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Abstract:

There has been a significant increase in the incidence of thoracic aortic aneurysms, currently making aneurysmal disease the 18th most common cause of death within the United States. This disease is typically an insidious one, with the aorta growing an average of only 0.1 cm per year. Consequently, most patients remain asymptomatic until late stages of the disease when dissection or worse, rupture has occurred. Under extremely rare circumstances, thoracic aortic aneurysms can present clinically due to mass effect. Airway, esophageal and vascular compression, secondary to aortic arch dilation has been previously documented. The condition can be acquired or congenital, and can also manifest as double aortic arch, aberrant subclavian artery, and pulmonary artery sling. Additionally, Kommerell's diverticulum, a bulbous configuration at the proximal descending aorta of left or right arch configuration, is a rare cause of tracheobronchal compression. Regardless of its form, mass effect due to thoracic vascular abnormality such as aneurysm can lead to Tracheomalacia, and ultimately airway collapse, under chronic conditions. Clinical presentation can vary from cough, to hoarseness of voice, chest pain, and in the case of our patient, progressive dyspnea which was initially mistaken for acute exacerbation of chronic heart failure.

Case Presentation:

A 92-year old female presents via EMS to the Jefferson Washington Emergency Department in acute respiratory distress. The report given by paramedics states that the woman had been experiencing progressively worsening shortness of breath for approximately the past three weeks; however, her condition had recently worsened to the point where she was experiencing dyspnea with minimal exertion, conversation and at rest. On arrival they found her sitting on her couch, struggling to breathe. The patient was demonstrating extreme work of breathing, with accessory muscle use and speaking in broken sentences. On auscultation they could appreciate what they thought to be rales in all lung fields. In addition, the patient displayed generalized edema. Believing her to be fluid overloaded and in acute decompensated heart failure, they placed the patient on 15L NRB and subsequently administered two doses of sublingual nitroglycerin. At that time the patient did report symptomatic improvement. On ED arrival the patient was still in respiratory distress, but she was hemodynamically stable and her pulse oximeter was measuring oxygen saturation levels approximating 98%. Upon hearing report from EMS, the patient was immediately placed on BiPaP and given 80 mg IV furosemide. Again, the patient endorsed symptomatic improvement. The clinical consensus among the ED team at that time was that EMS had been correct in their assessment given the patient's clinical improvement with pre-load reduction. It was only after the portable chest x-ray was obtained that the treatment team's understanding of the situation drastically changed. What suddenly became apparent was a gross deformation of the patient's mediastinum, which was both dramatically enlarged and anomalous. A STAT CT angiogram of the patient's chest was subsequently ordered. It revealed the following: 6 cm aneurysm of the aortic arch extending into the origin of the right brachiocephalic artery; a mural thrombus was noted in the aneurysm; a severe narrowing of the trachea due to compression by the thoracic aortic aneurysm; mass effect upon the esophagus; diffuse subcutaneous edema suggesting anasarca. It was immediately apparent that the patient needed emergent vascular intervention and was subsequently transferred to Jefferson Center City ICU. However, it was ultimately decided by the interventional cardiology team that, given the patient's age, and risk associated with the stenting, that the patient was placed on palliative care.

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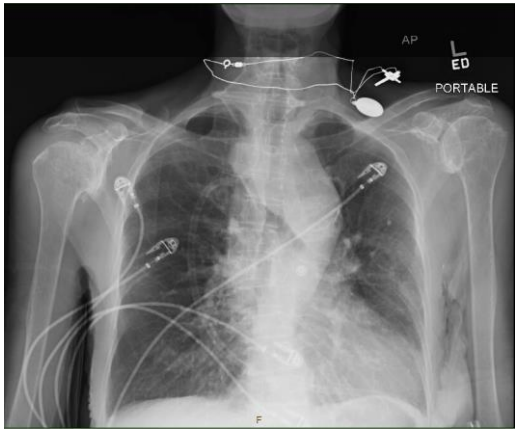

Figure 1: Chest X-Ray showing thoracic aneurysm compressing esophagus and trachea

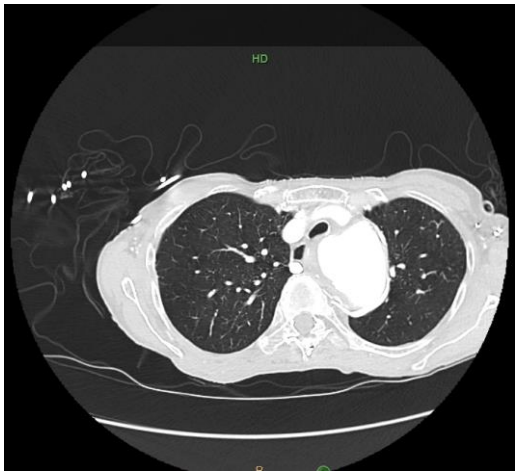

Figure 2: CT Angiogram showing thoracic aneurysm with esophageal and tracheal compression

Discussion:

Aneurysms occur when the wall of a given blood vessel weaken, causing it to enlarge or dilate. Aneurysms can form in any blood vessel in the body but are most commonly found in the aorta. The thoracic aorta is sub-divided into four different segments: aortic root, ascending aorta, aortic arch and the descending aorta, with thoracic aortic aneurysms (TAAs) typically involving one or more of these segments. Diameter of the thoracic aorta depends on may factors, including: patient age, sex, and body habitus. Consequently, an aneurysm is defined as an enlargement of the aorta that is 1.5 times expected size dependent upon patient characteristics and there are no specific cut-offs to achieve status as a TAA due to this high degree of variability. Underlying causes and treatment options for each segment are different. The most common location for TAA's is the aortic root (20%) and the ascending aorta (44%), with the most common cause of TAA being a process called cystic medical degeneration. In this disease, the elastic fibers comprising the vascular wall of the aorta degenerate, weakening the wall and leading to dilation and aneurysm. This pathologic state occurs typically between the years of 60 and 70. Smoking and high blood pressure are also associated with the formation of thoracic aneurysms. In younger patient's, TAA is more often attributed to genetic causes, which include but are not limited to: Marfan and Ehlers-Danlos syndromes, as well as Turner's syndrome in females. In addition, TAA's have been shown to be associated with bicuspid aortic valves, a congenital condition in which they aortic valve has two leaflets instead of three. Finally, inflammation of the aorta, whether due to infection (syphilis) or autoimmune (Takayasu or Giant Cell Arteritis), causes TAA by destroying collagen and elastic tissues in the walls of the aorta. The majority of those individuals who are afflicted by thoracic aneurysm are asymptomatic. Consequently, most TAA's are found incidentally when patients are undergoing imaging for another reason; however, under certain circumstances thoracic aneurysms can present as heart murmurs due to leakage of the aortic valve. The most serious complication of TAA is aortic dissection and/or rupture. Aortic rupture is associated with a high mortality rate but symptoms can include tearing chest pain or back pain with simultaneous low blood pressure or shock. With regard to diagnosis and measurement of TAA, computed tomography angiography (CTA) or magnetic resonance angiography (MRA) are the imaging tests of choice. In certain instances echocardiography can assist in the diagnosis of TAA when the aortic root or ascending aorta is involved; however, its ability to assess the more distal segments of the aorta remain limited. Treatment of TAA depends on the segment involved and its underlying cause. By reducing stress on the wall of the aorta, medications such as beta blockers (propranolol and metoprolol) have been shown to slow the rate of growth of thoracic aneurysms. In addition, aspirin and statin are typically added to the patient's regimen to reduce the risk of heart attack and stoke. Surgical intervention is recommended for TAA's greater than 5.5 cm in most cases. Surgical options for TAA are dependent upon which segment of the aorta has been affected. For ascending aortic aneurysms, sternotomy with cardiopulmonary bypass is required. Ultimately, the diseased segment is replaced with a tube graft. For TAA's involving the aortic root and consequently valve function, the Bentall procedure is performed where a tube graft with a prosthetic valve attached to one end is employed. Thoracic aneurysms involving the descending aorta, thoracic endovascular repair (TEVAR) is comparatively a significantly less invasive option than that of traditional open aortic repair.

Conclusions:

The diagnosis of aortic aneurysm is often an incidental one. Similarly, the case we presented involved a 91 year old who was diagnosed with 6 cm aortic aneurysm while searching for evidence which coincided with the patient's clinical presentation of CHF exacerbation. Rather than finding cardiomegaly and bilateral pleural effusions, our ED team found a significantly enlarged/dilated thoracic aorta which was compressing the patient's trachea, esophagus and brachiocephalic artery. Many other cases in which mass effect due to thoracic aneurysm have been documented. In those individuals the condition can be acquired (smoking, hypertension), infectious (syphilis) or congenital (Marfan, Ehlers-Danlos), and can also manifest as double aortic arch, aberrant subclavian artery, and pulmonary artery sling. Additionally, Kommerell's diverticulum, a bulbous configuration at the proximal descending aorta of left or right arch configuration, is a rare cause of tracheobronchal compression. Regardless of its form or its underlying cause, mass effect due to thoracic vascular abnormality such as aneurysm can lead to Tracheomalacia, and ultimately airway collapse, under chronic conditions.