Aortic Aneurysm: Awareness, causes and management.

This article was written by council members of the Society of Vascular Nurses

Leanne Atkin, Lecturer Practitioner/Vascular Nurse Specialist, School of Human and Health Sciences, University of Huddersfield and Mid Yorkshire NHS Trust, E mail: l.atkin@hud.ac.uk

Michael van Orsouw, Vascular Matron Guys and St Thomas' NHS Foundation Trust Email: Michael.vanorsouw@Gstt.nhs.uk

Emma Bond, Vascular Nurse Specialist, Glan Clwyd Hospital, North Wales. Email: emma.bond2@wales.nhs.uk

Rachael Lear, Advanced Vascular Nurse Practitioner, St. Mary's Hospital, Imperial College Healthcare NHS Trust. Email: rachael.lear@imperial.nhs.uk

Introduction

The term aneurysm describes a localised widening or ballooning of a section of in a artery, an increase of greater than 50% of normal size is defined as aneurysmal, (Upchurch & Criado, 2009). Aneurysmal changes can be found in any artery but are most commonly seen in the aorta, (Nienaber & Fattori, 2012). Aneurysms can be asymptomatic or symptomatic but may be life threatening if there is sudden dissection or rupturing. This article describes the differences between abdominal and thoracic aneurysms and explores the incidence, causes and treatment of thoracic aneurysmal disease, highlighting the importance of vigilant post-operative care.

Definition of aortic aneurysm and Incidence

Aneursyms are classified by location; 80% of all aneurysms are Abdominal Aortic Aneurysms (AAA) with 10% categorised as Thoracic Aorta Aneurysms (TAA), (Nienaber & Fattori, 2012; Fournier & Zanoff, 2012). The aorta is anatomically divided into two sections: the thoracic aorta which runs from the heart to the diaphragm and the abdominal aorta which continues from the diaphragm to the aortic bifurcation. The thoracic aorta is further divided into 3 sections: the ascending aorta (which contains the aortic root, sinuses of valsalva & a tubular segment); the aortic arch and the descending aorta, (Topol, 2007). Approximately 60% of all thoracic aneurysms involve the aortic root or the ascending thoracic aorta, 10% involve the arch, 40% involve the descending aorta, and the remaining 10% involves both the thoracic and abdominal aorta (thoraco-abdominal), (Isselbacher, 2004).

Thoracic Aneurysm Symptomology

The majority of aneurysms are asymptomatic on diagnosis, having been found incidentally on imaging procedures for other health concerns. In the case of thoracic aneurysms if they do cause symptoms for the patient this is normally related to the direct compression of other intrathoracic structures. Such compression can cause breathing difficulties due to tracheal deviation, difficulty swallowing due to compression of the oesophagus, or hoarseness of voice due to compression of the laryngeal nerve, (Stoetling et al, 2012). Occasionally patients who have an aneurysm of the ascending aorta complain of deep, aching chest pain and/or back pain which can radiate to the neck and jaw. In the case of descending aortic aneurysms the pain is more commonly found in the back, between the shoulder blades. Those who have symptomatic thoraco-abdominal aneurysms may complain of lower back pain. Rapidly expanding thoracic aneurysms can cause severe chest pain and should be viewed as a precursor to rupture, (Nienaber & Fattori, 2012).

Rupture can at times be the first indication of the presence of an aneurysm, resulting in massive haemorrhage into the mediastinum, the pleural space, which can affect the oesphophagus and the bronchial tree. If the site of rupture is in the ascending aorta this may result in a haemopericardium and cardiac tamponade. A rarer symptom is that of haemoptysis, which can indicate a rupture of the descending thoracic aorta, the haemoptysis is the result of the aorta become adhered to one of the lungs, (Nienaber & Fattori, 2012).

In addition to aneurysms the aorta can also suffer from dissection, although uncommon, aortic dissection begins with tear in the inner layer of the aortic wall normally within the thoracic aorta. Dissection occurs when the innermost layer of the aortic wall (the intima) weakens. This results in a tear or intimal flap, which allows blood to be channeled into the wall of the aorta. The pressure of the blood separates the arterial layers leading to the development of a false lumen. Aortic dissection is often described using the DeBakey classification (fig 1), which clearly defines the severity of the dissection and its location. Patients suffering acute dissection often complain of acute chest or back pain and in some cases cardiac tamponade can occur, (Baliga et al, 2007). The cause of aortic dissection is unclear and remains under investigation. However, there are several risk factors associated with aortic dissection: these include hypertension, connective tissue disorders, atherosclerosis, cystic medial disease, aortitis and trauma, (Foster & Turley, 2016).

Causes

The mechanisms for initiating and stimulating the progression of aneurysms remains poorly understood, (Golledge & Norman, 2010). Different factors are thought to drive aortic dilation and

rupture at different aortic sites, (Norman and Powel 2010). The exact cause of most aortic aneurysms is unknown, (Sakalihasan et al., 2005). However, in some specific cases the cause of the aneurysm can be identified. These include: trauma; acute infection (brucellosis, salmonellosis); chronic infection (tuberculosis); inflammatory diseases (Behçet and Takayasu disease) and connective tissue disorders, (Sakalihasan et al., 2005). There are several syndromic and non-syndromic genetic conditions that are associated with the development of thoracic aortic aneurysms including Marfan's syndrome, Loeys-Dietz syndrome, Turner syndrome, bicuspid aortic valve, and other genetic mutations, (Hiratzka et al 2010).

Risk Factors

Using medical profiles of patients undergoing treatment for aortic dissection, ruptured aneurysms and electively treated aneurysms has allowed the identification of specific factors which increase the risk of developing aneurysmal disease. These include:

Smoking: This is a risk factor across the entire range of aortic diseases, although with different risk magnitudes for different diseases, (Landenhead et al 2015). The abdominal aorta appears to be particularly susceptible to smoking, (Jovin et al 2012). The prevalence of AAA's in tobacco smokers is more than four times that of life-long non-smokers, (Sakalihasan et al., 2005). Smoking is also a risk factor for aortic dissection and thoracic aortic aneurysm, (Landenhead et al 2015). Smoking is the main modifiable risk factor that has been associated with the development, expansion and rupture of AAA, (Brady et al., 2004). However no such association has been proven for thoracic aortic aneurysm, (Sidoff et al 2014).

Hypertension: In a study by Landenhead et al., (2015) hypertension was present in 86% of individuals who subsequently developed aortic dissection. Howard et al., (2013) suggest that half of aortic dissection events could be prevented by pharmacological intervention in hypertensive subjects. Studies have also demonstrated significant association between AAA and hypertension, (Kent et al., 2010). However, hypertension is not significantly associated with thoracic aortic aneurysms, (Landenhead et al., 2015).

Age and sex: Increasing age and the male sex have been shown to increase the risks of AAA, TAA and aortic dissection, (Forsdahl et al., 2009; Landenhead et al., 2015). In men the incidence of AAA is approximately 25 per 100,000 at age 50, increasing to 78 per 100,000 in those older than 70 years, (Hay et al., 2009). This is compared to a much lower prevalence in women, 13 per 100,000 aged over 65, (Scott et al., 2002).

Atherosclerosis: Atherosclerosis is associated with all types of aneurysmal disease, with a large proportion of patients having a history of coronary disease or peripheral arterial disease. However, whether there is any association in terms of causation of the aneurysms is still under debate, (Norman and Powel 2010). This link may be simply due to having similar risk factors for the development of both conditions. Furthermore, aneurysms rarely form in locations that are particularly prone to atherosclerosis (e.g., superficial femoral artery), (Norman and Powell, 2010) which suggests association rather than causative, (Kent et al., 2010). Interestingly diabetes does appear to offer some protection against the formation of AAA, (De Rango et al., 2014) and thoracic aneurysms, (Landenhead et al 2015).

Imaging

Preferred diagnostic modalities are Computerized Tomography (CT) or Magnetic Resonance Imaging (MRI). Contrast-enhanced CT or CT angiography can be used to ascertain the extent of aneurysmal dilatation, (Ramanath et al. 2009). It is recommended that patients in whom thoracic aneurysms are suspected undergo CT imaging of the entire aorta, as up to 20% of patients with thoracic aortic aneurysms also have distal involvement, (Booher et al., 2011).

Treatment options/decision making

Treatment options for thoracic aortic aneurysm are open surgical repair, endovascular stent grafting or conservative management, (Sastry et al., 2015). In order to make decisions regarding the treatment of thoracic aortic aneurysms, the location, size and shape of the aneurysm must be established. The risk of rupture largely (but not exclusively) depends on aneurysm size, though a range of factors influence the risks of intervention. All patents with aortic aneurysms are discussed within a Multi-Disciplinary Team (MDT) meeting, these meetings include inputs from vascular interventional radiologists, vascular consultants, anaesthetist consultants and specialist nurses. The MDT plays a key role in reviewing the risks and benefits of intervention on an individual patient basis and formulates a collective recommended treatment pathway.

Intervention is normally considered once the TAA reaches 5.5cm or is growing at a rate of more than 0.5cm per year. Below this threshold patients will undergo surveillance of their aortic diameter and will receive antihypertensive medication to reduce the risk of rupture or dissection, repeat imaging

is completed on a 3, 6 or 12 months basis depending on current size. Patients will be optimised in terms of risk factor reduction which includes best medical therapy to reduce overall cardiovascular risk, this may include antiplatelet medication, statin treatment, optimisation of diabetic control, and smoking cessation. Ultimately the size at which treatment is recommended is individually assessed and can be higher in patients who have significant comorbidities or lower in certain patients, for example a patient with Marfan's or Loeys-Dietz syndrome or in patients who are symptomatic, (Goldfinger et al., 2014).

The location of the aneurysm is an important factor when making treatment decisions. Aneurysms of the ascending aorta are usually treated surgically, by replacing the aneurysmal portion of the aorta with a Dacron graft in the operating theatre setting under general anaesthetic. For aneurysms of the descending aorta exceeding 5.5cm and for thoraco-abdominal aortic aneurysms, endovascular therapy is now the treatment of choice, as the surgery is less invasive with a shorter recovery time, (Svensson et al., 2008). These patients are usually treated in an interventional radiology suite and the team performing the procedure includes both a radiologist and radiographer in addition to the surgical, anaesthetic and scrub teams. However, around 25% of patients are not anatomically suitable for endovascular repair. A common reason for patients in this cohort to be turned down for endovascular repair is a lack of favourable 'landing zones' for the stent above and below the aneurysmal portion of the aorta. Unfavourable landing zones increase the risk of endoleak (blood flow into the aneurysm sac), (Booher & Eagle, 2011). Patients who present with arch aneurysms may be treated surgically, but for those who are deemed particularly high-risk, hybrid options exist that involve endovascular stent grafting of the thoracic aorta (TEVAR) (Figure 2), with extra-anatomic bypass (carotid-carotid or carotid-subclavian) to permit stent-graft coverage of the origin of the carotid artery, (Goldfinger et al., 2014). These hybrid procedures are performed in specialist complex aortic centres, so the patient may need to travel further afield to receive specialist treatment. For patients who are treated with endovascular techniques, lifelong follow-up in the form of regular CT surveillance is required to check for the development of endoleaks, device migration, or stent-graft failure, (Sharma & Kyriakides, 2007).

Complications

Morbidity and mortality rates after thoracic aortic aneurysm repair are high, complication rates depends on patients ability to undergo extensive surgery, patients at higher risk of morbidity and

mortality include the elderly, patients with significant co-morbidities especially renal disease, respiratory insufficiency and cardiac disease. Patients are therefore closely monitored post-operatively in either intensive care or high dependency settings. One of the most feared consequences of thoracic aortic aneurysms repair is paraplegia, due to changes in the blood supply to the spinal cord this can be as a result of ischaemia or reperfusion. Paraplegia is not only associated with severe disability and poor quality of life, but also with decreased survival rates, (Svensson et el. 1991). Paraplegia can affects up to 22% of patients undergoing thoraco-abdominal aortic aneurysm repair, though the risk for those undergoing isolated thoracic repair is reduced, (Bicknell et al., 2009). Various techniques have been shown to reduce the risk of spinal cord ischaemia, including revascularisation of intercostal arteries supplying the cord and spinal cord cooling, (Bicknell et al., 2009). Post-operatively, nurses working in critical care settings play a key role in maintaining a high mean arterial pressure (often through inotropic support) and in managing cerebral spinal fluid (CSF) drainage, in order to optimise cord perfusion. Spinal cord perfusion is directly associated with mean arterial pressure and CSF pressure. Cross clamping of the aorta or coverage of the arteries supplying the cord with an endograft results in spinal cord ischaemia, leading to increased CSF production and a rise in CSF pressure. CSF drainage is therefore a key adjunct to prevent spinal cord compartment syndrome secondary to spinal cord oedema, (Murakami et al., 2004).

Conclusion

The formation of aneurysms is multifactorial and complex. Aneurysms can involve different parts of the aorta and currently there are a variety of treatment options available. The MDT plays a vital role in assessing each patient individually to ascertain treatment options and calculate anaesthetic risk. This is balanced against risk of rupture and risk of complications aiming to establish a collective decision of which is the most effective treatment and at what time/size this should be performed. Patients undergoing intervention to repair thoracic aneurysms are at risk of life changing complications. Increasing practitioner knowledge and understanding of aneurysm formation, risk factors and surgical intervention can help to ensure patients receive optimal nursing care reducing overall risk of complications and mortality.

Figure 1

Types of Aortic Dissection

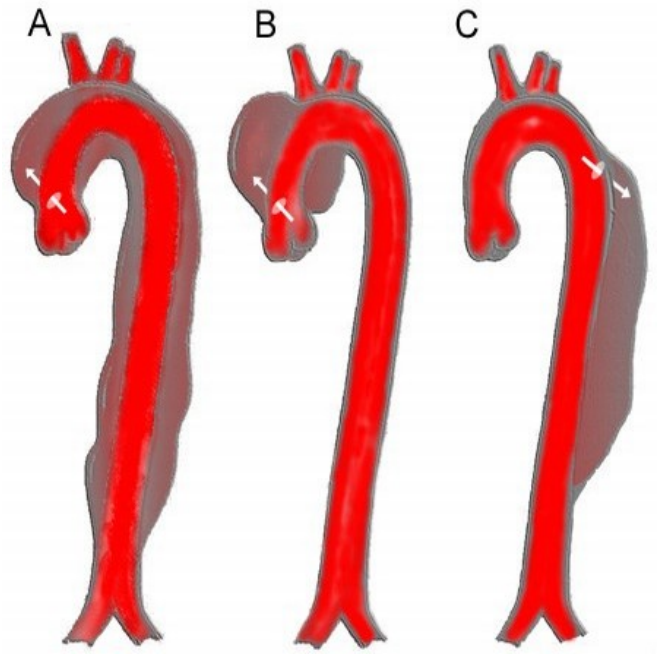

Figure 2 - TEVAR

References

Baliga, R. R., Nienabar, C. A., Isselbacher, E. M. & Eagle, K. A. (2007). *Aortic Diseection and Associated Syndromes.* Springer. USA

Bicknell CD, Riga CV, Wolfe JHN (2009) Prevention of Paraplegia during Thoracoabdominal Aortic Aneurysm Repair, *Eur J Endovasc*; 37: 654-660.

Booher AM, Eagle KA, (2011) Diagnosis and management issues in thoracic aortic aneurysm, *Am Heart J*; 162: 38-46.e1.

BRADY, A., THOMPSON, S., FOWKES, F., GREENHALGH, R. & POWELL, J.(2004). Abdominal aortic aneurysm expansion: risk factors and time intervals for surveillance. Circulation, 1101, 16-21.

DE RANGO, P., FARCHIONI, L., FIORUCCI, B. & LENTI, M. 2014. Diabetes and Abdominal Aortic Aneurysms European Journal of Vascular and Endovascular Surgery, 47, 243-261.

FORSDAHL, S., SINGH, K., SOLBERG, S. & JACOBSEN, B. 2009. Risk Factors for Abdominal Aortic Anurysm A 7- year Prospective study The Tromso study 1994-2001. Circulation, 119, 2202-2208.

Foster S and Turley R (2016) Thoracic Aortic Aneursysm and Aortic Dissection. Available at: https://www.urmc.rochester.edu/encyclopedia/content.aspx?contenttypeid=85&contentid=p08258 Accessed 28/2/16

Goldfinger JZ, Halperin JL, Marin ML, Stewart AS, Eagle KA, Fuster V, (2014), Thoracic Aortic Aneurysm and Dissection, *J Am Coll Cardiol*; 64:1725-39

Golledge, J., & Norman, P. (2010). Atherosclerosis and abdominal aortic aneurysm: Cause, response or common risk factors? *Arteriosclerosis, Thrombosis, and Vascular Biology*, *30*(6), 1075–1077. http://doi.org/10.1161/ATVBAHA.110.206573

Golledge J, Muller J, Daugherty A, Norman P (2006) Abdominal aortic aneurysm: pathogenesis and implications for management. Arterioscler Thromb, Vasc Biol. 2006 Dec; 26(12):2605-13.

Fournier & Zanoff, 2012). Understanding Aortic Aneurysms. Available at: http://www.nursingcenter.com/cearticle?tid=1373102 Accessed 28/2/16

HAY, N., MCCRACKEN, F., RICHARDSON, J., GEORGE, E. & BARNETT, D. 2009. Endovascular stent-grafts for the treatment of abdominal aortic aneurysms: NICE technology appraisal guidance. Heart, 95, 1798-800.

Hiratzka l., Bakis G., Beckman J., Besrin R., Carr V., Casey D., Eagle K., Hermann L., Isselbacher E., Kazerooni E., Kouchoukos N., Lytle B.,Milewicz D., Reich D., Sen S., Shinn J., Svensson L., Williams D., (2010) ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM Guidelines for the Diagnosis and Management of Patients With Thoracic Aortic Disease: Executive Summary *Journal of the American College of Cardiology* 55(14) 1510-1539

Howard D., Banerjee A., Fairhead J., Perkins J., Silver L., Rothwell P., (2013) Population-based study of incidence and outcome of acute aortic dissection and premorbid risk factor control: 10-year results from the Oxford Vascular Study. Circulation. ;127:2031–2037.

Isselbacher, E. M. (2004). Diseases of the aorta. In Braunwald E, Zipes DP, Libby P, Bonow RO (Eds). Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 7th edition. WB Saunders. Philadelphia

Jovin I., Duggal M., Ebisu K., Paek H., Oprea A., Tranquilli M., Rizzo J., Memet R., Feldman M., Dziura J., Brandt C., Elefteriades J.,(2012) Comparison of the effect on long-term outcomes in patients with thoracic aortic aneurysms of taking versus not taking a statin drug. Am J Cardiol. ;109:1050-1054.

KENT, K., R., Z., EGOROVA, N., RILES, T., MANGANARO, A., MOSKOWITZ, A., GELIJNS, A. & GRECO, G. 2010 Anlaysis of risk factors for abdominal aortic aneurysm in a cohort of 3 million individuals. Journal of Vascular Surgery, 52, 539-548.

Landenhead M., Engström G., Gottsäter A., Caulfield M., Hedblad B., Newton-Cheh C., Melander O., Smith G., (2015) Risk Profiles for Aortic Dissection and Ruptured or Surgically Treated Aneurysms: A Prospective Cohort Study *Journal of the American Heart Association* 21 (4) 1

Murakami H, Yoshida K, Hino Y, Matsuda H, Tsukube T, Okita Y (2004) Complications of cerebrospinal fluid drainage in thoracoabdominal aortic aneurysm repair, *J Vasc Surg*; 39: 243-5.

Nienaber, C. A. & Fattori, R. (2012)). *Diagnosis and Treatment of Aortic Diseases*. Springer. USA.

Norman PE, Powell JT.(2010) Site specificity of aneurysmal disease. Circulation. 121:560–568.

Ramanath VS, Oh JK, Sundt TM, Eagle KA. (2009)Acute aortic syndrome and thoracic aortic aneurysm. *Mayo Clin Proc*; 84(5): 465-81

Sastry P, Hughes V, Hayes P, Vallabhaneni S, Sharples L, Thompson M, Catarino P, Moorjani N, Vale L, Gray J, Cook A, Elefteriades JA, Large SR, on behalf of the ATTAA collaborators (2015) The ETTAA study protocol: a UK-wide observational study of 'Effective Treatments for Thoracic Aortic Aneurysm', *BMJ Open*; 5:e008147. doi:10.1136/bmjopen-2015-008147.

Scott RAP, Bridgewater S, Ashton HA. Randomised clinical trial of screening for abdominal aortic aneurysm in women. Br J Surg . 2002;89: 283–285

Sharma P, Kyriakides C, (2007) Surveillance of patients post-endovascular aneurysm repair. *Postgrad Med J.* 83(968): 750-753.

Sidoff D., Choke E., Stather P., Bown M., Thompson J., Sayers R., (2014)Mortality from thoracic aortic Diseases and associations with cardiovascular risk factors Circulation available at http://circ.ahajournals.org/content/early/2014/11/13/CIRCULATIONAHA.114.010890 accessed 12th February 2016

SAKALIHASAN, N., LIMET, R. & O., D. 2005. Abdominal Aortic Surgery. The Lancet (British edition), 365, 1577-89.

Stoetling, R. K., Hines, R. L. & Marschall, K. E. (2012). *Stoelting's Anaesthetia and Co-Existing Disease*. 6th Ed. Elsevier. Philadelphia

Svensson LG, Patel V, Robinson MF, Ueda T, Roehm Jr JO, Crawford ES. (1991) Influence of preservation or perfusion of intraperatively identified spinal cord blood supply on spinal motor evoked potentials and paraplegia after aortic surgery. *JVascSurg*, 13(3):355e65.

Svensson LG, Kouchoukos NT, Miller DC, Bavaria JE, Coselli JS, Curi MA, Eggebrecht H, Elefteriades JA, Erbel R, Gleason TG, Lytle BW, Mitchell RS, Nienaber CA, Roselli EE, Safi HJ, Shermin RJ, Sicard GA, Sundt TM 3rd, Szeto WY, Wheatley GH 3rd, Society of Thoracic Surgeons Endovascular Surgery Task Force (2008) Expert consensus document on the treatment of descending thoracic aortic disease using endovascular stent-grafts. *Ann Thorac Surg*; 85(1 Suppl):S1-41

Topol, E. J. (2007). *Textbook of Medicine.* 3rd Ed. Lippincott Williams & Wilkins. Philadelpia.

Upchurch, G. R. & Criado. (2009). *Aortic Aneurysms: Pathogenesis and Treatment. Humana Press. USA.*