# DISSECTING THORACIC AORTIC ANEURYSM WITH CLASSIC IMAGING FINDINGS AND REVIEW OF LITERATURE

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### **ABSTRACT**

Background: Thoracic aortic aneurysm is an increasingly recognized condition that is sometimes diagnosed incidentally on imaging examinations performed to evaluate other unrelated conditions. Symptomatic presentations of thoracic aortic aneurysms may be due to mass effect on the airway, esophagus, recurrent laryngeal nerve and the thoracic spine. Alternatively, they may present with the dreaded complication of aortic dissection or rupture.

Occasionally abnormalities of the aortic contour or size can be detected on routine chest x-ray. However, it is difficult to confidently diagnose thoracic aortic aneurysm on chest x-rays as mediastinal masses may mimic aortic aneurysms. Computed tomography (CT) or magnetic resonance (MR) aortography, with the advantage of obtaining 3D volumetric data, remains the gold standard of imaging with sensitivity and specificity approaching 100%. Objective: To emphasize beauty of cross-sectional images in unraveling confusing opacities on sheet radiographs and its sensitivity in identifying

Objective: To emphasize beauty of cross-sectional images in unraveling confusing opacities on chest radiographs and its sensitivity in identifying potentially lethal chest pathologies.

Method: This is a case report of a 58-year-old man with breathlessness and features of congestive cardiac failure. Preliminary chest X-ray revealed a huge soft tissue opacity in the left upper zone of the lung which was conformed as aneurysmal dilatation of the thoracic aorta on chest computed tomography. Conclusion: This case elucidates the importance of cross-sectional imaging in unravelling confusing opacities on chest radiographs and its sensitivity in identifying potentially lethal chest pathologies.

Keywords: Aortic Aneurysm, Opacity, Hypertension, Computed tomography.

## INTRODUCTION

Aortic aneurysm is a localized or diffuse dilatation involving all layers of the aortic wall, exceeding the expected aortic diameter by a factor of 1.5 or more.<sup>1</sup> The thoracic aorta consists of four main segments which are: the aortic root, the ascending aorta, the aortic arch, and the descending aorta; and any of these segments may be involved in thoracic aortic aneurysm. The incidence of thoracic aortic aneurysms (TAAs) is estimated to be increasing and there are around 10.4 cases per 100 000 person-years.2 The etiology is multifactorial with an interplay of both genetic and environmental factors acting in consonance to initiate a series of arterial wall degeneration known as medial degeneration, characterized by disruption and loss of elastic fibers and increased deposition of proteoglycans.1-3

The most common cause of aneurysms is atherosclerosis, a condition manifested by hypertension-

induced arterial wall weakening and rupture.<sup>4</sup> Other causes include trauma, infection including tuberculosis and syphilis, non-infectious arteritis e.g giant cell arteritis, Takayasu's arteritis and Behcet disease. Syndromes such as Marfan's syndrome, Ehlers-Danlos syndrome, Loeys-Dietz syndrome and Turner's syndrome are genetic causes.<sup>1-3</sup>

Thoracic aortic aneurysm may be asymptomatic and discovered incidentally during radiologic investigations for related or unrelated medical conditions. Therefore, in the appropriate clinical setting, high index of suspicion is required when evaluating patients with solitary mediastinal mass incidentally identified on a chest radiograph.

We present a case of TAA incidentally identified on a plain chest radiograph in our institution and then reviewed the literature.

### CASE REPORT

Patient is a 58-year-old African male, who is a known hypertensive and being managed for hypertensive heart disease and atrial fibrillation by the Cardiology unit, was re-admitted on account of difficulty in breathing on moderate exertion as well as bilateral ankle swelling of 1-week duration secondary to poor drug compliance. He did not have a Marfanoid habitus or any feature suggestive of connective tissue disease. He drank 2 bottles of alcoholic beverage per day for about ten years, however he stopped taking alcohol about 10 years ago. He also smoked cigarettes in his teenage years.

As part of patient's work up, a chest x-ray (Figure 1) was done which revealed a huge well defined homogeneous soft tissue opacity in the left upper lung



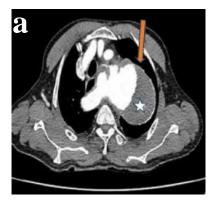
**Figure 1:** Frontal chest radiograph showing a huge well-defined homogeneous soft tissue opacity in the left upper zone bowing and displacement of the trachea to the right.

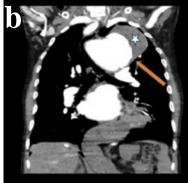
zone. There was associated bowing and displacement of the trachea to the right. No calcification was seen within the opacity. There was cardiomegaly however, the pulmonary vasculature was not engorged. The visualized lung fields were clear. These findings raised a suspicion for bronchogenic carcinoma. He was referred for a computed tomography of the chest to further evaluate this suspicious mass.

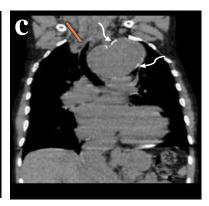
Chest computed tomography was done using a 16slice Phillips helical CT machine 9 days after the chest x-ray. This showed a huge outpouching arising from the distal aortic arch and proximal descending aorta. It had a smooth outline and measured 10.9cm by 6.9cm in its longitudinal and anteroposterior dimensions respectively, while its neck measured 3.6cm in diameter (Figure 2). It showed an anteromedial contrast opacified segment and a surrounding predominantly isodense area suggestive of a thrombus. Multiple atheromatous plaques are also seen within the rim and there are mass effects on the trachea and oesophagus. The heart was enlarged with a left ventricular preponderance. Cardiothoracic ratio = 57%. The pulmonary vasculature was prominent, however not dilated and no filling defect was seen within them.

A focal lobulated mildly enhancing isodense structure with a crescentic inferior hyperdense part was noted in the apical segment of the right lower lobe of the lung. It measured 2.3cm by 1.6cm in longitudinal and anteroposterior dimensions respectively and was in keeping with a solitary pulmonary nodule with associated calcification. The remaining lung fields were however clear. No lymphadenopathy. No pleural or pericardial effusion. The ribcage and overlying soft tissue were within normal limits.

The bone window images showed degenerative changes of the thoracic spine. There was no pressure erosion of the anterior vertebral bodies and no suspicious osseous lesion.







**Figure 2**. Axial contrast enhanced chest CT (a) and coronal reformat (b) showing the large TAA (arrow) with intramural thrombus (\*). The non-enhanced coronal reformat (c) shows mass effect on the trachea (arrow) and atherosclerotic plaques on the aneurysmal wall (curved white arrows).

### **DISCUSSION**

Thoracic aortic aneurysm refers to a localized or diffuse dilatation involving all layers of the aortic wall, exceeding the expected aortic diameter by a factor of 1.5 or more<sup>1</sup>.

Several genetic and environmental factors have been identified to play a causative role however, the most common cause is atherosclerosis<sup>4</sup>. Trauma, infections including tuberculosis and syphilis; non-infectious arteritis e.g giant cell arteritis, Takayasu's arteritis and Behcet disease, are other causes, while genetic syndromes such as Marfan's syndrome, Ehlers-Danlos syndrome, Loeys-Dietz syndrome and Turner's syndrome are identified associations<sup>1-3</sup>.

The most common location for TAAs is in the aortic root and/or ascending aorta, though they can also occur in the descending thoracic aorta and, less frequently, in the aortic arch<sup>3</sup>. In our case, the aneurysm involved the proximal descending aorta.

Commonly TAAs are identified incidentally on chest imaging. Symptomatic presentation may be due to mass effect on the airway, esophagus, recurrent laryngeal nerve and the thoracic spine. Alternatively, they may present with the dreaded complication of aortic rupture<sup>5</sup>.

Occasionally abnormalities of the aortic contour or size can be detected on routine chest x-ray however, it is difficult to confidently diagnose TAAs on chest radiographs as mediastinal masses may mimic aortic aneurysms. Asymptomatic TAA is commonly detected as a mediastinal widening or a calcified soft-tissue mediastinal mass on a chest radiograph taken for some other reason; not infrequently, as in this patient, it is confused with a tumour<sup>1</sup>.

Ultrasound has a limited role in evaluation of TAAs, as its ability to demonstrate the thoracic aorta is confined to the proximal aortic segments. Computed tomography (CT) and magnetic resonance imaging (MRI), however, have unlimited access to thoracic structures thus, they have superior accuracy in diagnosing TAAs. Multi-detector computed tomography (MDCT) can easily detect TAAs with 100% accuracy and with its added advantage of direct visualization of aortic wall calcifications, which is important in planning a surgical or endovascular repair, supersedes MRI. However, MRI is preferred to CT for monitoring expansion rate of chronic TAAs because it does not use ionizing radiation<sup>1</sup>. Also, there are no dimensional gaps in MRI hence accurate lesion dimensions can be achieved.

Although digital subtracted angiography (DSA) used to be the gold standard for vascular imaging, it has been superseded by CT angiography and MR angiography, which can obtain 3D volumetric data, and able to assess the extraluminal soft tissues<sup>5</sup>. However, DSA provides direct visualization, and therefore remains invaluable, in endovascular repairs of TAAs.

Patients with TAAs are at higher risk for aortic dissection if they are hypertensive, especially when poorly or not controlled, have dyslipidemia, indulge in smoking, cocaine use and other stimulants that increase aortic wall stress<sup>6</sup>. Therefore, people with TAAs must be counseled about smoking cessation and must avoid exposure to passive smoking<sup>7</sup>. Also, dyslipidemia must be treated, and high blood pressure should be controlled using a beta blocker and angiotensin converting enzyme inhibitors or angiotensin receptor blockers<sup>6,8</sup>.

Surgical interventions are usually considered if there are symptoms suggesting TAAs expansion, ascending thoracic aorta or aortic sinus diameter is <sup>3</sup>5.5cm, degenerative TAAs, chronic aortic dissection, intramural hematoma, penetrating atherosclerotic ulcer, mycotic aneurysm, or pseudoaneurysm<sup>6</sup>. Asymptomatic patients with genetic conditions such as Marfan's syndromes should have elective surgery if their diameters are 4.0 to 5.0 cm depending on the disorder to prevent acute dissection or rupture<sup>6,9,10</sup>. If thoracic aortic dissection develops, aortic shear stress must be lowered, and a decision made on which patient should have surgical intervention or endovascular repair<sup>6</sup>.

#### **CONCLUSION**

The incidence of TAAs is increasing and clues to their presence may first be identified on a plain chest radiograph usually acquired for related or unrelated pathologies.

Though angiographic studies (CT Angiography, Conventional Angiography) are the imaging investigations of choice in the setting of aneurysms, chest CT can suggest the presence of an aneurysmal dilatation in the great vessels especially when large.

## Ethical statement

Written informed consent for the case to be published (including images, case history and data) was obtained from the patient.

### Conflict of interest

The authors have no conflicts of interest to declare

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