Abstract

Abdominal Aortic Aneurysm (AAA) is the most common type of true aneurysm and have a high incidence to rupture.

The introduction of endovascular grafting was a milestone in the treatment of patients with AAA as it provided a treatment option for those patients with large aneurysms with inoperable conditions because of the presence of significant medical comorbidities. By this innovation, an early benefit in quality of life can be achieved, as it relates to reducing hospital stay and recovery period in comparison with open surgery.

In our study**,** we enrolled 20 patients to describe initial and mid-term results of Endovascular Aortic Aneurysm Repair (EVAR) in patients with infra-renal abdominal Aortic Aneurysm.

Keywords:

- ✓ Aortic aneurysms
- ✓ Endovascular techniques
- ✓ EVAR

Introduction

Abdominal Aortic Aneurysm (AAA) is the most common type of true aneurysm and have a high capability to rupture, which makes it a significant health care problem **(Abu Rahma , 2000).**

The prevalence of abdominal aortic aneurysms (AAAs) has been increasing rapidly during the past decade as aneurysmal rupture had been estimated as the 13th most common cause of death in the Western world **(Thompson and Bell, 2000).**

The two primary methods for AAA repair are open and endovascular repair which has proven to be safe and effective treatment **(Rutherford et al., 2004) .**

Over the last decade, the management of Aortic Aneurysms has changed dramatically because of the progress of the technique of endovascular aneurysm repair. Patients and physicians have embraced endovascular aneurysm repair as a method of choice to treat high risk patients with Aortic Aneurysms **(Greenberg et al., 2004).**

Endovascular repair has several advantages over conventional surgery. First, it is a minimally invasive technique, which is of major importance in patients with comorbid factors, who are at high risk for conventional surgery. Operative trauma, blood loss and significant disturbances of hemodynamics, and ventilatory condition associated with endovascular repair are minimal compared with the open procedure. Moreover, hospital stay and recovery time have been reduced significantly since the introduction of EVAR, approximately from 10 to 3 days for hospital stay and from 6 months to 11 days for recovery time **(Becquemin et al., 2000).**

However, on the other hand, the endovascular repair has few complications as intraoperative embolization, the tendency to kink in the

unsupported graft, stent graft migration, early and late Endoleak and endotention, rupture after aneurysm repair, structural failure of the device itself, and secondary interventions including late conversion to open repair **(Buth and Harris, 2005).**

The Aim of work

The aim of this study is:

• To outline a frame for detection and diagnosis of patients with abdominal aneurysms via different clinical and radiological methods.

• To consider the patients suitable for EVAR according to the patient selection criteria.

• To highlight the benefits and advantages of endovascular procedures.

• Assess the possible complication of endovascular procedures and how to avoid it

Anatomy of the Aorta

The aorta is the main trunk of vessels which convey the oxygenated blood to the tissues of the body for their nutrition. It commences at the upper part of the left ventricle, where it is about 3cm in diameter, and after ascending for a short distance, arches backward and to the left side, over the root of the left lung; it then descends within the thorax on the left side of the vertebral column, passes into the abdominal cavity through the aortic hiatus in the diaphragm, and ends, considerably diminished in size (about 1.75 cm. in diameter),opposite the lower border of the fourth lumbar vertebra ,

by dividing into the right and left common iliac arteries. Hence it is divided into several portions; the ascending aorta**,** the arch of the aorta**,** and the descending aorta**,** which is divided into the thoracic and abdominal aorta **(Giorgio et al.,1995).**

Abdominal Aorta:

❖ Course:

The abdominal aorta begins at the median, aortic hiatus of the diaphragm anterior to the inferior border of the twelfth thoracic vertebra and the thoracolumbar intervertebral disc. It descends anterior to the lumbar vertebrae to end at the lower border of the fourth lumbar vertebra, a little to the left of the midline, by dividing into two common iliac arteries. It diminishes rapidly in caliber from above downward, because its branches are large; however, the diameter of the vessel at any given height tends to increase slightly with age. The angle of aortic bifurcation varies widely, particularly in the elderly. It has been suggested that the relationship between aortic size and shape is a possible causative factor in the development of abdominal aortic aneurysm (**Newman et al., 1971**).

This may be caused by the reflection of transmitted pressure waves, which occurs at junctions between vessels. At the aortic bifurcation, pressure oscillations and possibly turbulence may be set up as a result of differences in the luminal diameters of the common iliac arteries, and so give rise to reflected waves that may injure the

intima of the distal abdominal aorta. The role of the relative calibers of the iliac arteries remains uncertain (**Shah et al., 1978**).

❖ Relations:

The upper abdominal aorta is related anteriorly to the coeliac trunk and its branches. The coeliac plexus and the lesser sac lie between it and the left lobe of the liver and lesser omentum. Below this, the superior mesenteric artery leaves the aorta, crossing anterior to the left renal vein. The body of the pancreas, with the splenic vein on its posterior surface, extends obliquely up and to the left across the abdominal aorta, separated from it by the superior mesenteric artery and left renal vein **(Standring et al., 2008)**.

Below the pancreas, the proximal parts of the gonadal arteries, and the third part of the duodenum, lie anteriorly. In its lowest part it is covered by the posterior parietal peritoneum and crossed obliquely by the origin of the small intestinal mesentery **(Standring et al., 2008)**.

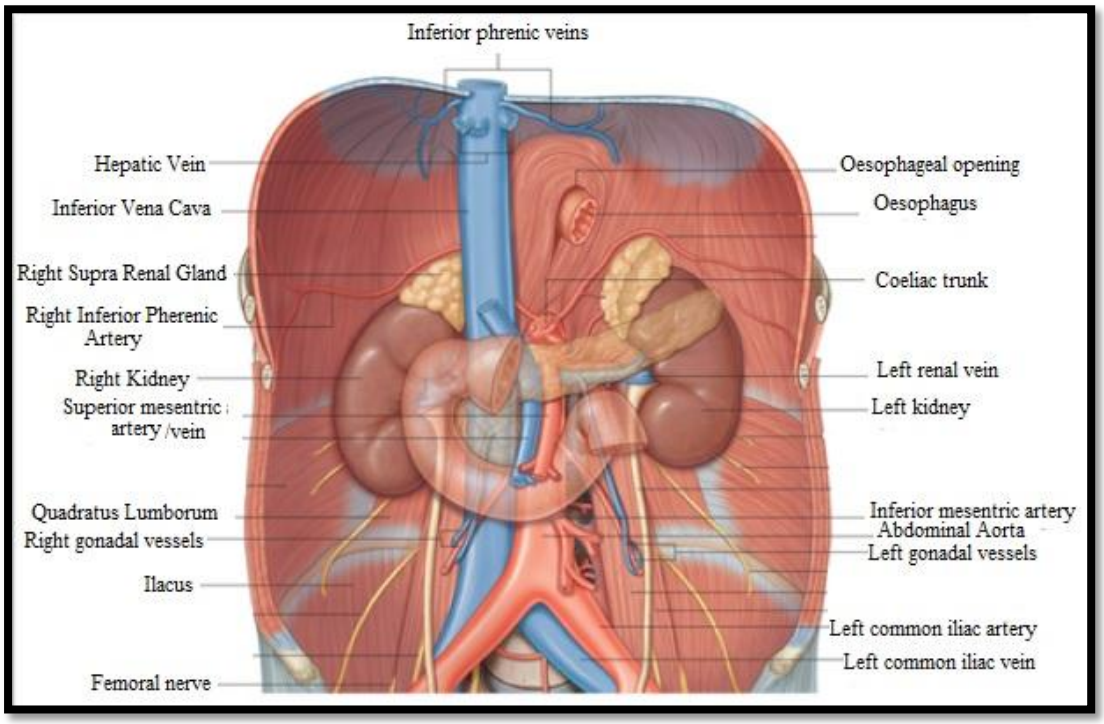


Figure (1): The abdominal aorta, the inferior vena cava and their main branches **(Standring et al., 2008)**

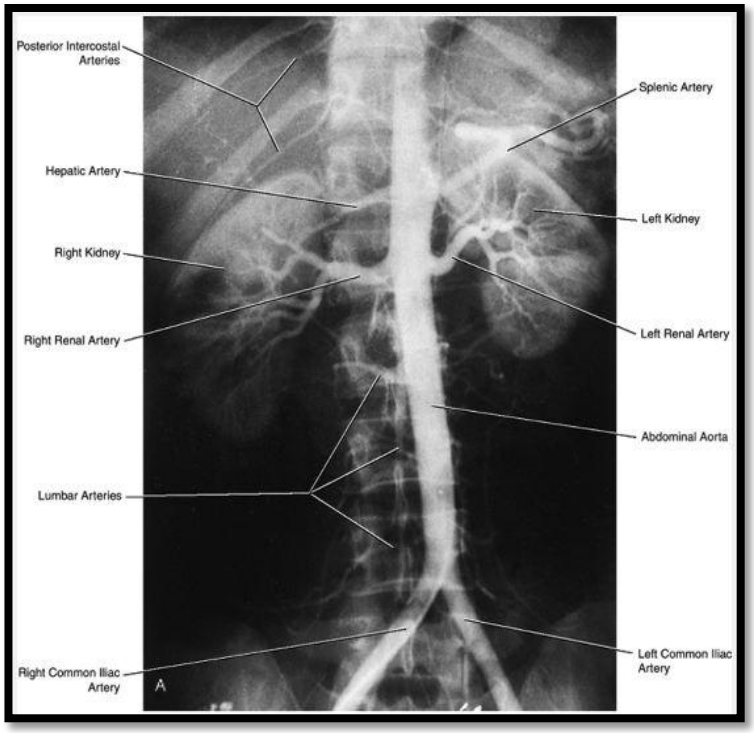


Figure (2): Angiography of the abdominal aorta and main branches **(Uflacker, 2007).**

Posterior to the abdominal aorta, the thoracolumbar intervertebral discs, the upper four lumbar vertebrae, intervening intervertebral discs and the anterior longitudinal ligament. Lumbar arteries arise from its dorsal aspect and cross posterior to it. The third and fourth (and sometimes second) left lumbar veins also cross behind it to reach the inferior vena cava. The aorta may overlap the anterior border of the left psoas major **(Standring et al., 2008)**.

On the right, the aorta is related above to the cisterna chili and thoracic duct, the azygos vein and the right crus of the diaphragm, which overlaps and separates it from the inferior vena cava and right coeliac ganglion. Below the second lumbar vertebra, it is closely applied to the left side of the inferior vena cava. This close relationship occasionally allows the formation of an aorto-caval fistula, particularly after aneurysmal disease **(Standring et al., 2008)**.

On the left, the aorta is related above to the left crus of the diaphragm and left coeliac ganglion. Level with the second lumbar vertebra, it is related to the duodenojejunal flexure and the left sympathetic trunk, the fourth part of the duodenum and the inferior mesenteric vessels **(Standring et al., 2008)**.

❖ Branches:

The branches of the aorta are described as anterior, lateral and dorsal. The anterior and lateral branches are distributed to the viscera. The dorsal branches supply the body wall, vertebral column,

vertebral canal and its contents. The aorta terminates by dividing into the right and left common iliac arteries **(Standring et al., 2008)**.

Anterior group

1- Coeliac trunk (coeliac axis):

The coeliac trunk is the first anterior branch and arises just below the aortic hiatus at the level of T12/L1 vertebral bodies. It is 1.5-2 cm long and passes almost horizontally forwards and slightly right above the pancreas and splenic vein. It divides into the left gastric, common hepatic and splenic arteries. The coeliac trunk may also give off one or both of the inferior phrenic arteries. The superior mesenteric artery may arise with the coeliac trunk as a common origin. One or more of the superior mesenteric branches may arise from the coeliac trunk **(Standring et al., 2008)**.

2- Superior mesenteric artery:

The superior mesenteric artery originates from the aorta 1cm below the coeliac trunk, at the level of the L1-2 intervertebral disc. It lies posterior to the splenic vein and the body of the pancreas. The left renal vein separates it from the aorta. It runs inferiorly and anteriorly, anterior to the uncinated process of the pancreas and the third part of the duodenum **(Standring et al., 2008)**.

3- Inferior mesenteric artery:

The inferior mesenteric artery is usually smaller in caliber than the superior mesenteric artery. It arises from the anterior or left

anterolateral aspect of the aorta at about the level of the third lumbar vertebra, 3 or 4 cm above the aortic bifurcation and posterior to the horizontal part of the duodenum **(Standring et al., 2008)**.

Lateral group

1- Suprarenal artery:

The middle suprarenal artery arises from the lateral aspect of the abdominal aorta, level with the superior mesenteric artery **(Standring et al., 2008)**.

2- Renal artery:

The renal arteries are two of the largest branches of the abdominal aorta and arise laterally from the vessel just below the origin of the superior mesenteric artery. The right is longer and usually arises slightly higher than the left. It passes posterior to the inferior vena cava, right renal vein, head of the pancreas and second part of the duodenum. The left renal artery arises a little lower down and passes behind the left renal vein, the body of the pancreas and the splenic vein **(Standring et al., 2008)**.

3- Gonadal artery

The gonadal arteries are two long, slender vessels that arise from the aorta a little inferior to the renal arteries. Each passes inferolaterally under the parietal peritoneum on psoas major **(Standring et al., 2008)**.

Dorsal group

1- Inferior phrenic arteries:

The inferior phrenic arteries usually arise from the aorta, just above the level of the coeliac trunk. Occasionally they arise from a common aortic origin with the coeliac trunk, from the coeliac trunk itself or from the renal artery **(Standring et al., 2008)**.

2- Lumbar arteries:

The lumbar arteries arise in series with the posterior intercostal arteries. There are usually four on each side. They arise from the posterolateral aspect of the aorta, opposite the lumbar vertebrae. A fifth, smaller, pair occasionally arise from the median sacral artery, but lumbar branches of the iliolumbar arteries usually take their place **(Standring et al., 2008)**.

3- Median sacral artery

The median sacral artery is a small branch that arises from the posterior aspect of the aorta a little above its bifurcation **(Standring et al., 2008)**.

Etiology of Aortic Aneurysms

A- Cystic Medial Degeneration

1- Atherosclerosis:

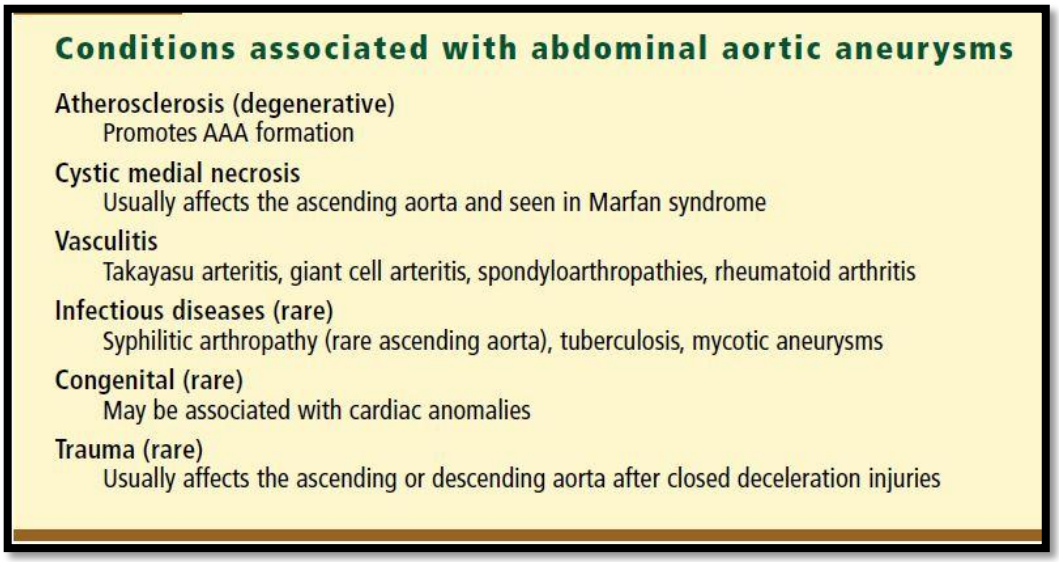
Atherosclerosis is one of the most common cause of aneurysmal degeneration in the ascending aorta **(Bickerstaff et al., 1982).** Atherosclerosis is less commonly seen in ascending aortic aneurysms than in descending thoracic or abdominal aortic aneurysms and it has long been theorized that the development of invasive atheromas results in destruction of elastic fibers and smooth muscle cells in the media, resulting in weakening and dilation. This process was proposed as the primary etiology of descending thoracic and abdominal aortic aneurysms, and the second most common cause of ascending aortic aneurysms **(Galloway et al., 1898).**

These theories are challenged by the concept that atherosclerosis is a concomitant process that infiltrates a diseased media with altered barriers. This would explain the divergent course of the atherosclerotic abdominal aorta towards obstructive versus aneurysmal disease **(Svensson et al., 1997).**

2- Hypertension and age:

Cystic medial degeneration is known to occur to some extent with aging, but this process is accelerated by hypertension. Hypertension leads to intimal thickening, degradation of the extracellular matrix, loss of elastic fibers, and smooth muscle cell necrosis. As a consequence, the aortic wall becomes stiff and progressively dilates **(Guo et al., 2001).**

Table (1): Conditions associated with abdominal aortic aneurysms:



Conditions associated with abdominal aortic aneurysms

Atherosclerosis (degenerative)
Promotes AAA formation

Cystic medial necrosis
Usually affects the ascending aorta and seen in Marfan syndrome

Vasculitis
Takayasu arteritis, giant cell arteritis, spondyloarthropathies, rheumatoid arthritis

Infectious diseases (rare)
Syphilitic arthropathy (rare ascending aorta), tuberculosis, mycotic aneurysms

Congenital (rare)
May be associated with cardiac anomalies

Trauma (rare)
Usually affects the ascending or descending aorta after closed deceleration injuries

(Almohameed et al., 2005)

B- Connective tissue disorders:

When cystic medial degeneration occurs at younger ages, it is classically associated with recognized connective tissue disorders, such as Marfan syndrome or less commonly, Ehlers– Danlos syndrome or Turner syndrome **(Guo et al., 2001)**.

(i) Marfan syndrome

It is an autosomal dominant variably penetrant inherited disorder of the connective tissue in which cardiovascular, skeletal, ocular, and other abnormalities may be present to a variable degree. The prevalence is estimated to be around 1 in 3000–5000 individuals. It is caused by mutations in the gene that encodes fibrillin-1 (FBN1) on chromosome 15. More than 300 mutations in FBN1 have been described. The phenotype presents to a highly variable degree because of varying genotype expression **(De Paepe et al., 1996).**

The clinical features are the result of weaker connective tissues due to defects in fibrillin-1, a glycoprotein and principal component of the extracellular matrix microfibril. The diagnosis of Marfan syndrome is made on clinical grounds, and it is not always simple because of the variability in clinical expression. A multidisciplinary approach is needed to diagnose and manage patients afflicted with this syndrome. The presence of major criteria in two separate systems and involvement of a third (minor or major) are needed to establish the diagnosis **(De Paepe et al., 1996).**

The most common cardiovascular features are aortic root aneurysm and mitral valve prolapse. These anatomical abnormalities may cause aortic rupture, aortic dissection, aortic insufficiency, and mitral insufficiency **(De Paepe et al., 1996).**

(ii) Ehlers–Danlos Syndrome

Is one of the disorders that involves the skin and joints causing hyperelasticity and fragility of the skin and hypermobility of the joints. It may also involve the cardiovascular system. Vascular Ehlers–Danlos syndrome is a rare autosomal dominant inherited disorder of the connective tissue resulting from mutation of the COL3A1 gene encoding type III collagen. Affected individuals are prone to serious vascular, intestinal, and obstetrical complications. These problems are rare during infancy but occur in up to 25% of affected persons before the age of 20 years and in 80% before the age of 40. Spontaneous rupture without dissection of large and medium-caliber arteries such as the abdominal aorta and its branches, the branches of the aortic arch, and the large arteries of the limbs accounts for most deaths. Intestinal perforation,

usually involving the colon, is less fatal. Pregnancy is a high risk. Aortic root dilation was present in 28% in a series of 71 patients with Ehlers–Danlos syndrome aortic dissection is uncommon **(Germain, 2002).**

Diagnosis is based on clinical findings including specific facial features, thin translucent skin, tendency to bleed, and rupture of vessels and/or viscera. Diagnosis can be confirmed either by biochemical assays or by molecular biology studies demonstrating mutation of the COL3A1 gene. Diagnosis should be suspected in any young person presenting with arterial or visceral rupture or colonic perforation. There is currently no specific treatment for this syndrome **(Germain, 2002).**

(iii) Turner syndrome

A sex aneuploidy syndrome in which chromosomes XO occur in 1:5000 live female birth cardiovascular problems include bicuspid aortic valve, coarctation, thoracic aortic aneurysm and dissection (**Pyeritz, 2002)**

(iv) Loeys-Dietz syndrome (LDS)

A recently described autosomal dominant aortic aneurysm syndrome that has widespread systemic involvement. A spectrum of mutations in transforming growth factor ß receptors 1 and 2 (TGFBR1 or TGFBR2, respectively) cause the LDS which histologically results in a loss of elastin content and disarrayed elastic fibers in the aortic media **(Loeys et al., 2006).**

C-Familial thoracic aortic aneurysm syndrome :

Most pedigrees have suggested an autosomal dominant mode of inheritance, but some have suggested a recessive mode and possibly X-

linked inheritance as well. In an analysis using a large database of patients with thoracic aortic aneurysms, it has been found that at least 19% of patients without Marfan syndrome had a family history of a thoracic aortic aneurysm **(Coady et al., 1999).**

D- Aneurysms Associated with Aortic Dissection:

Patients who survive acute dissection of the aorta often have or will develop an associated aneurysm. This aneurysmal dilation most commonly originates from the false lumen of the aortic dissection. This outer wall of the false lumen consists of only the weak outer media and adventitia. The rate of expansion is higher than in other types of aortic aneurysms, as the barrier to dilation and rupture is only this outer one-third of the media and the adventitia **(Dapunt et al., 1994).**

E- Inflammatory Aneurysms:

(i) Mycotic aneurysms:

The term mycotic denotes aneurysms originating on the basis of infection. Mycotic aneurysms of the aorta are rare but can be fatal if they are not diagnosed early. Bacterial seeding of the aortic wall can occur by hematogenous spread to the intima or the vasa vasorum, lymphatic spread, or direct extension from an adjacent infected focus. The endothelial lining of the aorta is generally high resistant to infection, but disruption of this barrier by atherosclerosis reduces resistance to infection. Aneurysms are usually saccular and well localized with staphylococcus aureus and salmonella species being the predominant organisms **(Malouf and Chandrasekaran, 2003)**.