guideline development to assist South African Vascular Surgeons in the management of Vascular disorders. In 2002, at a meeting convened in Pretoria by Prof J C van Marle, the very first VASSA Guideline Development Meeting was held, with the topic being Aortic Aneurysms. This meeting was held to address the very real difficulty associated with the then relatively recent introduction of EVAR to the South African Vascular landscape. The reluctance of Funders to reimburse for the devices, combined with the enthusiasm with which EVAR was embraced by South African Surgeons, led inevitably to a conflict which threatened the use of EVAR. The problem of a perceived lack of Level 1 evidence, and a complete lack of South African Guidelines were the root causes of this impasse.

After publication of the Guidelines in 2002, Funders realised that the cost of EVAR, while high, was not going to discourage use of the devices, and – inevitably – pressure from patients and surgeons alike led to an increased willingness to fund these procedures. However, not unreasonably, there has been an increasing pressure from Funders on Surgeons and VASSA members to justify the continued use of existing EVAR devices, as well as to justify reimbursement for ever more advanced and expensive devices in the evolving management of Aneurysmal disease.

VASSA and its members have a strong commitment to the practice of excellent Vascular Surgery. VASSA members have always remained at the forefront of Vascular Surgical knowledge, and have thus always been eager to introduce new technology and procedures to the country in order to

improve the standards of care offered to our patients. Combined with this has been an equally strong commitment to seeking the best evidence to support such advances, and a willingness to learn about such procedures from the foremost pioneers of these new technologies.

VASSA is committed to the ideal of Best Patient Care Always but recognises the financial strictures of our working environment. Third party payers may understand the economics of healthcare, but frequently have only a basic understanding of the latest techniques, procedures, devices and standards of care available to treat patients. VASSA believes that the benefits to Patients offered by a co-operative relationship between Surgeons and Funders will be massive. VASSA also recognises that we, as Vascular Surgeons "prescribing" expensive care to patients, have a responsibility to practice due diligence, and thus to practice within boundaries which are acceptable to all involved parties. The generation of evidence based Guidelines fulfils all the requirements of this mandate.

The intention is that this Guideline should be added to and updated in the future. Sections which will be added include Takayasu's Disease, and a stetement concerning Training, Facilities and Accreditation.

VASSA is thus proud to present the following South African Guideline on the Management of Aneurysmal Disease.

James Tunnicliffe

Martin Forlee

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Jakkie Odendaal

(Editorial Committee)

The Process of Guideline Development.

As a result of lessons learnt in the generation of previous Guidelines, it was decided to trial a different process to generate this Guideline.

The Editorial Committee drew up a list of topics to be covered in the Guideline. These topics were distributed to the allocated Authors. A deadline for the return of the paper for each topic was set three months prior to the actual Guideline Meeting. The Authors were given 3 months to research their allocated topics, write the paper, and prepare their slide presentations. The papers and presentations were then collated by the Editorial Committee, and the presentations inspected to ensure that there was no significant overlap of information. Any concerns at this stage were raised with the author(s) concerned and the required changes made.

The Authors were instructed to use the matrix shown in Fig. 1 to draw up their Guidelines on each topic according to their reading of the available evidence.

The Editorial Committee then perused the evidence as provided by the Authors in their respective Bibliographies. Once satisfied as to the scientific rigour, the Editorial Committee informed the Authors accordingly.

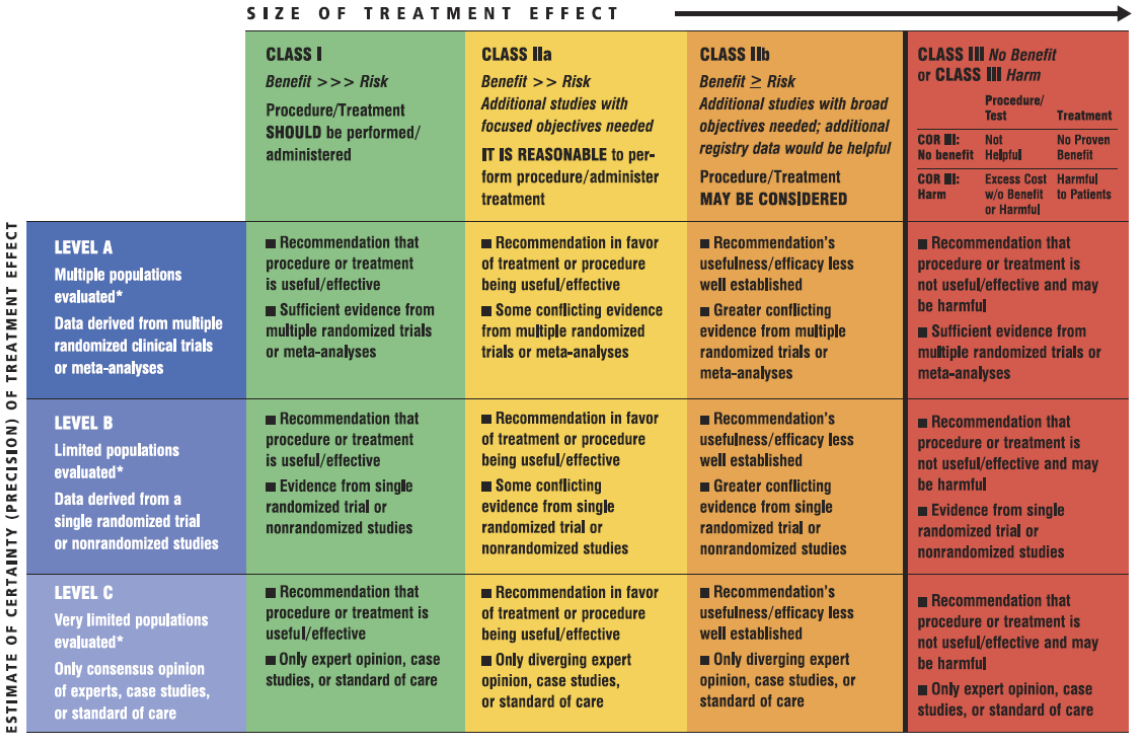

Fig 1. Class of Recommendations and Levels of Evidence[1]

The actual Guideline Development required the input of all interested parties. To that end the draft papers and bibliographies were distributed to all the delegates 2 weeks before the meeting at which the papers would be discussed. This was to give all the delegates the opportunity to do any necessary research to drive discussion on any of the included topics. This was considered an essential step in order to allow all relevant evidence to be considered, and was central to the success of the process, and thus the credibility of the resulting Guideline.

The meeting at which the papers were all presented and discussed took place without the presence of any representatives of the Medical Device industry or Medical Funders in order to remove any possibility of bias or reticence in the discussion of the Guidelines proposed. Each paper was discussed, the levels of evidence examined (and changed by agreement of all delegates where

relevant). The completed Guidelines were then edited after the meeting to reflect any and all changes made to the papers at the meeting. This process took somewhat longer than expected as a result of unexpected formatting difficulties relating to the initial documents presented to the Editorial Committee.

The end result is the following Guideline, complete with the bibliographies employed by all the Authors. The Editorial Committee believes that this Guideline is a definitive document that can be referred to by Vascular Surgeons, other Health Care professionals, Funders, Patients and other interested parties looking for guidance in the management of Aneurysmal Disease in South Africa. The Guideline is not an exhaustive document – it would simply not be possible to produce a detailed Guideline covering absolutely all minutiae of Aneurysmal disease in a reasonable time period. This Guideline does not seek to supplant other respected International Guidelines either, but it certainly does aim to provide a uniquely South African perspective for South African users of it.

The Authors (all of whom gave up their valuable time to develop this Guideline) are responsible for the quality of the Guidelines written, and the Editorial Committee and VASSA thank them for their efforts.

Small abdominal aortic aneurysm

Nadraj G Naidoo

An abdominal aortic aneurysm is defined as a permanent focal dilatation of the abdominal aorta. The following related definitions apply:

- Sub-aneurysmal abdominal aorta: 25mm-29mm in diameter
- Small abdominal aortic aneurysm: 30mm-54mm in diameter
- Medium abdominal aortic aneurysm: 40mm-54mm in diameter (this represents the group that many felt would benefit from repair and were subsequently randomised in small AAA trials)
- Large abdominal aortic aneurysm: 55mm or larger in diameter

This section will focus on the management of small abdominal aortic aneurysms (AAA) i.e. AAA < 55mm in diameter.

Epidemiology and screening:
Dominant risk factors associated with AAA include male gender, smoking and family history. Other compelling associated risk factors include advancing age, high cholesterol levels, coronary artery disease, any atherosclerosis viz. peripheral arterial disease, and hypertension.[1] Obesity is a weak risk association for AAA. Caucasians are more affected than other ethnic groups. Female gender and diabetes are negatively associated with AAA. However, females with AAA tend to be 10 years older than their male counterparts. These AAAs tend to rupture at smaller diameters and have higher case fatalities with ruptures. Approximately 90% of AAA are degenerative (non-specific) in aetiology and infra-renal in location. No compelling data exists for small “atypical” AAA viz. infected AAA; HIV-associated AAA; Intimo-medial mucoid degenerative AAA; etc. Growth of the AAA is related to diameter, and averages ~ 2-3mm per annum for small AAA

The most compelling complication of AAA is rupture with an overall mortality of ~ 80%. Approximately 40% of patients will receive treatment with an estimated 50% case fatality. Rupture risk relates exponentially to AAA diameter > 55mm (rupture risk of 10% or more per annum). Considering that ~ 70% of ruptured AAA were previously undetected, and that the operative mortality for elective repairs ranges from 3% - 5%, there is considerable value in screening for AAA in patients at risk.

Screening
Early screening programmes (randomised and non-randomised trials) have been shown to be safe, feasible, cost-effective and have reduced AAA-related mortality consistently. [5,6,7,8] Approximately 60% - 70% of patients on surveillance in screening programmes, however, will require intervention in 3-5 years. The U.S. Preventive Task Force estimated that AAAs affects 3.9% to 7.2% of men and 1.0% to 1.3% of women aged 50 years or older. [23] Contemporary screening programmes document a decreasing prevalence of AAA, which may reflect decrease in risk factors such as smoking, statin use, etc.: 1.8% for the NHS AAA screening programme [10] and 1.7% in the Swedish AAA screening programme. [11]
A more recent study reported screening of individuals with cardiovascular disease (CVD) risks (patients undergoing coronary angiogram; requiring DUS for carotid or peripheral arterial disease; etc.) employing various screening strategies may be more feasible in parts of the world were national screening programmes do not exist. [9]

Screening of sub-aneurysmal aorta

A recent multicentre observational study identified 1696 patients, with sub-aneurysmal aorta, in eight screening programmes in Europe (prevalence of 2.1% in 65 year old males). They reported that 67.7% developed a AAA in 5 years (0.9% had a diameter of 5.4mm) and 26.2% developed a AAA > 5.4mm in diameter at 10 years.[2] In a recent Swedish study ~ 53% (21/40) of patients 65 years or older with sub-aneurysmal aorta developed AAA after 5 years; none were > 5.4 mm in diameter and there were no AAA events recorded. [3]

A few individuals (<0.2%) of screening detected abdominal aorta < 25mm in diameter will develop AAA over 13 years or more, and may confound screening viability programmes with late ruptures. [4, 5]

Identification of small AAA represents an opportunity to optimise cardiovascular risk. Smoking and high blood pressure are independently associated with an increased risk of AAA growth and rupture.

Repair of Small AAA

The rupture risk of a small AAA is ~ 0.6% -1% per annum. Considerable controversy exists regarding the optimum treatment of small AAA between 4.0cm and 5.4cm in diameter. Two options exists: immediate repair vs. deferred repair i.e. surveillance and repair when threshold diameter for intervention is reached (5.5cm). Two randomised trials (RCT) compared immediate open repair to surveillance. [12,13,14, 15,16,17] Two recent studies also compared endovascular aneurysm repair (EVAR) of small AAA to surveillance. [18.19.20]

These four good quality RCTs reported an early survival advantage in favour of surveillance because of the high 30 day operative mortality rate in the immediate repair arm. However these RCTs did not report any meaningful survival advantage between the surveillance and immediate repair arms during the three to eight year follow-up period. A pooled analysis of the two RCTs comparing immediate repair to surveillance demonstrated that neither patient age nor aneurysm size between 4.0 and 5.4cm, altered clinical outcomes. [21]

A recent Cochrane review of all four RCTs failed to show benefit of immediate repair vs. surveillance. There were conflicting results regarding quality of life. The authors concluded that neither immediate open surgical repair nor immediate EVAR can be supported based on current evidence. [22]

Surveillance protocols

The UK MASS trial recommended annual surveillance scans for 3.0 – 4.4 cm screening detected AAAs. They also recommended three monthly surveillance scans for 4.5cm – 5.4cm AAAs. Referral to a vascular unit was recommended when the AAA diameter reached 5.5 cm, aortic expansion was 10mm or more in one year or when symptoms attributable to the AAA developed. [24] Other recommendations have also evolved over time. [25.26]

Rescreening for those with original normal aortic diameters is not encouraged based on the low yield.

Future directions:

All things considered it is very unlikely that a national screening programme for AAA will be feasible in South Africa. Our experiences with management of AAA in Caucasian patients are similar that reported in the Western literature and AAA guidelines from these countries are likely to be appropriate here. While, anecdotally, we see more AAA in the mixed ethnic patients compared to other non-Caucasian ethnic groups in the Western Cape, screening in

this group remains to be defined. Aneurysm screening in black, Indian, Asian or HIV positive patients remains to be defined.

Recommendations:

1. Screening for AAA is recommended for the following:
    a. 65 – 75 year old Caucasian men who ever smoked (**Class IIa, Level A**)
    b. < 65 year old Caucasian men at high risk (cardiovascular disease; peripheral arterial disease; family history) (**Class IIa; Level B**)
    c. Women >65 years, with first degree family history of AAA. (Class IIa, Level B)
    d. Screening for other ethnic groups or in females cannot be supported based on current evidence (Class IIb, Level B)
2. Medical treatment
    a. Smoking cessation strategies must be implemented (Class I; Level B)
    b. Tight blood pressure control needs to be maintained (Class I; Level B)
    c. Weight loss should be encouraged in obese patients (Class IIb; Level B)
    d. Statins and antiplatelet agents must be prescribed in all at risk patients (Class IIa; Level B)
    e. Roxithromycin; Doxycycline and other novel medications cannot be recommended based on current evidence (Class III; Level B & C)
3. Surgical repair of small AAA, 4 – 5.4 cm in diameter, cannot be recommended currently (**Class I; Level A**)
4. Endovascular repair of small AAA, 4 – 5.4 cm in diameter, cannot be recommended currently (**Class I; Level A**)
5. Surveillance Protocol (Class IIa, Level B)
    a. Aorta diameter <25mm; No surveillance, Discharge.
    b. Aorta diameter 25 – 29mm repeat ultrasound at 5 years.
    c. Aorta diameter 30 – 44mm, repeat ultrasound 2 yearly
    d. Aorta diameter 45 – 49mm repeat ultrasound annually
    e. Aorta diameter 50 – 54mm, repaet ultrasound 6 monthly
6. Rescanning for original normal aortic diameters (at 65 years baseline) cannot be supported based on current evidence
7. Indications for repair of AAA on surveillance:
    a. Onset of symptoms or complications
    b. AAA diameter of 5.5 cm or larger
    c. Eccentric saccular AAA diameter > 3cm
    d. Common iliac aneurysm diameter > 3cm
    e. Rapid AAA growth:
        i. > 5mm increase in diameter in six months
        ii. >10mm increase in diameter in one year

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Preoperative work up and planning

Jay Pillai

History and physical examination

The risk of developing an aneurysm is high in those with a positive family history and smokers. The risk is lower in diabetic patients, African Americans (can be extrapolated to Africa) and diabetic patients.

The risk of rupture is increased by persistent smoking, COPD, female gender, hypertension and in transplant patients. Previous abdominal surgery may influence the choice of aneurysm repair.

An abdominal aneurysm may be present in 60% and 80% of patients with popliteal and femoral aneurysms, respectively. Patients with abdominal aneurysms have a 15% chance of having either a femoral or popliteal aneurysm. Clinical assessment of the femoral and popliteal arteries is therefore recommended (**Class11b, Level B;**).

Co Morbid Diseases

Cardiac Disease

Open repair is associated with a higher risk of cardiac events and mortality. EVAR should be considered in all patients with an estimated cardiac risk of between 3% and 7% (**Class 2b, Level B**) .The presence of active cardiac conditions and functional capacity should be assessed in all patients (**Class 2b, Level B**). Active cardiac conditions (unstable angina, cardiac failure, significant arrhythmias) should be treated prior to EVAR (**Class 2b, Level B**). Non - invasive stress testing should be considered in high risk patients if it is felt that it will change operative strategy or outcomes (**Class 1, Level B**). A 12 lead ECG is recommended in all patients and an echocardiogram in high risk patients (**Class I, Level B**). Coronary revascularization should be considered prior to EVAR in high risk patients (acute ST elevation MI, unstable angina, triple vessel or main stem disease) (**Class 2b, Level C**). β-Blockers, statins and aspirin are recommended perioperatively (**Class 2b, Level B**).

Pulmonary Disease

Aneurysm prevalence is higher in patients with COPD. Smoking is linked to prevalence, expansion rates and rupture. COPD increases the perioperative mortality and morbidity of open surgery (**Class 2b, Level B**).

Lung function tests are indicated prior to EVAR (**Class 2b, Level C**).

Smoking cessation is recommended for at least 2 weeks prior to EVAR (**Class 2a, Level C**)

Bronchodilators are indicated for 2 week prior to EVAR (**Class 2a, Level C**)

Renal Dysfunction

Preoperative renal dysfunction increases morbidity and mortality after open surgery and after EVAR. Severe renal dysfunction in EVAR patients is associated with a greater length of hospital stay, congestive cardiac failure and organ dysfunction. Perioperative hydration appears to be beneficial but the exact volume, type of fluid and timing of fluid administration appears to be uncertain. The use of multiple strategies to decrease the risk of progressive renal dysfunction or renal failure appear to be beneficial in patients undergoing open surgery and may be extrapolated to patients undergoing EVAR. Contrast induced nephropathy is defined as a 25% increase in the serum creatinine levels. In patients undergoing EVAR with

severe renal impairment multiple strategies may be indicated. These include adequate perioperative hydration with normal saline, administration of Vit C, n acetyl cysteine; mannitol and fenoldapam (**Class 2b, Level B**). Angiotensin receptor blockers and angiotensin converting enzyme inhibitors should be avoided on the day of the procedure and recommenced the following day after adequate volume replacement (**Class 2b, Level B**). There appear to be no difference in outcomes when low osmolar and iso– osmolar contrast is used. Using a low volume of contrast is recommended (**Class 2a, Level B**).

Diabetes

Diabetes appears to have an early effect on mortality and morbidity in patients undergoing EVAR. Diabetes may represent a marker for others comorbidities and glucose control in the perioperative period of less than 10 mmol /dl is recommended (**Class 2b, Level B**).

Ultrasound as a screening modality detects the presence of an aneurysm with a sensitivity and specificity that approaches 100%. It is however Imprecise in measuring aneurysm size and growth rates. CT is more reproducible than ultrasound and it is the standard modality for operative planning.

CT imaging generally assists in predicting aneurysm rupture. Although aneurysm diameter represents the most important parameter to determine risk of rupture, some limitations exist as 13% of aneurysms less than 5 cm have been associated with rupture. It is recommended that patients should be assessed individually and multiple parameters be assessed. These include aneurysm shape, wall stress, expansion rate; sac thrombus, diastolic blood pressure and wall stiffness changes. Other patient related factors may also need to be considered (COPD, use of anticoagulants, uncontrolled HT, and gender. Anatomic suitability for EVAR in the future versus age and life expecting may also be considered (**Class11b, Level B**).

AAA Elective: EVAR vs OPEN

Philip Matley

Endovascular Aortic Aneurysm Repair (EVAR), first introduced in 1990 was initially used for patients considered unfit for open repair but in most vascular centres has now become the procedure of choice for all patients requiring intervention, who have suitable anatomy, unless they are extremely unfit or have a life expectancy of less than two years, in which case expectant management is usually recommended.

The pre-requisties for safe EVAR are suitable skills, training & experience; a suitable facility including excellent imaging systems; a range of endografts and a reasonable case load.

EVAR has been compared to open surgical repair (OSR) in four randomised trials as well as a large non-randomised Medicare study:

The EVAR 1 study [1,2] randomised 1252 Patients between 1999-2004 with a 30 day mortality of 1.8% for EVAR versus 4.3% for OSR, a 2.4 times survival advantage. There was however no difference in total mortality or aneurysm related mortality in the long term. There was a “catch up” of total mortality by 2 years and AAA related mortality by 6 years. More re-interventions were recorded in the EVAR group and the authors considered EVAR to be more costly. They concluded that EVAR was not superior to OSR. However, endovascular practice has changed significantly since 2004. By today’s standards the endovascular repairs in EVAR 1 were performed by relatively inexperienced operators using old technology with out-dated secondary treatment and out-moded criteria for re-intervention.

Many endograft problems including migrations with Type I endoleaks and disconnections were left untreated before rupture occurred [3]. Of the 22 EVAR patients with late rupture, 17 had recognised problems that were not treated and 15 had increasing sac expansion but no intervention. The 22 ruptures in 626 EVAR patients in EVAR 1 compares very unfavourably with modern registry data including an experience of 1 rupture in 974 EVAR procedures recoded [4]. 288 “complications” were noted in the EVAR group vs 66 in the OSR group but all endoleaks were considered as complications notwithstanding the fact that 156 of 288 were type II, the vast majority of which would be considered benign today. No data was provided on readmissions for bowel obstruction, incisional hernia or wound complications after OSR and the costs of laparotomy complications in the long term were not considered. A large number of CT scans used for surveillance in the EVAR group contributed to the high costs, most of which would be considered to be unnecessary today. The conclusions of the authors that EVAR is associated with more complications, more re-interventions and greater cost must be questioned in the light of current practice and the evidence from more recent randomised trials.

The DREAM trial [5] randomised 351 Patients between 2000-2003, recording a 30 day mortality of 1.2% for EVAR and 4.6% for OSR. Two year survival rates were 89.6% and 89.7% respectively with aneurysm related deaths being 2.1% for EVAR and 5.7% for OSR. During the 2 year follow-up, 7.8% of OSR patients required surgery for incisional hernias. Although there was a clear early survival benefit, the authors concluded that there was no long-term survival advantage for EVAR over OSR.

The OVER Trial [6] randomised 881 Patients between 2002-2008. EVAR was performed with a 30 day mortality of 0.5% compared to 3.0% for OSR. The survival advantage of EVAR was maintained to 3 years with AAA related deaths of 2.3% in the EVAR group versus 3.7% in the OSR group. Survival was significantly better with EVAR if the patient was younger than 70 yrs. 148 secondary Interventions were required in 98 EVAR patients (9 conversions; the rest endovascular) whereas105 procedures were required in 78 OSR patients including 48 incisional hernia repairs, 11 laparotomies for bowel obstruction, 7 amputations and 4 wound procedures. The authors concluded that the outcomes of EVAR are improving with a survival benefit to 3 years for EVAR and a similar rate of re-interventions for both procedures.

The ACE Trial [7] randomised 299 low-risk fit patients between 2003 – 2008. The 30 day mortality was 0.6% for EVAR versus 1.3% for OSR with a 3-year survival rate of 82.1% and 85.1% respectively. Re-interventions were reported in 16% of the EVAR group and 2.4% of OSR group but re-interventions for incisional hernia or bowel obstruction were not included. Minor complications were similar. EVAR was not considered to be superior to OSR in this study with an aneurysm related mortality of 4% for EVAR and 0.7% for OSR.

The large Medicare study [8] retrospectively analysed 22 830 matched patients between 2001 – 2004. The 30 day mortality was 1.2% for EVAR and 4.8% for OSR with convergence of the survival curves at 3 yrs. Re-interventions were required in 9% of the EVAR group but laparotomy complications were recorded in 9% of the OSR patients.

A 2014 Cochrane meta-analysis [9] concluded that EVAR is associated with significantly better 30-day mortality rates; no difference in long-term survival beyond 3 years; no difference in long-term AAA related mortality; no difference in complications (including strokes and renal impairment); no difference in health related quality of life (HRQoL) or sexual dysfunction and a higher incidence of pulmonary complications with OSR. Although there is a higher risk of re-intervention for EVAR, this usually involves a further endovascular procedure. Re-interventions following OSR usually require repeat laparotomy or incisional hernia repair. The authors suggested that there is probably no difference in costs, with any costs of re-intervention following EVAR being balanced by the shorter operative times, reduced transfusion and ICU requirements and the costs of treatment for laparotomy-related complications in OSR patients.

The importance of strict compliance with the instructions for use (IFU) of the various endografts has been emphasized by several authors. In a study of 10 228 patients post EVAR in in the USA [10], a breach of the IFU correlated strongly with sac enlargement and risk

of rupture. OSR is likely to be recommended in patients who are fit enough for this if the vascular anatomy is unfavourable for endovascular treatment.

Studies comparing the costs of EVAR versus OSR have reported conflicting results relecting the vast differences in the health systems of various countries. Although EVAR1 suggested higher cost for EVAR this was not the case in the OVER study or the Medicare review. Two cost studies from Canada [11] and Ireland [12] have both concluded that EVAR is cost-effective. A South African cost study from the Discovery Health Medical Scheme data base presented to the Vascular Society of Southern Africa by Matley in 2015 [13] comprised 496 patients undergoing elective AAA repair between 2010 and 2014. No significant cost difference was demonstrated with total costs being R230 629 for EVAR versus R234 392 for OSR. The costs of long-term follow-up or re-interventions beyond the first hospital admission were however not included.

Summary

Four randomised trials and a large Medicare review have uniformly demonstrated a highly significant reduction in 30-day mortality for EVAR versus OSR. With modern devices and increasing endovascular experience world-wide, 3-year survival rates appear to be as good or better with EVAR than OSR. Complication and re-intervention rates are similar for the two procedures with re-interventions for EVAR patients usually involving an endovascular procedure rather than open surgery. EVAR appears to be no more expensive than OSR and this would appear to be true in South Africa as well. In general, EVAR is highly patient acceptable when compared to OSR and is the usual treatment recommended by specialist vascular surgeons for patients requiring intervention, as long as the individual vascular anatomy allows strict compliance with the IFU for available endografts. For patients who do not have ideal anatomy for EVAR and who are considered to be fit enough to undergo open surgical repair, this should be preferred over endovascular treatment.

Recommendations

1. **EVAR is associated with a significantly lower 30-day mortality when compared to open repair (Level A evidence).**

2. **The long-term differences between EVAR and OSR in terms of re-intervention rates and survival are small. Patients requiring intervention who have suitable anatomy for EVAR can be offered endovascular repair as the preferred method of treatment. (Class IIa, Level A)**

3. **OSR is recommended for surgically fit patients with anatomy unsuitable for EVAR, or anatomy that falls outside device IFU (Class IIa, Level A)**

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EVAR for Ruptured Abdominal Aortic Aneurysm

James Tunnicliffe

Introduction:

EVAR was first introduced for the management of Infra-renal Abdominal Aortic Aneurysms in the 1990s with the first EVAR for Ruptured AAA (rEVAR for rAAA) carried out in 1994 [5,6]. Over the years applicability of EVAR has increased, technology and devices have improved, and additional devices have become available to extend the applicability of EVAR[1-3, 5-7]. It has been shown that the immediate morbidity and mortality of AAA repair was significantly reduced compared with open repair[7-16]. As a result, rEVAR has been considered in the hopes that the appalling morbidity and mortality of rAAA repair would decrease with EVAR [1,2].

Evidence:

There are four RCTs of EVAR for rEVAR (a – d), and one large Meta-analysis (e).

a) Nottingham Trial (2006) [10]

Single Centre RCT run over 26 months; well defined exclusion and inclusion criteria. First generation devices and no Hybrid room were significant limitations.

Well matched groups of OSR and EVAR patients. 103 suspected rAAA during study period were followed. 71 were NOT randomised – usually because of haemodynamic instability, lack of consent, or logistics failure. 32 were randomised – 17 to OSR, 15 to EVAR. Of the EVAR group, 1 died before surgery, 14 underwent CTA, and 1 of these was not anatomically suitable for EVAR. Of the 13 EVAR commenced, 1 was converted to OSR, 1 was converted to Axilli0Bifemoral graft, leaving 11 completed EVAR. Of the 17 randomised to OSR, 3 died before surgery. 15 (including the one crossover from EVAR) underwent OSR. 2 of these were abandoned, and 1 was converted to Axillo-Bifemoral graft, leaving 12 completed OSR.

Results: EVAR and OSR mortality and Morbidity were not statistically different. EVAR group had lower blood loss, lower autologous blood requirement, and lower colloid requirements.

b) AJAX Trial (2007)[11]

Designed to assess suitability of EVAR to rAAA repair, and applicability of rEVAR to the population. Multicentre RCT based in Holland.

Results: 256 consecutive patients referred to 3 Trial Centres
128 patients – other diagnosis
128 rAAA — 23 treated at referring hospital
105 admitted to Trial Centre
19 Unstable: OSR
83 CTA: 45 unsuited to EVAR
38 EVAR
Suitability for EVAR: 45.8%
Applicability (Amsterdam) 29.7%
(Specialist) 35.5%
Exclusion: 82% because of neck unsuitability
Limitations of Study: All Aorto-Uni-iliac devices and no mortality/morbidity outcomes reported

c) IMPROVE Trial (2014)[13]

Multicentre RCT, 29 in UK, 1 in Canada. Random allocation to EVAR (n=316) or OSR (n=297), with Primary outcome of 1 year all cause mortality, and secondary outcomes of QoL, Cost Benefit and time to first re-intervention.

	EVAR	OSR
1 yr all cause mortality	41.1%	45.1%
1 yr AAA mortality	33.9%	39.35
Re-intervention d31 – 1yr	4.2%	3.7%
Mean LOS	17d	25d

There was a trend toward benefit of EVAR in females, but no overall difference.

QoI scores favoured EVAR at 3 and 12 months, and Cost and QALY benefit of EVAR over OSR was £3877

d) ECAR Trial (2015)[12]

Multicentre (14 centres) RCT of EVAR vs OSR. Well defined inclusion and exclusion criteria. 100% data and trial protocol compliance.

Primary endpoint of 30d mortality

Secondary endpoints of 30d post-operative morbidity, length of ICU stay, blood transfusion requirements, in-hospital deaths, and morbidity/mortality rates at 6 months and 1 year. All rAAA NOT randomised during trail period were recorded in a parallel registry; Cost analysis carried out on all patients.

Results: 524 patients over 5 year trial period. 107 patients (32.8%) randomised (56 EVAR, 51 OSR). Registry of 116 EVAR and 301 OSR during same period.

Of trial patients, groups well matched, with only delay to surgery being significantly different, favouring OSR over EVAR. The causes of death were similar in both groups, with a trend towards fewer complications in the EVAR group. There was a statistically significant reduction in blood transfusion requirements, Pulmonary complications and ICU stay in the EVAR group. Composite M&M rates at 30 days, 6 and 12 months were not statistically different. After case-mix correction there was no significant cost difference between the groups.

e) Meta-Analysis (2014)[17]

135734 patients spread over 2 RCT, 5 prospective cohort studies and 11 retrospective studies, looking at 30d mortality and length of stay.

Results: rEVAR patients had significantly lower peri-operative mortality (OR=0.62, 95% CI = 0.58 – 0.67, $p<0.001$) and had significantly shorter hospital stay (-2.0 to -19.10 days: 95% CI = -9.23 t0o -1.26, $p=0.10$)

GUIDELINES for use of EVAR in Ruptured Abdominal Aortic Aneurysm

1. EVAR for rAAA is as good as OSR for anatomically suitable AAA

Level of Evidence	**A**
Class of Recommendation	**IIa**

2. rEVAR may show benefit – reduced Blood requirements, reduced complications, reduced LOS

Level of Evidence	**A**
Class of Recommendation	**IIb**

3. OSR for rAAA by experienced surgeon is better than rEVAR by inexperienced operator

Level of Evidence	**B**
Class of Recommendation	**IIb**

4. OSR and EVAR for rAAAshould ideally be done in Centres of Excellence

Level of Evidence	**A**
Class of Recommendation	**IIa**

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Ch-EVAR, FEVAR, B-EVAR

Pradeep P. Mistry

The use of complex endovascular technologies to treat aneurysms involving the visceral and iliac vessels has increased in popularity in the last 10 years. A variety of these technologies are now available in South Africa. These techniques can be broadly classified as fusing devices (i.e. branched or fenestrated repairs) or layering of devices (i.e. chimney or sandwich repairs).

The Chimney EVAR (Ch-EVAR)

The chimney technique was intended as a technique to raise the proximal sealing zone allowing the endovascular device to be used for juxtarenal(JAA) and pararenal (PAA) aneurysms. It was also later used as a bailout procedure for inadvertent renal vessel coverage during EVAR(1). It can also be used to extend the proximal sealing zone in order to allow treatment of Type 1a endoleaks. The best evidence for Ch-EVAR comes in the form of two meta-analysis which report 10.7% (10/93) and 10.2 % (24 /234) early endoleak and 4.3% and 3.4% early mortality(2,3). In 2015, a meta-analysis of 236 patients showed a the type I endoleak and mortality rates of 11.8% and 13% respectively after a mean follow up of 12 months(4). Despite good feasilbility studies and early results concerns exist over longer term outcomes with particular emphasis on stent thrombosis and reinterventions for endoleaks.

Ch-EVAR is reasonable option for the management JAA and PAA endovascular repairs, in situations where F-EVAR would cause unacceptable cost, manufacturing delays, or tortuous anatomy deemed unsuitable for F-EVAR, or in patients deemed not fit for OSR. **(Class IIb, Level C).** Currently an IFU exists for ChEVAR for the Endurant (Medtronic) device only. These procedures can be technically challenging, particularly in emergency settings and when performed by inexperienced operators.

Fenestrated EVAR (F-EVAR) and Branched EVAR (B-EVAR)

Clinical studies coupled with an appreciable number of years of clinical use, have demonstrated the various benefits of F-EVAR as compared to conventional open surgical repair in high risk patients with para and juxtarenal aneurysms. These benefits encompass significantly decreased morbidity and mortality rates, and significantly decreased intensive care and in-hospital times as described in detail in a review conducted by Health Quality Ontario in 2009(5).

In a systematic review of a cohort of studies F-EVAR had lower 30-day mortality than open repair (1.4% vs. 3.6%) and a lower late-mortality.(6). Primary visceral vessel patency was 96.6% for F-EVAR (823/852 vessels), decreasing to 92% at 1 year (423/460 vessels)(6). Two meta-analysis evaluating F-EVAR results have included 660 and 629 patients respectively. The 30-day pooled proportion mortality was 2% and 2.1% .Target vessel patency rates ranged from 90.5 to 100%, whilst type I/III endoleaks ranged between 3 and 4-6% (7,8). Durability of branches in fenestrated and branched endografts has been thoroughly assessed by Mastracci et al. (19) on 650 patients. At a mean follow up of 3 years, only 30 (1.7%) target vessel stent occlusions were reported. Kaplan-Meier estimated freedom from reintervention at 5-years was 89% (9).

Despite the absence of RCT literature exhibits solid data regarding feasibility, early, mid- and long-term outcomes after F-EVAR. FEVAR is the procedure of choice for the management JAA and PAA endovascular repairs in patients high risk for open repair **(Class I, Level B).**

FEVAR should only be practiced by centres with the necessary experience of EVAR and complex aortic interventions. (**Highly recommended**)

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Iliac Artery Aneurysms (IAA) – Management Including Iliac Branch Devices

B Dube

The definition of iliac artery aneurysms (IAA) varies widely in the literature but some consensus guidelines suggest 18mm as the cut-off. IAA is classified as aorto-iliac or solitary with the former being the most frequently observed. Their natural history has not been prospectively studied but cohort studies have shown virtually no rupture with aneurysms less than 4cm with most ruptures occurring above 7cm. Rupture is associated with significant morbidity and mortality (up to 40%), therefore an early diagnosis and treatment is crucial. Most authors recommend repair of IAA when symptomatic, complicated or if they attain a size of 3-4 cm.

Ultrasound scan is a useful initial assessment modality and also for surveillance of small IAA. On the other hand, Computed Tomographic Angiography (CTA) is essential for pre-treatment planning. Endovascular iliac aneurysm repair (EVIAR) is now the preferred modality for treatment of most IAA. Retrospective comparative studies have shown less morbidity and mortality with similar short term durability to open repair.

Internal iliac artery (IIA) embolisation and coverage are a recognised adjunct to EVIAR but bilateral IIA loss may result in devastating pelvic and spinal cord ischaemia in high risk subjects. Most societal guidelines recommend preservation of at least one IIA as routine and deviation from such may be done on an individualised basis. There are various techniques for IIA preservation which include, the Bell-bottom technique, the Sandwich technique, the Trifurcation and Hybrid techniques. Such methods are cheaper and have high (> 80%) initial technical success rate but are limited by higher endoleak rates and lower iliac limb patency rates in cohort studies.

The iliac branch device (IBD) and iliac branch endo-prosthesis (IBE) have been developed to improve IIA preservation during EVAIR. These are highly specialised components with various anatomic criteria that limit suitability. Registry data and short term cohort studies have shown equivalent technical success rates to previous techniques but with associated lower endoleak and iliac limb occlusion rates.

1. Definition

1.1 The definition of an IAA is controversial but previous reporting standards suggest that a common iliac aneurysm (CIA) is that > 18mm in diameter. This definition is generally applied to all the iliac vessels. **(Class IIa, Level B)** [1,2]

2. Screening for IAA

2.1 Aorto-iliac and Solitary IAA have a low prevalence and thus routine screening is not justified. **(Class IIb , Level C)** [3,4]

3. Diagnostic Modality

3.1Ultrasound scan can be utilised as the initial diagnostic modality and for follow-up of small IAA. This modality has good correlation with Computed tomography angiography (CTA), but is of very little use in pre-treatment planning. **(Class IIb, Level C)** [3,4]

3.2 CTA is the most essential modality for pre-treatment planning. It is used to evaluate the abdominal aorta as well as assessment for bilateral involvement. **(Class IIa, Level B)** [5,6]

4. Indication for repair of IAA

4.1The average diameter of reported ruptured IAA is 7 - 8cm and virtually no rupture has been reported at diameters of 3.8 – 4cm. Based on this natural history, IAA should be repaired when they attain a diameter of 3 - 4cm ,when symptomatic or complicated. **(Class IIa, Level B)** [7-9]

5. Modality of Treatment

5.1 Endovascular iliac aneurysm repair (EVIAR) has emerged as the preferred modality of treating IAA as it is associated with lower morbidity and mortality and has equivalent short term durability to open surgery. **(Class IIb, Level B)** [10-13]

5.2 Open surgery is the preferred modality for treating ruptured IAA in unstable patients or when immediate relief of compressive aneurysm effect is required. **(Class IIb, Level C)** [14]

5.3 During open surgery for AAA, associated CIAA > 2.5cm or 1.8 – 2.5cm in patients with good life expectancy (> 8 years), simultaneous iliac aneurysm repair is recommended. **(Class IIa, Level B)** [15,16]

6. EVIAR Considerations

6.1 Stent graft repair for IAA is feasible if there is at least 15mm proximal and distal landing sealing zones. **(Class IIb, Level C)** [19,20]

6.2 In cases where the EIA distal landing is required, the IIA origin should be embolised as opposed to simple stent-graft coverage. In such cases, embolisation of the IIA origin with preservation of outflow vessels is essential to minimise pelvic ischaemia.**(Class IIb, Level C)** [19,20]

6.3 When treating IAA, it is reasonable to preserve at least one IIA to minimise the sequelae of pelvic ischaemia especially in high risk groups. **(Class IIb, Level C)** [21]

6.4 In the absence of high risk features, both IIA can be sacrificed in the management of complex IAA. High risk features include; previous thoraco-abdominal aortic aneurysm repair, contralateral IIA stenosis/occlusion, impaired collateral circulation from inferior mesenteric artery and profunda femoris arteries. The outcome of such a procedure is not affected by staged or simultaneous IIA embolisation. **(Class IIa, Level B)** [22,23]

6.5 Currently available techniques of IIA preservation include; Bell-bottom, Sandwich, Trifurcation and Hybrid techniques. The iliac branch devices (IBD) and iliac branch endo-prosthesis (IBE) are associated with equivalent technical success, lower iliac limb occlusion and lower type 1b endoleaks as compared to the former techniques. **(Class IIb, Level B)** [24-28]

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Post EVAR Follow-up

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Various studies have shown that complications continue to arise for at least 8 years after EVAR procedures[1,2].

The principal concerns during the follow-up period are graft-related endoleak, aneurysm enlargement and migration of the stents at the aortic and iliac landing zones, and modular disconnections. Methods for surveillance are plain radiography (AXR), duplex ultrasonography (DU), contrast-enhanced computed tomography (CTA) and magnetic resonance imaging (MRI).

Plain XRays
Standard XRays are easy obtainable and using AP and lateral views are very accurate in assessing for stent fractures, modular disconnections and device migration. Plain XRays, however, are not a stand-alone modality in follow-up as they cannot assess for leaks and increasing aneurysm diameter[5].

Colour Duplex Ultrasonography
Colour duplex ultrasonography (CDU) and contrast enhanced duplex ultrasonography (CEUS) have been shown to be effective in identifying endoleaks and increasing aneurysm diameter[6]. Studies have shown that they are as accurate in this regard as compared to CT angiography[6,7]. Based on the lack of information about stentgraft integrity and migration, CDU and CEUS is not a stand-alone follow-up modality for surveillance after EVAR.

CT Angiography
CTA with delayed images is the most widely used modality for follow-up after EVAR and currently the best method for detecting endoleaks. CTA is the gold standard for measurement of the AAA diameter. The Eurostar study[8] suggested that delayed-phase CT with 3 mm slices was probably the best technique to demonstrate collateral reperfusion. The major concerns of the frequent use of CTA are contrast agent-induced nephrotoxicity [9], cumulative amount of
exposure to ionizing radiation with potential lifetime cancer risk [10], and cost. It has been reported that post EVAR surveillance can result in exposure to approximately 144-205 mSv over a five year period which in a high output unit can equate to one cancer per year attributable to EVAR surveillance[11]. CTA can almost be a stand-alone modality for lifelong follow-up after EVAR but with the potential risk of radiation and nephrotoxicity.

Magnetic Resonace Imaging
MRI and MR angiography are an alternative to CTA. Reliability of MRI for the measurement of aortic diameter and detection of endoleaks is comparable to that of CTA[12]. MRI has the advantage over CTA due to the lack of exposure to the ionizing radiation and low nephrotoxicity of MRI contrast medium. Disadvantages of MRA are the cost and lack of widespread availability. Patients with pacemakers and certain types of stent grafts are also unable to undergo MRI scans.

Recommendations for post EVAR surveillance
All patients should have appropriate imaging (Duplex or CTA) at 30 days post procedure. In selected cases considered at risk for problems based on intra-operative findings and completion imaging, CTA should be performed. **Class IIb, Level A**.

All patients should have a duplex ultrasound at 6 months, and CTA only if there is evidence of sac expansion. **Class IIb, Level B**.

At 12 months and annually thereafter, if there is no endoleak and a stable or shrinking AAA, a Duplex Ultrasound is recommended. If the patient's body habitus preclude an adequate DU, then a non-contrast CT with plain radiographs can be substituted. **Class IIb, Level B.**

Any enlarging aneurysm after prior imaging studies have suggested complete aneurysm sac exclusion, should prompt imaging with CTA and plain X-Rays. The presence of a new endoleak without sac expansion does not require further imaging at that time, but warrants closer follow-up (repeat Duplex at 3 months**). Class IIb, Level B.**

Follow-up with DU, non-contrast CT imaging, and plain radiographs seems reasonable for patients with renal insufficiency at any time after EVAR. **Class III, Level C.**

For follow-up after EVAR in young patients, MRI should be preferred to CT, if there are no contraindications, to reduce radiation exposure. **Class IIa, Level C**

In the EVAR 1 trial[3], a lower aneurysm-related mortality rate after EVAR did appear to be maintained at 4-year follow-up (4% in the EVAR group versus 7% in the OR group), but in terms of overall mortality this was cancelled out by excess mortality from other causes at around 28% in both groups. Comparable results were found in the DREAM trial[4], with lower aneurysm-related deaths at 2 years in the EVAR group (2.1% vs. 5.7%) but comparable survival for OR (89.6%) and EVAR (89.7%) groups. For this reason it is recommended that all patients receiving an EVAR should be kept on the best medical treatment including statins, aspirin, ACE-inhibitor or β-blockers if considered appropriate for secondary prevention of cardiovascular disease. **Level IIa, Recommendation B.**

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Type 2 Endoleak

A.T.O.Abdool-Carrim, T.T.Monareng

Definition, Incidence and Classification:

Type 2 Endoleak is defined as continued perfusion of the aneurysm sac despite endograft deployment.[2] Type 2 is the most common type of endoleak occurring in 10 – 44 % of patients and accounts for approximately half of all endoleaks. [3,4]

Type 2 endoleaks arise secondary to backflow from collateral arteries, usually the inferior mesenteric and lumbar arteries.[5] They also arise from other aortic collaterals such as the median sacral artery or accessory renal arteries.[5]

Endoleak may be **simple** as defined by an inflow and outflow vessels or "**complex**" and involve a nidus of vessels. The "complex" leaks are thought to behave like arterio-venous malformations, recruiting vessels over time and may therefore be difficult to embolize.[5]

Type 2 endoleak can be subdivided into early (occurring within 30 days of EVAR), persistent (those looking longer that 6 months) or late (those occurring after 1 year)

The management of Type II Endoleak has divided opinion with some advocating a conservative approach,[2] while others recommend early intervention.

Imaging of Type II Endoleak:

Current European guidelines[6] recommend a base line CTA with plain abdominal x-rays be performed within 30 days of EVAR. If no endoleak is noted, repeat imaging can be performed one year later.

Duplex ultrasound can also be utilized as it is more accessible with less radiation and contrast-induced nephropathy, but it is less sensitive and low flow leaks may be missed.[5] Body habitus and bowel gas are limiting factors for duplex ultrasound.

Contrast enhanced ultrasound can increase the sensitivity and studies have shown detection rates similar to CT Angiography.[7,8,9] Contrast enhanced Ultrasound may be better than duplex ultrasound in detecting endoleak.[10]

MRA has superior resolution and is highly accurate in diagnosing and classifying endoleaks.[11] MRA, however, is not easily available, costly and time consuming.

Dual-energy CT[12,13] maybe used to detect endoleak with good accuracy at reduced radiation exposure, but experience and availability are limiting factors.

Type II Endoleak Intervention:

Current European Society for Vascular Surgery guideline[6] recommends intervention when the sac diameter expansion is more than 10mm. Type II endoleak appears to be an independent risk factor for sac expansion[2,14,15,16]. The significance of this expansion on risk

of aneurysm rupture, however, remain unclear. Sac expansion with Type II Endoleak is a possible contributor to late rupture after EVAR.

Risk factors for development of Type II endoleak are the number of patent lumber arteries, diameter of arteries, patent IMA, proportion of aneurysm lined by thrombus, maximum thrombus thickness and patients > 80 years.[2]

Is there a role for pre-operative or intra-operative embolization of these patent vessels to prevent subsequent Type II endoleak? Many studies have shown that pre-operative / intra-operative embolization and coiling of aortic side branches to be feasible with variable success rates, however these procedures prolong operative time and costs. Given that many Type II endoleaks thrombose spontaneously, these extra costs and risks may outweigh any benefit[17,18,19,20,21,22,23,24]. Endovascular sac sealing may have a role in the prevention of type II endoleaks.

Conservative Management of Type II Endoleak:

Many studies [5,25,26,27] have shown that type II endoleaks are benign. The Eurostar registry drew data from 2000 patients and found a 1.8% rupture rate after 2 years with no significant difference between those with and without Type II endoleak.[28] Spontaneous resolution of Type II endoleak occurs in the range of 35 – 80% [2,15]

A conservative approach to Type II Endoleak is thus acceptable in cases with minimal sac expansion, especially in those occurring within 6 months of EVAR.

Treatment of Type II Endoleak after EVAR

Several methods for dealing with type II endoleak have been advocated:

1. Trans arterial embolization:
   - selective embolization of IMA via middle colic
   - selective embolization of lumber artery via ilio-lumbar artery

   This technique is most commonly used. A recent systematic review[5] reported an overall success rate of 62.5%. Therapy should be aimed at achieving stasis of flow within the aneurysm sac either with the use of coils or glue, and embolization of the feeding vessel.

2. Trans lumbar and direct sac embolotherapy
   - the aneurysm sac is punctured under CT Fluoroscopic guidance, the endoleak identified and embolised.

   A recent systematic review[5] showed a 76% success rate. Higher complication rates have been reported with this technique and it is therefore reserved for when trans-arterial embolization fails.

3. Transcaval approach
   Success with this technique is reported to be 96% level [29,30]. These are mainly single centre studies.

4. Laparoscopic Approach:
   This involves the laparoscopic ligation of IMA 2cm from wall of sac. The lumbar arteries and aortic neck are clamped with a laparoscopic clamp and the sac opened. Thrombus is evacuated to confirm no back bleeding. Level of evidence is low (Level C)

5. Open Surgical Approach:
   Via a midline laparotomy, the sac is exposed, opened ("sacotomy") and the bleeding vessel ligated without aortic cross clamping. The most common indication for open surgery was endoleak, especially Type II comprising of 26.8% with overall mortality of 3.2 %.[31]
   Endograft explantation and open conversion is required in some patients but is associated with higher mortality.

6. Newer techniques:

   Endovascular Aneurysm sealing (Nellix).[3]

   Polymer filled endobags with balloon expandable stents covered with expanded PTFE. These endobags are filled with polyethylene glycol based polymer containing 1% radio opaque contrast, which fills the aneurysm sac completely, sealing the lumen. Early results are encouraging as Type II Endoleak reported in 2% of patients at 5 months. Further studies and longer term results are needed.

   Peri-operative sac sealing

   Prevention of Type II endoleak may be justified in some instances where patients are at risk of developing Type II endoleak. A single centre randomized study[33] performed on aneurysmal sac embolization plus EVAR versus Standard EVAR in patients at risk of developing type II Endoleak was performed. Results showed 100% technical success and no aneurysm rupture or death. There was a higher type II endoleak rate in standard EVAR versus sac embolization group. Both groups showed similar resolution of Type II Endoleak in follow-up. Persistent endoleak was higher in standard 14.5% versus 6% in embolised groups. There were higher re-intervention rates in standard group. A significant reduction in costs was noted in the embolised group. The sac volume in follow-up increased in standard EVAR group and showed sac volume reduction in the embolised group. This study confirmed a significant higher freedom from re-intervention for Type II related Endoleak and also showed sac volume shrinkage at 2 years. Further studies needed before this technique can be routinely advocated.

Recommendations:

Imaging of Type II Endoleak

- Duplex ultrasound: at 6 months and yearly. **(Class IIa / Level B)**
- Contrast enhanced duplex ultrasound: at 6 months and yearly **(Class IIa / Level B)**
- CT angiography: at 1 month and yearly level of evidence **(Class IIa / Level B)**
- MR angiography: at 1 month and yearly level of evidence **(Class IIa / Level B)**

Conservative Management
Observation of type II Endoleak → developing early (<6 months) –acceptable level of evidence level 1a -- follow-up with serial CT angiogram and ultrasound scan
With sac enlargement >10mm- intervention indicated – **(Class IIa / Level B)**

Intervention for Type II Endoleak
Intervention is indicated when sac diameter has enlarged > 10 mm from previous scan otherwise observation and 6 monthly CT Angiography **(Class IIa / Level B)**

Peri-operative – prevention
In patients at high risk of developing type II endoleak – pre operative embolization of lumbar and inferior mesenteric arteries is acceptable **(Class IIa / Level B)**

Newer technique such as EVAS have early data that is very encouraging but longer term studies and larger numbers are needed.

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Management of Type 1, 3 and 5 Endoleaks Following Endovascular Repair of Abdominal Aortic Aneurysms

Bhavesh Natha

Summary

Endoleaks are common problems encountered in up to 25% of patients after endovascular aneurysm repairs (EVAR). They can be detected intra-operatively or many years after the procedure – hence emphasising the need for life long surveillance after an EVAR. Type 1 & 3 endoleaks are most strongly associated with aneurysm sac rupture and hence when detected they need to be treated. Endotension is a diagnosis made after excluding all other endoleaks and the type and timing of management is not very clearly defined.

The reasons for developing endoleaks are multifactorial. Device design & selection, aorta & aneurysm anatomy, and disease progression are a few areas where problems can occur. The number of endoleaks documented in the literature seems to be increasing and this can possibly be attributed to the increasing use of EVAR to treat aneurysms and the use of EVAR devices out of instructions for use (IFU). Fortunately, the vast majority of patients found to have endoleaks can be treated via endovascular steps and only a small portion requires conversion to open surgery.

The strength/quality of scientific evidence for managing Type 1,3 & 5 endoleaks is weak. There are numerous single centre retrospective reviews and extrapolated data from the follow-up arm of older randomised studies that offer recommendations on when and how to manage these endoleaks. Despite the low scientific strength from these studies designs, these endoleaks do need to be managed carefully due to their potential disastrous sequela.

Review of literature

Definition

Endoleak: Persistent blood flow in the aneurysm sac extrinsic to the endograft and is the most common complication after endovascular aneurysm repair (EVAR). Incidence reported up to 25%.

Classifications of Endoleaks

Type 1: leak at graft ends (inadequate seal)

- **1a: Proximal leak**
- **1b: Distal leak**
- **1c: Iliac occluder leak**

Type 2: sac filling via branch vessel

- 2a: single vessel
- 2b: two or more vessels

Type 3: leak through defect in graft fabric

- **3a: junctional separation of modular components**
- **3b: fractures of holes in graft**

Type 4: generally porous graft

Type 5: endotension

Type 1 Endoleaks

Primary type 1 endoleaks tend to occur early after EVAR (intra-op or in early post-operative follow-up). Causes:

- Aortic Anatomy: inappropriate anatomy; significant calcification/thrombus of the neck or distal landing zone
- Planning & Technique: choice and design of stent graft used; malpositioning of stent graft; under dilation of the stent graft

Secondary type 1 endoleaks tend to be diagnosed at later surveillance follow-ups. In some case of aortic remodelling there can be progressive dilation of aortic neck. Depending on stent graft design and characteristics or unfavourable infra-renal aortic neck this can lead to graft migration or lack of proximal sealing. In other cases, with increased sac retraction, the iliac stent graft limb can shorten and lead to type 1b endoleak.

Type 1c endoleaks occur due to retrograde perfusion of the aneurysm sac from an incompletely occluded iliac artery when using and aorto-uniiliac stent graft on the contra-lateral side.

Type 3 Endoleaks

Caused by structural failure of the implanted device. Type 3 endoleaks are caused by a structural failure of the implanted device, including junctional separation of modular components (due to migration or changes in vessel morphology with aneurysm shrinkage), holes in the fabric, and fabric tears due to graft strut fracture or erosion.

Type 5 Endoleak

Endotension corresponds to continued aneurysm expansion in the absence of a confirmed endoleak. The expansion of the aneurysm in a type 5 endoleak may be due to an undiagnosed endoleak, presumably with very slow flow and suboptimal imaging (e.g. no delayed helical CT acquisition). In some cases of endotension where open surgical repair of the aorta was performed, a seroma was all that was identified. Thus some authors propose that the sac expansion related to a type 5 endoleak might be ultrafiltration process through the PTFE graft pores.

Management

Both type 1 & 3 endoleaks allow direct communication between the aorta and the aneurysm sac leading to systemic arterial pressure within the sac. These endoleaks have been demonstrated in the majority of aneurysm ruptures after an EVAR repair. Thus the general consensus is that all type 1 & 3 endoleaks must be repaired aggressively when diagnosed. Currently the majority of these cases can be treated via endovascular techniques.

Correct identification of the underlying cause of the endoleak will help decide on the appropriate treatment strategy.

When and how to treat a type 5 endoleak (endotension) is a little less clear. An aneurysm sac can be pressurized via a low flow endoleak or indirectly via a clot (virtual endoleak), this explains why some AAA enlarge even when no endoleak can be detected and why endotension may occur without an endoleak.
Numerous techniques to measure aneurysm sac pressure have been used but accuracy is very variable in predicting what pressures lead to rupture. Thus most authors follow a pragmatic approach. If the aneurysm sac enlarges by >10mm the patient should be considered for treatment.

Treatment strategies for Type 1 endoleaks:

Balloon Angioplasty: This is usually the first step employed in attempting to treat a type 1a or 1b endoleak. Angioplasty of the seal zones is aimed at ensuring good opposition between the stent graft and the arterial wall and 'ironing' out any creases in the stent graft fabric.

Balloon expandable bare-metal stent: If angioplasty is not successful deployment of a balloon expandable bare-metal stent (most commonly used is the Palmaz stent) may be helpful in obtaining a tight proximal and distal seal when indicated. If the stent graft has migrated or undersized this modality will not be effective.

Stent graft extension: In cases where there has been migration distally in the proximal neck or shortening at the distal landing zone a stent graft extension may be an option. Proximally if there is adequate space below the renal artery ostia a tube proximal stent graft cuff can be deployed. If the renal artery ostia are too close, either a fenestrated stent graft cuff or conversion to a chimney EVAR or Fenestrated EVAR can be considered in the appropriate setting.

Type1b endoleaks refractory to simple angioplasty are generally treated with distal stent graft modular extension. Occasionally provisions may have to be made to extend the stent graft into the external iliac artery (necessitating coiling and coverage of the internal iliac artery) or use of a branched iliac device with need to be considered.

EndoAnchors: These helical anchors can be used to tack the proximal end of the stent graft to the aortic neck. They can be used prophylactically in patients with undergoing an EVAR that are known with a hostile aortic neck or be used therapeutically to treat type 1a endoleaks in certain situations. In limited studies they have been shown to be effective in successfully treating type 1a endoleaks where balloon angioplasty has failed and have also been used in conjunction with proximal stent graft cuff extensions. Thick circumferential calcification or thrombus >2mm thick, in the aortic neck, are contra-indications to EndoAnchor use.

Coils & Cyanoacrylate embolization: Few case studies have demonstrated some success in treating type 1a endoleaks that were not successfully managed by angioplasty and not amenable to other techniques. The gap between the proximal stent graft and aortic wall was cannulated and the type 1a endoleak identified. This space was then embolised and packed with coils or cyanoacrylate. The published studies demonstrated a good 6-month follow-up success rates. Theoretically the cyanoacrylate is less likely to allow recanalization than the coils over the long term, but this has not been demonstrated.

Open surgical repair: In patients who are fit and either not amenable or failed endovascular modalities, open surgical repair is an option. The vast majority of the patients with a type 1a endoleak will require supra-renal aortic clamping in order to explant the endograft and repair the aneurysm. This is associated with well-established potential peri-operative complications.

Treatment strategies for Type 3 endoleaks:

Stent Graft deployment: Tears or fracture and modular separation are generally successfully treated by deploying another stent graft to bridge the gap between the two components that have separated or torn.

Open Surgical repair: In cases with severe angulation or severely diseased Iliac where stent graft repair fails, conversion to open surgery is an option.

Treatment strategies for Type 5 endoleaks:

Appropriate imaging: Appropriate imaging (CT Angiography and delayed phase and possibly direct angiography) should be performed to rule out another type of endoleak that could be leading to sac expansion

Stent Graft deployment: Realignment of stent graft is one method demonstrated to help halt sac expansion from endotension

Open Surgery: Conversion to open surgery and possible sac evacuation and aneurysmorraphy or possible graft explanation and open aneurysm repair is the other alternatives for patient with rapidly enlarging aneurysm sac secondary to endotension.

Strength of Data

The evidence relating to timing and choice of treatment for managing type 1, 3 and 5 endoleaks is based upon numerous single centre small retrospective reviews. There is some evidence inferred from the data obtained from the larger randomised controlled studies long-term follow-up and from the various vascular registries.

There are no randomised controlled trials focusing on the management of type 1,3 and 5 endoleaks.

Future Direction

Current trial assessing the long-term benefit of EndoAnchors for prophylactic use in difficult aortic necks and treatment for type 1a endoleaks

Recommendations

1. All Type 1 endoleaks should be treated. (**Class IIa, Level C**)
2. All Type 3 endoleaks should be treated. (**Class IIa, Level C**)
3. An enlarging abdominal aortic aneurysm after endovascular abdominal aortic repair without evidence of an endoleak and with an increase in diameter >10 mm should be repaired. **(Class IIb, Level C)**

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Management of iliac limb complications after EVAR

Pierre Mouton

Summary

Limb thrombosis occur in newer devices, with an incidence of 0%–5%. Most limb thromboses occur within 2 months after EVAR and are a result of kinking of components or poor outflow. Delayed limb occlusion may result from endograft migration or from development/progression of atherosclerotic occlusive disease in the outflow arteries.

Introduction

Aortic endograft occlusion is a known complication after EVAR, with significant variability in incidence of 0% to 7.2%. Newer generation endografts have been associated with a lower incidence of graft occlusion, but it remains one of the major causes of secondary interventions and re-hospitalization after EVAR.[1]

Most occlusions occur within 2 months after EVAR[1-3]. A technical reason for occlusion can be found in 60% of patients.[1,11]

Predictors and causes

Prevention is better than management. Numerous causes for graft obstruction have been suggested. This include extension to the external iliac artery, tortuous iliac vessels, smaller limb diameter and/or low profile devices with less radial force, AUI endograft, younger age, presence of thrombus in the native aorta, the type of device, excessive oversizing and twisting of the limb during deployment.[1-4,10]

There are 5 main predictors for limb occlusion, accounting for more than 70% of all causes. These include stent graft landing in the external iliac artery, external iliac artery <10mm in diameter, aneurysm size less than 59mm, stent graft kinking or an endoleak requiring correction.[5]

Prevention intra-operative

A more liberal intraoperative and early postoperative intervention strategy may reduce the occlusion rates and improve outcome. Completion angiography, including rotational views, should be performed after removal of the stiff guide wire and imaging checked for kinking, stenosis or irregularities. Direct pressure measurements at the sheaths after all endovascular material is removed may also aid in identification of any hemodynamic obstruction to flow. Appropriate treatment with angioplasty and/or stenting may then be applied to correct these findings.[1,5,6]

Postoperative surveillance

With follow-up of the patient after the procedure, the patient needs to be evaluated for potential graft limb compromise. This should include a thorough lower-extremity pulse

examination and/or determination of ankle-brachial index. Similarly, development of claudication, lower-extremity ischemia, or a decreased ankle-brachial index following EVAR should be further assessed with imaging. Following identification of high risk patients, closer surveillance during the initial 6-month post procedure period with CT angiogram at 1 and 6 months, may reduce the rate of limb occlusion.[2,5,7,12]

Management of occlusion

The majority of patients require an intervention, either by open surgery or an endovascular procedure. Management will depend on the time of presentation and the clinical picture of presentation. Early occlusion will be more related to kinking or stenosis and this is more amenable to endovascular management. Late occlusion can be due to peripheral vascular disease deterioration or device migration and frequently managed with open surgery.[7-9,11]

Endovascular procedure will include pharmaco-mechanical thrombolysis, which may expose the underlying cause. This can be managed with angioplasty and/or bare metal stenting.[7-9,11]

Open surgery can include surgical thrombectomy, which should be used with caution, as the balloon thrombectomy may cause disruption of the endograft. Other options involve mainly an extra-anatomic bypass, with femoral to femoral bypass the most favored.[7-9,11]

Recommendations

1. The most important pre-operative predictor for limb occlusion is a planned landing zone in the external iliac artery. Choose the least risky option for repair. e.g. open surgery or AUI device. **(Class IIa, Level B)**

2. Intraoperative completion angiography, including rotational views, should be performed after removal of the stiff guide wire and imaging checked for kinking, stenosis or irregularities. Appropriate treatment with angioplasty and/or stenting may then be applied to correct these findings. **(Class IIa, Level C)**

3. To evaluate for potential graft limb compromise, follow-up should include a thorough lower-extremity pulse examination and/or measurement of ankle-brachial index. Similarly, development of claudication, lower-extremity ischemia, or a decreased ankle-brachial index following EVAR should be further assessed with imaging. **(Class IIa, Level C)**

4. In patients with high risk factors for limb occlusion, closer surveillance post procedure with 1 month and 6 month follow up CT is warranted. **(Class IIb, Level B)**

5. The treatment options for an occluded limb following EVAR include thrombectomy or thrombolysis followed by secondary endovascular or open limited surgical intervention, or extra-anatomic bypass surgery (e.g., femoral–femoral or axillo-femoral bypass graft). **(Class IIa, Level C)**

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Inflammatory Abdominal Aortic Aneurysm

J de V Odendaal

Inflammatory abdominal aortic aneurysms (IAAA) are a distinct clinico-pathological and radiological entity with current perspectives favouring an immune mediated aetio-pathogenesis involving non-specific, degenerative abdominal aortic aneurysms (AAA).[1]

It is characterized by unusually thick anterior and lateral walls with a shiny white appearance, with associated dense fibrosis which involves adjacent structures.[2] The classical triad for clinical diagnosis includes abdominal/back pain, weight loss and a raised ESR. Computed Tomographic Angiography (CTA) remains the mainstay in diagnosis. The true natural history is unknown but involves enlargement and rupture.[3]

The duodenum and proximal jejunum is nearly always adherent to the aneurysm.[4] Early experience regarding extensive adhesiolysis was complicated by duodenal, ureteric and inferior vena cava injuries leading to a 3 fold increased mortality.[5, 6] Goldstone suggested a modified operative approach through a transperitoneal incision with as little dissection as possible.[7] Classic textbook teaching suggests that the retroperitoneal route is better because inflammation is worse anterolaterally, with posterior wall sparing.[6] In most large series the transperitoneal route was used.[4, 6, 9, 10, 11, 12] Surgeons are encouraged to use the route they are most familiar with.

The ureters are involved in 30- 50% of patients with IAAA.[3, 4] Although ureterolysis can be done safely,[13] complications such as avascular necrosis and ureteric leak have been reported.[14] Multiple previous reports have confirmed regression of the inflammatory mantle after open surgery without ureterolysis with normalization of renal function.[6,9, 15, 16] Routine ureterolysis is not indicated, but the ureters may remain persistently entrapped [4, 10] Renal function, peri-aortic fibrosis (PAF) and ureteric involvement should be monitored and treated post-operatively with stenting and steroids/tamoxifen if necessary.[17]

The role of ureteric stents is controversial. The procedure is not without risk and can cause ureteric injury, infection and bacteraemia.[16] Indications are variable and include patients with severe or bilateral obstruction. Some have used it routinely.[12]

Morbidity and mortality rates for elective surgery is now comparable with non-inflammatory aneurysms,[6] but more anastomotic aneurysms occurs[11, 18].

The technical difficulties encountered during open surgery makes EVAR an attractive option. The PAF regress less frequently after EVAR with persistent post-procedure hydronephrosis noted in up to 60% of cases.[19, 20] EVAR has less morbidity than open surgery.[21] A recent Cochrane review found that there is insufficient evidence to make a firm recommendation regarding preferred treatment.[22] EVAR is a suitable treatment, but open surgery should be considered in patients with hydronephrosis deemed low risk for surgery.[12]

Recommendations:

1. Pre-operative ureteric stenting should be done at the discretion of the treating surgeon, acknowledging that the role remains controversial. **(Class IIb, Level B)**

2. When open surgery is performed, the surgeon should utilize the operative approach (trans-peritoneal / retroperitoneal) they are most familiar with without attempting extensive adhesiolysis. **(Class IIa, Level B)**

3. Routine ureterolysis is not recommended during open surgery.
   (Class IIa, Level B)

4. EVAR is a suitable treatment in patients without hydronephrosis who are anatomically suited for the procedure. **(Class IIa, Level B)**

5. The role of EVAR in patients with hydronephrosis is controversial, but can be performed at the discretion of the treating physician. **(Class IIb, Level B)**

6. Renal function and regression of PAF / hydronephrosis should be monitored in all patients after EVAR and open surgery. Additional ureteric stenting or steroid/tamoxifen therapy may be considered when renal indications develop. **(Class IIa / Level B)**

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The Diagnosis and Treatment of Post Catheterisation Aneurysms

Nishen Paruk

Vascular access site complication incidence varies between 0.8% to 1.8% of diagnostic cardiac catheterisation and up to 9%1 of percutaneous coronary interventions (therapeutic). Complication rates for diagnostic and therapeutic peripheral vascular interventions are not known. Groin femoral arteries are used for vascular access in the majority of interventions with increasing use of radial artery access for coronary interventions. The potential for access site complications has increased with the need for larger sheath sizes in the treatment of thoracic and abdominal aortic lesions and trans-catheter aortic valve repair. It is likely that the incidence of vascular access site complications is greatly underestimated. Vascular closure devices, radial artery access, access under fluoroscopy or ultrasound guidance have all been used to minimise the risk of these complications. A pulsatile mass is an obvious indication of a pseudoaneurysm but with 60% of pseudoaneurysms missed on physical examination alone, diagnostic duplex ultrasound should be obtained whenever the diagnosis is suspected[2]. When deciding on the particular mode of treatment, the size of the pseudoaneurysm, antiplatelet drug and anticoagulation treatment, patient symptoms and specific features of the pseudoaneurysm should be taken into account.

Small pseudoaneurysms ≤2.0cm in diameter will resolve spontaneously in 50% of cases depending on coagulation status[3]. If treatment is necessary then options include “blind compression” or real time ultrasound guided compression, real time ultrasound guided para aneurysmal saline injection, real time ultrasound guided intra-lesional thrombin injection, percutaneous transarterial closure of the arterial defect using a covered stent or open surgical repair. Larger pseudoaneurysms may be treated with open surgical repair. However, there are now many successful reports of ultrasound guided compression therapy or direct thrombin injection. Complications of therapy including distal arterial thromboembolism and pseudoaneurysm recurrence need to be monitored for and timeous treatment instituted.

Due to the paucity of reporting of post catheterisation pseudoaneurysms following non coronary catheterisations, recommendations are based predominantly on data from coronary interventions and American and European heart association guidelines rather than Vascular and Endovascular society published literature. A comprehensive literature search of Pubmed, Google scholar and the Cochrane Central Register of controlled trials for the period 2005 to 2016 was undertaken. There are no specific recommendations for radial artery access complications and suggestion is to extrapolate from femoral artery access complication guidelines. Evidence based recommendations in this literature review are in keeping with the “Compilation of 2005 and 2011 ACCF/AHA Guideline Recommendations” published in 20134 and supported by the European Societies for Ultrasound in Medicine and Biology guidelines[5].

Several approaches have been considered to prevent complications of transradial and transfemoral access for interventions. These include preinterventional ultrasound vessel mapping, risk stratification and real time ultrasound guided access.

Iatrogenic post catheterisation pseudoaneurysms will continue to complicate arterial cannulation procedures. With the ever increasing trend toward minimally invasive procedures, vascular surgeons will be involved more frequently in the management of this complication. Efforts aimed at preventing pseudoaneurysms are evolving with strong evidence available for the use of ultrasound guided compression and thrombin injection in the management of this complication. There is inadequate evidence [9,10,11] available to support the use of "blind compression", real time ultrasound guided compression or ultrasound guided thrombin injection as being the superior treatment modality. Open surgery is reserved for large recurrent aneurysms or complicated aneurysms including those causing femoral nerve compression or skin signs with impending rupture[12]. There is no consistent evidence supporting the routine use of fluoroscopy or risk scoring systems in the prevention of post catheterisation pseudoaneurysms. There is weak evidence supporting the routine use of ultrasound guidance in the prevention of post catheterisation pseudoaneurysms.

Recommendations:

Diagnosis & Treatment

1. Patients with suspected femoral pseudoaneurysms should be evaluated by duplex ultrasonography. **(Class I, Level B)**[4]

2. Initial treatment with ultrasound guided compression or thrombin injection is recommended in patients with large and/or symptomatic femoral artery pseudoaneurysms**. (Class I, Level B).**[4,9,10,11]

3. Surgical repair is reasonable in patients with femoral artery pseudoaneurysms >2.0cm in diameter or that persist or recur after ultrasound guided compression or thrombin injection. **(Class IIa, Level B)**[4]

4. Revaluation by ultrasound 1 month after the original injury is useful in patients with asymptomatic femoral artery pseudoaneurysms <2 cm in diameter. **(Class IIa, Level B)**[4]

Prevention

1. Real time ultrasound guidance helps to reduce the complication rate of radial and femoral arterial access for cardiac and vascular interventions.
   (Class IIb< Level B)[6,7,8]

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Percutaneous Access for TEVAR and EVAR

Pieter Zwanepoel

Suture-mediated closure devices were developed to facilitate rapid and secure common femoral artery (CFA) haemostasis after endovascular procedures up to 10F sheaths. Owing to the utility of these closure devices, their use has been adapted to CFA closure in conjunction with large-sheath endovascular aortic aneurysm repair (EVAR) up to 27 Fr in size.[1]

Torsello et al. reported the first large, non-randomized series in order to assess the feasibility of percutaneous endovascular aneurysm repair (PEVAR).[1] A prospective randomized pilot study published in 2003 comparing endovascular suture technique with conventional cutdown, revealed technical success with percutaneous vessel closure ranging from 71% to 96% with improvement over time and experience.

Analysing the results of percutaneous technique in a large single centre experience, Eisenack et al. found a primary technical success of 96.1% in 500 consecutive patients. The need for early conversion correlated with femoral artery calcification (OR 74.5, 95% CI 17.8 to 310.7; $p < 0.001$) and operator experience (OR 43.2, 95% CI 9.8 to 189.0; $p < 0.001$). The risk of late complications was significantly higher in the presence of a groin scar (OR 48.8, 95% CI 9.2 to 259.0; $p < 0.001$), while sheath size and obesity played a minor role in influencing the results.[2]

The main risk factors for failure of the closure device are represented by calcified femoral arteries, scarred groin, and hostile anatomy such as high femoral bifurcation and femoral arterial grafts.[2345] Limited operator experience have also been identified as predictors of technical failure.[26]

In 2014 the first multicentre randomized controlled trial study results were reported in the PEVAR trial. This study was conducted to assess the safety and effectiveness of percutaneous EVAR. Two different suture mediated closure systems were used, either the 8FR Perclose Proglide (PG) or 10Fr Prostar XL (PS) system. The trial was designed to assess non-inferiority of PEVAR versus standard open femoral access (FE).[7]

Procedural technical success was 94% (PG), 88% (PS), and 98% (FE). One-month primary treatment success was 88% (PG), 78% (PS), and 78% (FE), demonstrating non-inferiority vs open femoral access for PG ($P = .004$) but not for PS ($P = .102$). Failure rates in the access closure sub-study analyses demonstrated non-inferiority of PG (6%; $P = .005$), but not of PS (12%; $P = .100$), vs open femoral access (10%). Compared with open femoral access, PG and PS yielded significantly shorter times to haemostasis and procedure completion and favourable trends in blood loss, groin pain, and overall quality of life. Initial non-inferiority test results persist to 6 months, and no aneurysm rupture, conversion to open repair, device migration, or stent graft occlusion occurred.[7]

Several nonsignificant trends favouring one or both PEVAR groups were observed: (1) reduced mean blood loss; (2) fewer PEVAR patients requiring transfusion; (3) hospital discharge on average a half-day earlier; and (4) fewer PEVAR patients prescribed analgesics for groin pain (15% vs 30%, FE). No differences were observed in times to ambulation or normal diet.[7]

A significant disadvantage in the use of these techniques is the very significant cost that it adds to the overall procedure and this needs to be balanced against the potential savings from

reduced need for transfusion and possible earlier discharge.

Recommendations

1. Percutaneous access for EVAR or TEVAR is a safe alternative compared to standard open femoral artery access
(Class IIa, Level B)

2. Percutaneous access may be contra-indicated in calcified arteries, scarred groins and hostile anatomy such as high femoral bifurcation and femoral arterial grafts.
(Class IIa, Level B)

3. Percutaneous access should preferably be used only by adequately trained and experienced operators. **(Class IIb, Level C)**

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Visceral and renal artery aneurysms – management

H Louwrens

Renal artery aneurysms (RAA) are rare, with the widely accepted incidence of 0,1% in the general population.[1]Computed tomography series report a higher incidence of 0,7% acknowledging that its most likely overestimating RAA prevalence. [2] The natural history of these aneurysms remain elusive and therefore indications for surgical intervention is controversial. The growth rate is that of slow to no growth with between 0,06-0,086 cm/year reported with no difference between calcified and non- calcified RAA.[3-4] They occur predominantly in women in the 5th to 6th decade, lack the traditional risk factors of aneurysmal disease except hypertension and 75% are asymptomatic at diagnosis. [3-8]

Traditional indications for repair included all symptomatic RAA, RAA in women of childbearing age, all recent onset false aneurysms and asymptomatic RAA of more than 2cm.[3-8] Repair of symptomatic and false RAA are mostly uncontested indications as well as those diagnosed in women of childbearing age due to increased risk of rupture with a high mortality of mother and fetus.[9-10] The cut off of 2cm for asymptomatic RAA are thought to be aggressive among certain authors and multiple series document safe observation of RAA between 2 and 3cm.[3-4,11]

Traditional open surgical repair of RAA has made way in recent years for less invasive endovascular techniques such as coiling and the use of stentgrafts, with very similar morbidity and mortality in well selected patients.[12,6]

Splanchnic artery aneurysms are similarly uncommon with an incidence of 0,1% and also have an unclear natural history, no standards for surveillance and no consensus on indications and best type of surgical treatment.[13-14]

Splenic artery aneurysms (SAA) are the most common splanchnic aneurysms and have a strong association with multiparity, portal hypertension and livertransplantation.[13,15-18] Patients with portal hypertension undergoing liver transplantation has a high rate of rupture of SAA peri-operatively and should undergo repair regardless of size. [16-17]As with RAA there is an increased risk of rupture in pregnancy and repair is indicated in all women of childbearing age with the diagnosis of a splenic artery aneurysm. [18] Observation of asymptomatic SAA of less than 2,5cm have revealed growth in only around 10% of patients with no ruptures, so the natural history of true SAA seems to be rather indolent.[13,19] Treatment has evolved from open surgery with or without splenectomy to endovascular exclusion by embolization or stent grafting as first line treatment of most patients with a lower perioperative mortality and acceptable long term outcomes.[20-21]

Pseudoaneurysms of the other splanchnic vessels have a high rupture rate and is on the increase especially in the hepatic arteries due to an increase in interventions for biliary disease and more cross sectional imaging after trauma. [22] These aneurysms should all be treated regardless of size and both open and endovascular repair are durable but mortality and morbidity are lower with an endovascular approach .[23-24]

Treatment of true aneurysms of the rest of the splanchnic vasculuture should be individualized according to the location, size, etiology and condition of the patient.

Recommendations

Renal artery aneurysms

Repair if symptomatic, in women of childbearing age and recent onset pseudoaneurysms **(Class IIa, Level B)**

Repair of asymptomatic aneurysms can be considered from 2cm and should be offered at 3cm **(Class IIb, Level B)**

Can be repaired with endovascular or open techniques **(Class IIa, Level B)**

Splenic artery aneurysms

Repair if symptomatic, in women of childbearing age, in liver transplant recipients and recent onset pseudoaneurysms **(Class IIa, Level B)**

Repair if asymptomatic at >2,5cm **(Class IIb, Level B)**

Repair with endovascular techniques if possible **(Class IIb, Level B)**

Repair all splanchnic pseudoaneurysms with endovascular techniques if possible **(Class IIa, Level B)**

Repair all pancreaticoduodenal and gastoduodenal aneurysms at diagnosis, with endovascular techniques if possible **(Class IIa, Level C)**

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Infected Aneurysms

N Govender, TV Mulaudzi

Classification:

Based on aetiology:
1. Microbial arteritis with aneurysm formation due to non-cardiac origin bacteraemia or contiguous spread of a localised infection
2. Post traumatic infected pseudoaneurysms
3. Infections of pre-exisiting aneurysms (bacteraemia or contiguous spread)
4. infected (mycotic) aneurysms from septic emboli

	Microbial arteritis	**Post traumatic Infected Pseudoaneurysms**	**Infection of pre-existing aneurysm**	**Infected Aneurysms from Cardiac source**
Etiology	Bacteraemia, contiguous spread	Narcotic addiction, trauma	Bacteraemia, contiguous spread	Endocarditis
Age	>50	<30	>50	30 -50
Incidence	Common	Common	Unusual	Rare
Common Location	Aorta, Iliac artery, intimal defects	Femoral Carotid	Infrarenal aorta	Aorta, visceral, intracranial, peripheral
Common Bacteriology	Salmonella	Staphylococcus aureus	Staphylococcus	Gram positive cocci

Wilson SE et al [1]

Diagnosis:

Presentation depends on anatomical location, virulence of the organism and duration of the infection.
General findings include fever, chills and leucocytosis.

- For aortic aneurysms – back pain or abdominal pain
- Peripheral aneurysms – distal embolization, pulsatile mass and overlying cellulitis [2]

In some patients non-specific signs and symptoms make diagnosis difficult. High index of suspicion should be maintained as patient survival depends on prompt diagnosis and management[3]

Infected aneurysm should be suspected when:

- Aneurysm found in conjunction with positive blood or tissue cultures
- Erosion of vertebrae adjacent to an aortic aneurysm
- Rapid aneurysm growth
- An uncalcified aneurysm in an older patient
- Initial manifestation of an aneurysm after bacterial sepsis in immunocompromised patients[3,4]

Laboratory findings

Leukocytosis and elevated ESR are common but non-specific findings in infected aneurysms.
Negative blood cultures are not sufficiently sensitive to rule out the diagnosis of infected aneurysm[5].
Intraoperative cultures should always be obtained with sampling of the arterial wall and thrombus for aerobic and anaerobic bacteria, as well as fungal cultures to help direct postoperative antibiotic therapy[6].

Imaging

Radiological studies are useful for obtaining diagnosis and planning surgical reconstruction.
Ultrasonography is helpful in peripheral aneurysms but not specific and cannot confirm the diagnosis[2]
CTA is readily available and has become the standard test for infected aneurysms.
CTA findings consistent with infected aneurysms include

1. Lack of calcification
2. Saccular , multi-loculated or eccentric aneurysms
3. Soft tissue inflammation or surrounding mass
4. Air within the aneurysm or surrounding tissue
5. Periarterial fluid collection
6. Pseudoaneurysm formation
7. Contained rupture
8. Rapidly enlarging/evolving aneurysms[7].

Positron Emission tomography (PET) alone or in combination with CT can be used for the detection of arterial infection but has not received widespread acceptance[8].

MRI and MRA may prove helpful when CT contrast media are contraindicated, as both modalities are highly sensitive for inflammation[9].

Indium -111 labelled white cell scan has not always been accurate in primary infection[10].

Antibiotic Treatment

Antibiotics should be administered as soon as possible and should be continued after surgical treatment

Duration of antibiotics is not well established and can vary from weeks to lifelong courses in cases of highly virulent organisms or multidrug resistant pathogens[4].

Operative treatment

The following general principles apply to infected aneurysms
Control of haemorrhage through proximal and distal control should be obtained, and intra-operative gram stain and tissue culture should be sent for aerobic, anaerobic fungal culture.
Operative control of sepsis should be obtained with resection including the aneurysm and all the surrounding necrotic or infected tissue.
Pre and post-operative antibiotic therapy should be broad spectrum, and include vancomycin and an agent that covers gram negative organisms (especially Salmonella) until organism specific antibiotic therapy can be instituted. The duration of therapy is at the discretion of the surgeon.
Revascularisation through non infected tissue planes should be used with extra- anatomic bypass, or an in-situ reconstruction may be performed with autogenous conduit (most desirable), cryopreserved allografts, or prosthetic grafts (least desirable)
Selection and need for reconstruction are guided by several factors:
1. Surgeons experience
2. Patients surgical risk and comorbidities
3. Anatomic location of the aneurysm
4. Availability of autogenous conduit
Ideally the graft should be covered with well vascularised tissue, and if necessary omental or muscle flaps should be used to augment coverage[11].

Endovascular Repair

Endovascular repair has gained acceptance over the years for treatment of abdominal and thoracic aortic aneurysms and there are increasing numbers of reports of these techniques being used in infected aneurysm patients. A meta-analysis of 48 patients with infected aneurysms treated with endovascular aneurysm repair (EVAR) found that ruptured aneurysms and fever at operation were the two most significant predictors of poor prognosis and persistent infection.

Long term outcomes of EVAR for infected aneurysms are lacking, and the concern for possible extension of a persistent infection into more proximal segments of the aorta is certainly valid because further surgical treatment would be more challenging. Salmonella has been associated with persistent and extended infection, and the virulence of the organism involved should be considered when choosing the method of repair[12].

Some authors have demonstrated that reoperation for infected aneurysms, after having been inadequately treated by endovascular means, is more likely to be unsuccessful or more complicated , leading to higher operative mortality[13-15].

These data give credence to the idea of using EVAR as a short -term temporary bridge to open reconstruction rather than definitive therapy.

Recommendations

Classification of aneurysms based on aetiology facilitates antibiotic management with regards to likely pathogens and surgical management **(Class IIb, Level C)**

The presentation of an infected aneurysm will depend on anatomic location, the virulence of the organism, and the duration of infection. In some cases diagnosis can be difficult with non-specific symptoms. Patient survival depends on early diagnosis and definitive management, a high index of suspicion should be maintained in certain clinical scenarios **(Class IIb, Level C)**

CTA is the standard test for investigating infected aneurysms, with specific findings suggestive of infected aneurysm. MRA/MRI may prove helpful in scenarios where contrast media is contraindicated. **(Classs IIb, Level C)**

Broad spectrum antibiotics should be the initial choice and then narrowed to culture and sensitivity directed therapy once this is available. Blood culture and tissue culture should be obtained before initiating antibiotics **(Class IIb, Level C)**

A minimum of six weeks intravenous antibiotics is recommended **(Class IIb, Level C)**

In high risk patients or in cases where location of recurrent infection would be lethal eg. aortic repairs, most physicians err toward lifelong suppressive therapy **(Class III, Level C)**

Operative control of sepsis should be obtained with resection including the aneurysm and all the surrounding necrotic or infected tissue. **(Class IIa, Level B)**

Revascularisation through non-infected tissue planes should be used with extra-anatomic bypass, or an in-situ reconstruction may be performed with an autogenous conduit (most desirable), cryopreserved allografts, or prosthetic grafts (least desirable) **(Class IIa, Level C)**

If an endovascular method of repair is chosen, it should generally be used as a bridge to more definitive therapy in patients who can tolerate an open reconstruction once they are clinically stable. **(Class II, Level B)**

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Aorto-Enteric Fistulae

Cloete NJ

Primary aorto-enteric fistula (PAEF)

Primary aorto-enteric fistula is a rare life-threatening condition which is defined as a spontaneous erosion and communication between the abdominal aorta and the gastro-intestinal tract[1]. An aorto-duodenal fistula is the most common presentation (60 %) due to the close anatomical relationship between D3/4 and the abdominal aorta. The usual presentation is a massive upper GI bleed, with a delay in diagnosis due to a low index of suspicion. The aetiology is variable, but usually results from pathological changes of the aorta, such as degenerative or mycotic aneurysms. Other causes include gallstone erosion [2], pancreatic carcinoma [3] and duodenal diverticulum[4].

First described in 1829, it remains a rare entity with a reported autopsy incidence rate of 0.04 – 0.07[5]. Other reports quote less than 200 cases found in a literature review. Classically upper GI bleed (64 %), abdominal pain (32%) and palpable abdominal mass (25 %) form the triad of AEF[6], but this is only true in 11% of cases. Less common symptoms include back pain, melaena, fever and syncope. Identification of the “herald bleed” is crucial to early diagnosis and appropriate treatment. However a review by Steffes and O'Leary found that in 29 % of patients, the time between the initial bleed and death was more than one week .The investigations utilised to confirm diagnosis is debateable. A recent review found that Computed Tomographic Angiography (CTA) is the modality of choice, as opposed to upper endoscopy or catheter angiography[7]. Air within the sac wall and contrast in the GI tract strongly suggests the presence of an AEF.

Untreated AEF results in a mortality rate of 100 %. Surgical repair is the only treatment option available currently. Hypotensive resuscitation with a SBP of 60 – 80 mmHg may reduce the risk of recurrent bleeding before definitive surgery. Broadly speaking the treatment options can be divided into open surgical procedures and endovascular procedures. Numerous studies and literature reviews have shown a higher mortality rate with extra-anatomical repairs compared to in-situ repairs[8]. In-situ repairs using ePTFE grafts or silver impregnated polyester grafts may be indicated in unstable patients. Closure of the intestinal defect alone is not recommended. Operative specimens for culture are mandatory to guide appropriate antimicrobial therapy. Endovascular repair has been advocated as a bridging option prior to definitive repair in selected cases.

Secondary AEF

Secondary aorto-enteric fistula is a well described complication of open AAA repair .The incidence of AEF is higher with inflammatory aneurysms compared to degenerative aneurysms. AEF after stentgraft repair have been reported to occur in 0.36 % of cases[9]. The lower incidence is postulated to be due to the abolishment of contact between a suture line and the GI tract. AEF in the post EVAR setting represents stentgraft infection from either local direct septic focus or haematological seeding. Other mechanisms reported include stent migration and kinking. Ratchford et al reported on the association between type I endoleaks and AEF, postulating that continued sac expansion predisposes to this complication[10]. Norgren et al also noted that type IV endoleaks may be implicated especially with the earlier types of stentgrafts.[11] The operative repair options are similar to those discussed in the above text.

Aorto-caval Fistula

Aorto-caval fistulae are a rare entity. It is reported to be associated with less than 1% of abdominal aortic aneurysms. With ruptured aneurysms it is reportedly found in 2 – 7 % of cases[12]. Operative mortality rate ranges from 16 – 66 %[13].

The typical clinical presentation, include confusion, lethargy, abdominal pain and backache. A palpable abdominal mass with a machinery murmur are classical findings on examination. Occasionally high output cardiac failure intervenes with elevated central venous pressure, pulmonary oedema, lower limb oedema, pulsatile veins and gastro-intestinal or genito-urinary bleeding. The diagnosis can be confirmed with ultrasound or CTA. Traditionally ACF have been repaired with open surgery with a non-absorbable suture repair from within the aneurysm sac. Since 1998 endovascular repair has been utilised as an alternative option particular for high risk surgical patients. The proposed survival benefit has not come to realisation as borne out with the results of the IMPROVE study[14]. Patients with inflammatory AAA deserve a mention as the incidence of ACF with rupture appear to be more prevalent compared to other presentations[15]. Endoleaks are the major drawback of this modality. A recent review of 67 patients found an endoleak rate of 50% in the group treated with endovascular repair, which is significantly higher than previously reported[16]. With respect to the reported endoleaks less than half resolved spontaneously. A more aggressive approach was required to treat the endoleaks usually within the same admission. Novel interventions have been utilised, include placement of an Amplatzer ductal occluder or intracaval stentgraft placement[17]. The potential morbidity associated with deep venous stenting has to be borne in mind, with the risk of ilio-caval thrombosis and need for anticoagulation raising concerns. This contemporary review reported a complication and mortality rate of 46% and 19% (adjusted to 3.8 % if delayed diagnosis is factored in) for endovascular repair, and 36% and 12% for open repair respectively.

Recommendations

Open surgical repair remains the preferred option. **(Class IIb, Level C)**

Endovascular treatment has merit; the frequency of endoleaks and the complexity of their management must to be considered carefully. **(Class IIb, Level C)**

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Management of the infected aortic prosthesis

J van Marle

Open repair of abdominal aortic aneurysms has a reported graft infection rate of 0.5-3%; the incidence of graft infection increases significantly from 1 to nearly 3% in case of femoral extension of the abdominal graft[1,2]. Graft infection following endovascular aneurysm repair is less than after open repair with a reported incidence of < 1%[3,4,5]. Graft sepsis is a serious complication with a reported mortality of 15-45% and limb loss rate of up to 30% after open repair[6]. The mortality following surgical treatment of infected endografts varies from 0-19%[7,8,9] .The majority of graft infections will present within one year of the primary intervention[7,9,10].

Pathogenesis

Prosthetic infection can be caused in a number of ways including peri-operative contamination, hemotogenous seeding, translocation of sepsis from a distant infectious source, and erosion of graft into adjacent bowel. Almost 2/3 of patients who present with graft sepsis have a remote source of infection potentially responsible for hemotogenous seeding[11]. Secondary interventions increase the risk of graft sepsis[8,9,12,13] . The most common organism responsible for graft sepsis is Staphyllococcus aureus.[8] Other species include S.epidermidis, Enterobacteriaceae, Enterococci and Streptococci[8,9,12,14].

Clinical presentation

Aortic graft infection may present with acute sepsis (1/3), chronic sepsis (1/3) and aorto-enteric fistula (1/3) [8,10,12]. The most common symptoms are fever, weightloss and malaise together with signs of systemic illness. Other complications include haemorrhagic shock due to anastomotic breakdown, retroperotineal effusion, septic embolisation and complications of groin sepsis following aorta bifemoral bypass including cellulitis, abscess formation, pseudo-aneurysm, haemorrhage and cutaneous fistulae.

Diagnosis

Diagnosis of graft sepsis is based on a combination of clinical findings and radiological imaging, supported by a positive culture of the causative organism. Two separate sets of bloodcultures should be taken, and where possible aspiration of peri-prosthetic collections should be performed through an uncontaminated route. Positive cultures are found pre-operatively in 2/3 of cases of suspected graft infection[10].

CT imaging is essential in the initial diagnostic workup of patients where graft sepsis is suspected. Indications of possible sepsis include peri-graft fluid, tissue infiltration, fluid collection with or without gas, psoas or groin abscesses and aortic rupture[15]. FDG-PET is more sensitive in diagnosing infectious processes and fused FDG-PET/CT may be better at differentiating between infected and non-infected grafts[16]. It is especially indicated where

clinical findings are suggestive of infection in the absence of positive cultures[17]. Labelled whiteblood cell scintigraphy and galium scintigraphy may be used as ancillary methods to establish vascular graft infection.

Management

The principles of management are the same for graft infection after open repair and endovascular repair[2,18]. Treatment consists of excision of the septic prosthesis, thorough debridement of all infected tissues, vascular reconstruction and targeted antibiotic therapy. Vascular reconstruction following excision of the infected graft can be by extra-anatomic bypass or in-situ reconstruction. Specific strategies are determined by various factors including the extent of the infection, causative organisms, graft patency, limb viability and patient stability.

Extra-anatomic bypass

The infected graft or endoprosthesis is removed, the aortic stump debrided to normal appearing tissue and the stump closed. The suture line is butressed with anterior spinal ligament and covered with an omental patch. Extra-anatomic bypass (axillo-bi-femoral bypass(AXBF), axillo-uni-femoral bypass(AXUF), or axillo-popliteal bypass(AXP) is performed via a non-infected field. The AXBF has the best patency with the inverted-C as the preferred configuration for the femoral crossover limb[19]. PTFE graft seems to have a lower re-infection rate than Dacron grafts (3% vs 15%) and external support is highly recommended to improve graft patency[20]. Staging the procedure by removing the septic graft 24-48 hours after the extra-anatomic bypass, reduces the mortality (from 26-13%) and limb amputation rate (from 46-11%)[21,22]. In case of patient instability due to haemorrhage or severe sepsis, revascularisation is done immediately after graft excision (sequential procedure).

In more recent studies, in-situ vascular reconstruction have provided better outcomes with decrease in mortality and improved limb salvage[7,8,12]. Autogenous reconstruction using the deep veins of the lower limbs (superficial femoral and popliteal veins) is safer and more effective than extra-anatomic bypass grafting with a lower mortality (9%), better patency and decreased risk of amputation (2%) and lowest risk of secondary infection (1%)[23,24,25,26]. The operation, however, is time consuming and complex with significant bloodloss and is therefore not indicated in unstable and high risk patients.

Revascularisation using in-situ graft replacement may be used in patients with minimal infection: localised/segmental graft infection without invasive peri-graft sepsis and primarily caused by coagulase-negative staphylococci (S-epidermidis) [1,27]. Silver impregnated grafts, PTFE or Dacron grafts soaked in Rafampin (60mg/ml for 30min) are used for the in-situ reconstruction[28]. Re-infection rates of 4-18% have been reported.

Extra-cavity infections

The groin represents the most frequent site of vascular graft infection with an incidence of about 5% and significant morbidity and morality[29]. Management of groin infections following aorta-bifem bypass is determined by the extent of the infection i.e. graft body vs anastomotic sites, degree of infection and type of infection (S.epidermidis vs MRSA and gram-negative or polymicrobial infections), graft patency and limb viability. In cases of limited infection involving only the body of the graft, and caused by less virulent organisms e.g.

Staphylococcus epidermidis, the management consists of repeated wound debridements, irrigation and local woundcare including vacuum assisted closure therapy[30,31]. When the anastomosis is involved or there is infection involving gram-negative organisms or MRSA, the infected portion of the graft should be resected to normal, uninfected graft with good tissue incorporation. Vascular reconstruction can be either in-situ autogenous vein, or Rafampin-soaked graft[25,27,28]. Covering the vascular reconstruction with muscle flaps has been shown to enhance healing and decrease the rate of recurrent infection[32,33]. In cases of severe contamination an extra-anatomic bypass should be performed using either an ax-uni-fem or trans-obturator bypass to normal uninfected superficial femoral artery. The trans-obturator bypass offers a durable means of revascularisation in the presence of a septic groin[34].

Conservative management

There is a limited role for conservative management of graft sepsis. This consists of local drainage and prolonged antibiotic therapy. Reported mortality ranges from 36.4-100% and this modality should only be considered in patients with prohibitive surgical risk[7,8,9,12,14].

Antibiotic therapy

Antibiotic therapy should be targeted according to culture and sensitivity of the causative organisms. Pre-operative identification of infective organisms should be attempted in all cases by taking bloodcultures and aspiration of peri-prosthetic collections. Empirical antibiotic therapy should be used in cases where it is not possible to wait for surgical microbiological results e.g. severe sepsis, septic shock, aneurysmal rupture or anastomotic disruption. The antibiotics selected should be bactericidal, have good tissue distribution, including diffusion into biofilm, should have activity against slowly metabolising strains and have a good safety profile. Empirical antibiotic therapy for PVGI are given in table 1[35].

Table 1: Empirical antibiotic therapy for PVGI (CIII)

Clinical situation	**No allergy to β Lactams**	**Allergy to Penicillin**
1. PVGI, no severe sepsis, no history of MDR* bacterial infection	Piperacillin / Tazobactam + Vancomycin / Daptomycin ± Gentamycin	Cefotaxime / Ceftriaxone / Cefipime / Aztreonam + Metronidazole + Vancomycin / Daptomycin ± Gentamycin
2. PVGI with severe sepsis or previous	Imipenem / Meropenem / Doripenem	Fosfomycin

infection with ESBL – GNB*	+ Vancomycin / Daptomycin ± Gentamycin	+ Metronidazole + Vancomycin / Daptomycin ± Gentamycin
*MDR: Multi-drug resistant *ESBL-GNB: Extended specimen beta-lactamase producing Gram-negative bacillus		

Surgical removal of all infected material should be performed as quickly as possible to facilitate the efficacy of the anti-infective therapy. Antibiotic therapy should be re-evaluated post operatively and adjusted according to the intra-operative sampling results to target only the relevent pathogen(s). Guidelines and recommendations drawn up by a multi-disciplinary working group have recently been published[35]. A summarised version is presented here and the reader is referred to the full text for a detailed discussion.

Table 2: Antibiotic therapy for PVGI caused by Methicillin-sensitive Staphylococcus spp[35].

1. Cloxacillin / Oxacillin III,B [Pen.allergy: Cefazolin / Vancomycin / Daptomycin III,B]
   [C/I to β Lactams: Vancomycin / Deptomycin III, B]
2. Gentamycin for 3 days III, C
3. Replace Gentamycin with Rifampicin on day 4 III, B
4. Day 15: Relay with oral Rifampicin & Fluoroquinolone III, C
5. Duration of treatment: 6 weeks III, C

Table 3: Antibiotic therapy for PVGI caused by Methicillin-resistent Staphylococcus spp.[35]

1. Vancomycin III B and Daptomycin III, C [Vancomycin MIC ≥1.5 μg/l, use Daptomycin III, B]
2. Gentamycin 3 days III, C
3. Replace Gentamycin with Rifampicin III, B
4. Day 15: Relay with oral Rifampicin and Fluoroquinolone III, C
5. Duration of treatment: 6 weeks

Table 4: Antibiotic treatment of PVGI caused by Enterobacteriaceae[35].

1. Ceftriaxone / Cefotaxime III, B [allergy to Penicilline: Aztreonam III, C]
2. Relay with Fluoroquinolone III, C
3. Duration of treatment: 6 weeks

Streptococcal infection[35]: Amoxycillin is the treatment of choice for streptococcal infections sensitive to this drug. Gentamycin is added for 3 days. In case of allergy or decreased sensitivity to Betalactams, Vancocin is the drug of choice. Duration of therapy is 6 weeks. Switching to oral Amoxycillin can be considered after 14 days.

Enterococcal infection[35]: Amoxycillin is recommended. Gentamycin is given for 3 days. Vancocin or Tycoplanin alone is recommended in case of allergy or resistance to Amoxycillin. Duration of treatment 6 weeks.

Pseudomonas infection[35]: Treatment is based on a Betalactam (Ticarcillin, Ceftazidine, Piperacillin or Tazobactam) and a Carbapenim (excluding Ertapenim). Amicacin or Tobramycin is combined for the 1st 3 days. Ciprafloxacin/Fosfomycin is used as a relay after 3 weeks.

Poly-microbial infection: Sensitivity testing should be performed on each cultured bacteria. A multi-drug regimen may be required to cover all the bacteria.

Each vascular unit should refine and adapt these recommendations using a multi-disciplinary approach and considering the most prevalent organisms and local drug resistance.

Duration of treatment: it is recommended that post-operative antibiotic therapy should be given for 6 weeks for optimal treatment (C3). It should be administered parenterally, but when using compounds with good bio-availability, e.g. Rifampicin or a Fluoroquinolone, oral administration after 2 weeks is possible.

Recommendations

1. Antibiotic prophylaxis against PVGI is required prior to vascular interventions, endoscopic procedures and dental procedures where bleeding is expected. **(Class III, Level B)**

2. Generalized sepsis, groin drainage, pseudo-aneurysm formation or ill-defined pain after OSR or EVAR should prompt evaluation of PVGI. **(Class III, Level B)**

3. GIT bleeding after OSR or EVAR should prompt evaluation for an aorto-enteric fistula. **(Class III, Level B)**

4. CT imaging is essential in the initial work-up of a patient where PVGI is suspected. **(Class IIb, Level B)**

5. FDG-PET CT is indicated in cases of equivocal CT findings in the absence of positive cultures in patients where there is a strong suspicion of PVGI. **(Class IIb, Level B)**

6. In patients with extensive PVGI, the infected graft should be excised with stump closure with reconstruction with an AXBF **(Class IIb, Level B)**

    a. Stable patients: staged procedure should be performed. **(Class IIa, Level B)**

    b. Unstable patients: sequential procedure is preferable. **(Class IIb, Level B)**

7. In patients with limited contamination, in-situ reconstruction with autogenous fem-pop vein is recommended, if the patient is stable and can endure the prolonged invasive procedure. **(Class IIa, Level B)**

8. In case of high risk or unstable patients with limited contamination, in-situ reconstruction using Silver or antibiotic impregnated or PTFE grafts can be used. **(Class III, Level C)**

9. Antibiotic therapy should be targeted according to MC&S of the causative organisms. **(Class I, Level A)**

10. Pre-operative empirical antibiotic therapy should be started in cases where it is not reasonable to wait for intra-operative specimens, but the antibiotic therapy should be adjusted and de-escalated as soon as final documentation of the infection is available. **(Class III, Level C)**

11. Empiric Antibiotic therapy should be adjusted and de-escalated as soon as final documnetation of the infection is available. **(Class III, Level B)**

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HIV-RELATED ANEURYSMS

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The first report of an arterial aneurysm in the context of the human immunodeficiency virus (HIV) infection was described by Sinzobahamvya in Zimbabwe[1]. HIV-related aneurysms (HRA) is usually the hallmark of advanced disease with unique characteristics that includes clinical presentation at a young age, multiplicity of aneurysms, and a variable spectrum of disease at numerous anatomical locations, most frequently in the carotids and femoral vessels. It poses specific challenges of questionable aetio-pathogenesis, increased infection risk and unpredictable life expectancy. The role of HAART (highly active anti-retroviral therapy), whilst promoting longevity, has not been well defined in the natural history of aneurysmal disease. The diagnostic evaluation for HRA is the same as HIV naïve patients with duplex ultrasound studies (DUS) preferred as a first line imaging tool. Definitive imaging is required for defining the magnitude of pathology and treatment planning. Clinical evaluation should include cardiovascular risk profiling, flag other co-morbidities and atherosclerotic risk factor assessment, define the patient`s immune status including viral loads, incorporate screening for opportunistic infections, and define nutritional status. Cumulative evidence regarding HIV aneurysms has been confined to case reports and series, the majority of which were conducted in the pre-HAART era.

Optimal therapy in these patients is still debatable. Treatment of HRA is presently based on anecdotal experience, and repair involves conventional surgery, endovascular intervention, or hybrid repair depending on available expertise. There are no comparative studies between these modalities. Patients are managed along conventional vascular surgical guidelines. There is currently no consensus guidelines on HIV-related aneurysms to inform best practice. Long-term results of intervention are speculative, attributable to poor patient compliance with follow-up. The exact indications for endovascular intervention requires further study. Symptomatic or complicated (HRA) are treated in surgically fit patients irrespective of immune status. Non-operative management is reserved for patients with full – blown AIDS. Attempts to risk stratify patients post-intervention have yielded variable results.

RECOMMENDATIONS

Screening[2-5]

1. There is no evidence in favour of mass screening for aneurysms in Adult HIV patients **(Class III, Level C)**

2. Patients who have demonstrated a HIV-related aneurysm in one specific vascular territory should have imaging of other vascular territories **(Class II, Level C)**

Risk stratification[6-10]

1. HAART should not be interrupted perioperatively as it may confer an increased cardiac risk **(Class IIa, Level B)**
2. The use of the Revised Cardiac Risk Index may underestimate the true cardiac risk in HIV positive patients undergoing vascular surgery. **(Class IIb, Level B)**

3. HIV positive patients with traditional atherosclerotic risk factors should receive optimum medical therapy. **(Class IIa, Level B)**

4. Selected statins should not share the same metabolic pathway as protease inhibitors. **(Class IIa, Level C)**

5. The evidence for statin administration in HIV positive patients without traditional risk factors is weak. **(Class III, Level C)**

6. Albumin levels < 3.5g/dl and a low CD4 count correlates with increased perioperative morbidity and mortality in patients with HIV undergoing abdominal vascular surgery. (Class II a /Level C) A CD4 count of less than 200 cells/mm^3 have an increased perioperative morbidity. **(Class IIb Level C)**

Central Aneurysms[3,5,10-14]

1. Surgical repair of thoracic and thoraco-abdominal aneurysms is safe and effective in the short term **(Class IIa, Level C)**.

2. Repair of HIV-related aortic aneurysms should be performed in symptomatic patients only. **(Class IIa, Level C)**

3. Surgical repair of HIV-related abdominal aortic aneurysms, in surgically fit patients, is efficacious but with higher mortality than non-HIV counterparts. **(Class IIa, Level C)**

4. Endovascular Abdominal Aortic Aneurysm repair (EVAR) is safe and efficacious in the short term. **(Class IIa, Level C)**

5. Elective endovascular intervention for Iliac artery aneurysms is feasible with low perioperative morbidity and mortality. **(Class IIa, Level C)**

6. Visceral and renal artery aneurysms should be managed as per non-HIV recommendations **(Class IIb, Level C)**

Peripheral aneurysms[13-21]

1. All patients with symptomatic aneurysms should have therapeutic intervention unless they are surgically unfit **(Class Iib, Level C)**

2. In asymptomatic patients with HIV aneurysms, there are no natural history studies or evidence correlating aneurysm size with rupture or thrombo-embolic risk. Decision to intervene should be individualized **(Class Iib, Level C)**

3. There is no conclusive evidence to guide the choice between open surgery or endovascular management, however both appear safe and effective for select patients in the short term **(Class Iib, Level C)**

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Popliteal Aneurysms – Elective Management

K Michalowski

Degenerative (atherosclerotic) popliteal artery aneurysms (PAAs) are the most common peripheral aneurysms. They constitute 70% of true lower extremity aneurysms, with an estimated incidence of <0,01% in hospitalized patients. 50% of popliteal aneurysms are found bilaterally and 50% are associated with aortic aneurysms. They represent a challenging treatment paradigm with a variety of presentations and management options.

Maximal diameter of the popliteal aneurysms, which should be consider as an indication for elective repair, is recommended at 2 or 3 cm. Asymptomatic PAAs smaller than 3 cm without the thrombus can be observed, with a little chance of thrombosis.

Observation should only be offered to asymptomatic patients with PAAs of <2.0 cm (annual Duplex Doppler).

Elective repair of asymptomatic PAAs >2 cm is indicated with mural thrombus or evidence of previous thromboembolism.

Intervention is mandatory for all symptomatic PAAs.

Symptoms are caused by thrombosis of the aneurysm itself or emboli to the distal circulation, or by local mass effect. Rupture is very rare.

The primary objective of treatment is to exclude the aneurysm from the circulation. This can be achieved by an open or an endovascular approach.

Open surgical repair can be conducted by medial and posterior approach. Early elective repair is recommended because these patients have no surgical mortality, a low rate of complications, and no limb loss at 5 years. GSV and endoaneurysmorraphy or ligation is recommended with a 5-year patency rate of 93% and a limb salvage rate of 100%.

It seems that PAA requiring intervention in females is associated with higher long-term mortality.

Endovascular repair is a technique that uses a covered stent to exclude the aneurysm and therefore is equally constrained, primarily by anatomy. Technical success rates range from 94% to 100% and primary patency rates are 75% to 94% at 1 year and 59% to 87% at 2 years, with limb salvage rates of 85% to 100%. Current evidence only supports the use of stent grafts if anatomy is suitable for elective repair of PAAs in those with surgical risk and in elderly.

Recommendations

Patients with palpable popliteal mass should undergo an ultrasound examination to exclude popliteal aneurysm **(Class I, Level B)**.

All patients with diagnosed PAAs should be screened for contralateral disease and for abdominal aortic aneurysm **(Class I, Level B)**.

Asymptomatic patients with PAAs < 2cm should be offered surveilance including ultrasound **(Class I, Level B)**.

Asymptomatic patients with aneurysms of any size with mural thrombus, or evidence of previous thromboembolism should undergo repair to reduce the risk of thrombotic complications or limb loss **(Class I, Level B)**.

All symptomatic patients irrespectively of size of aneurysm should undergo repair **(Class 1, Level B)**.

Open repair with surgical bypass, aneurysmorrhaphy or ligation of the aneurysm, or with interposition graft, remain the treatment of choice. **(Class I, Level B)**.

Current evidence on endovascular stent-grafting of PAAs is limited but can be considered for patient with surgical risk and the elderly **(Class IIa, Level C)**.

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Management of the Occluded Popliteal Artery Aneurysm

Martin Forlee

Summary

Popliteal artery aneurysms are the commonest peripheral artery aneurysm following the aorto-iliac segment.

Up to 59% of PAA's are symptomatic with approximately 25% of cases presenting as acute limb ischaemia. Rupture is extremely rare. The amputation rate after acute thrombo-embolism is as high as 30% due to obliteration of the runoff vessels.

Duplex Doppler is the initial investigation of choice. CTA and MRA or on table angiography are essential to assess inflow and outflow anatomy.

Infra-inguinal reconstruction using autogenous vein is the gold standard. Catheter directed thrombolysis is suggested to restore distal runoff in patients who can tolerate further ischaemia. Exclusion of the aneurysm with a stent graft is feasible in high risk surgical patients, but sudden graft occlusion remains a concern.

Introduction

Popliteal artery aneurysms (PAA) are the most common peripheral arterial aneurysms after the aorto-iliac segment and account for 70% of cases, with an estimated incidence of 0.1% to 2.8%.

A recent literature review analysed 5459 limbs in 4247 patients with popliteal aneurysms (average size 30 mm). 59% of patients were **symptomatic**. The presenting symptoms were acute limb ischaemia (25%), claudication (26%) and rupture (3%).[1] (Table 1). Asymptomatic PAAs cause complications in 15%-25% at 1 year and 60%-75% at 5 years, if left untreated[8].

Clinical Presentation	Average	Range	n
Asymptomatic	41 %	0-87 %	1295/3146
Symptomatic	59 %	13-100%	1851/3146
- Acute limb ischemia	25 %	3-100 %	718/2871
- Claudication	26 %	0-100 %	680/2567
- Rupture	3 %	0-17 %	54/1806
Elective	72 %	0-100 %	1496/2077
Eme	%	0-100 %	658/2077

Table 1: Clinical presentation[1]

Limb loss with acute presentation is not infrequent and the amputation rate after acute thrombo-embolism can be as high as 30% despite emergency interventions, because of thrombo-embolisation of the runoff arteries [4].

Imaging

Duplex ultrasound can be used to examine both the ipsilateral and contralateral popliteal arteries and is more sensitive than physical examination in diagnosing PAA. Furthermore, it provides information on the presence and velocity of flow, presence of mural thrombus, and patency of outflow arteries. Computed tomographic arteriography and magnetic resonance arteriography are useful adjuncts because they provide information on outflow and inflow as well as provide imaging of the artery in the popliteal space[8].

Management of Acute Limb Ischaemia

The severity of symptoms can be classified according to the presence of sensory or motor loss[3]. (Table 2)

The classification of acute ischaemia is very important, because it determines the urgency of the treatment. Thrombolysis, with or without additional surgery, has been advised for the treatment of acute ischaemia Rutherford class I-IIa. When sensory loss or motor deficit is present (class IIb-III), there is no time for thrombolysis and surgery should be performed immediately[2].

Grade	Clinical	Description
I	Viable	Normal motor and sensory
IIa	Threatened: marginal	Minimal sensory loss, normal motor
IIb	Threatened: immediate	Sensory loss above toes and motor loss
III	Non viable	Profound sensory and motor loss with no arterial or venous Doppler signals

Table 2: Rutherford Classification of Acute Limb Ischaemia

A treatment algorithm (adapted from Robinson et al[8]) based on the severity of the clinical presentation is proposed in Figure 1. The gold standard of treatment is infra-inguinal reconstruction using autogenous vein. Thrombolysis is useful in patients with no discernible runoff and who are able to tolerate the time needed for this procedure. Smith cautions the use of thrombolysis in the presence of patent runoff vessels as it can cause clinical deterioration due to distal thrombo-embolization[9]. Patients with adequate runoff should thus

proceed to definitive surgical repair. Patients with class IIb-III ischaemia with no runoff should undergo trifurcation exploration and fogarty thrombectomy +/- on table thrombolysis.

Ravn et al. reported 571 patients with 717 legs treated in Sweden in 1987-2002: preoperative thrombolysis improved run-off and reduced the risk of amputation when the patient presented with acute ischaemia[6], and open repair (OR) with a posterior approach (often using the inlay technique) had better long-term results because of the reduced risk of late expansion[7].

A systematic review of 895 patients presenting with acute thrombosed popliteal aneurysms with ischaemia compared different strategies of treatment: pre-operative thrombolysis followed by exclusion of the PAA with bypass surgery or surgery alone (crural thrombectomy + bypass surgery). Pre-operative and intra-operative thrombolysis resulted in a significant improvement in 1-year primary graft patency rates, but did not result in a significant reduction for amputations compared with surgery alone.

Is endovascular management feasible ?

A report from the Swedish Vascular registry reporting on 592 interventions in 499 patients showed that endovascular repair has significantly inferior results compared to open surgery, especially in the group of patients who present with acute limb ischaemia[5].

Trinidad Hernandez compared outcomes of elective and emergency endovascular popliteal artery aneurysm repair in 31 limbs. 12/31 (39%) limbs were emergencies with 11 presenting with acute limb ischaemia and 1 with rupture. The authors concluded that emergency endovascular repair was feasible, but was associated with major adverse event rate of 35.5% including 30-day stent occlusion (29%), endoleak (13%), and stent fracture (3.2%)[4].

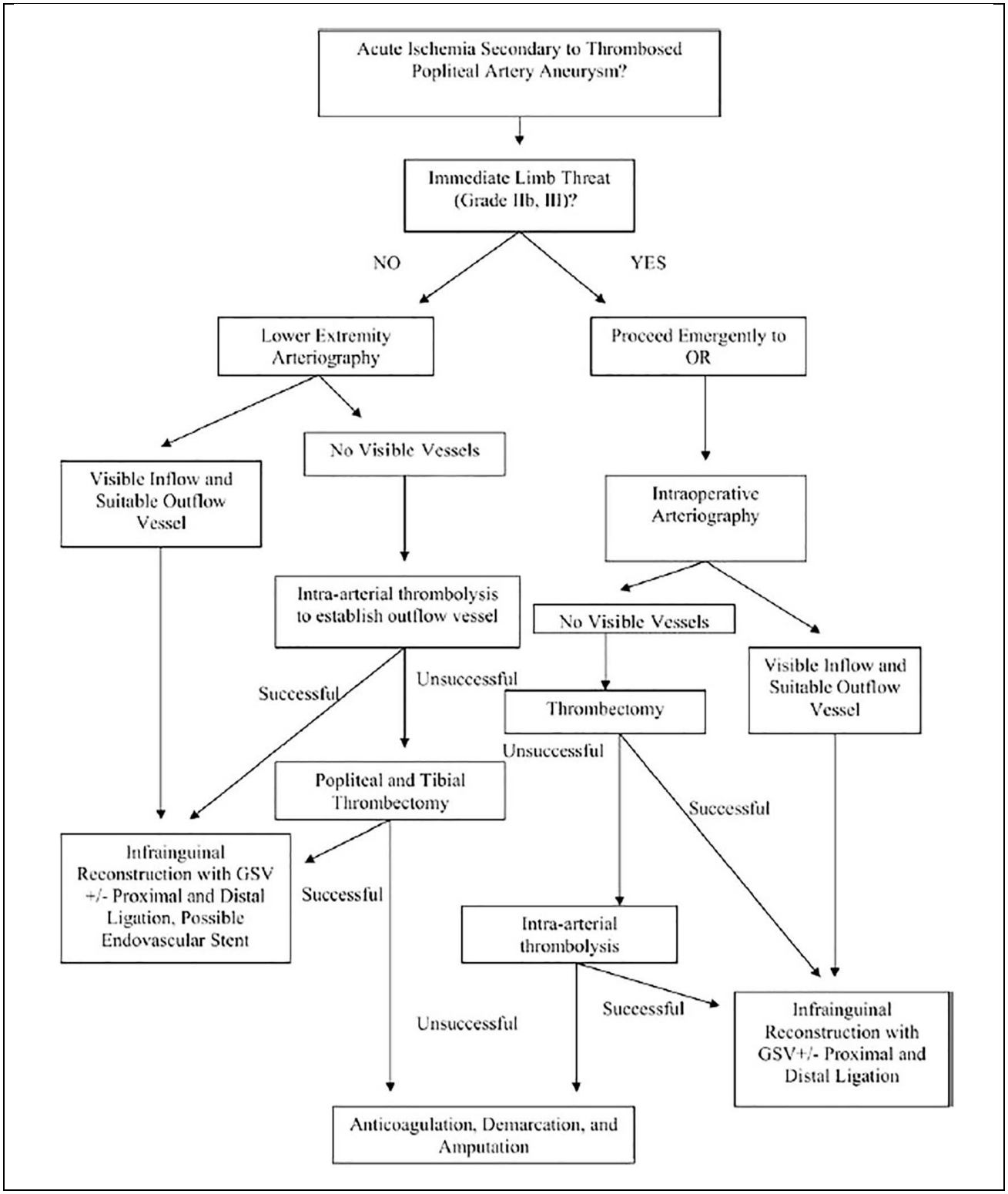

Fig 1: Suggested Treatment Algorithm[8]

Recommendations:

1. Duplex Doppler is the investigation of choice to confirm the diagnosis. Pre-operative CT or MR angiography or intra-operative digital subtraction angiography should be used to determine inflow vessel and outflow vessel anatomy. **(Class I, Level C)**

2. In patients with acute ischaemia and popliteal artery aneurysms and absent runoff, catheter directed thrombolysis or mechanical thrombectomy (or both) is suggested to restore distal runoff and to resolve emboli. **(Class IIa, Level B)**

3. Open surgery with infra-inguinal reconstruction using autogenous conduit is the treatment of choice **(Class I, Level B)**

4. Stent grafting is a feasible option in patients who are at high risk for open surgical repair **(Class II, Level B)**

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Extracranial Carotid Artery Aneurysms

Laura Redman

Summary

Extracranial carotid aneurysms are rare and the natural history is unknown.[1] There is no substantial evidence to make definitive guidelines. The cause has also changed over the years from infectious causes (such as syphilis and tonsillitis) to atherosclerotic degeneration, trauma, dissection, previous surgery and in the South African setting – HIV and TB.

ExtraCranial Carotid Aneurysms ECAA's are rare occurrences making up <1% of all carotid pathology and only defined by case series.[2]

The majority of presentations are symptomatic – with approximately half being local symptoms and a third related to cerebral ischaemia. Most ECAA's occur in the internal carotid artery followed by the carotid bifurcation. The natural history is seemingly unfavourable and surgical intervention is usually recommended

Definition[2]

This is also a controversial subject when it comes to carotid aneurysms as the accepted definition of an aneurysm is at least a 50% diameter increase in relation to the expected normal diameter of the aneurysm.

However, the carotid bulb is 40% greater in diameter than the distal ICA. It has been suggested that a bulb aneurysm is thus defined by 200% of the ICA or 150% of the CCA.

Carotid imaging techniques[3]

Extracranial aneurysms may be accurately assessed by duplex ultrasound if they situated are low enough in the neck.

Higher aneurysms or those associated with dissection either need investigation with MRA or CTA.

MRA has the advantage of being able to distinguish fresh from old thrombus and assess the circle of Willis. CTA has the advantage of assessing bony relations and determining surgical accessibility.

The use of catheter directed angiography is reserved for patients in whom ligation is planned. A balloon occlusion test can be performed at this time to assess tolerance to ligation. A stump pressure is performed at the same time in order to assess adequate cerebral blood flow – a pressure of >50% of mean systemic pressure is indicative of adequate blood flow.[3]

ICA occlusion should be done for 30 minutes and any neurological symptoms assessed. Neurological events are presumed to be due to thromboembolic events and emphasise the need for anticoagulation from 6 weeks to 3 months.

The accuracy of ICA occlusion and future prediction is debatable.

Recommendations for carotid imaging techniques

- Appropriate imaging of the entire extra-cranial vascular tree by CTA, MRA and/or Duplex Doppler is indicated. **(Class Iia, Level C)**

Natural History

The natural history of ECAA's can only be estimated from reviews. Initial reviews showed poor surgical outcomes, however, the majority of these aneurysms were mycotic.

As many neurological symptoms have also been described, it is thought that the natural history is unfavourable and most likely intervention should always be undertaken.

Management[1,3,,5]

Surgical

1. Ligation
   This is limited to rupture, infection and inoperable patients. Patients with a hostile surgical necks from neck irradiation and neck dissection may selectively require ligation if endovascular management is not an option.
   Ligation is seldom necessary with the advance of surgical techniques

   If ligation is planned, ICAA occlusion testing should be performed and if this fails, extracranial to intracranial bypass can be attempted. This, however,has poor results and has to be carefully considered

2. Resection
   This is the standard operation performed for ECAA's.

3. Endovascular Repair
- Endovascular intervention is an important modality for very distal aneurysms where surgery may be difficult. It is also an option for the hostile surgical neck. Endovascular techniques include: bare metal stents with and without coils and covered stents.

4. Conservative management
   This should be limited to non-operable lesions.

Recommendations

- All Carotid aaneurysms should be repaired in fit patients. **(Class IIa, Level C)**
- Endovascular techniques should be reserved for very large aneurysms, very distal aneurysms, hostile necks and patients not fit for open surgery

(Class IIa, Level B)

Medical

Medical management after surgical repairs has not been well defined although the reasoning to prevent embolic phenomena has resulted in most units using similar medications under varying protocols.

Recommendations

- Primary surgical repair is treated with aspirin post operatively. **(Class IIb,Level C).**
- Endovascular treatment: Clopidogrel 5 days pre-operatively, or loading dose of 300mg if emergent. Post-operatively dual antiplatelet therapy, clopidogrel for 6 weeks and aspiring lifelong. **(Class IIb, Level C)**

Post-operative and long term surveillance

Surgical intervention is the choice of management and results in a stroke and mortality rate of 10-12% and a cranial nerve injury rate of 6%. Transient cranial nerve injuries are reported at 20%.

These results seem favourable compared to non-operative intervention which carries a higher mortality and stroke rate.[4]

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Brachiocephalic, Subclavian and Upper Limb aneurysms

Summary

Upper limb aneurysms are very uncommon, with the most frequent being iatraogenic brachial pseudoaneurysm from cardiac cateterisation. With the advent of radial punctures, these are also likely to increase. HIV again, needs to be considered on the South African setting. Trauma is another more common cause in our country than other countries. With regards to the brachiocephalic and subclavian arteries, infectious disease processes such as HIV and TB may cause aneurysmal disease as well as inflammatory disease such as Takayasu's disease. Brachiocephalic and subclavian aneurysms only account for 1% of all peripheral aneurysms. Up to half of the aneurysms in these locations due to atherosclerosis, have aneurysms elsewhere and this should always be investigated. True subclavian aneurysms may be due to atherosclerosis. Thoracic Outlet syndrome is also a common cause of subclavian artery aneurysms. Axillary artery aneurysms are usually due to trauma, most typically from sporting injuries. Other causes of upper limb aneurysms include occupation syndromes such as Hypothenar Hammer syndrome.

Generally, all upper limb aneurysms should be treated due to unfavorable outcomes, predominantly related to thromboembolic phenomena, nerve compression and lastly rupture.

Surgical outcomes are good overall. Endovascular intervention has an important role in the more proximal arteries.

Definition

The accepted definition for a true aneurysm is "permanent localized dilatation of an artery having at least a 50% increase in diameter compared to the expected normal diameter of the artery in question". With regards to the upper limb, this varies according to the artery involved: The palmar arch artery is 1-3 mm, the radial artery is 2-4mm, the ulnar artery is 2-4mm, the interosseous artery is 2-3mm, the brachial artery is 5-7mm, the axillary artery is 6-8 mm, the subclavian artery is 6-10mm and the brachiocephalic artery is 8-12 mm.

Upper Limb Imaging Techniques [1,2,3]

Imaging will depend on the location of the suspected aneurysm. Generally, most lesions outside of the thorax can initially be imaged with duplex Doppler ultrasound.

Brachiocephalic and subclavian artery lesions.

Although duplex Doppler has been advocated by some, the imaging is limited due to the bony thoracic cage. CTA or MRA are the non-invasive tests of choice and before any revascularization is considered imaging of the brain should be performed to exclude any infarcts – these may render the patient more susceptible to reperfusion injury. Catheter directed angiogram should be reserved for inconclusive non-invasive testing.

The heart should also be evaluated as 40% of these patients have coronary artery disease. These investigations may include ECG and echo and further testing decided upon thereafter.

Axillary artery aneurysms are very important to detect and may be imaged with duplex Doppler ultrasound, CTA or MRA. If a patient has had significant trauma to the shoulder or

arm with either an abnormal pulse or a normal pulse examination with brachial plexus palsy, then he/she should be imaged with arteriography.

Brachial artery aneurysms may be diagnosed on duplex ultrasonography if they are simple.

Arteriography may be needed to assess aneurysm extension, sites of embolism and occlusion and planning for vascular reconstruction.

Hand lesions need to be identified with a catheter directed angiogram in order to assess the aneurysm and commonly associated occlusions and stenosis as well as determining if the superficial palmar arch is complete. This was thought to be the gold standard of treatment and the aortic arch as well as upper extremity can be evaluated for other pathology.

However, high resolution contrast enhanced MRA has also been used recently as non-invasive technique as well as CTA.

Recommendations

Brachiocephalic and subclavian aneurysms should be imaged by CTA or MRA **(Class IIa, Level B)**

Brain Imaging is recommended in neurologically symptommatic patients, or where there is a high risk of intra-cranial involvement **(Class IIa, Level B)**

Brachiocephalic and subclavian aneurysm should have a cardiac workup (including an ECG and echocardiogram) as part of the pre-operative planning **(Class IIa, Level B)**

Shoulder or arm trauma with an abnormal pulse, or a normal pulse but a brachial plexus palsy should be imaged using CTA or MRA **(Class I, Level B)**

Brachial artery aneurysm may be imaged with Duplex Doppler **(Class IIb, Level B)**

Natural History[1, 7, 8]

Aneurysmal disease in brachiocephalic, subclavian and upper limb arteries may lead to distal embolisation with resultant vascular symptoms ranging from claudication to rest pain and ulceration.

Compressive symptoms from brachial plexus and recurrent laryngeal nerve may occur. There is also a risk of rupture.

With regards to axillary artery aneurysms, due to the excellent collateral circulation, distal ischaemia is rare. If there is hameorrhage into the axillary sheath, brachial plexus compression may occur. Delayed treatment of this pathology has very poor outcomes.

Urgency in repair is predominantly due to the thromboembolic complications followed by nerve compression and then rupture.

Clinical Assessment[1]

Symptoms from brachiocephalic and subclavian aneurysms include pain from expansion or rupture in the upper chest or neck, embolic phenomena causing ischaemia of the cerebral circulation or upper extremity and nerve or organ compression (recurrent laryngeal, brachial plexus, oeseophagus and trachea). Erosion into the lung may also occur.

Axillary artery aneurysms may rupture into the axillary sheath causing brachial plexus compression and neuralgia. Distal ischaemia may be evident form emboli.

Brachial artery aneurysms usually result in a obivious mass and may compress the median nerve as well as cause distal ischaemia form embolisation.

Palmar artery aneurysms may result in Raynaud's syndrome or ischaemia from emobilisation.

Classification

Brachiocephalic aneurysms have been classified as follows:

Group A	No involvement of the origin of the artery
Group B	Involvement of the origin of the brachiocephalic, not involving aorta
Group C	Involvement of the brachiocephalic artery and aorta

The majority of subclavian aneurysms are in the proximal portion of which the cause is usually degenerative and the distal aneurysms almost half due to thoracic outlet syndrome.

Since the 80's the number of subclavian artery aneurysms have increased due to changes in pathology, economics and detection. Increases in trauma, atherosclerosis, post radiation, thoracic outlet and HIV have all been documented

Axillary artery aneurysms due to trauma typically occur in the 3rd portion of the axillary artery.

Management[1,4,5,6, 7]

1. Brachiocephalic

Treatment of brachiocephalic lesions depend on the cause.

Surgical treatment is advised in all symptomatic and asymptomatic patients if fit for surgery as the consequences are not only related to rupture but thromboemobilic complications. Ligation without establishing arterial continuity should be avoided due to resultant ischameic symptoms in up to 25% of patients. Endovascular intervention is suitable for inflammatory conditions to avoid resection and anastomosis in diseased vessels.

Recommendations

Brachiocphalic Aneurysms

Open surgical treatment

Type A and Type B: Resection or endoaneurysmorrhaphy and interposition grafting. **(Class IIa, Level B)**

Type C: Brachiocephalic and aortic graft replacement with or without cardiopulmonary bypass. **(ClassIIa, Level B)**

Endovascular

Endovascular intervention should be chosen in high-risk patients. **(Class IIa, Level C)**

Endovascular intervention for inflammatory conditions must be combined with medical treatment **(Class IIb, Level C)**.

2. Subclavian Aneurysms

Options depend on location. Proximal aneurysms are usually due to degeneration and distal aneurysm due to thoracic outlet syndrome.

Ligation and extra-anatomical bypass for unusual aneurysms. If due to thoracic outlet syndrome – removal of bony structure included.

Endovascular and hybrid

Morbidity and mortality rates of open surgical repair and endovascular repair are within similar ranges. Complications seem to be less severe with endovascular or hybrid repairs compared to open. Systemic complications are more associated with open surgical repair. Endovascular repair is usually the preferred choice nowadays due the decreased cardiopulmonary complications.[6]

Recommendations

Subclavian Aneurysms

Surgical Management

Resection and interposition grafting should be performed in fit patients **(Class 1, Level B)**

Ligation of unusual or difficult or septic aneurysm may be undertaken **(Class IIb, Level C)**

Endovascular and hybrid

Treatment of choice for the intrathoracic first part of left subclavian. **(Class IIa, Level B).**

3. Axillary Artery Aneurysm

Recommendations

Surgical Treatment

Open repair using interposition vein graft is the preferred surgical choice. **(Class II, Level C)**

Endovascular management should be reserved in this region for very poor operative risk **(Class IIb, Level C).**

4. Brachial Artery Aneurysm

Surgical Treatment

Open repair using interposition vein graft is the preferred surgical choice. **(Class IIa, Level C)**

Endovascular Treatment

Endovascular management should be reserved for very poor operative risk **(Class IIb, Level C).**

Concomitant medical therapy is appropriate for conditions such as Takayasu's TB and HIV.

Post-operative Outcomes

Surgical outcomes are generally good with low morbidity and mortality. Endovascular outcomes do not have record of long term results but early outcomes are good and comparable.

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