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Thoracoabdominal aortic aneurysm life-threatening events following endovascular aortic repair

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THORACOABDOMINAL AORTIC ANEURYSM LIFE-THREATENING EVENTS FOLLOWING ENDOVASCULAR AORTIC REPAIR

by

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DEDICATION

I would like to dedicate this work to my grandpa, Andrea. Thank you for showing me what hard work looks like.

ACKNOWLEDGMENTS

I would like to thank my mentor for this project, Dr. Marc Schermerhorn and his team, in particular Dr. Christina Marcaccio and Dr. Priya Patel. I would also like to thank Dr. Karen Symes for her help on this project and support of me in pursuit of my degree and Dr. Gwynneth Offner for her guidance.

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ABSTRACT

Objective

This study aimed to evaluate the perioperative morbidity and mortality related to endovascular abdominal aortic aneurysm repair (EVAR), complex EVAR, and thoracic endovascular aortic repair (TEVAR), as well as the details of these procedures in order to mitigate the risks in the future. We characterized the odds of adverse outcomes and complications including death from the Society for Vascular Surgery's Vascular Quality Initiative (VQI) registry.

Methods

We performed a retrospective cohort study of patients who underwent infrarenal EVAR, complex EVAR, or TEVAR using data from the VQI registry between January 2011 and September 2021. Patients with missing data on landing zone were excluded, as were emergent cases. We collected data on baseline demographics, smoking status, race/ethnicity, comorbid conditions, such as body mass index (BMI), renal function, and cardiac disease. We also collected data on the procedural details, including adjuvant access. The primary outcome was post-operative thoracoabdominal aneurysm life-altering events (TALE). TALE was defined as a composite endpoint of postoperative

death, permanent postoperative dialysis, permanent postoperative paralysis, and/or postoperative stroke. Secondary outcomes included identifying anatomic and procedural characteristics associated with post-operative TALE.

Results

We identified all patients who underwent infrarenal EVAR (N=62143), complex EVAR (N=3665), and TEVAR (N=8981) in the VQI from January 2011 to September 2021. Patients who underwent repair following rupture or who had missing landing zone data were excluded. Thus, the final cohorts for each type of repair were: EVAR, N=58327; complex EVAR, N=3537; TEVAR, N=8335. Rates of TALE were 1.4% among EVAR patients, 4.6% among complex EVAR patients, and 10% among TEVAR patients. The rates of perioperative mortality were 1.1% in EVAR patients, 4.5% in complex EVAR patients, and 5.6% in TEVAR patients. Stroke occurred in 0.2% of EVAR patients, 1.3% of complex EVAR patients, and in 3.8% of TEVAR patients. Both transient and permanent dialysis were observed in 0.20% of EVAR patients. Dialysis was observed transiently in 1.2% of complex EVAR patients and permanently in 1.1% of complex EVAR patients. Transient dialysis was observed in 1.5% of TEVAR patients and permanently in 1.1% of TEVAR patients. Discharge to a skilled nursing facility (SNF) was observed in 5.8% of EVAR patients, 14% of complex EVAR patients, and 18% of TEVAR patients.

After adjusted analysis, symptomatic repair in EVAR patients was associated with higher odds of TALE (OR 3.4; 95% CI [2.7-4.1]), as were certain comorbidities such as

chronic kidney disease (CKD) (OR 2.1; 95% CI [1.7-2.5]) and cerebrovascular disease (CVD) (OR 2.1; 95% CI [1.5-2.9]). Female sex was associated with higher odds of TALE in EVAR patients (OR 1.5; 95% CI [1.3-1.9]). Larger aneurysm diameter was also associated with higher odds of TALE in the EVAR group, particularly diameter >65 mm as compared with <55 mm (OR 2.0; 95% CI [1.6-2.5]).

For complex EVAR patients, female sex was associated with higher odds of TALE (OR 2.1; 95% CI [1.5-2.8]) as were certain comorbidities, such as CKD (OR 1.8; 95% CI [1.3-2.4]). When compared with no adjuvant access, the use of left upper extremity adjuvant access was associated with higher odds of TALE in complex EVAR patients (OR 1.6; 95% CI [1.1-2.4]). Similar to EVAR, larger aneurysm diameter trended towards an association with higher odds of TALE for complex EVAR (OR 1.5; 95% CI [0.95-2.3]). When compared with landing zone 9, more proximal landing zones were associated with higher odds of TALE: zones 3-5 (OR 2.4; 95% CI [1.2-4.4]), zones 0-2 (OR 3.5; 95% CI [1.1-11]).

When compared with proximal landing zone 3-5 in TEVAR patients, proximal landing zone 0-2 was associated with higher odds of TALE (OR 1.8; CI 95% [1.5-2.1]). When compared with no adjuvant access, the use of right upper extremity adjuvant access during TEVAR trended towards an association with higher odds of TALE (OR 1.2; CI 95% [0.80-1.8]) as did the use of left upper extremity adjuvant access (OR 1.1; 95% [0.83-1.3]). Additionally, when compared with no adjuvant access the use of multiple adjuvant access sites was associated with higher odds of TALE for TEVAR patients (OR 2.0; CI 95% [1.2-3.3]).

Conclusion

TALE was observed in 10% of TEVAR, 6.8% of complex EVAR, and 1.4% of EVAR patients. Factors that were commonly associated with TALE include symptomatic repair, more proximal landing zone, use of adjuvant access for complex EVAR and TEVAR, and wider aortic diameter. While TALE was observed after all three types of repair, higher rates were observed in patients who underwent complex EVAR and TEVAR.

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LIST OF ABBREVIATIONS

AAA .....Abdominal Aortic Aneurysm

ACE.....Angiotensin-Converting Enzyme

AKI ..... Acute Kidney Injury

ARB ..... Angiotensin Receptor Blockers

CAD .....Coronary Artery Disease

CHF..... Congestive Heart Failure

CKD ..... Chronic Kidney Disease

CT ..... Computed Tomography

DM ..... Diabetes Mellitus

EVAR..... Endovascular Aortic Aneurysm Repair

HDL .....High-Density Lipoprotein

HTN .....Hypertension

IEL ..... Internal Elastic Lamina

LSA .....Left Subclavian Artery

MRI .....Magnetic Resonance Imaging

SMA.....Superior Mesenteric Artery

SVS ..... Society for Vascular Surgery

TAA ..... Thoracic Aortic Aneurysm

TAAA ..... Thoracoabdominal Aortic Aneurysm

TEVAR .....Thoracic Endovascular Aortic Repair

USPSTF .....United States Preventative Services Task Force

INTRODUCTION

The Circulatory System

The circulatory system is a complex network of blood vessels that are responsible for the transport of blood throughout the body. The heart is a muscular organ that pumps blood through this network of blood vessels and serves as the functional center of the circulatory system. In describing blood flow relative to the heart, proximal refers to blood vessels that are more centrally located (i.e., closer to the heart), and distal refers to vessels that are located farther from the heart. Blood travels from the heart to distal tissues to provide nutrients and oxygen necessary to keep the cells alive, and transports hormones from the organs where they are synthesized to their target sites. Blood is also responsible for removal of waste products from the body, such as carbon dioxide.

There are several types of blood vessels that have different roles within the circulatory system. The aorta arises directly from the heart and is the largest artery in the body. Arteries branch from the aorta and carry nutritious, oxygen-rich blood away from the heart toward end organs and tissues. Arteries gradually become smaller as they travel distally and eventually transition into arterioles. Arterioles are similar in function to arteries, but split into increasingly smaller vessels. Arterioles transition to capillaries, which are the smallest functional units of the circulatory system and are the most distal. The main function of capillaries is to exchange from the blood what the tissues need (i.e., oxygen and nutrients) for what the tissues need to eliminate (i.e., carbon dioxide). Capillaries transition to venules, which are slightly larger and transport blood proximally

back toward the heart. Venules carry blood that contains the waste products that the tissues eliminated. Venules gradually become larger and transition into veins, which merge to form the vena cava. The vena cava returns blood directly to the heart. To accommodate these various roles within the circulatory system, blood vessel types differ in layers of musculature as well as types of connective tissue, but all vessels share certain components, including the endothelium.

Anatomy of the Aorta

The aortic root is the most proximal portion of the aorta and arises directly from the left ventricle of the heart. The root then transitions to the ascending aorta, which gives rise to the aortic arch[1]. The aortic arch gives rise to three branches: the brachiocephalic artery, the left common carotid artery, and the left subclavian artery[2]. Though the anatomy of these branch vessels can vary, the brachiocephalic artery is commonly the most proximal branch and bifurcates into the right subclavian artery, which provides blood to the right upper extremity, and the right common carotid artery, which provides blood to the brain[3]. Distal to the brachiocephalic artery, the left common carotid artery provides blood to the brain, and the left subclavian artery provides blood to the left upper extremity. This aortic arch anatomy is displayed in Figure 1[3].

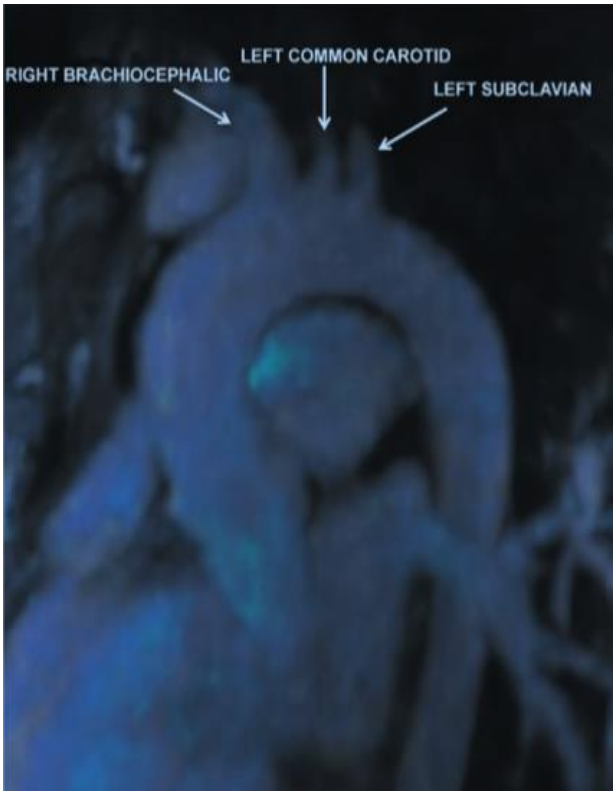

Figure 1. MRI (Magnetic Resonance Imaging) of the Aortic Arch Branches.

From: Hanneman et al., 2017[3]

The aortic arch transitions to the descending aorta just distal to the left subclavian artery[1]. The descending aorta continues inferiorly through the thoracic cavity to the diaphragm[4]. As it descends, the aorta gives rise to several small arteries, the posterior intercostal arteries, which provide blood flow to the middle of the spinal cord[5]. Together, the aortic root, ascending, arch, and descending aorta comprise the thoracic aorta. Once the aorta travels through the diaphragm, it then becomes the abdominal aorta as seen in Figure 2[6]. There are multiple branches that arise from the abdominal aorta and give rise to other arteries that supply blood to the kidneys, bowel, and other organs. These branches include the celiac trunk, the superior mesenteric artery (SMA), the left and right renal arteries, and the inferior mesenteric artery[6]. Smaller branches also arise directly

from the aorta, the lumbar arteries, which provide blood flow to the lower portion of the spinal cord[5]. Just distal to the inferior mesenteric artery, the abdominal aorta bifurcates into the left and right iliac arteries, which provide blood to the pelvis and lower extremities[7]. This aortic bifurcation occurs at the level of the fourth lumbar vertebra and is the termination of the abdominal aorta[6].

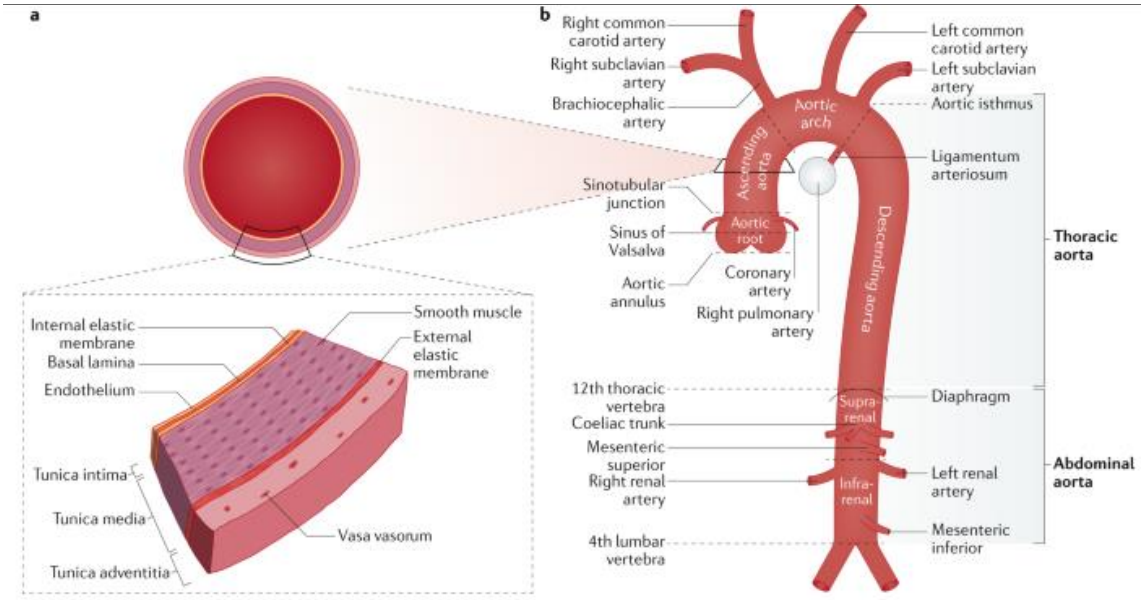

Figure 2. Overview of the Aorta. This image shows the layers of the aorta (pictured on the left) as well as the progression of the aorta from the root to the end of the abdominal aorta, where it bifurcates. This image also shows the arteries that branch from the aorta.

From: Bossone and Eagle, 2020[6]

Arteries have three structural layers, the tunica intima, tunica media, and tunica adventitia as seen in Figure 2[6]. The tunica intima is the inner-most layer and includes an endothelial layer that lines the lumen of the vessel. The endothelium is comprised of a single layer of simple squamous cells. These cells are thinnest in the capillaries and thickest in the aorta[8]. The endothelium releases factors that regulate dilation and constriction of the vessels as well as factors that control coagulation. The endothelium

also allows passage of leukocytes from surrounding connective tissue to pass through and enter the blood stream[9]. Endothelial cells can be non-fenestrated, meaning they are joined at tight junctions and have limited permeability, fenestrated continuous, meaning the cells are joined by tight junctions but they have small windows throughout each cell and are permeable to smaller molecules, or fenestrated discontinuous, which have much larger pores throughout this layer and are highly permeable[8]. Beyond the endothelium, the tunica intima also includes smooth muscle cells, connective tissue, and scarce leukocytes. Connective tissues and muscle are important components for maintaining structural integrity and allowing for optimization of the functions of arteries. Arteries have a higher content of muscle and elastic fibers than other areas of the circulatory system[8].

The internal elastic lamina (IEL) lies just outside the tunica intima and is comprised of elastin, which is organized cylindrically and separated by smooth muscle cells. This pattern allows for the formation of fenestrations, which are permeable to flow along a pressure gradient. While the IEL is permeable to macromolecules, it does control the diffusion of these molecules, particularly in larger arteries[8].

The tunica media is made up of smooth muscle cells alternating with elastic fibers. These two components create units that allow the aorta to stretch to accommodate mechanical stress associated with pulsatile increases in blood flow. The muscle and elastic units also provide tension in the aorta walls, which is necessary to maintain blood pressure and propel blood forward. The strength provided by these units maintains the integrity of the aorta walls[10]. Similar to the IEL in the tunica intima, the tunica media is

encircled by a thin layer of elastic fibers called the external elastic lamina. This layer separates the tunica media and the tunica adventitia[8].

The outermost layer of the aorta is the tunica adventitia. This layer contains collagen, which provides stiffness to the aorta. This stiffness increases with age in relation to the increase in collagen density of the aorta[11]. Within the adventitia is the vasa vasorum, a network of nerves and small blood vessels that provide blood flow the walls of the aorta itself[8].

Physiology of the Aorta

As blood flows through the aorta, the friction generated by its movement applies shear stress to the aortic wall. The magnitude of shear stress is directly proportional to vascular pressure. While the endothelium bears the majority of the shear stress, this stress can also apply to the tunica media[12]. This occurs through the transmural pressure gradient, meaning that the blood exerts stress on the endothelium, and subsequently, the endothelium exerts stress onto the tunica media. This transfer of stress also relies on the fenestrations in the IEL. The fenestrations amplify the stress as there is a lack of a barrier between the intima and media at those sites[13]. The shear stress is dependent on the viscosity and flow rate of blood as well as the vessel radius as defined by the equation $\tau = 4\mu Q/\pi r^3$ where μ is viscosity, Q is flow rate, and r is the vessel radius. Thus, the shear stress is directly proportional to the flow rate and viscosity and inversely proportional to the radius[8].

The pulsatile stretching of the aorta occurs in concurrence with systole, which is when the heart contracts and ejects blood into the aorta. The bolus of blood causes the aorta to stretch to accommodate this volume of blood. The aorta then recoils after the blood flows through in sync with diastole, which is when the heart is relaxed and filling with blood. This stretching is referred to as circumferential stress. Circumferential stress is applied to the entirety of the vessel wall and as such leads to changes to the morphology and orientation of both endothelial cells and smooth muscle cells, which leads to collagen and elastin production[8].

Aortic Aneurysms

An aneurysm is an increase in diameter of a blood vessel by greater than 50%. The aorta varies in diameter depending on location and has the largest diameter at the aortic root and ascending aorta, then gradually decreases in diameter as it descends into the abdomen. Aortic diameter also varies by sex, with female individuals generally having smaller aortic diameters compared with males[14]. Moving from proximally to distally in the chest, the average aortic diameters are as follows: aortic root 3.61 cm for females and 3.77 cm for males, ascending aorta 2.86 cm for both females and males, descending aorta 2.55 cm for females and 2.69 cm for males. At the level of the diaphragm, the average aortic diameter is 2.42 cm for females and 2.56 cm for males. Moving distally in the abdomen, the average aortic diameters are as follows: supraceliac abdominal aorta 2.2 cm for females and 2.61 cm for males, suprarenal abdominal aorta 1.87 cm for females and 2.13 cm for males, infrarenal abdominal aorta 1.53 cm for

females and 1.73 cm for males. These data were collected by analyzing computed tomography (CT) imaging[15].

Aortic aneurysms can occur at any location along the aorta and can vary in length. Thoracic aortic aneurysms (TAAs) involve the thoracic aortic, abdominal aortic aneurysms (AAAs) involve the abdominal aorta, and thoracoabdominal aneurysms (TAAAs) involve both the thoracic and abdominal aorta. AAAs are most common and occur approximately nine times more frequently than thoracic aneurysms[16]. Aortic aneurysms may also involve one or multiple branches of the aorta[17]. The natural history of aortic aneurysms is that they progress over time, with increasing aortic diameter as the vessel wall weakens. This can lead to aneurysm rupture, which is associated with exceedingly high mortality. The larger the aneurysm diameter, the more likely it is to rupture[18–20].

True aneurysms involve all three layers of the vessel wall and may be either fusiform or saccular. Fusiform aneurysms are the more complex of the two types, but are also more common[21]. Fusiform aneurysms affect the entire circumference of the vessel, while saccular aneurysms affect a focal portion of the vessel as seen in Figure 3[17,22]. Saccular aneurysms are at a higher risk for rupture than fusiform aneurysms and are more likely to rupture at smaller diameters[23].

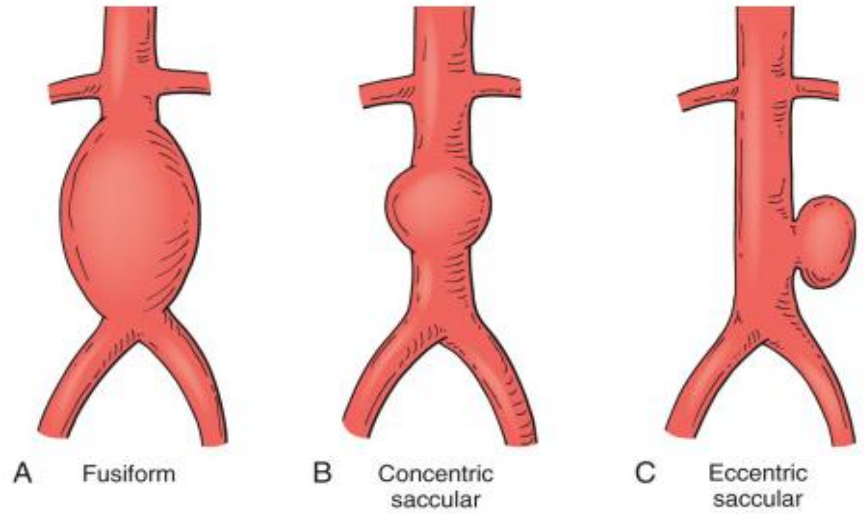

Figure 3. Fusiform and Saccular Aneurysms. A demonstrates how fusiform aneurysms affect the aorta circumferentially. B and C show how saccular aneurysms affect the aorta at focal points only.

From: Lawrence, Rigberg, 2019[17]

Pseudoaneurysms differ from true aneurysms in that pseudoaneurysms involve disruption of at least one of the three aorta wall layers, but may involve disruption of two or all three layers. This results in blood accumulation within these layers or within connective tissue outside of these layers, forming the aneurysm. Pseudoaneurysms are at very high risk for rupture[24]. Aortic pseudoaneurysms can form postoperatively, particularly at prior anastomosis sites. Causes of postoperative aortic pseudoaneurysms include technical error and infection, particularly graft infections[25].

As previously mentioned, elastin is present in the aorta walls within the internal elastic lamina, interspersed with smooth muscle in the tunica media, and in the external elastic lamina. Elastin together with collagen, which is present in the tunica media and adventitia, are responsible for the strength necessary to maintain the aorta wall integrity[26]. Aneurysm formation begins with the loss of elastin or the fragmentation of elastin, which weakens the vessel wall. The pressure exerted on the vessel walls by the blood flow

induces stress on a weakened wall and causes dilation. Though loss of collagen, is more likely the main cause for rupture. Studies have shown that collagen degradation resulted in vessel dilatation of 10-23%, while elastin degradation resulted in 6-10% dilation[27].

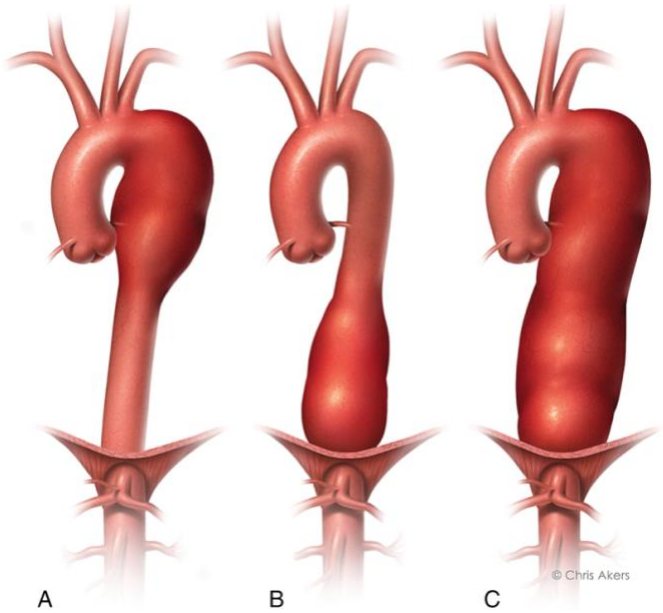

Figure 4. Types of Thoracic Aortic Aneurysms. A shows a TAA that involves the proximal portion of the descending thoracic aorta, just distal to the left subclavian artery. B shows a TAA that involves the distal portion of the descending thoracic aorta, originating at the 6th intercostal space and terminating at the end of the descending aorta, just proximal to the diaphragm. C shows a most extensive thoracic aorta involving the entire descending thoracic aorta.

From: DeAnda et al., 2022[7]

Abdominal aortic aneurysms (AAAs) occur between the portion of the aorta inferior to the diaphragm and superior to the bifurcation into the iliac arteries. Of these, 85% are infrarenal AAAs, meaning that the aneurysm is located distal to the renal arteries[22,28]. AAAs can affect one portion of the abdominal aorta or span the entirety of the abdominal aorta[7,11]. Complex AAAs are those that involve origins of the renal and mesenteric arteries[29]. Thoracic aortic aneurysms can occur in one or multiple locations anywhere from the origin of the aorta at the aortic root to the descending

supradiaphragmatic aorta as seen in Figure 4[11]. TAAs are most common in the aortic root and ascending aorta, and are least common in the aortic arch[30]. Thoracoabdominal aortic aneurysms (TAAA) span both the thoracic aorta and abdominal aorta as seen in Figure 5[31]. The majority of TAAAs occur due to elastin breakdown and subsequent weakening of the vessel wall. However, approximately 15-20% of TAAAs occur secondary to aortic dissection, a disease process in which a tear develops in the intimal layer of the aorta wall. This tear causes blood to flow between and separate the tunica intima and media, which weakens the aortic wall and increases risk of aneurysm formation[31–33].

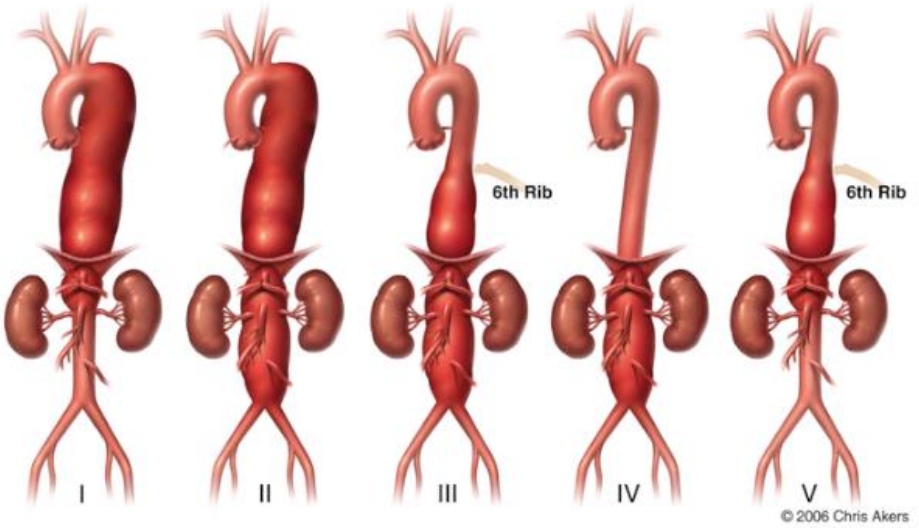

Figure 5. Types of Thoracoabdominal Aortic Aneurysms. I shows a TAAA that involves the entire descending thoracic aorta as well as the proximal portion of the abdominal aorta that terminates proximal to the renal arteries. II shows a TAAA that involves the entire descending thoracic aorta and the entire abdominal aorta. III shows a TAA that involves the distal portion of the descending thoracic aorta and the entire abdominal aorta. IV shows an AAA that involves the entire abdominal aorta but does not extend proximally to involve the thoracic aorta. V shows a TAAA that involves the descending thoracic aorta as well as the suprarenal abdominal aorta.

From: DeAnda et al., 2022[7]

Epidemiology

Abdominal aortic aneurysms occur in 2-8% of people living in developed countries. In Western populations, the incidence of AAA is approximately 2.5 to 6.5

aneurysms per 1000 person-years and increases with age[34,35]. In a prospective population-based study performed in the United Kingdom between 2004 and 2014, the incidence of AAA was approximately 55 per 100,000 person-years for males 65-74-years-old, 112 per 100,000 person-years for males 75-85-years-old, and 298 per 100,000 person-years for males 85-years-old and older[36]. Overall, the average age that people develop an AAA is 75-years-old[31]. Compared with AAAs, TAAAs occur in a younger population, with the mean age of TAAA patients closer to 65-years-old[31,37]. The overall incidence of TAAAs is 10.4 cases per 100,000 people in the United States[38]. Similar to TAAAs, the average age for development of TAA is lower than that of AAA. One study showed that the average age for developing a TAA in males was approximately 62.4 and for females with 67.7[39].

Males are more likely to develop aortic aneurysms than females, and this difference increases with older age. For AAA, males are affected at a 4 to 1 ratio[40], with prevalence rates of approximately 4.6% for males and 1.2% for females in patients over the age of 45[41]. TAAAs are also more common in males compared with females, though the difference in disease prevalence is not nearly as high as that of AAAs for younger patients[31,37]. At age 65, males are only twice as likely to develop TAAA as females. However, this sex-based discrepancy increases with age, and by age 75, males are six times more likely to develop TAAA than females[31,37,42]. While the prevalence of TAA is also higher in males than females, the difference is even less than that for TAAA, with a ratio of approximately 3 to 2[43]. Importantly, the prevalence of aortic aneurysm disease may be underestimated in population-based studies as a result of the criteria used to

define aneurysm disease. For example, many studies define an AAA as an aortic diameter of at least 3 cm. Because females typically have smaller aortic diameters than males, this AAA diameter threshold may exclude some females from the diagnosis even if they do in fact have dilation of the aorta greater than 50%[14,20].

While the overall outcomes for patients with AAA have improved, there continue to be disparities that affect racial and ethnic minority populations. Two different studies both showed that in people over the age of 45, Whites are twice as likely to have AAA than Blacks[20,44]. Despite aneurysmal disease being more prevalent in Whites, Blacks and Hispanics typically present at a further stage in aneurysm disease progression where they are symptomatic or the aneurysm has already ruptured. Presentation at later stages in disease progression is associated with higher rates of morbidity and mortality and overall worse outcomes. Additionally, aneurysmal disease is typically associated with male sex and older age, however, Black patients often develop aneurysmal disease at a younger age and while aortic aneurysms are still more common in males, Black females are disproportionately affected at a higher rate as compared to White females[45,46].

Risk Factors

There is a strong correlation between tobacco use and development of aortic aneurysms, which is true for both current and former smokers. Overall, smoking incurs a 4-fold increase in risk of AAA[35]. However, for those who currently smoke 25 or more cigarettes per day, there is a 15-fold increase in the risk of developing an AAA compared with non-smokers,[47] and 90% of AAA patients have a history of smoking[48]. In addition to

the number of cigarettes smoked per day, duration of smoking history also contributes to increased risk of AAA[49]. There is also an association between smoking and development of TAAAs and TAAs. One study showed that 19% of the risk of developing a TAA was attributable to smoking[50]. In addition to increased risk of developing an aortic aneurysm, smoking is also associated with aneurysm enlargement and increased risk of rupture[31]. The association between smoking and aneurysm progression is likely multifactorial, though it may be in part due to development of chronic obstructive pulmonary disease (COPD), which has separately been linked to increased risk for aneurysm rupture[31,51].

Other cardiovascular risk factors including hypertension (HTN), low levels of high-density lipoprotein (HDL cholesterol), and high levels of total cholesterol have also been shown to be associated with AAA[49,52]. Patients with hypertension are 30-40% more likely to develop an aortic aneurysm than those with normal blood pressure. One study showed that patients taking antihypertensive medications had a 70-80% increased risk of developing an aortic aneurysm[53]. Patients who had levels of HDL cholesterol of 1.83 mmol/L or greater had 70% lower incidence of AAA than subjects with HDL cholesterol less than 1.25 mmol/L[49,52]. While these comorbidities are associated with increased risk of AAA, interestingly, diabetes mellitus (DM) is associated with a lower risk of developing a AAA[47,54].

A family history of an aortic aneurysm in a first degree relative, which includes parents, siblings, and children, is a significant risk factor for aortic aneurysm development[15]. One retrospective study of 20,182 patients with AAA showed that 10% of those patients had a family history of AAA[55]. For TAA and TAAA, 20% of patients have

a first degree relative who had an aneurysm[31,56]. In addition to family history of an aortic aneurysm specifically, connective tissue disorders like Marfan syndrome and Ehlers-Danlos syndrome can be hereditary and increase the risk of aneurysmal disease, as these tissues are necessary for maintaining aortic wall integrity[57].

Clinical Presentation

Patients who present with an intact aortic aneurysm are often asymptomatic[7,31,57,58]. However, approximately 16% of patients with AAA who undergo surgery, do present with associated symptoms[59]. For these patients, the nature and severity of symptoms depends upon several factors including the location and diameter of the aneurysm. Patients with aneurysms in the chest may present with pain in the anterior chest or upper back, while those with aneurysms in the abdomen may present with pain in the abdomen, low back, or pelvis. In addition to localized pain, symptomatic patients may have tenderness to palpation over the aneurysm, which is most easily assessed on physical exam for aneurysms located in the abdomen. These patients may also have a palpable pulsatile abdominal mass. Patients may also present with more non-specific symptoms related to compression of surrounding structures or organs, particularly in the abdomen. Patients who have aortic root aneurysms may have a heart murmur heard on auscultation[30,60].

Patients who have a ruptured aortic aneurysm commonly present with more significant symptoms than those with an intact aneurysm. Patients with ruptured TAAs most commonly present with severe chest or back pain and those with ruptured AAAs

typically experience abdominal and back pain, as described above[22,30]. In addition to pain, patients with ruptured aneurysms may have altered mental status or even be unconscious due to hypotension and shock, depending on the duration and location of rupture[60].

Ruptured TAAs are associated with high morbidity and mortality that is nearly 100% in patients who are left untreated. Even in those patients who undergo emergent repair, the survival rate does not increase significantly[61]. Ruptured AAA is associated with 85-90% mortality[62]. Overall, ruptured aortic aneurysms result in hypovolemic hypotension and may cause multisystem organ failure, which is associated with 50-70% mortality, and is not associated with a repair type[48,63]. In a study of 215 patients with ruptured AAA, 42% of patients died prior to arrival at the hospital and 29% died in the hospital prior to operation[64]. Of the remaining 28% of patients who underwent emergent surgery, only 57% survived the postoperative period[64]. A later study demonstrated 50% postoperative survival rate of ruptured AAA repair[65]. For AAA, rupture from the anterolateral wall has a very low survival rate and most patients do not survive the duration of transportation to a medical facility due to significant hemorrhage into the peritoneal cavity. Posterolateral rupture has a slightly higher chance of survival because bleeding into the retroperitoneal space may allow for a temporary seal on the rupture, which limits blood loss. While this temporary seal may keep the patient stable for a period of time, the tear will eventually progress and the patient will hemorrhage and become unstable[22].

Diagnosis

There are several ways to assess for an aortic aneurysm. First, aneurysms involving the abdominal aorta (TAAA and AAA) can sometimes be detected on physical exam by palpating the patient's abdomen in the supraumbilical region. The sensitivity of this method increases with aneurysm size and decreases with larger body habitus. The sensitivity for detecting an AAA based on aneurysm diameter is as follows: 29-61% for aneurysms measuring 3.0-3.9 cm, 50-69% for 4.0-4.9 cm, and 76-82% for aneurysms with a diameter for 5.0 cm or more[22,66,67]. In contrast, aneurysms that are isolated to the thoracic aorta (TAA) are unlikely to be detected on physical exam.

Most aortic aneurysms are diagnosed either intentionally or incidentally based on cross-sectional imaging. First, ultrasound imaging can be used to evaluate the aortic root and abdominal aorta, but visualization of the aortic arch and descending thoracic aorta is limited with this method of imaging[3]. Ultrasound is 95% sensitive for detecting AAA and has 100% specificity[68,69]. However, ultrasound studies may be limited due to patient body habitus and central adiposity as well as by the operator and generally have lower clarity than other imaging modalities, as seen in Figure 6[70].

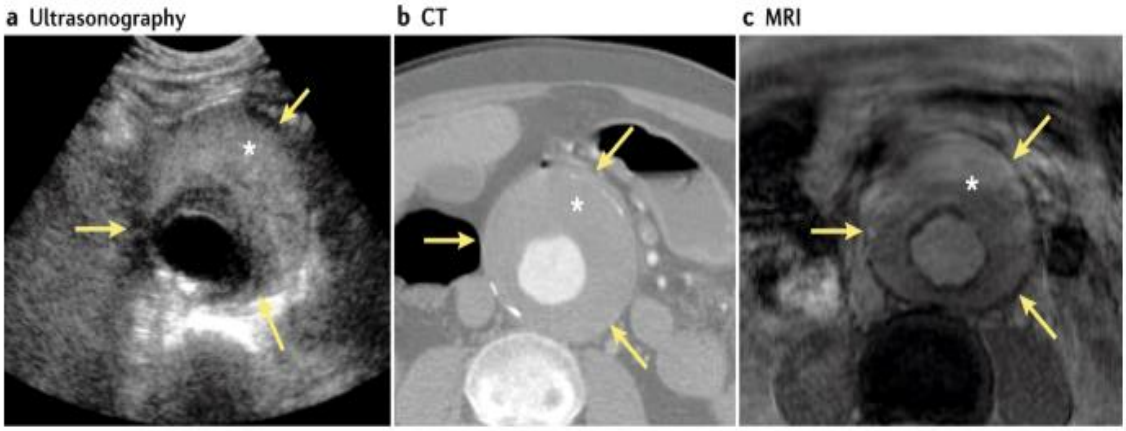

Figure 6. AAA as Seen with Different Imaging Modalities. These three images show AAAs with parietal thrombus, indicated by the asterisk, as seen with different imaging modalities. Image A is using US, Image B is using CT, and Image C is using MRI.

From: Sakalihasan et al., 2018[60]

Computed tomography (CT) and Magnetic Resonance Imaging (MRI) are alternative imaging methods that provide full visualization of the aorta and have higher sensitivity compared with ultrasound, as seen in Figure 6, but these imaging modalities are also more expensive[30,56,60,70]. CT imaging is used more commonly used than MRI, as it is less costly and takes significantly less time to perform[56,71]. However, CT imaging exposes patients to high doses of radiation and often includes the use of iodinated contrast dye, which can pose risk of allergic reactions or renal complications[22,56,60,70]. MRI imaging offers some advantages over CT imaging, as this modality avoids radiation exposure and does not require use of iodinated contrast. However, MRI cannot be performed on patients who have certain metal implants, such as a defibrillator, and is not available at all facilities.

Screening

The United States Preventative Services Task Force (USPSTF) recommends a one-time screening ultrasound for AAA in all males aged 65 to 75 years with a smoking history, and selective screening in those who are non-smokers but have other relevant risk factors based on personal medical history and family history[58]. Currently, the USPSTF cites insufficient evidence to recommend AAA screening in females aged 65 to 75 years with a smoking history and specifically recommends against screening in those without a smoking history at any age as the risks of screening outweigh the benefits[58]. However, the Society for Vascular Surgery (SVS) recommendations for AAA screening differ from the USPSTF. For those with a family history of AAA, SVS recommends a one-time screening ultrasound for males 55 years or older and females 65 years or older. SVS also recommends a one-time screening ultrasound for all males and females 65 years or older with a history of smoking[48,58]. The Centers for Medicare and Medicaid Services will reimburse for a one-time screening ultrasound in patients who are at risk for AAA, which they define as any patient with a family history of AAA or male patients aged 65-75 who have a history of smoking[72]. Despite the current screening methods and protocols, many AAA are not detected through ultrasound screening. In one study, about 50% of people with a AAA diagnosis were never screened[41]. Instead, most asymptomatic AAAs are identified as incidental findings during evaluation for unrelated pathologies[73].

For TAA, screening is recommended in patients who have a family history, in a first degree relative, that could be concerning for development of aortic pathology in the patient[56]. There are no broad guidelines for TAA screening in the general population[56].

Patients who have a familial history of TAA are more likely to develop an aortic aneurysm at a younger age than those who develop other types of aortic aneurysms and have no family history of aortic pathology[74].

Aneurysm Surveillance

For aortic aneurysms that do not meet criteria for repair at the time of diagnosis, imaging surveillance is indicated to monitor for aneurysm growth. Aortic aneurysms grow at an average of 2.3 mm per year and this rate increases as the aneurysm size increases[75,76]. The larger the aneurysm, the more frequently it should be evaluated with concern that it could progress to a rupture. For AAA, aneurysms that measure 5.0-5.4 cm in diameter should be imaged every six months, aneurysms measuring 4.0-4.9 cm should be imaged annually, aneurysms measuring 3.0-3.9 cm should be imaged every three years, and aneurysms that measure 2.5-2.9 cm should be reimaged in ten years[48]. The ideal imaging modality is ultrasound, however, CT may also be used for surveillance and is necessary to evaluate the aneurysm preoperatively, should the patient require surgery[48,77–80]. The data are lacking for recommendations regarding surveillance of TAAs and TAAAs, though the general recommendation is to perform serial imaging approximately every 6-12 months. This is variable and depends on the size and expansion rate of the aneurysm[31].

Risk Factor Modification

In addition to aneurysm surveillance, management of aortic aneurysms includes risk factor modification. This includes smoking cessation, when relevant, and optimizing management of medical comorbidities. Antihypertensives are one of the main medications used to manage progression of aortic aneurysms as hypertension is one of the main risk factors that can lead to increased dilation and rupture of the aneurysm. These medications may include beta blockers and angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARB). Low-dose aspirin is recommended for patients with aneurysms as well as it improves cardiovascular health and reduces adverse cardiovascular events, particularly in those with coronary artery disease (CAD)[48]. Patients should also be prescribed a high-intensity statin, which helps to manage cholesterol and has been shown to reduce overall cardiovascular morbidity and mortality in this population[31,69,81]. Aside from management strategies that targets risk factors for aortic aneurysms, there are no current medications that have been proven to prevent or reduce aneurysm progression or rupture[69].

Indications for Aneurysm Repair

The overall goal in management of aortic aneurysms is to detect and repair aneurysms prophylactically to prevent rupture. For asymptomatic patients with intact (non-ruptured) aneurysms, the decision of when to proceed with aneurysm repair involves weighing the risk of aneurysm rupture against the risk of operative repair, taking into account patient anatomy, comorbidities, candidacy for surgery, life expectancy, and

goals of care. The risk of aneurysm rupture depends on several factors including stability of the aneurysm, growth rate, and aneurysm diameter, as seen in Figure 7[18,19,69,82]. Patients who present with ruptured aortic aneurysms require emergent repair[60].

Aneurysm Size	1-yr Incidence of Rupture
	%
<5.5 cm	≤1.0
5.5–5.9 cm	9.4
6.0–6.9 cm	10.2
≥7.0 cm	32.5

Figure 7. Risk of Rupture of AAA. This figure shows the risk of rupture of AAA annually in relation to aneurysm size. These statistics are based off a study in which the participants were mostly male.

From: Lederle et al., 2002; Lederle et al., 2002; Powell et al., 2007; Kent, 2014[18,19,69,82]

Seventy-five to eighty percent of AAA are repaired as elective procedures[60]. For AAA, the risk of rupture is less than 1% annually when the aortic diameter is less than 5.5 cm in males, but this risk increases drastically to 14% for aneurysms with a diameter of 6.0 cm or greater[69,83]. Females have a higher risk of rupture at smaller aneurysm diameters compared with males due to a generally smaller aortic diameter at baseline as seen in Figure 8[40,84]. One study showed the risk of rupture for a female with an AAA measuring 5.0-5.9 cm was 3.9% annually and increased 22.3% annually for aneurysms that were 6.0 cm in diameter or larger[83]. Based on these data, current guidelines recommend elective repair of AAA once aneurysms reach a diameter threshold of 5.5 cm in males and 5.0 cm in females[69,81]. Elective AAA repair is also recommended in patients who have a 0.5 cm increase in aneurysm dilation over a 6-month period regardless of the absolute aortic diameter, as this growth rate is associated with increased rupture risk[69,85].

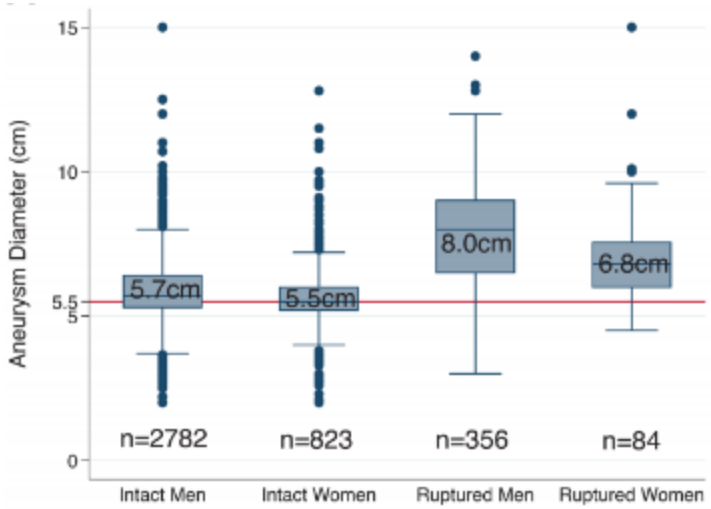

Figure 8. Aortic Diameters for Intact vs Ruptured Aneurysms in Men and Women. This graph shows the average diameters for aortic aneurysms that are intact or ruptured for both men and women. It demonstrates that women are more likely to have an aneurysm rupture at a smaller diameter, even if the diameter of an intact aneurysm is comparable to that of men.

From: Lo et al., 2014[84]

Indications for prophylactic repair of aneurysms involving the thoracic aorta differ based on the segment. It is recommended to repair an ascending TAA when the diameter is between 5.5 cm and 6.0 cm, while the descending aorta should be repaired when it measures between 6.0 cm and 6.5 cm[57,86]. Repair of TAAA is recommended when the aneurysm reaches a diameter between 5.2 cm and 5.6 cm, or when the aneurysm is double the size of the normal diameter of the aorta at the particular segment[31].

For patients who present with intact symptomatic aortic aneurysms, the decision to repair the aneurysm differs than that of asymptomatic patients. Aneurysm symptoms are indicative of impending rupture, and the time between symptom onset and aneurysm rupture is unpredictable. As such, current guidelines recommend prompt repair of

symptomatic aneurysms[48]. Ruptured aortic aneurysms are life-threatening and require immediate repair[60,69,83].

Aneurysm Repair

Aortic aneurysm repair can be performed with either an open surgical approach or an endovascular approach. Endovascular repair is less invasive and involves accessing the aorta through other vasculature such as the femoral and iliac arteries[69]. Endovascular repair can also be performed using only local anesthesia rather than general anesthesia, which can be beneficial in some patients. However, not all patients are appropriate candidates for endovascular repair. Furthermore, open repair offers some long-term advantages that should be considered, particularly in younger patients. Although open repair is more invasive and requires a larger incision and general anesthesia, accumulating evidence suggests that open repair is more durable than endovascular repair[69]. As a result, patients who undergo open repair require less frequent surveillance imaging than those who undergo endovascular repair. Additionally, individuals who have complications from endovascular repair, may need revision surgery with open repair[60,69]. Overall, the optimal repair strategy for a given patients depends on multiple factors including patient age, comorbidities, operative risk, anatomy, aneurysm progression, as well as patient preference[69].

Abdominal Aortic Aneurysm Repair

Open AAA repair requires a surgeon to make a large incision in the abdomen or along the left flank to access the abdominal aorta. The surgeon then places a synthetic graft in the section of the aorta containing the aneurysm. Depending on the patient's anatomy and the extent of the aneurysm, open repair may involve placement of a tube graft that is sutured to the proximal and distal aorta, as seen in Figure 9, or placement of a bifurcated stent graft that is sutured to the aorta proximally and extends to the iliac or femoral arteries distally. Similar to an endovascular graft, a graft placed via open repair excludes the aneurysm from blood flow, thereby reducing the pressure applied to the vessel walls and decreasing the chance of rupture.

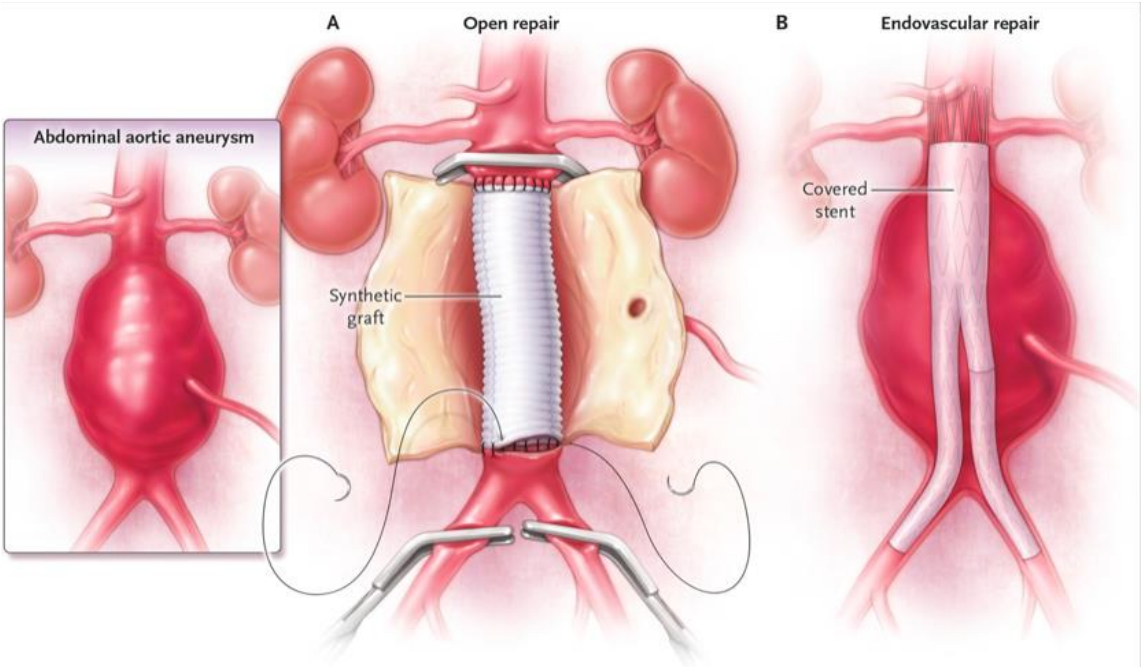

Figure 9. Comparison of Open vs. Endovascular Repair for AAA. Image A shows how in an open repair, the graft is placed within the aorta, underlying the section containing the aneurysm, and anastomosed to the surrounding aorta. Image B shows the presence of a stent within the aorta underlying the aneurysm.

From: Kent, 2014[69]

Endovascular aortic aneurysm repair (EVAR) involves placement of a bifurcated graft within an AAA. This graft extends proximal and distal to the aneurysm, as seen in Figure 9[60]. During EVAR, vascular access is typically obtained through the bilateral femoral arteries. Under fluoroscopy, the graft is fed through the vasculature using catheters and guidewires, then inserted into the portion of the abdominal aorta containing the aneurysm[22]. The stents are deployed, which secures the graft to the non-aneurysmal portions of the aortic wall. This graft excludes blood flow from the existing aneurysm and thus decreases pressure on the aneurysm wall, which decreases the chance of rupture.

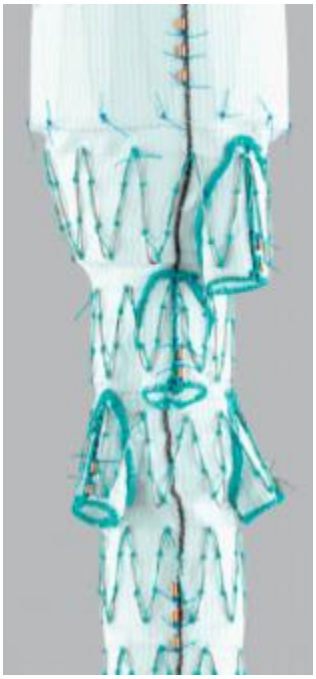

Figure 10. Branched Stent Graft.

From: Robinson et al., 2019[87]

In order to maintain blood flow to abdominal aortic branches, particularly the renal and mesenteric arteries, endovascular grafts may have branches or fenestrations, this is termed complex EVAR[7,29]. Branched stent grafts have branches off the main graft

in accordance with the aortic branches, as seen in Figure 10, which maintains perfusion through these vessels. Stents are placed through the branches to prevent flow into the aneurysm sac[87]. Fenestrations are openings in the graft that allow for blood to flow through the vessels that branch off the aorta, as seen in Figure 11. A stent is then placed through the fenestration and into the vessel to exclude the aneurysm from blood flow[87]. These grafts may require custom creation, which takes at least several weeks, making this not a suitable option for patients requiring emergent repair. This requires highly specialized facilities, which are not necessarily widely available to all patients. Furthermore, this method does not have a significant amount of long-term data available to assess the longevity of this method[88].

Figure 11. Fenestrated Graft.

From: Robinson et al., 2019[87]

Thoracic Aortic Aneurysm Repair

The mechanism of repair of TAAs depends largely on the segment of the thoracic aorta that is affected. Open repair of thoracic aortic aneurysm involving the aortic root and/or arch requires a sternotomy and is very invasive[30]. In these cases, the aortic valve may or may not be affected[30]. If the valve is affected, the repair involves replacement of

the valve and anastomosis of the graft to the non-aneurysmal portion of the ascending aorta or aortic arch. If the valve is not affected, it can remain intact and the graft can originate at the aortic annulus[57,89]. The patient will require an open procedure with cardiopulmonary bypass support[30]. Aortic arch aneurysm repair is further complicated by the need to maintain blood flow to the branch vessels. As such, open aortic arch repair typically involves placement of an aortic graft that includes three branches, which are ultimately re-anastomosed to the brachiocephalic, left common carotid, and left subclavian arteries[57]. Open repair of the descending aorta entails replacing the aneurysmal segment of the aorta with a tube graft. This is typically performed via a left thoracotomy. This repair carries an increased risk of postoperative spinal cord ischemia as this portion of the aorta gives rise to intercostal arteries, which supply the spinal cord[57].

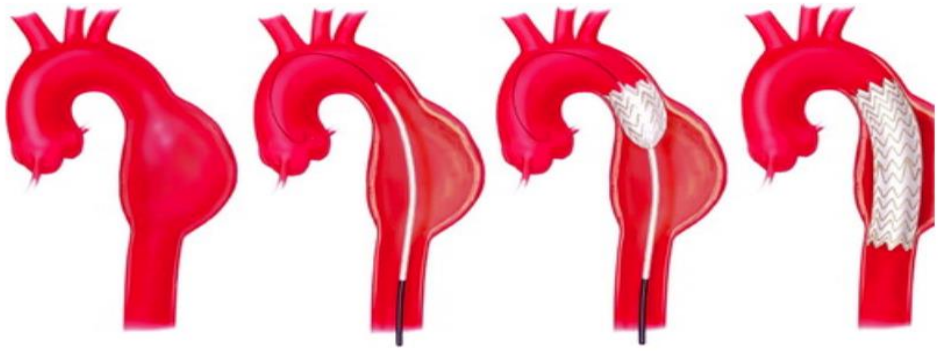

Figure 12. TEVAR. This image shows a stent graft being placed in the descending thoracic aorta via TEVAR procedure.

From: Isselbacher, 2005[57]

Thoracic Endovascular Aortic Repair (TEVAR) is a much less invasive alternative to open repair of thoracic aortic aneurysms. Similar to EVAR, during a TEVAR procedure, a stent graft is introduced through the femoral arteries, advanced to

the affected portion of the descending aorta, and deployed at this location to exclude the aneurysm, as seen in Figure 12[30,57]. For this procedure, the aorta is segmented into landing zones, which correspond to portions of the aorta and the associated branches, as seen in Figure 13. Once the graft is deployed and fixed in place within the aorta, the parts of the aorta where proximal and distal ends of the graft are fixed are termed the proximal and distal landing zones. For TEVAR procedures, the landing zones are 0-5[90]. Depending on the location of the aneurysm, there is concern that the graft would occlude the posterior intercostal arteries, which can lead to spinal cord ischemia[90].

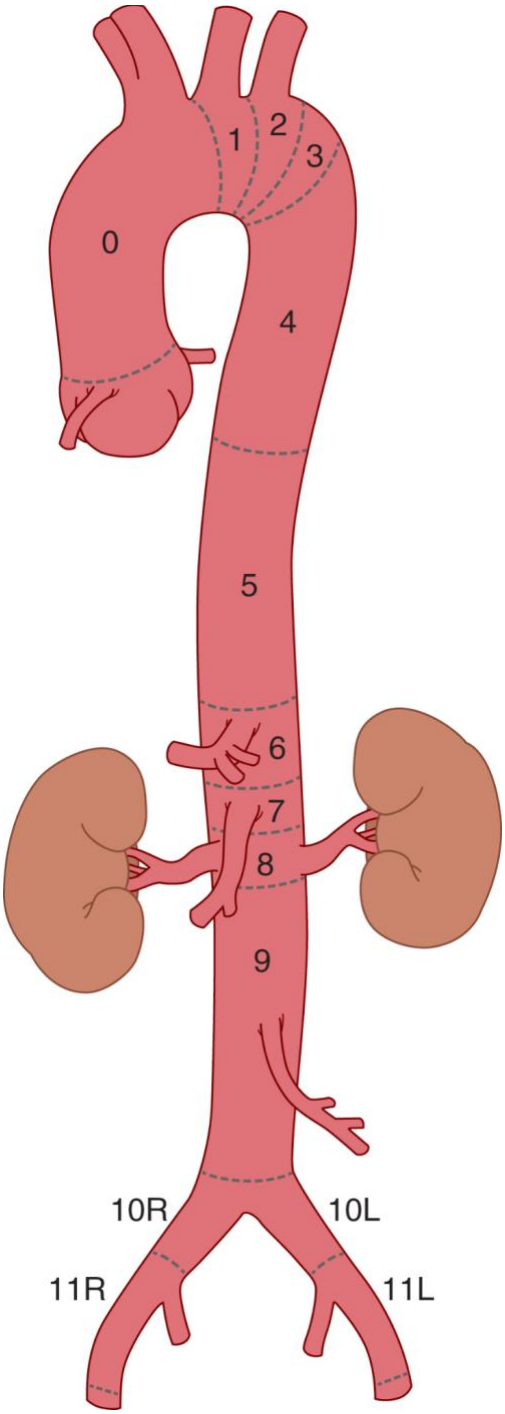

Figure 13. Landing Zones of the Aorta.

From: DeAnda et al., 2022[7]

Thoracoabdominal Aortic Aneurysm Repair

TAAAs are especially complex cases for repair due to the extent of these aneurysms and frequent involvement of aortic branch arteries that provide blood flow to the abdominal organs and spinal cord. Blood flow provided by these arteries may become compromised during surgery as well as postoperatively, which could cause many different sequelae effecting multiple organ systems. Therefore, surgical approach is based on the extent of the aneurysm. TAAA repair may be performed with either an open or endovascular approach, as is the case for other types of aortic aneurysm repairs. However, TAAAs account for only 10% of all aortic aneurysms, and as such, there are far less data regarding the repair of TAAAs[37,88,91].

Open TAAA repair involves exposure of the thoracoabdominal aorta via a thoracotomy incision that is extended into the abdomen. This approach also requires division of the diaphragm. A graft is used to replace the aneurysmal segment of the aorta, as described in prior open operative approaches. The graft may contain branches to allow for bypasses to important branch arteries to maintain blood flow[81,88]. These grafts are typically used to repair aneurysms that are present in the abdomen and have significant extension into thoracic aorta[92]. Typically, open repair is best suited for younger patients, particularly those whose anatomy would make an endovascular approach difficult[88].

Endovascular repair is less invasive, thus making it a better option for patients who are high-risk operative candidates[93]. Endovascular repair for TAAA involves access through the femoral arteries to place a stent graft across the aneurysm and exclude it from blood flow, similar to the endovascular approaches for AAA and TAA repair described

above. For endovascular TAAA repair, the stents may occupy any of the landing zones depending on the span of the aneurysm[90]. Since open repair for TAAAs requires such a large incision, endovascular repair for this type of aneurysm is particularly less invasive and benefits patients who may not be appropriate candidates for open repair. Endovascular repair also eliminates the component of visceral ischemia, which occurs during reconstruction of open TAAA repair[88]. However, due to the span of TAAAs, endovascular repair carries the risk of covering the posterior intercostal arteries, which would hinder blood supply to the spinal cord, ultimately leading to higher risk of postoperative spinal cord ischemia compared with EVAR[88]. Similar to complex EVAR, for TAAAs involving parts of the abdominal aorta that contain the renal and mesenteric artery origins, grafts used in endovascular repair of TAAAs may have fenestrations or branches to maintain blood flow to these visceral vessels, as seen in Figure 14[7,29].

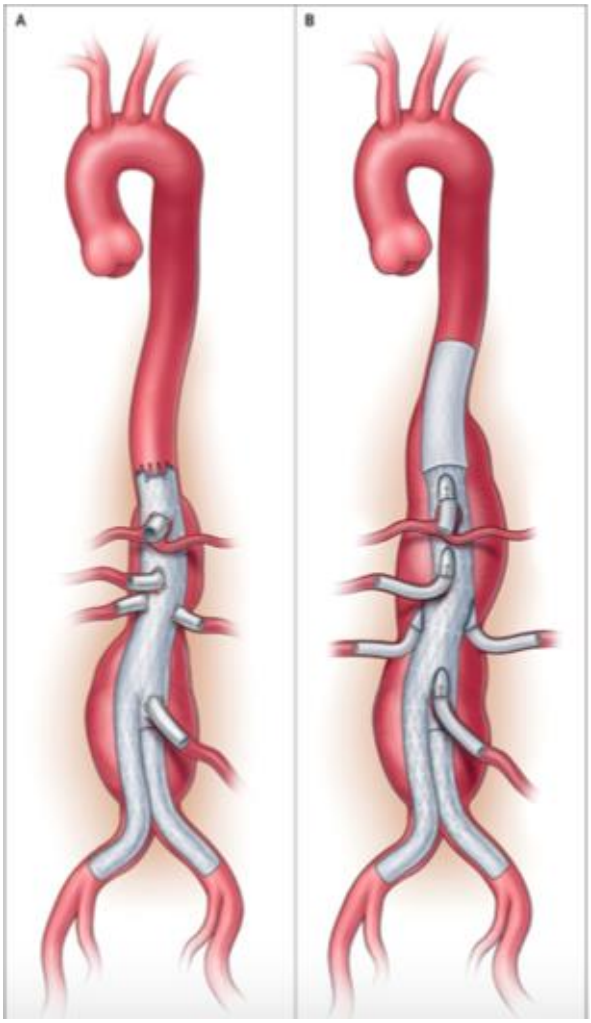

Figure 14. Thoracoabdominal Aneurysm Graft Types. This image shows two types of grafts used in endovascular repair of TAAAs. Image A shows a fenestrated TAAA graft, Image B shows a branched TAAA graft.

From: Schanzer, Oderich, 2021[81]

For some patients, a hybrid repair option involving both open and endovascular techniques may be available. This repair method involves an open repair of the abdominal aorta and uses this access to endovascularly repair the thoracic aorta. This limits the length of the incision and eliminates the need for thoracotomy and some other procedures necessary during this surgery, such as single-lung ventilation. This repair method may be suitable for patients who are not candidates for a full open surgical repair

based on comorbidities and other factors and for those who have anatomy which could cause complications with endovascular repair[88]. It may also be a reasonable option for patients who have had a TAAA rupture as it provides more access than purely endovascular repair and does not require a custom graft.

Outcomes and Complications

Complications may arise from aneurysm repair and vary depending on aneurysm and repair type. Morbidity and mortality associated with open AAA repair is higher than that associated with EVAR[48,94]. The mortality rate for open repair of AAA is 4.6%[60]. The complication rate of open AAA repair is 25% and the postoperative length of stay is 13 days[60]. One study showed that more than 20% of patients who underwent open AAA repair required surgery for laparotomy-related complications[48,94]. Endovascular repair typically results in lower perioperative morbidity and mortality[69]. Specifically, endovascular repair is associated with lower perioperative and two-year mortality, though the mortality rates after three years between the two groups are comparable[94]. One study showed a 30-day mortality rate of 1.2% in patients who underwent EVAR, which is less than half the mortality rate associated with open repair of AAA. This same study also showed significantly decreased complication rates of 12% with EVAR. The length of post-EVAR hospitalization is also shorter than with open repair, with a median length of stay of 2 days as compared with 7 days following open repair[95].

The mortality rate for open TAA repair is 15%[96]. Perioperative paralysis is also a concern for open repair with a rate of approximately 7.1%[97]. A retrospective study

showed a complication rate of 38% at 2-year follow-up[98]. Similar to mortality rate associated with open AAA and EVAR, TEVAR has a significantly lower rate of perioperative mortality than open repair. One study showed 30-day morbidity and mortality rates for patients who underwent TEVAR to be 20% and 5%, respectively[99].

Several studies have shown perioperative mortality rates between 7.0 and 16% with open repair of TAAA[81,92,100–102]. The rate of permanent spinal cord injury varies depending on the extent of the aneurysm and ranges 5-14% in those who had open TAAA repair[88]. Renal complications are also a concern with open TAAA repair with a rate of developing renal failure requiring dialysis of 7.4%[88,103]. One study demonstrated that the perioperative mortality for patients who underwent endovascular TAAA repair was 1.1%, significantly lower than that of open repair[100].

Neurologic complications such as stroke and paralysis as well as renal complications requiring dialysis cause significant changes to the patient's daily life and affect quality of life. Stroke is one of the main complications of TEVAR and occurs in 2.9% of patients in the perioperative period[104]. For TEVAR, more proximal landing zones are associated with higher rates of stroke. Patients who undergo EVAR and complex EVAR experience stroke much less frequently, approximately 0.1% and 0.9%, respectively[104]. Complex EVAR procedures may require adjuvant vascular access, which can be obtained via arteries in arms or the carotid arteries. The use of adjuvant access was showed to be associated with higher rates of stroke perioperatively. The stroke rate in patients who had arm access was 2.5% as compared with 0.3% in those who did not have arm access[104]. Additionally, adjuvant access which may be required for TEVAR

procedures is associated with increased risk of stroke, with use of multiple adjuvant access sites associated with a 2.7-fold increase in stroke risk[104]. The rate of stroke in patients who have undergone TAAA repair is 2.2%, this is consistent across open and endovascular repairs[92].

Paraplegia may be due to the graft interfering with the posterior intercostal arteries that supply the spinal cord with blood flow[86]. Paraplegia may be transient or permanent[92]. The odds of suffering a stroke perioperatively increases approximately 6-fold for patients aged 55-64 years and 4-fold for patients aged 65 years or older[86]. Similar to TAA, the risk for paraplegia is related to the aneurysm location and interference with spinal cord blood supply. For TAAAs, this includes the posterior intercostal arteries, hypogastric arteries, the left subclavian artery, as well as the lumbar arteries[86].

Renal failure is a possible complication of EVAR, as the left and right renal arteries branch directly off the abdominal aorta. This segment of the aorta may be aneurysmal and the grafts or surgery itself could interfere with the blood flow to the kidneys[105]. Renal failure could require the patient to undergo dialysis, which may be transient or permanent[106]. While renal failure requiring dialysis is a concern with AAA repair, the risk of this renal complication related to TAA surgery is less than 1%[86].

An endoleak, which is continued blood flow to the aneurysmal sac despite the presence of the graft, is a complication of endovascular repair and can lead to aneurysm rupture[60,69]. This complication may occur either at the time of repair or many years later, which is why patients who have undergone endovascular repair require lifelong monitoring. Endoleaks may resolve spontaneously or may require surgical intervention to

prevent aneurysm rupture and are most common following EVAR[48,105,107]. In addition to endoleaks, EVAR complications include continued aneurysm growth and graft infection[60,69]. Graft infections occur in only 0.3% of patients and the risk of infection is comparable between endovascular and open repair techniques[108,109].

Colonic ischemia occurs when there is interference of blood flow to the bowel. This can result in bowel perforation, which is associated with a 90% mortality rate. Colonic ischemia occurs in 4% of patients following open AAA repair and 1.2% of patients following EVAR[110]. Abdominal compartment syndrome may occur after either open or endovascular repair of ruptured AAA, particularly in patients with a large retroperitoneal hematoma and subsequent hemodynamic instability. This complication can result in multisystem organ dysfunction without prompt diagnosis and surgical intervention[48,111].

Recently, Rocha et al. introduced the concept of a thoracoabdominal aneurysm life-altering events (TALE) to describe major, life-altering complications that are observed after open TAAA repair[112]. This composite endpoint includes postoperative mortality, permanent paraplegia, permanent dialysis, and stroke[112]. The risk of these complications may be affected by multiple factors including comorbidities as well as urgency of repair and surgical techniques[112]. Although the concept of TALE was developed to evaluate outcomes following open TAAA repair, this endpoint is also relevant for patients undergoing endovascular repair and may differ based on the anatomic extent of the repair. Therefore, we examined rates of TALE and its individual

components among patients who underwent endovascular repair of TAA, TAAA, or AAA.

METHODS

Database

This retrospective cohort study was completed using data from the Society for Vascular Surgery's Vascular Quality Improvement (VQI) registry collected between January 2011 and September 2021. The VQI registry contains data that is prospectively collected from 18 regional quality groups from the United States and Canada, with more than 560 participating centers. These data include over 350 pre-defined variables such as patient demographics, comorbidities, procedural characteristics, and in-hospital outcomes as well as long-term mortality data. More information can be found at www.vqi.org. The VQI Research Advisory Committee and the Institutional Review Board at Beth Israel Deaconess Medical Center approved this study and waived the need for informed consent due to the retrospective and deidentified nature of the data.

Procedure Description

We identified patients who underwent infrarenal EVAR, complex EVAR, and TEVAR for aortic aneurysm disease. Infrarenal EVAR was defined as EVAR with a non-fenestrated, non-modified device with a proximal landing zone in aortic zone 9. Complex EVAR procedures are defined as endovascular aortic repair of abdominal aortic aneurysms that involve origins of the renal and/or mesenteric arteries, this can be done with several types of devices. TEVAR was defined as having a distal landing zone of

aortic zone 5 or above. Patients with missing data on landing zone were excluded. Additionally, data regarding patients who underwent emergent repair for ruptured aneurysms were excluded as the cause of adverse outcomes are not necessarily procedure related and may be due to underlying hemodynamic instability in these patients.

Patient Cohort

We identified all patients who underwent infrarenal EVAR (N=62143), complex EVAR (N=3665), and TEVAR (N=8981) in the VQI from January 2011 to September 2021. Patients who underwent repair following rupture were excluded, for EVAR: N=3807, for complex EVAR: N=90, and for TEVAR: N=599. There were no EVAR patients who were excluded due to missing landing zone. Those excluded due to missing landing zone were as follows: for complex EVAR, N=38, and for TEVAR, N=47. Thus, the final cohorts for each type of repair were: EVAR, N=58327; complex EVAR, N=3537; TEVAR, N=8335.

We collected data on baseline demographics and comorbid conditions. Race/ethnicity was self-identified by patients and categorized as, non-Hispanic White, Black, Hispanic, or Other in this study. We also collected data on comorbidities including body mass index (BMI), renal function, and cardiac disease. Body mass index (BMI) was calculated preoperatively using the standard weight/height^2 (kg/m^2) formula. Renal function was measured by calculating the estimated glomerular filtration rate (eGFR) using the Chronic Kidney Disease Epidemiology Collaboration formula[113]. Chronic kidney disease (CKD) is defined as eGFR <30 or the need for hemodialysis. Coronary

artery disease (CAD) was defined as myocardial infarction (MI), stable or unstable angina. Smoking was also considered in this study. The categories for smoking are as follows: never smoker, current smoker, or former smoker, defined as quitting at least 30 days prior to the aneurysm repair. Adjuvant access locations were categorized as right or left carotid artery access, right arm access (axillary or brachial artery), left arm access (axillary or brachial artery), or multiple sites. Left subclavian artery (LSA) treatment was defined as none, open bypass, stent placement, branch/fenestration, or occlusion/coverage. Urgent procedures were defined as those performed within 24 hours of presentation to the hospital or repair for a symptomatic aneurysm.

Several outcomes were evaluated at different time points. Postoperative mortality was defined as death within 30 days of repair or during hospitalization if hospitalization was greater than 30 days. Postoperative dialysis was defined as need for dialysis postoperatively in patients who did not require dialysis pre-procedure. Dialysis was considered transient if it was not required at the time of patient discharge and considered permanent if still required at discharge. Postoperative paralysis was defined as lack of palpable muscle contraction. Paralysis was considered transient if the symptoms resolved prior to hospital discharge and was considered permanent if the symptoms were present at discharge. Postoperative stroke was defined as new neurologic symptoms lasting 24 hours or more. Neurologic symptoms included motor or sensory loss, speech abnormality, or any other hemisphere specific symptoms. Thoracoabdominal aneurysm life-altering event (TALE) was defined as a composite endpoint of postoperative death,

permanent postoperative dialysis, permanent postoperative paralysis, and/or postoperative stroke.

Statistical Analysis

The statistical analysis for this project was carried out by Dr. Priya Patel. Baseline characteristics and postoperative outcomes were compared across all three cohorts (EVAR, complex EVAR, TEVAR). Categorical variables were compared using Chi-square test and were presented as frequencies and percentages. Continuous variables were compared using analysis of variance or Kruskal Wallis test. Continuous variables were presented as mean and standard deviation or median and interquartile range. A descriptive analysis is provided for operative characteristics of each cohort. Due to differences in operative technique meaningful comparisons could not be provided across the three cohorts. The primary outcome was post-operative TALE. Secondary outcomes included anatomic and procedural characteristics associated with post-operative TALE.

To identify anatomic and procedural characteristics associated with TALE, we created mixed-effects multivariable logistic regression models with clustering at the center and physician level. Variables included in each model were selected a priori. Baseline and demographic characteristics included in each model were age, sex, race (White, Black, Hispanic, Other), poor renal function (eGFR<30 mL/min/1.73m^2), prior stroke, prior carotid artery revascularization, prior aortic repair, urgency, and aortic diameter. Additional anatomic and operative specific variables were added to each model based on the repair type. The model for EVAR additionally included proximal aortic

extension cuff and concomitant iliac aneurysm. The model for complex EVAR additionally included repair technique, adjuvant access (none, right arm, left arm, right or left carotid artery, or multiple sites), and proximal landing zone. Due to collinearity between proximal landing zone and left subclavian artery treatment, two separate models were created for the TEVAR cohort. The first model included disease pathology, adjuvant access, and proximal landing zone, in addition to the previously described baseline and demographic variables. The second model for the TEVAR cohort substituted proximal landing zone with left subclavian artery management. Results of the multivariable models were presented as odds ratio (OR) and 95% confidence interval (CI), with an OR>1 representing an increased odds of TALE.

This study adhered to the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) standards for observational studies[114]. All tests were two-sided and p-value <.05 was considered statistically significant. All variables had <5% missing data. All analyses were performed using STATA version 16 software (StataCorp LP, College Station, Texas, USA).

RESULTS

Patient Cohort

In our study of 70199 patients, 58327 patients underwent infrarenal EVAR, 3537 patients underwent complex EVAR, and 8335 patients underwent TEVAR. Patients who underwent TEVAR were on average the youngest (mean age of 66) as compared with EVAR (mean age of 73) and complex EVAR (mean age of 74) (p<.001). There was a higher proportion of female patients in the TEVAR cohort (40%) compared with EVAR (19%) and complex EVAR (27%) (p<.001). Overall, most procedures were elective, though TEVAR patients had a higher proportion of symptomatic patients (31%) compared with EVAR (9.5%) and complex EVAR (5.7%) (p<.001).

Table 1. Demographics of the patient population involved in this study. Race/ethnicity was self-reported.

Factor	EVAR	Complex EVAR	TEVAR	p-value
N	58327	3537	8335	
Age, mean (SD)	73 (8.6)	74 (7.8)	66 (13)	<0.001
Female	11149 (19%)	948 (27%)	3329 (40%)	<0.001
Race/Ethnicity				<0.001
Non-Hispanic White	50548 (89%)	2980 (88%)	5123 (64%)	
Black	3228 (5.7%)	231 (6.8%)	2057 (26%)	
Hispanic	1744 (3.1%)	120 (3.5%)	482 (6.0%)	
Other	1016 (1.8%)	70 (2.1%)	317 (4.0%)	
BMI (IQR)	27 (24, 31)	27 (24, 30)	28 (24, 32)	<0.001
Urgency				<0.001
Elective	52592 (91%)	3336 (94%)	5740 (69%)	
Symptomatic	5515 (9.5%)	201 (5.7%)	2592 (31%)	

Most patients were prior smokers, though more TEVAR patients were never-smokers (31%) than EVAR (14%) and complex EVAR (10%). The proportion of current-

smokers was similar across all three groups 32% for EVAR, 34% for complex EVAR, 30% and for TEVAR. Hypertension was seen in the majority of patients in this study, 84% of EVAR patients, 89% of complex EVAR patients, and 91% of TEVAR patients. Diabetes was present in 21% of EVAR patients, 18% of complex EVAR patients, and 16% of TEVAR patients. Chronic kidney disease (CKD) was present in 34% of EVAR patients, 41% of complex EVAR patients, and 37% of TEVAR patients. COPD was present in 34% of EVAR patients, 40% of complex EVAR patients, and 26% of the TEVAR patients. History of stroke was present in 3.3% of EVAR patients, 4.1% of complex EVAR patients, and 4.7% of TEVAR patients.

Table 2. Risk factors and comorbidities and their presence in each repair type group.

Factor	EVAR	Complex EVAR	TEVAR	p-value
Smoking status				<0.001
Never	7900 (14%)	369 (10%)	2553 (31%)	
Prior	31666 (54%)	1981 (56%)	3310 (40%)	
Current	18611 (32%)	1187 (34%)	2462 (30%)	
Hypertension	48078 (84%)	3132 (89%)	7480 (91%)	<0.001
Diabetes	11972 (21%)	644 (18%)	1302 (16%)	<0.001
Insulin Dependent Diabetes	2242 (3.9%)	87 (2.5%)	268 (3.2%)	<0.001
CKD (eGFR ≤30 or HD)	19876 (34%)	1448 (41%)	3054 (37%)	<0.001
COPD	19582 (34%)	1403 (40%)	2182 (26%)	<0.001
CHF (Moderate-Severe)	1114 (1.9%)	83 (2.3%)	172 (2.1%)	0.15
Coronary Artery Disease	17107 (29%)	1053 (30%)	1353 (16%)	<0.001
History of Stroke	1475 (3.3%)	146 (4.1%)	388 (4.7%)	<0.001
Prior CEA/CAS	2512 (4.4%)	172 (4.9%)	210 (2.5%)	<0.001
Prior CABG	9659 (18%)	638 (18%)	647 (7.8%)	<0.001
Prior PCI	12500 (23%)	834 (24%)	949 (11%)	<0.001
Prior Aortic Aneurysm Repair	1010 (1.8%)	560 (16%)	2004 (24%)	<0.001

Anatomic Characteristics

Table 3. Aneurysm diameters for each repair type group, urgency of procedure, and adjuvant access sites used for each repair type.

	EVAR	Complex EVAR	TEVAR
N	58327	3537	8335
Aneurysm diameter			
<55mm	26291 (45%)	751 (21%)	4751 (57%)
55-65mm	21665 (37%)	1764 (50%)	2104 (25%)
>65mm	9091 (16%)	1053 (30%)	1510 (18%)
Proximal aortic extension	6626 (15%)	-	-
Concomitant Iliac Aneurysm	14465 (26%)	-	-
Urgency			
Elective	52592 (91%)	3336 (94%)	5740 (69%)
Symptomatic	5515 (9.5%)	201 (5.7%)	2592 (31%)
Adjuvant Access			
None		2458 (70%)	6287 (76%)
Right arm		213 (6.0%)	264 (3.2%)
Left arm		831 (24%)	1264 (15%)
Carotid (right or left)		3 (0.1%)	374 (4.5%)
Multiple		28 (0.4%)	127 (1.5%)

Table 4. Proximal landing zone based on repair type.

Proximal landing zone	EVAR	Complex EVAR	TEVAR
Zone 0		4 (0.1%)	319 (3.8%)
Zone 1		5 (0.1%)	444 (5.3%)
Zone 2		25 (0.7%)	2875 (35%)
Zone 3		109 (3.1%)	3157(38%)
Zone 4		203 (5.7%)	1322 (16%)
Zone 5		663 (19%)	218 (2.6%)
Zone 6		479 (14%)	
Zone 7		1262 (34%)	
Zone 8		610 (17%)	
Zone 9	58327 (100%)	177 (5.0%)	

Postoperative Outcomes

Rates of TALE were 1.4% among EVAR patients, 4.6% among complex EVAR patients, and 10% among TEVAR patients. TEVAR had the highest rates of complications in all categories. The rates of perioperative mortality were 1.1% in EVAR patients, 4.5% in complex EVAR patients, and 5.6% in TEVAR patients. Stroke occurred in 0.2% of EVAR patients, 1.3% of complex EVAR patients, and in 3.8% of TEVAR patients. Both transient and permanent paralysis were complications of complex EVAR and TEVAR, however these rates were not statistically significant. Renal complication rates were overall low. Both transient and permanent dialysis were observed in 0.20% of EVAR patients. Dialysis was observed transiently in 1.2% of complex EVAR patients and permanently in 1.1% of complex EVAR patients. Transient dialysis was observed in 1.5% of TEVAR patients and permanently in 1.1% of TEVAR patients. Lastly, discharge to a skilled nursing facility (SNF) was observed in 5.8% of EVAR patients, 14% of complex EVAR patients, and 18% of TEVAR patients.

Table 5. Complications and outcomes for each repair type.

Outcomes	**EVAR**	**Complex EVAR**	**TEVAR**	
N	58327	3537	8335	p-value
TALE	845 (1.4%)	242 (6.8%)	844 (10%)	<0.001
Perioperative Mortality	647 (1.1%)	158 (4.5%)	465 (5.6%)	<0.001
Stroke	126 (0.2%)	45 (1.3%)	318 (3.8%)	<0.001
Paralysis, Transient		41 (1.2%)	155 (1.9%)	0.006
Paralysis, Permanent		61 (1.7%)	177 (2.1%)	0.15
Dialysis, Transient	114 (0.2%)	41 (1.2%)	120 (1.5%)	<0.001
Dialysis, Permanent	121 (0.2%)	39 (1.1%)	87 (1.1%)	<0.001
Discharge to SNF	3386 (5.8%)	484 (14%)	1472 (18%)	<0.001

Adjusted Analysis

EVAR

After adjusted analysis, symptomatic repair was associated with higher odds of TALE (OR 3.4; 95% CI [2.7-4.1]), as were certain comorbidities, CKD (OR 2.1; 95% CI [1.7-2.5]) and cerebrovascular disease (CVD) (OR 2.1; 95% CI [1.5-2.9]) (Table 6). Female sex was associated with higher odds of TALE (OR 1.5; 95% CI [1.3-1.9]). Larger aneurysm diameter was also associated with higher odds of TALE, particularly diameter >65 mm as compared with <55 mm (OR 2.0; 95% CI [1.6-2.5]) (Table 6).

Table 6. EVAR.

EVAR	OR	95% Confidence Interval	p-value
Age (by decade)	1.51	1.34, 1.70	<.001
Female sex	1.53	1.26, 1.87	<.001
Race/Ethnicity			
White (ref)			
Black	0.95	0.67, 1.35	0.763
Hispanic	0.89	0.53, 1.49	0.651
Other	0.92	0.50, 1.71	0.797
Smoking History			
Never (ref)			
Prior smoker	0.79	0.61, 1.01	0.058
Current smoker	0.91	0.68, 1.21	0.521
Comorbidities			
CAD	1.54	1.29, 1.85	<.001
COPD	1.49	1.24, 1.79	<.001
CKD	2.09	1.74, 2.52	<.001
CVD	2.09	1.49, 2.92	<.001
CHF	1.60	1.30, 1.90	<.001
Prior CEA/CAS	1.44	1.06, 1.96	0.02

Prior Aortic Repair	1.09	0.66, 1.81	0.729
Symptomatic Repair	3.35	2.73, 4.12	<.001
Aortic Diameter			
<55mm (ref)			
55-65mm	1.17	0.95, 1.44	0.144
>65mm	2.02	1.61, 2.53	<.001
Proximal Extension	1.46	1.18, 1.81	<.001
Iliac aneurysm repair	1.18	0.96, 1.45	0.108

Complex EVAR

After adjusted analysis, female sex was associated with higher odds of TALE (OR 2.1; 95% CI [1.5-2.8]) as were certain comorbidities, such as CKD (OR 1.8; 95% CI [1.3-2.4]). When compared with no adjuvant access, the use of left upper extremity adjuvant access was associated with higher odds of TALE (OR 1.6; 95% CI [1.1-2.4]) (Table 7). Similar to EVAR, larger aneurysm diameter trended towards an association with higher odds of TALE (OR 1.5; 95% CI [0.95-2.3]). When compared with landing zone 9, more proximal landing zones were associated with higher odds of TALE: zones 3-5 (OR 2.4; 95% CI [1.2-4.4]), zones 0-2 (OR 3.5; 95% CI [1.1-11]).

Table 7. Complex EVAR.

Complex EVAR	OR	95% Confidence Interval	p-value
Age (by decade)	1.18	0.97, 1.45	0.103
Female sex	2.07	1.53, 2.80	<.001
Race/Ethnicity			
White (ref)			
Black	1.24	0.76, 2.03	0.387
Hispanic	0.92	0.42, 2.02	0.839
Other	1.80	0.77, 4.24	0.175
Smoking History			

Never (ref)			
Prior smoker	0.98	0.60, 1.59	0.927
Current smoker	1.23	0.73, 2.08	0.44
Comorbidities			
CAD	1.56	1.16, 2.11	0.004
COPD	1.23	0.91, 1.67	0.174
CKD	1.78	1.32, 2.40	<.001
CVD	1.50	0.83, 2.72	0.182
CHF	1.10	0.80, 1.60	0.461
Prior CEA/CAS	1.84	1.09, 3.10	0.022
Prior Aortic Repair	1.05	0.72, 1.53	0.801
Symptomatic Repair	1.51	0.92, 2.46	0.1
Aortic Diameter			
<55mm (ref)			
55-65mm	1.02	0.68, 1.55	0.909
>65mm	1.49	0.95, 2.32	0.083
Adjuvant Access			
None (ref)			
Right Arm	1.29	0.71, 2.34	0.412
Left Arm	1.59	1.07, 2.36	0.021
Carotid (right or left)	--	--	--
Multiple (Arms or Carotid)	2.67	0.94, 7.57	0.064
Proximal Landing Zone			
Zone 9 (ref)			
Zone 0-2	3.53	1.13, 11.01	0.03
Zone 3-5	2.35	1.24, 4.44	0.009
Zone 6-8	0.88	0.46, 1.67	0.691

TEVAR

After adjustment, when compared with proximal landing zone 3-5, proximal landing zones 0-2 trended towards higher odds of TALE (OR 1.8; CI 95% [1.5-2.1]) (Table 8). When compared with no adjuvant access, the use of right adjuvant access trended towards an association with higher odds of TALE (OR 1.2; CI 95% [0.80-1.8]) as did the

use of left adjuvant access (OR 1.1; 95% [0.83-1.3]). Additionally, when compared with no adjuvant access the use of multiple adjuvant access sites was associated with higher odds of TALE (OR 2.0; CI 95% [1.2-3.3]) (Table 8).

Table 8. TEVAR.

TEVAR	OR	95% Confidence Interval	p-value
Age (by decade)	1.09	1.02, 1.17	0.016
Female sex	1.02	0.86, 1.20	0.843
Race/Ethnicity			
White (ref)			
Black	1.09	0.90, 1.33	0.362
Hispanic	1.09	0.77, 1.55	0.624
Other	1.28	0.87, 1.89	0.209
Smoking History			
Never (ref)			
Prior smoker	1.02	0.83, 1.26	0.824
Current smoker	1.19	0.95, 1.47	0.126
Comorbidities			
CAD	1.32	1.08, 1.62	0.008
COPD	1.13	0.93, 1.36	0.22
CKD	1.68	1.43, 1.98	<.001
CVD	1.03	0.72, 1.47	0.886
CHF	1.10	0.88, 1.30	0.426
Prior CEA/CAS	1.58	1.02, 2.44	0.04
Prior Aortic Repair	0.78	0.63, 0.95	0.016
Symptomatic Repair	3.10	2.61, 3.69	<.001
Aortic Diameter			
<55mm (ref)			
55-65mm	1.18	0.95, 1.46	0.134
>65mm	2.27	1.84, 2.80	<.001
Adjuvant Access			
None (ref)			
Right Arm	1.22	0.80, 1.84	0.356
Left Arm	1.05	0.83, 1.33	0.669
Carotid (right or left)	0.69	0.45, 1.06	0.094

Multiple (Arms or Carotid)	1.96	1.18, 3.25	0.01
Proximal Landing Zone			
Zone 3-5 (ref)			
Zone 0-2	1.80	1.50, 2.10	<.001

Table 9. Left Subclavian Artery Reintervention

Landing Zone	None	Bypass	Stent	Branched/ Fenestrated	Total
Zone 0	45	184	6	24	259
Zone 1	57	219	16	22	314
Zone 2	403	1,379	99	182	2063
Zone 3	228	78	31	20	357
Zone 4	60	40	14	8	122
Zone 5	6	7	2	1	16
Total	799	1,907	168	257	3131

DISCUSSION AND CONCLUSIONS

TALE was seen in 2.8% of all patients who underwent endovascular aortic aneurysm repair. In particular, TALE was observed in 10% of TEVAR, 6.8% of complex EVAR, and 1.4% of EVAR patients. Factors that were commonly associated with TALE include symptomatic repair, more proximal repair, use of adjuvant access for complex EVAR and TEVAR, and wider aortic diameter. While TALE was observed after all three types of repair, higher rates were observed in patients who underwent complex EVAR and TEVAR. Additionally, among patients who underwent EVAR and TEVAR, TALE occurred more frequently for patients with symptomatic aneurysm.

Aortic diameter >65mm was associated with higher odds of TALE after EVAR and TEVAR, it also trended towards a higher odds of TALE after complex EVAR. A prior study investigated the relation of aneurysm size to outcomes of EVAR by comparing three groups of AAA patients with aneurysm diameters measuring 4-5.4 cm, 5.5-6.4 cm, and 6.5 cm or larger. The outcomes evaluated included aneurysm-related deaths as well as post-EVAR rupture. Patients who had aneurysms 6.5 cm or larger had the highest rates of aneurysm-related death at midterm follow-up and the risk of aneurysm-related death increased from 1% in the first three years to 8% in the fourth year. This study demonstrated that larger aneurysms were associated with higher rates of rupture and death[115], which is consistent with the data in our study. A different study of EVAR patients evaluated perioperative and five-year complication and mortality rates. This study also grouped patients according to aneurysm diameter with parameters of <5 cm, 5-

5.4 cm, 5.5-5.9 cm, and 6.0 cm and larger. This study concluded that aneurysms >6 cm are associated with higher complication and mortality rates[116].

Adjuvant access was obtained in complex EVAR and TEVAR patients, most often via the left arm. Complex EVAR and TEVAR patients who had left arm access or multiple access sites had higher rates of TALE as compared with patients who did not require adjuvant access. Carotid adjuvant access was obtained in 4.5% of TEVAR patients, but was not associated with higher rates of TALE. One study evaluated the relationship between stroke and procedural characteristics of complex EVAR and TEVAR. This study showed an increased risk of stroke with use of adjuvant access in both complex EVAR and TEVAR patients, particularly arm access in complex EVAR patients and multiple access sites in TEVAR patients. More specifically, it showed an 8.4-fold increase in odds of postoperative stroke in patients who had adjuvant arm access during repair as well as association between use of multiple access sites and higher risk of stroke[104]. Our study was consistent with these findings, as TALE includes stroke as an outcome. The increased stroke risk in patients with adjuvant access may be due to the increased risk of plaque dislodgement from wire manipulation within the aortic arch or arch branch vessels. This plaque can then embolize and lead to stroke[104,117]. Additionally, debris and air emboli can also be introduced with adjuvant access and may also lead to stroke[104,118]. To reduce the risk of stroke, access through the femoral arteries alone may be a more suitable option when feasible and carries lower risk of stroke[104].

A history of CKD prior to aneurysm repair was associated with higher odds of TALE in all three repair groups. One study showed similar findings in a group of patients

who had endovascular repair of pararenal aortic aneurysms as well as TAAAs. Results of this study showed that a history of CKD was associated with higher rates of mortality and higher odds of renal function decline. Additionally, those who had pre-existing moderate or severe CKD were more likely to experience an acute kidney injury (AKI) following repair as compared with those who had no history or mild history of CKD[119]. Another prior study evaluated outcomes of EVAR and TEVAR in patients with a history of CKD compared with patients without a history of CKD. This study showed that the rates of perioperative mortality in the CKD group nearly doubled that of the non-CKD group. The CKD group was also associated with higher complication rates, including major cerebrovascular events[120]. These studies are consistent with the results of our study and show a higher risk of adverse events after endovascular aneurysm repair in patients with CKD as compared with patients without CKD.

A history of congestive heart failure (CHF) was associated with TALE in EVAR patients but not in complex EVAR and TEVAR patients. This finding in EVAR patients is consistent with prior work showing that CHF was associated with adverse events after EVAR[121]. However, the adverse events of this study were similar but more broad than that of our study and included any in-hospital myocardial infarction, dysrhythmia, CHF exacerbation, stroke, pneumonia, respiratory failure, renal failure, lower extremity ischemia, bowel ischemia, or reoperation. CHF has also been shown to be associated with perioperative mortality after EVAR[122], but whether CHF is associated with the other TALE complications (stroke, paralysis, or dialysis) remains unknown. This may partly explain why CHF was not associated with TALE after complex EVAR or TEVAR, as

TALE after EVAR was driven more by mortality than by stroke, paralysis, or dialysis compared with TALE after complex EVAR and TEVAR.

Proximal landing zones were associated with higher odds of TALE in complex EVAR patients and TEVAR patients. Complex EVAR patients had higher odds of TALE with landing zones between 0-5 and TEVAR patients had higher odds with zones 0-2. A prior study involving TEVAR patients evaluated both location of the proximal landing zone, as well as the length of the aorta the graft spanned in relation to adverse outcomes. This study showed that proximal landing zones extending to the aortic arch were associated with stroke[123]. A different study saw similar results and thought this may be due to increased wire manipulation near the arch[104]. Longer graft lengths were also associated with adverse outcomes, primarily paralysis. This could be due to increased numbers of posterior intercostal arteries without blood flow due to the graft[123].

Limitations of the study include missing data from the VQI registry on postoperative paralysis in EVAR patients, though this is a rare complication. Other limitations of the registry are the time span of data collected in reference to surgery. The VQI does not include long-term results.

In conclusion, among patients who underwent endovascular repair of aortic aneurysms in the Vascular Quality Initiative registry, rates of perioperative TALE varied by anatomic location of repair. Specifically, rates of TALE were highest after TEVAR at 10%, followed by 6.8% after complex EVAR, and 1.4% after infrarenal EVAR. Factors that were commonly associated with TALE included symptomatic repair, more proximal landing zone, use of adjuvant access for complex EVAR and TEVAR, wider aortic

diameter, and CKD. Overall, patients who experience TALE may endure significant changes to their lifestyles and thus, changes in quality of life. These factors should be considered when determining a patient's suitability for surgery as well as the opportune time of intervention.

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