Thoracic aortic interventions

Editor: Martin Veller

The best scientific basis for any guideline or clinical recommendations is level 1 evidence derived from multiple prospective randomised trials and meta-analyses. Very often this is not available, and as a consequence recommendations have to be based upon large retrospective series, non-randomized studies or the experience of experts. In an effort to establish guidelines for South Africa, a meeting of South African vascular surgeons (listed at the end of this document) all of them members of the Vascular Society of Southern Africa (VASSA) was held in March 2011 – generously supported by the Crossroads Institute of South Africa and Baroque Medical. During this "Carotid and Thoracic Aortic Consensus Meeting all aspects concerning the management of these conditions was intensively discussed with additional input of two Belgium experts in this field (Patrick Peeters and Koen Keirse, Imelda hospital). These guidelines are therefore based upon the extensive discussions and lectures during this Consensus Meeting, as well as the latest publications and recommendations that were and have become available in the literature. Participants were also encouraged to evaluate and use guidelines developed by other societies and bodies and to if appropriate adapt these to South African circumstances. The intention is to cover the subject fully and as a result some recommendations will per force need to be repeated in some of the sections in order to ensure that each section comes to an appropriate conclusion.

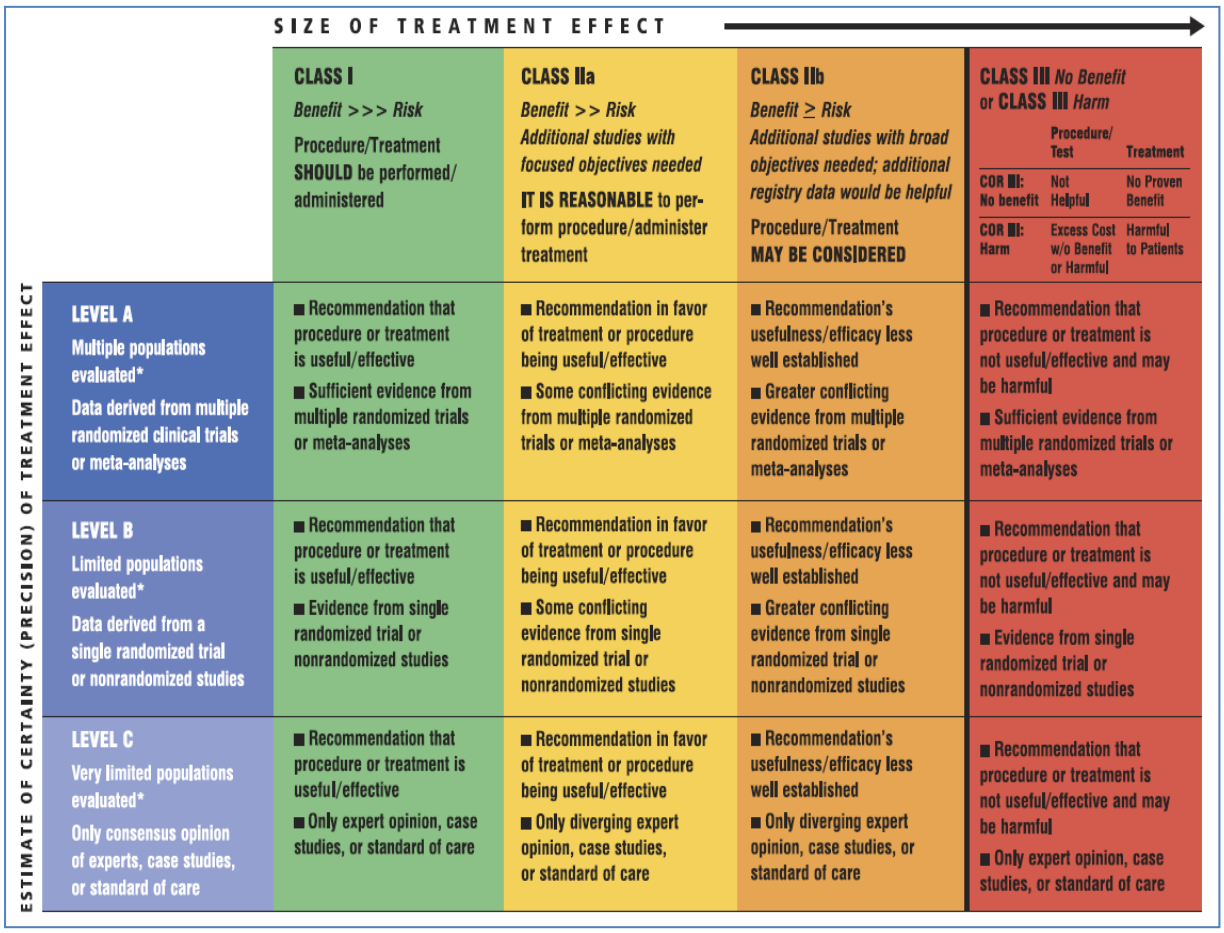
It is essential to note that these guidelines are not intended to be absolute dictates, but should provide a framework within which the reasonable physician can and should practice, and which will require exceptional circumstances to practice outside thereof. These guidelines when published will be the official guidelines of VASSA.

Presently, many new prospective trials exist or are being planned whose results may eventually change current practice. Undoubtedly future technological, pharmaceutical and other therapeutic developments and progress in the understanding of the diseases will become available. These guidelines will therefore have to be revised on a regular basis and it is envisaged that similar meetings will be held on a regular basis for this purpose.

Index:

Levels of Evidence and Class of Recommendations

It was agreed by the participants that many methods of evaluating the quality of data and making guideline recommendations on the basis of this information exist. A consistent easily applicable system is essential and as a consequence the method currently being used in most American cardiovascular guidelines would be adopted by VASSA[1]. This is reflected below:



ESTIMATE OF CERTAINTY (PRECISION) OF TREATMENT EFFECT	SIZE OF TREATMENT EFFECT →			
	CLASS I *Benefit >>> Risk* Procedure/Treatment **SHOULD** be performed/ administered	**CLASS IIa** *Benefit >> Risk* *Additional studies with focused objectives needed* **IT IS REASONABLE** to perform procedure/administer treatment	**CLASS IIb** *Benefit ≥ Risk* *Additional studies with broad objectives needed; additional registry data would be helpful* Procedure/Treatment **MAY BE CONSIDERED**	**CLASS III** *No Benefit* or **CLASS III** *Harm* Procedure/Test / Treatment COR III: No benefit — Not Helpful / No Proven Benefit COR III: Harm — Excess Cost w/o Benefit or Harmful / Harmful to Patients
LEVEL A Multiple populations evaluated* Data derived from multiple randomized clinical trials or meta-analyses	■ Recommendation that procedure or treatment is useful/effective ■ Sufficient evidence from multiple randomized trials or meta-analyses	■ Recommendation in favor of treatment or procedure being useful/effective ■ Some conflicting evidence from multiple randomized trials or meta-analyses	■ Recommendation's usefulness/efficacy less well established ■ Greater conflicting evidence from multiple randomized trials or meta-analyses	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Sufficient evidence from multiple randomized trials or meta-analyses
LEVEL B Limited populations evaluated* Data derived from a single randomized trial or nonrandomized studies	■ Recommendation that procedure or treatment is useful/effective ■ Evidence from single randomized trial or nonrandomized studies	■ Recommendation in favor of treatment or procedure being useful/effective ■ Some conflicting evidence from single randomized trial or nonrandomized studies	■ Recommendation's usefulness/efficacy less well established ■ Greater conflicting evidence from single randomized trial or nonrandomized studies	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Evidence from single randomized trial or nonrandomized studies
LEVEL C Very limited populations evaluated* Only consensus opinion of experts, case studies, or standard of care	■ Recommendation that procedure or treatment is useful/effective ■ Only expert opinion, case studies, or standard of care	■ Recommendation in favor of treatment or procedure being useful/effective ■ Only diverging expert opinion, case studies, or standard of care	■ Recommendation's usefulness/efficacy less well established ■ Only diverging expert opinion, case studies, or standard of care	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Only expert opinion, case studies, or standard of care

From: TG Brott, JL Halperin, S Abbara, et al. Guideline on the management of patients with extracranial carotid and vertebral artery disease. J Am Coll Cardiol. 2011; 57: e16-94.

References:

1) TG Brott, JL Halperin, S Abbara, et al. Guideline on the management of patients with extracranial carotid and vertebral artery disease. J Am Coll Cardiol. 2011;57:e16-94.

A. Imaging and work – up: who should be investigated

Lynne Tudhope

Thoracic aortic diseases are usually only diagnosed when an acute and often catastrophic complication occurs. The only way to detect thoracic aortic disease and determine the risk for any future complications is by means of radiological imaging, computer tomography (CT) or magnetic resonance imaging (MRI), or in some cases, echocardiographic examination.

As radiologic imaging technologies have improved in accuracy, their use as a diagnostic modality has increased. However, so has the potential risk for radiation over-exposure and contrast induced nephrotoxicity.

Because the results of treatment for stable, asymptomatic conditions are far better than treatment during the acute and often fatal presentations of thoracic aorta disease, the identification of patients at increased risk is extremely important if high morbidity and mortality rates are to be eliminated. The selection of the most appropriate imaging study is dependent on both patient factors and the availability of a specific imaging modality. The identification of individuals with genetic alterations or mutations that predispose to aortic disease is especially important.

Recommendations for aortic imaging techniques to determine the presence and progression of thoracic aorta disease

Class 1 recommendations:

- Morphological abnormalities of the aorta must be identified and reported separately even when the aortic diameter is within normal limits (Level of Evidence C)
- Any aneurysm, dissection, traumatic injury and/or aortic rupture should be immediately referred to the relevant speciality (Level of Evidence C)
- Standard reproducible anatomic landmarks for reporting the diameter of the aorta should be used. These should be measured perpendicular to the axis of blood flow. (Level of Evidence C)
- Both CT and MRI measure the external diameter perpendicular to the blood flow axis. The widest diameter of the aorta root, usually at mid sinus level, should be the landmark for root measurement. (Level of Evidence C)
- In contrast, echocardiography measures the internal diameter of the aorta perpendicular to the blood flow axis. Here too the mid sinus level of the aorta root provides a standard anatomic landmark. (Level of Evidence C)

- It is imperative that radiation exposure is kept to a minimum whenever possible. (Level of Evidence B)

Class 2a recommendations:

- The aortic diameter of any patient can be related to the patient's age, gender and body size. Tables are available for comparison. (Level of Evidence C)

Essential elements that should be reported in imaging of the thoracic aorta

- The anatomic location of aorta abnormality
- The maximum diameter and length of any dilatation
- Internal filling defects indicative of possible thrombus or atherosclerosis
- Extension of the pathology/morphological abnormality into branch vessels
- Radiological evidence of rupture such as pericardial and pleural fluid, extravasation of contrast and/or mediastinal hematoma
- Comparison of previous radiological studies, when available, to determine any increase in size of the aorta
- The presence of intramural hematoma or a penetrating ulcer

Imaging modalities

Chest X-ray:

- Used as a screening test to identify findings consistent with a dilated aorta or thoracic haemorrhage
- Can detect abnormalities of shape and size of the aorta

CT:

- Imaging of the entire aorta is possible with CT. The entire vascular tree from the aorta root to the femoral arteries and distally can be visualised including the aortic wall and lumen and this routinely provides sufficient information to plan the required intervention, be it surgical or endovascular.
- After surgery and /or intervention, CT is the preferred modality for follow up evaluation because of the presence of metallic clips and endovascular prostheses.

- Pre and post contrast studies can delineate the extent of dissection flaps, identify areas of decreased perfusion and demonstrate extravasation associated with aortic rupture.

MRI:

- The sensitivity and specificity of MR in identifying and diagnosing thoracic aorta disease is equal to CT techniques, but it is not as freely available as CT.
- The advantage of MR over CT from a patient perspective is reduced radiation and non exposure to iodinated contrast agents. However, gadolinium usage also poses a risk for patients with renal insufficiency.

Echocardiography:

- This modality can be used to detect the presence of aorta pathology as well associated cardiac abnormalities
- One of its major limitations is the appearance of reverberation artefacts overlying the lumen of the aorta which may be mistaken for a dissection flap.
- Due to the fact that echocardiography measures the internal diameter of the aortic lumen, when there is intraluminal clot, wall inflammation or aortic dissection present, an inaccurate measurement of the true size of the aorta might be obtained using this modality.

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B. Thoracic aneurysms: who and how to treat

Cobus van Marle

An aneurysm of the descending thoracic aorta (dTA) is defined as a permanent, localised dilatation with an increase in diameter of at least 50% compared to that of the normal contiguous aorta. The normal diameter for the dTA ranges from 2.45cm to 2.64cm in women and 2.39cm to 2.98cm in men.[1] The descending aorta grows at a rate of about 0.19 cm/year.[2] The larger the aorta, the faster it grows and the higher the risk for rupture. Annual event rates of rupture, dissection or death in dTAA's larger than 6 cm are 15.6%.[3] The risk of these complications increases at an exponential rate after the aorta reaches a diameter of 6cm. It is therefore recommended that asymptomatic aneurysms with a diameter of $\geq$ 5.5 cm should be repaired unless life expectancy is limited or the quality of life is substantially impaired.[4] There is no evidence that asymptomatic aneurysms < 5.5 cm benefit from surgical repair unless there is an underlying connective tissue disorder or a positive family history for aortic rupture or dissection where smaller criteria may apply.[4] Symptomatic aneurysms should be treated regardless of size if there are no contra indications.

Endovascular stent-grafting

Data compiled from 17 published series of thoracic endovascular aneurysm repair (T-EVAR) containing 1342 patients report a peri-operative mortality of 5.7%,and a stroke and paraplegia rate of 2.9 and 1.5% respectively.[4] A systematic review and meta-analysis of the peri-operative results of endovascular stenting and open surgery for thoracic aortic disease found that T-EVAR reduced peri-operative mortality (OR: 0.44, 95% CI: 0.33-0.59) and neurological morbidity (OR: 0.42, 95% CI: 0.28-0.63) in stable patients undergoing endovascular repair of dTAAs.[5] In addition cardiac complications, transfusions, reoperation for bleeding, renal dysfunction, pneumonia and length of stay were reduced. The best available comparative information on the results of open surgery versus stent-grafting in patients with dTAAs was provided by 2 non-randomized controlled clinical trials. In the Gore TAG phase 2 trial operative mortality and spinal cord ischaemia were significantly lower in the T-EVAR group compared to the open surgical group, 2.1 vs 12% (p< 0.001) and 3% vs 14% (p<0.003) respectively.[6] ICU stay, total hospital stay and return to normal activity were twice as long in the open surgery group. In the Zenith TX2 study, the 30 day survival rate was statistically non-inferior for the T-EVAR group compared to the open group (98.1% vs 94.3% p<0.01) and the severe morbidity composite index and cumulative major morbidity scores were significantly lower at 30 days for T-EVAR compared to the open group.[7] The re-intervention rates were similar in both groups.

Patient selection

Patients with dTAA'a have a decreased life expectancy compared to an age and sex matched normal population. Survival after T-EVAR was dismal in stent-graft patients who were not considered fit for open surgery due to multiple co-morbidities.[8] Five and eight year actuarial survival for this patient cohort was 31% ± 6% and 28% ± 6% respectively compared with 78% ± 6%, and 38% ± 12% for those patients who were candidates for open surgery. It is therefore recommended that patients who are not candidates for open surgery, should not be considered for endovascular repair.

Ruptured descending thoracic aortic aneurysms (rdTAA)

Patients with rdTAAs who have been treated with T-EVAR have a reduced 30 day mortality compared to open surgery (11.4% - 18.9%) versus (22.2% - 33.3%).[9-11] The incidence of stroke and paraplegia was also significantly reduced after T-EVAR.

Infected aneurysms

T-EVAR has been used in infected dTAAs with early peri-operative mortality rates ranging from 0-11.5% and 3 year survival of 57.8%. Freedom from re-intervention was 81.2% at 2 years.[12-14] T-EVAR into the native aorta has a decreased risk for recurrent infection, compared to the T-EVAR for septic graft infection. Post-operative antibiotics is recommended for at least 6 weeks and preferably life-long where endografts are maintained in an infected field.[14] HIV-related aneurysms are not uncommon in South Africa and the results of surgical treatment for peripheral and abdominal aneurysms have been published.[15,16] There is only limited experience with endovascular repair, but it seems feasible that T-EVAR should be considered.

Recommendations

Symptomatic aneurysms should be repaired when feasible.[4,17] (Class 1, Level of Evidence A)

For patients with degenerative aneurysms with a diameter exceeding 5.5cm, endovascular grafting should be strongly considered when feasible. [4,17] (Class 1, Level of Evidence B)

Endovascular management is not indicated for asymptomatic aneurysms smaller than 5.5cm. (Class 3, Level of Evidence C)

Endovascular stent-grafting appears to be the preferred treatment of ruptured aneurysms when is method is feasible. (Class 2a, Level of Evidence B).

Endovascular treatment is reasonable in the treatment of infected aneurysms. (Class 2a, Level of Evidence C).

Patients who have a limited life expectancy or poor quality of life due to medical co-morbid conditions, should not be offered any vascular management of their aneurysms. (Class 3, Level of Evidence C).

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C. Aortic dissection: who and how to treat

Talib Abdool-Carrim

Type A or type B dissection patient, once diagnosed, must be admitted to an acute Intensive Care Unit as medical therapy is the mainstay of early treatment of aortic dissection.

Reduction of systolic blood pressure to between 100 and 120 mmHg with close monitoring of renal function and pain relief are priorities. This is achieved by using Morphine and intravenous beta-blocking agents (metaprolol, esmolol or labetalol) or in combination with vasodilating drugs such as nitroprusside or angiotensin converting enzyme inhibitors. Intravenous veraprimil or diltiazem may be used if beta-blockers are contraindicated.

Additionally the heart rate should be kept low: a heart rate of below 60 beats per minute significantly decreases secondary adverse events.[1]

In a series of 384 patients with type B dissection from the International Registry of Acute Aortic Dissection (IRAD), 73% were managed medically with an in hospital mortality rate of 10%.[2] Short term survival was 91% at 1 month and 89% at 1 year. The long term survival varies between 60 to 80% at 4 to 5 years and is around 40 to 45% at 10 years. Predictors of early mortality were malperfusion, hypotension and partial thrombosis of false lumen.

Treatment of Type A Aortic Dissection

Surgery is the usual treatment for type A Dissection. In the IRAD registry the overall mortality with open surgery was 25%. Mortality was much higher in patient with instability or malperfusion phenomena. Long term outcome are reasonable with 5 and 10 year survival at 54 – 71%. Stevens et al reported that 10, 15 and 20 year survival is 55%, 48% and 30% respectively.[3] Over the past 20 years there has been a reduction in the hospital mortality.

Endovascular treatment of type A dissection is still considered to be investigational and limited to some centres with extensive experience.

Controversies in surgical treatment with Type A Aortic Dissection

The management of the persistent false lumen in the distal aorta once the type A dissection has been repaired – by addressing the entry point with an interposition graft – but leaving the distal false lumen to occlude by thrombosis, remains unclear. The long term follow-up of acute dissection indicates that as many as 70% of false lumens do not thrombose and are at risk of expansion. For this reason some authorities place an frozen elephant trunk in the distal arch and

proximal descending aorta at the time of repair of acute type A dissection. This procedure does prolong the emergency operation but good results have been achieved when performed by experts. No randomised is however available and as a consequence the controversy continues.

Treatment of Type B Aortic Dissection

Uncomplicated type B dissection:

Patient with type B dissection are initially managed with medical therapy as already outlined. Survival rate 89% at one month and 84% at one year have been reported. Traditionally surgery is reserved for complications of dissection, namely malperfusion, rupture, ischaemic limbs, persistent or intractable pain, progression of dissection and uncontrolled hypertension.

Given the reasonable results for medical therapy for uncomplicated type B dissection medical therapy is still the gold standard as surgical intervention was associated with a mortality of 31%, also the risk of irreversible spinal cord injury and operative death can range from 14 – 67% respectively.

Endovascular therapy is emerging as a strong alternative to open surgery with less morbidity and mortality. Among those patients with acute type B dissection more than 60% of deaths were due to rupture usually due to false lumen patency. The endovascular stent graft may achieve closure of false lumen (or partial thrombosis) and therefore protect from false lumen enlargement.

The INSTEAD trial, the first prospective randomized trial of elective stent graft placement in survivors of uncomplicated chronic dissection (> 14 days), reported that thoracic stent graft (TEVAR) placement failed to improve 2 year survival rates and adverse events when compared with optimal medical treatment.[4] Based on the outcome of the INSTEAD trial chronic uncomplicated aortic dissection presently should be managed medically.

The ABSORB trial is still ongoing and no results have as yet been published.[10] This study is addressing the placement of TEVAR in acute (< 14 days) uncomplicated type B dissection versus best medical treatment. This trial hopefully will address the exact role of stent grafting in acute uncomplicated type B dissection.

Complicated Type B Aortic Dissection

Patients with complicated, unstable type B aortic dissection manifesting with renal or visceral ischaemia have an operative mortality of 50 to 88% respectively. Aortic stent grafting in this instance is an attractive alternative. Fattori et al on behalf of the IRAD Registry reported that in hospital complications occurred in 20% with TEVAR as compared to 48% with open surgery.[5] The in- hospital mortality with TEVAR was 11% and it was 33% after open surgery. A meta-analysis in 2008 by Parker et al

found TEVAR to have an in hospital mortality rate of 9% and major complications at 8.1%.[6]

However, at present there are no good randomized, long term studies evaluating TEVAR in acute, complicated type B aortic dissection.

Recommendations:[7]

- Acute thoracic dissection involving the ascending aorta (type A dissection or type B dissection with extension into the ascending aorta) should be urgently evaluated for emergency surgical repair. (Class 1, Level of evidence B)
- In acute (<14 days old), uncomplicated type B aortic dissection the current treatment is pharmacological blood pressure control primarily using beta-blockers. (Class 1, Level of evidence B)
- In acute, complicated type B aortic dissection an endovascular first therapeutic approach is recommended (Class 2a, Level of Evidence B). This may require Petticoat replacement of the stent or individual visceral artery stenting if malpefusion perfusion persists[8] (Class 2a, Level of Evidence C).
- Chronic type B aortic dissection with one of the following criteria may be considered for TEVAR: An increase in the aneurysm size (1 cm/year), a patent or partially patent false lumen, aneurysm size of 5.5cm at presentation, extension into ascending aorta. (Class 2a, Level of Evidence B)

Areas of Uncertainty:

- The role of TEVAR in type A aortic dissection which has been repaired but where a patent false lumen is found in the distal descending aortic dissection.
- Role of TEVAR in acute uncomplicated type B aortic dissection.

Future Direction:

- Results from the ongoing randomized study (ADSORB Trial) to determine if TEVAR is better than optimal medical therapy in acute uncomplicated Type B Dissection.
- Randomized study to address surgery versus TEVAR in complicated aortic dissection.
- Long term follow up of TEVAR used for aortic dissection.

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D. Thoracic aortic trauma: who and how to treat

Nad Naidoo

Thoracic aortic injuries, irrespective of mechanism of injury, are associated with a mortality rate of 85% at the scene of injury. Approximately 60 to 70% of patients reaching hospital are unstable on admission.

Penetrating injuries account for 90% of all thoracic vascular injuries. Open surgical repair of penetrating aortic injury remains the standard of care. Thoracic endovascular aortic repair (TEVAR) is reserved for patients who are not suitable for surgery.

The vast majority of blunt traumatic aortic injuries (BTAI) involve the isthmus (90%). These injuries account for approximately 10% to 20% of the mortality in motor vehicle accident patients. There is an in-hospital mortality rate of 20% to 25% during the triage period, and a 50% mortality rate during the first 24 hours. Patients generally have multiple injuries and the injury severity score (ISS) averages 40. Emergency surgical aortic open repair (OR) is associated with a mortality rate of between 15% and 25%, a stroke rate of approximately 5% and a paraplegia rate of between 8% and 20%. Evolving experience has shown that delaying definitive surgical treatment, in select cases, together with blood pressure control is safe and associated with better outcomes. This may allow competing, life threatening non-aortic injuries to be treated initially.

TEVAR is rapidly evolving as a safe and effective alternative to open surgical repair. Benefits include avoidance of a thoracotomy, single lung ventilation, extra-corporeal circulation, aggressive anti-coagulation, and aortic cross-clamping. It has been used to facilitate treatment in patients who are human immunodeficiency virus positive, and those with compelling aortic anomalies.

Coverage of the left subclavian artery is associated with arm ischaemia (6%), spinal cord ischaemia (4%), vertebro-basilar ischaemia (2%) and anterior circulation stroke (5%). A review of the literature together with recommendations has been recently published. A selective approach to coverage of the left subclavian artery is advised.[1]

Two recent meta-analyses have shown superior outcomes for TEVAR compared to OR.[2,3] Both studies have shown a statistically significant decrease in procedure-related mortality, 30 day mortality and paraplegia rate in favour of TEVAR. Tang et al have also shown a significant decrease in stroke rate and a trend towards a significant decrease in overall complication rate in favour of TEVAR.[3] There is a trend towards better cumulative survival rates beyond 5 years in patients who had a TEVAR.[4] In a recent prospective, non-randomized, multicenter study comparing TEVAR (125 patients) with OR (68) patients there was a statistically better mortality rate, less systemic complications, less local complications, less blood transfusions and reduced length of hospital stay in favour of TEVAR.[5] There were 32 device

related complications and 18 endoleaks (6 needed repair), the majority of these complications occurring in low volume centres.

Technical details regarding TEVAR for BTAI have evolved and current evidence including procedural practice guidelines has been published. Peri-procedural anticoagulation is not always necessary. Post deployment stentgraft balloon moulding is also not necessary.[7]

Inferior vena cava filters are not routinely deployed during this procedure.

A recent global survey of TEVAR (1180 cases) revealed a utility rate of ~ 10% for acute traumatic aortic disruption.[6]

There is no evidence for long-term anticoagulation or dual anti-platelet therapy for TEVAR in trauma.

A comprehensive literature review was undertaken on behalf of the Society of Vascular Surgery. A Clinical Practice Guideline document was recently published outlining the evidence and recommendations regarding TEVAR for thoracic aortic injuries.[7]

More recent data has enabled more practical attempts at classification of these lesions and defining the natural history of minimal aortic injuries (intimal tear < 10 mm without aortic contour defect) as benign. These lesions usually heal spontaneously without surgery or stentgrafting. Close follow-up, however, is indicated.[8]

Strength of the data

There are no randomized controlled trials comparing TEVAR vs. OR for thoracic aortic injuries. Many of the recommendations are at best grade 2 (few grade 1) based on level C evidence.

Areas of uncertainty

The long-term durability of the procedure and the device remain to be defined with long term follow-up.

Follow-up diagnostic modalities need to be defined. Serial plain 3 projection chest x-rays are a reasonable recommended (3 monthly for 1 year, 6 monthly in the second year, then annually). A MDCT-Angiogram is mandatory in the first 30 days post TEVAR. A repeat MDCTA is indicated at 6 months, then annually for 5 years. This is essential until we have more evidence about the 5 year follow-up results to inform a revision of this strategy.

Future directions

It is unlikely that a randomised controlled study would be performed considering the low annual yield even in dedicated trauma centres, as well as the ethical considerations. Both are complimentary strategies and patient management should be individualized depending on local resources, expertise and experience.

Recommendations:

- TEVAR is the current recommended first line of therapy for BTAI but should be performed with suitably sized devices. (Class 2a, Level of Evidence C)
- Aortic injuries should be treated in dedicated high-volume trauma centres where adequate resources and expertise is available. (Class 2a, Level of Evidence B)
- A multi-disciplinary approach is recommended. (Class 2a, Level of Evidence B)
- Initial non-operative management of intimal tears < 10 mm with anti-impulse therapy and aspirin. Close follow is indicated. (Class 2a, Level of Evidence B)

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E. Aortic hybrid repairs: current outcomes

Nad Naidoo

Aortic hybrid repair (AHR) refers to the application of a combination operative and endovascular treatment modalities to treat complex aortic pathologies, especially in high risk patients. These patients generally have inappropriate and/or inadequate landing zones for pure aortic stentgrafting. The components of AHR require aortic debranching with branch revascularization, with simultaneous or staged aortic stentgrafting. By definition AHR requires debranching and revascularization of 2 or more aortic branches. AHR may be applied to treat complex aortic arch disease or thoraco-abdominal aortic aneurysms (TAAA). Much of the experience to date relate to high risk patients, is very limited and restricted to major referral centres.

Good clinical outcomes were published by Zhou et al.[1] They reviewed 31 patients (16 arch, 15 TAAA, 11 had previous aortic repairs, 10 had staged procedures). Technical success was 100%, with a peri-operative mortality rate of 2.2% and peri-operative complication rate of 19.2%. There were no stroke or paraplegia documented in this series. Ham et al recently described their experience with 51 patients (27 arch, 24 TAAA, 39% symptomatic, 75% staged procedures).[2] The peri-operative mortality was 3.9%, morbidity rate 39%, stroke rate 4% and paraplegia 2%. Procedure-related complications were significant (26%). Technical success was 87%. Actuarial survival was 86% at 1 year and 67% at 3 years.

For arch AHR patients the endoleak rate was higher if the procedure was performed without cardio-pulmonary bypass.

A systematic review of AHR for complex arch disease identified 18 studies (195 patients).Mean technical success rate was 86% (range: 69% - 100%).[3] Type I or type III endoleak was seen in 9%. Conversion to open surgery was seen in 3%. Mean peri-operative mortality was 9% (range: 0% - 25%). Mean peri-operative morbidity was 14% (range: 0% - 50%), stroke rate 14% and paraplegia 0.5%. Interestingly these results approach those for pure open repairs.

Very few institutions have a large experience with AHR for TAAA. Chiesa et al described 41 patients (76% simultaneous procedures).[4] Technical success was achieved in all (no intra-operative deaths). Peri-operative mortality was 13%. Peri-operative morbidity was 32% and paraplegia rate was 2.4%. Similar results were documented by Quinones-Baldrich et al.[5]

Interestingly in high risk patients with previous aortic surgery, AHR for TAAA did not lead to an improvement in outcomes compared to pure open repair.

Patel et al compared AHR (23 patients) with open repair (77 patients) in patients with TAAA.[6] The AHR group had higher SVS scores, more COPD and more patients with

previous aortic surgery. When adjusted for SVS scores < 8, the AHR group had a higher composite 30 day mortality and/or permanent paraplegia rate (p: 0.03)

Two systematic reviews were published. Donas et al identified 13 studies (58 patients).[7] Mean TAAA diameter was 7.15 cm (range: 5 – 12 cm). The majority had type I, II or III TAAA. Primary technical success was 100%. Stentgraft related complications at 30 days were 18.9%. Overall endoleak rate was 20.6% (re-intervention required in 13.7%. Two patients developed stroke. No patient developed paraplegia. Peri-operative mortality was 10.7% (overall mortality was 15.5%). Peri-operative morbidity was 48.2%. None of the 3 ruptured TAAA patients survived. A more recent systematic review (15 studies, 108 patients) found similar results.[8]

Strength of Data

There are no large randomised controlled studies. Much of the experience relates to case reports and institutional case series (level C evidence). The outcomes of AHR in high risk patients are modest at best. There is no compelling evidence that is totally for or against AHR currently in high risk patients. The role of AHR in conventional risk patients is still to be defined.

Areas of Uncertainty

The role of AHR in conventional risk patients is still to be defined. Selecting out a sub-group that would benefit from AHR in high risk patients has been problematic. Whether these high risk patients' benefits from AHR compared to no treatment is unclear.

Future Directions

The long-term outcomes of AHR in high risk patients still need to be defined. A RCT comparing AHR vs. no treatment for TAAA in patients considered high risk for surgery is needed.

Recommendation

AHR should be performed in carefully selected patients in dedicated specialised high volume centres.

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Participants:

Talib Abdool-Carrim, University of the Witwatersrand, Johannesburg

Ruan Botha, University of the Free State, Bloemfontein

Charl Dreyer, Cape Town Medi Clinic, Cape Town

Martin Forlee, Kingsbury Hospital, Cape Town

Tiago Fernandes, University of the Witwatersrand, Johannesburg

Dirk le Roux, University of the Witwatersrand, Johannesburg

Lewis Levien, Fourways Hospital. Johannesburg

Philip Matley, Kingsbury Hospital, Cape Town

Kris Michalowski, Milnerton Medi Clinic, Cape Town

Pradeep Mistry, University of the Witwatersrand, Johannesburg

Thanyani Mulaudzi, University of Pretoria, Pretoria

Nad Naidoo, University of Cape Town, Caoe Town

Jay Pillai, University of the Witwatersrand, Johannesburg

Bala Pillay, University if Kwa-Zulu Natal, Durban

John Robbs, Entabeni Hospital, Durban

Lynne Tudhope, Montana Hospital, Pretoria

James Tunnicliffe, Kingsbury Hospital, Cape Town

Cobus van Marle, Unitas Hospital, Pretoria

Martin Veller, University of the Witwatersrand, Johannesburg

Cobus Vermaak, University of the Witwatersrand, Johannesburg