THORACIC AORTIC ANEURYSM (TAA) – A DIAGNOSTIC DILEMMA IN A PATIENT WITH HAEMOPTYSIS

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ABSTRACT

This case illustrates the difficulty in diagnosing TAA from an uncommon symptom of haemoptysis, especially in a patient with recent cardiac event and the possibility of having a lung malignancy as he is an ex-smoker. It appears that CT Angiography is the modality of choice here.

KEYWORDS: haemoptysis, thoracic aortic aneurysm, CT angiography, diagnostic imaging

BACKGROUND

With increasing age, smoking, and hypertension; the walls of the aorta weaken and expand, causing the ballooning of a segment known as aneurysm.¹ The usual symptoms of thoracic aorta aneurysm (TAA) are chest pain, back pain, cough, shortness of breath, and hoarseness of voice.²⁻³ These symptoms occur as the aneurysm compresses on surrounding structures. Rarely, haemoptysis develops when there is erosion into the respiratory system by the aneurysm.²⁻⁴ However, the majority of TAA still remain asymptomatic and an incidental finding on radiographs.² This case report illustrates hemoptysis as a rare symptom of TAA.

CASE REPORT

A 78 year old gentleman walked into Emergency Department with a complaint of left sided chest pain. The onset was sudden and it occurred during exertion. It was a crushing, non-radiating pain associated with palpitations. He also had episodes of haemoptysis associated with shortness of breath for the last two days.

His last admission was approximately a year prior, where he suffered from an acute extensive myocardial infarction (MI) complicated with cardiogenic shock. Intravenous thrombolysis was administered and he was then transferred to the Coronary Care Unit. Other comorbid factors include hypertension and stage III chronic kidney disease. He was also an ex-smoker. Past
surgical history revealed an appendicectomy done many years ago.

On presentation, he was alert and conscious; but tachypneic with a respiratory rate of 28 breaths per minute. His heart rate was 133 beats per minute and his blood pressure was 202/112 mm of Hg. Oxygen saturation was 87% under room air and there were bibasal crepitations on auscultation of his lungs. Both the first and second heart sounds were heard without murmur, while his abdomen was soft and non tender. There were no radio-radial or radio-femoral delays. Both femoral pulses and all distal pulses of the lower limbs were of strong volume. There were also no significant discrepancies in the blood pressures of all four limbs.

Routine blood investigations revealed a normal full blood count, coagulation profile, and liver function test. Cardiac enzymes were also not raised. Parameters for renal profile were normal except for his serum creatinine which was 200μmol/L. His first electrocardiogram (ECG) showed ST-segment elevation over leads V2 and V3, while the second had T-wave inversions over lead V2 with ST-segment elevation over lead V3. ECG from his previous admission was ST-segment elevation over leads I, aVL, V1 to V6.

A chest radiograph done showed a widened mediastinum. It was noted that this abnormality was already present in previous chest radiograph film performed during his previous admission for MI. This abnormality was reported as the unfolded aorta after consultation with the radiologist. However, a serial comparison of the chest radiograph films over the span of a year (figure 1) revealed a progressively increasing size of mediastinum. The prospect of lung malignancy and TAA as differential diagnoses was entertained. A decision for computed tomography angiograph (CTA) of the thorax and abdomen was then agreed upon.

CTA was performed and was reported as thoracic aneurysm beginning from the aortic arch distal to the left subclavian artery until the descending aorta, measuring 7.9 cm in width and 11 cm in length (figure 2A-A). There was associated left pleural effusion with heterogeneous density, suspicious of leaking TAA (figure 2A-B). Circumferential mural thrombus with atherosclerotic plaque was also seen, but there was no dissection (figure 2B, right arrow 1). The trachea was only slightly deviated to the right (figure 2B, left arrow 2). The aneurysm had caused splaying of the left pulmonary artery, and also pushed the left main bronchus infero-anteriorly; without disrupting both their patency (figure 3A-A,B). The heart was enlarged but there was no pericardial effusion. Another aneurysm was noted at the infra-renal abdominal aorta, measuring 5cm in both width and length (figure 3B). It was associated with mural thrombus but there was no dissection or leaking.

Surgical intervention was offered but the patient’s family opted for conservative management and admission for observation.

DISCUSSION

There are only a few literatures reporting haemoptysis as a presentation of TAA. This illustrates the infrequent presentation of haemoptysis in this condition which leads to diagnosis dilemma. Haemoptysis can represent a myriad of conditions.2-9 Major causes of haemoptysis have mostly remained the same over the decade. They include bronchitis, bronchiectasis, pneumonia, tuberculosis, lung carcinoma, and coagulopathy. TAA causing haemoptysis is very rare and only occurs in 6% of the cases.5 These patients usually have no other complaints.5,7
Haemoptysis occurs in TAA when the lower respiratory tract is breached by the aneurysm. A constantly pulsating large aortic aneurysm may erode the trachea and form a fistula. Via this fistula, blood from the aorta enters the airways and causes haemoptysis. It is possible for the aneurysm to erode any lower airway structure, especially if the blood pressure is high enough to cause strong pulsations. Based on CTA findings for this case, the aneurysm may have eroded the lung pleura with a resulting leak that caused a haemorrhagic pleural effusion. The process is further accelerated as the patient had uncontrolled hypertension which had also caused the TAA initially.

Blood investigations will be able to differentiate between infective causes of hemoptysis from a non-infective one. It will also be able to rule out systemic coagulopathy as the cause of haemoptysis. In TAA, the blood parameters will mostly be normal. ECG may be confusing as it will mimic MI especially if the aneurysm causes a coronary insufficiency. The ECG may also show changes which are non-specific.

REFERENCES


FIGURES

![Figure 1](image_url)

**Figure 1** Serial chest radiographs with progressive widening of the mediastinum, causing tracheal deviation to the right (white arrows). (A) Taken during presentation of MI late 2015. (B) Taken during follow up in 2016. (C) Taken during latest presentation.
Figure 2  (A) CTA of thorax revealed aneurysm of the aortic arch till descending aorta: A, contrast-filled lumen; B, associated heterogeneous density representing a leak into the pleura. (B) Right arrow. 1, Circumferential thrombus of the aneurysm. Left arrow. 2, Tracheal deviation to the right.

Figure 3  (A) Structures surrounding the aneurysm: A, Splaying of left pulmonary artery. B, Deviation of left main bronchus. Both structures maintain patency. (B) White arrows showing aneurysm of infra-renal abdominal aorta with mural thrombus.