INTRODUCTION

An arterial aneurysm is a pathologic entity defined by a gross anatomic change in the geometry of the vessel wall, by increasing in arterial diameter of at least 50% compared to a base line [1].

An aortic aneurysm forms when a part of the thoracic or abdominal aorta weakens and the wall stretches and expands, it becomes thinner, loses its elasticity and dilates like a balloon [2].

Anatomically, aneurysms that involve the ascending aorta may extend as proximally as the aortic annulus and as distally as the innominate artery, while that of aortic arch involve the segment of aorta between a line at a right angle proximal to the innominate artery origin and extending to a line drawn at a right angle distal to the origin of the left subclavian artery, whereas descending thoracic aorta begin beyond the left subclavian artery to the aortic opening of diaphragm at the level of T12 [3].

There are two gold-standard classification systems for aortic dissections: the DeBakey system and the Stanford system. They differ in that the former is based on the anatomy, and the latter on management of the patient.

In general, Stanford type A affect the ascending aorta but limited to the ascending aorta and arch only, while type B

affect the descending aorta. Type A is a surgical emergency, in contrast to type B, which is less lethal and can be treated conservatively with medical therapy and concomitant endovascular intervention [4].

The DeBakey classification of dissecting aneurysms of the aorta, type I has an intimal tear in the ascending aorta with dissection extending down the entire aorta, type II has an intimal tear in the ascending aorta with dissection limited to the ascending aorta, type III has an intimal tear in the proximal descending thoracic aorta with dissection either limited to the thoracic aorta up to diaphragm (type IIIa), or extending distally to the abdominal aorta or bifurcation (type IIIb)[5].

The majority of aneurysms are degenerative; other causes include traumatic, mycotic and pseudo-aneurysms. The mean age of diagnosis is 59–69 yr with a male predominance of 2:1–4:1. The 5-yr survival rates of patients with thoracic and abdominal aortic aneurysms not surgically treated is ε20 and 16–19%, respectively [6].

A recent population-based study suggests an increasing prevalence of thoracic aortic aneurysms (TAAs). The rise is probably artifactual, resulting from improvements in diagnosis. The incidence of TAAs is 10.4 per 100,000[7]. TAAs are relatively rare and comprise 2% to 5% of all degenerative

aneurysms, with the majority of aortic aneurysms affecting the abdominal aorta [8].

Aneurysms are often silent without symptoms until rupture occur. Patients are frequently being diagnosed with a TAAs on routine imaging for unrelated presentations [9].

Recent advances in imaging technologies have drastically boosted the role of pre-procedural imaging. The accepted diagnostic gold standard, digital subtraction angiography, is now being challenged by the state-of-the-art computed tomography angiography (CTA), magnetic resonance angiography (MRA) and trans-esophageal echocardiography (TEE). Among these, technological advancements of multidetector computed tomography (MDCT) have propelled it to being the default modality used, optimizing the balance between spatial and temporal resolutions and invasiveness. MDCT angiography allows the comprehensive evaluation of thoracic lesions in terms of morphological features and extent, presence of thrombus, relationship with adjacent structures and branches as well as signs of impending or acute rupture, and is routinely used in these settings[10].

Treatment of abdominal aortic aneurysms, thoraco-abdominal aneurysms, and thoracic aneurysms involves surgical repair in good-risk patients with aneurysms that have reached a size sufficient to warrant repair. Descending thoracic

aneurysms may be repaired with open surgery or, if appropriate, with endovascular stent grafting techniques[11]. Stent graft repair of descending thoracic aortic aneurysms should be performed if the predicted operative risk is lower than that of an open repair. Patient age, comorbidities, symptoms, life expectancy, aortic diameter, characteristics and extent of the aneurysm, and landing zones, should also be taken into consideration.

The high mortality of aneurysmal disease has led many to suggest surgical repair of the dilated aorta, even though the anatomical morphology of the thoracic aorta together with the characteristics of patients affected by TAAs pose a considerable challenge to surgeons. Open surgical repair using prosthetic graft interposition is the conventional treatment for TAAs mainly because of its feasibility and effectiveness in excluding the degenerated aorta from the systemic circulation. Open surgical repair of TAAs is associated with significant perioperative complications including 30- day mortality and paraplegia, with rates of 4.8% and 4.6% respectively. Stroke and renal failure are also important complications to be considered [12].

Despite noteworthy improvements in surgical procedures with extracorporeal circulation for peripheral organ preservation, the development of different techniques for spinal

cord protection, together with the refinement of prosthetic grafts for aortic repair, morbidity and mortality rates remain high [13].

Non-surgical repair may be an attractive therapeutic option for patients with TAAs. The success of the stent-graft placement performed in abdominal aortic aneurysms prompted experts to translate the technique to thoracic aneurysm repair. Dake was the first to report the use of stent graft in 13 patients with TAAs with positive outcomes over 11 months of observation[14].

AIM OF WORK

This essay aims at reviewing TEVAR versus conventional open surgery in patients with thoracic aortic aneurysms regarding the efficiency and outcome of both procedures.

Chapter (I):

ANATOMY OF THORACIC AORTA

1. Anatomy of thoracic aorta:

1.1. Sections of thoracic aorta:

The thoracic aorta is divided into 4 parts: the ***aortic root*** (which includes the aortic valve annulus, the aortic valve cusps, and the sinuses of Valsalva); the ***ascending aorta*** (which includes the tubular portion of the ascending aorta beginning at the sinotubular junction and extending to the brachiocephalic artery origin); the ***aortic arch*** (which begins at the origin of the brachiocephalic artery, and is the origin of the head and neck arteries; and the ***descending thoracic aorta*** (which begins at the isthmus between the origin of the left subclavian artery and the ligamentum arteriosum and courses anterior to the vertebral column, and then through the diaphragm into the abdomen)[15].

2.1. Branches of thoracic aorta *(figure 1):*

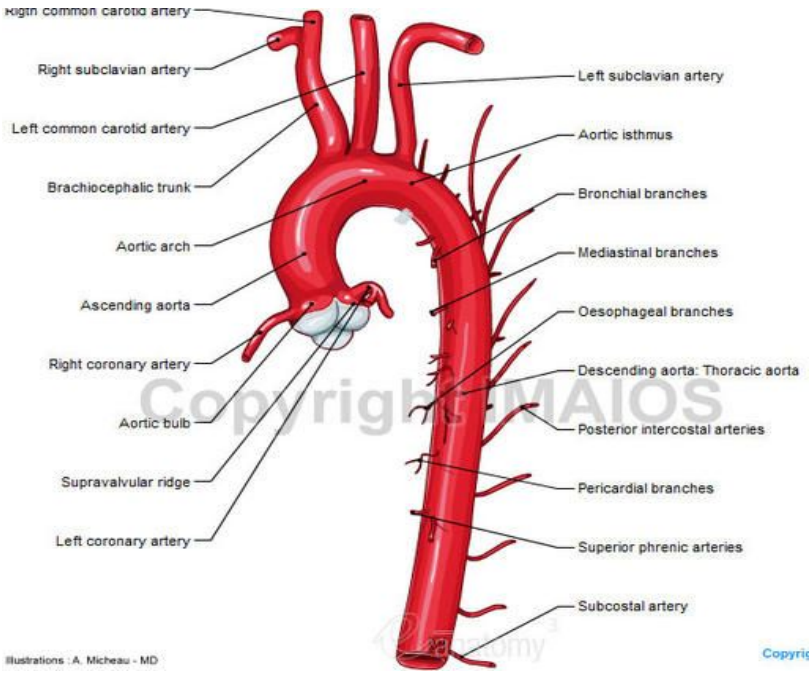

Figure (1): Branches of thoracic aorta

The only branches of the ascending aorta are the two coronary arteries which supply the heart; they arise near the commencement of the aorta from the aortic sinuses which are opposite the aortic valve [16].

Three branches arise from the convex aspect of the arch: the brachiocephalic trunk, left common carotid and left subclavian arteries. They may branch from the beginning of the arch or the upper part of the ascending aorta. The distance between these origins varies, the most frequent being approximation of the left common carotid artery to the brachiocephalic trunk[18]. An analysis of variation in branches

from 1000 aortic arches showed the usual pattern in 65%; a left common carotid shared the brachiocephalic trunk in 27%, and the four large arteries branched separately in 2.5%. The remaining 5% showed a great variety of patterns, the most common (1.2%) being symmetric right and left brachiocephalic trunks[17].

The descending thoracic aorta provides visceral branches to the pericardium, lungs, bronchi and oesophagus, and parietal branches to the thoracic wall.

Pericardial branches are few small vessels are distributed to the posterior aspect of the pericardium. **Bronchial branches** vary in number, size and origin. There is usually only one right bronchial artery. This arises either from the third posterior intercostal or upper left bronchial artery, and runs posteriorly on the right bronchus. The left bronchial arteries, usually two, arise from the thoracic aorta, the upper near the fifth thoracic vertebra, the lower below the left bronchus. They run posteriorly to the left bronchus and are distributed as on the right. **Phrenic branches** arise from the lower thoracic aorta and are distributed posteriorly to the superior diaphragmatic surface. They anastomose with the musculophrenic and pericardiacophrenic arteries[18].

There are usually nine pairs of **posterior intercostal arteries**. They arise from the posterior aspect of the descending thoracic aorta and are distributed to the lower nine intercostal

spaces. Right posterior intercostal arteries are longer, reflecting the aortic deviation to the left, they cross the vertebral bodies behind the oesophagus, thoracic duct and azygos vein, right lung and pleura. Left posterior intercostal arteries turn backwards on the vertebral bodies in contact with the left lung and pleura; the upper two are crossed by the left superior intercostal vein, and the lower by the hemiazygos and accessory hemiazygos veins[19].

Posterior intercostals arteries divide three times before reaching the spinal cord. The first branch is the spinal branch, which divides into the anterior and posterior radicular arteries and, farther on, bifurcates into the dorsal and vertebral branches. The last bifurcation of the spinal branch is constant for anterior and posterior supply of the vertebral canal, of the nerve roots and of the dura mater, at some levels only, and the anterior and posterior radicular arteries pass through the dura mater and reach the marrow. Generally, one of the anterior radicular arteries is dominant in terms of caliber and is known as the ***great anterior radicular artery, Adamkiewicz artery or arteria magna*** *(figure 2)*[20].

The artery of Adamkiewicz is most commonly found originating from a posterior intercostal artery branching from the aorta between the spinal levels of T9 and T12; however, its variability allows it to be found anywhere from the spinal levels

of T9 and L5. Immediately following its point of origin, the artery of Adamkiewicz extends through the intervertebral foramen of the corresponding spinal level. It then ascends the anterior surface of the spinal cord as many as two and a half vertebrae before it undergoes its characteristic "hairpin curve" after which it immediately anastomoses with the anterior spinal artery [19].

Great radicular artery of Adamkiewicz provides the major blood supply to the lumbar and sacral cord. When damaged or obstructed, it can result in anterior spinal artery syndrome, with loss of urinary and fecal continence and impaired motor function of the legs; sensory function is often preserved to a degree. It is important to identify the location of the artery when surgically treating an aortic aneurysm to prevent damage which would result in insufficient blood supply to the spinal cord [20].

Subcostal arteries are the last paired branches of the thoracic aorta, in series with the posterior intercostal arteries, and are below the twelfth ribs.

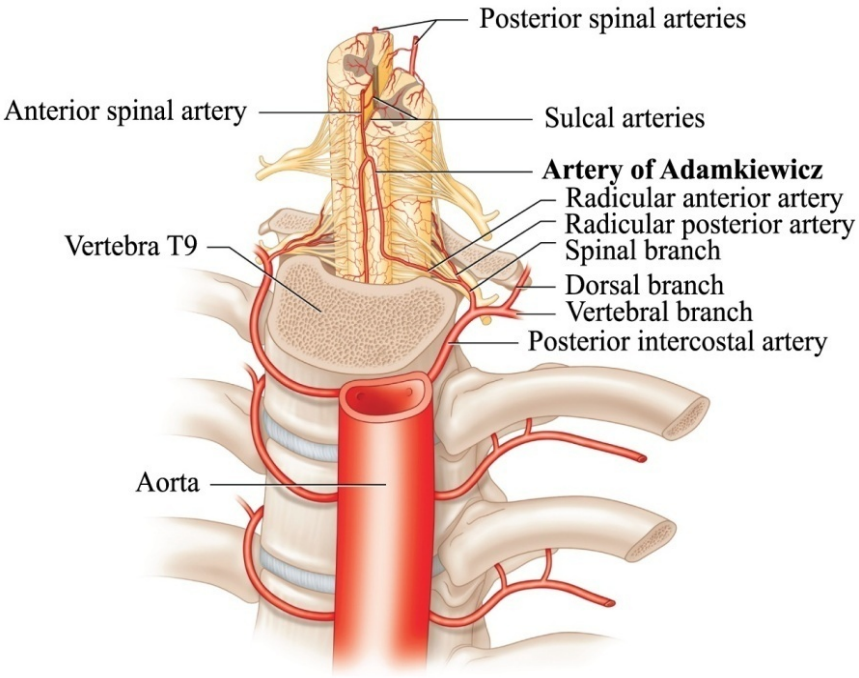

Figure (2): Schematic drawing of the blood supply of the spinal marrow [20].

Chapter (II):

PATHOLOGY OF AORTIC ANEURYSM

1. Pathological terms of arterial dilatation:

There are many pathological terms of arterial dilatation should be differentiated:

1.1. Aneurysm: is a permanent localized dilatation of an artery, having at least a 50% increase in diameter compared to the expected normal diameter of the artery in question. Although all 3 layers (intima, media, and adventitia) may be present, the intima and media in large aneurysms may be so attenuated that in some sections of the wall they are undetectable [21]. In patients with Marfan syndrome, when the cross-sectional area (in square centimeters) divided by the patient's height (in meters) exceeds a ratio of 10, this should then be considered significant and an indication that the patient requires surgical repair Thus, in some respect, the definition of an aneurysm is not absolute, but rather refers to the significant dilatation of the aorta [22].

1.2. Pseudoaneurysm (or false aneurysm): contain blood resulting from disruption of the arterial wall with extravasation of blood contained by periarterial connective tissue and not by the arterial wall layers. Such an extravascular hematoma that freely communicates with the intra-vascular space is known as a pulsating hematoma [23].

1.3. Aortic dissection: disruption of the media layer of the aorta with bleeding within and along the wall of the aorta. Dissection may, and often does, occur without an aneurysm being present. An aneurysm may, and often does, occur without dissection. The term dissecting aortic aneurysm is often used incorrectly and should be reserved only for those cases where a dissection occurs in an aneurysmal aorta[24].

1.4. Intramural hematoma (IMH): is a variant of classic aortic dissection. It results from hemorrhage that occurs in and is contained within the medial layer of the aortic wall, rather than from a primary tear in the intima. There is no communication between the hematoma and the aortic lumen. The clinical signs and symptoms associated with intramural hematoma resemble those seen in classic aortic dissection [25].

1.5. Penetrating atherosclerotic ulcer (PAU): is defined as an ulceration of an aortic atherosclerotic plaque penetrating through the internal elastic lamina into the aortic media [26] ***(****figure 3).*

1.6. Ectasia: is an arterial dilatation less than 150% of normal arterial diameter.

1.7. Arteriomegaly: is diffuse arterial dilatation involving several arterial segments with an increase in diameter greater than 50% by comparison to the expected normal arterial diameter [23]**.**

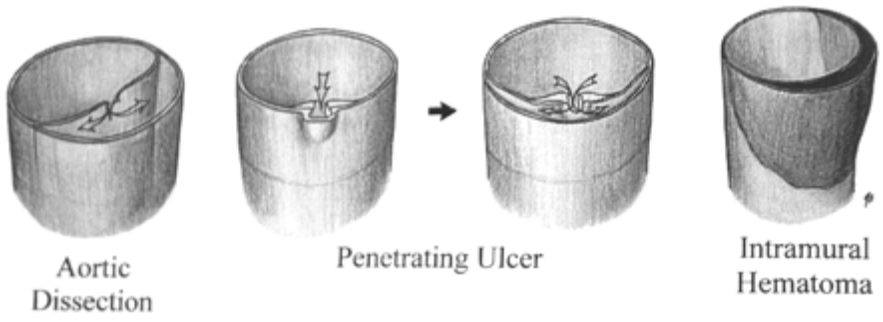

Figure (3): Aortic dissection, IMH, and PAU[26]**.**

2. Epidemiology:

Given the indolent nature of aneurysms, the true incidence of aneurysm disease is hard to estimate. On the other hand, lethal thoracic aortic dissections are also often misdiagnosed as myocardial infarctions [27].

The widespread use of imaging techniques in the current era has led to the earlier recognition of aneurysms, permitting the identification of more patients. Determining whether the apparent increase in number of patients seen is due to increased detection or to a genuine increase in the incidence of this disease has become a challenge. However, recent evidence suggests the latter[28].

Data available from population-based studies estimate the incidence of TAAs to be around 10.4 cases per 100,000 person-years[29]. Although findings from autopsy series vary widely, the prevalence of TAAs probably exceeds 3–4% in individuals older than 65 years. The incidence of aortic disease is certain to increase both as a result of progressive aging of the population and its associated rise in hypertension prevalence [8].