Chronic Posttraumatic Pseudoaneurysm of the Thoracic Aorta 55 Years after A Blunt Trauma - A Case Report With Review of Literature
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Abstract
Injuries of thoracic aorta due to blunt trauma carry very high mortality rates and studies estimated that less than 2% of people who sustain it remain alive if they were not diagnosed and treated appropriately. Moreover, even those lucky few live with the risk of rupture of the Pseudoaneurysm that can develop years to decades after the causative trauma culminating in the fatal internal hemorrhage. This paper reports a case of a 72-years-old Libyan male who sustained a blunt chest injury and multiple rib fractures 55 years ago that resulted in a large pseudoaneurysm of the arch of aorta.

Blunt traumas to the chest can cause injuries in the thoracic aorta. The majority of victims expire on the way to the hospital. However, if the surrounding tissue contains the hematoma or the injury of the wall of the aorta is partial and the adventitia layer remained intact, then the patient develops a pseudoaneurysm1. The percentage of those who survive this injury without diagnosis and treatment is 1% to 2% only 2. Usually, 80% to 90% of the victims with aortic injury die before reaching hospital, and without surgical intervention about 90% of those who reach hospitals alive die within 4 months of the injury, with more than half of these deaths occurring within the first 24 hours 3.

The most common etiology for thoracic aorta injury from a blunt trauma is a high speed deceleration injury, such as in high speed motor vehicle accidents and fall down from high places2,4,5, but other causes were reported like blunt chest trauma with fractured ribs due to fall down6 and crush injuries with rib fractures7. The isthmus of aorta is the most common site of injury 8. There are also other sites which are less common such as the arch of aorta, the ascending aorta and the descending aorta 8,10.

The patients who have posttraumatic pseudoaneurysms of the aorta remain asymptomatic for long durations and usually present with symptoms of compression due to the enlargement of the Pseudoaneurysm or by its rupture1,11. Late rupture of the aneurysm can occur after many years or even decades, and in one case the rupture occurred after more than 30 years of the initial trauma that caused the Pseudoaneurysm 8,12.

CASE REPORT:
Our patient is a 72-years-old Libyan male smoker who was referred to Sebha Medical Center from Traghen hospital with severe shortness of breath and fatigue. A full history was taken, and it revealed that about 55 years ago, a wall fell on the patient's chest and caused fractures of 8 ribs on the left side that prompted treatment as an inpatient for 4 months. Thirty years following the incident, he was examined during a routine medical checkup on general population of his village and the doctors doing the checkup referred him to Sebha Medical Center for further evaluation and he was found to have a cystic lesion in the chest and was advised to have surgical treatment as early as possible. However, he refused to undergo the surgical intervention. Thereafter, he left the hospital and remained asymptomatic for over two decades. For the last six years, he has been suffering from hoarseness of voice and

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progressive shortness of breath. Two days prior to admission, he became severely short of breath and fatigued. The patient was then admitted to Traghen Hospital following a sudden collapse while he was preparing himself for the prayer. He was referred to Sebha Medical Centre, with a chest x-ray showing a well-defined lesion in the left hemi-thorax, for further workup and management. There was no family history of similar disease or connective tissue diseases and the patient denied having any extramarital sexual relationships and did not have history of syphilis.

On examination, the patient was conscious and oriented. He was averagely built. His pulse rate was 82b/m and blood pressure 120/80 mmHg. He had no pallor, icterus, cyanosis, clubbing, lymphadenopathy or pedal edema. His JVP was not raised. On examination of the chest, there was an undue depression in the left infraclavicular region extending from 2nd intercostal space to 5th intercostal space. Examination of his heart revealed normal first and second heart sounds with no added sounds. On auscultation of his chest, he was found to have decreased breath sounds on the left side with expiratory rhonchi. His abdomen was normal and there was no obvious neurological abnormality.

The results of his CBC showed that WBC:4.7x10^3/µL, RBC:5.45x10^6/µL, HB:16.1g/dL, Platelets:245x10^3/µL, ESR:3mm/1hour, RBS:110mg/dL, Blood Urea:12mg/dL, Na:137.7mmol/L, K:4.19mmol/L, Cl:100.3 mmol/L.

The patient’s chest x-ray showed a smooth well-defined left-sided large homogenous calcified mediastinal lesion (Fig.1). Ultrasound examination of his abdomen and pelvis was normal. He was scheduled for contrast enhanced CT scan of chest. Before administration of contrast, a rounded lesion with calcified walls in the middle mediastinum with fluid-density contents was noted (Fig.2), its dimensions were 102x92x85 mm. After administration of contrast, the lesion is highly intense enhancing with partial filling defect indicating that the lesion is a vascular lesion with mural thrombus (Fig.3). Three dimensional images were reconstructed from the scan images to show the relation between the lesion and the arch of aorta (Fig.4).

DISCUSSION:
Cases of blunt thoracic trauma causing injury of the thoracic aorta are serious conditions with very high mortality rate; only about 2.5% of the victims survive without treatment. Those patients who survive develop pseudoaneurysms of aorta, which can rupture anytime causing massive bleeding and death. One of the factors that make diagnosing these injuries difficult is that these are usually associated with other serious life threatening conditions that mask this complication. Detection of acute aortic injuries on presentation gives the patients the opportunity to undergo open repair, endovascular repair or be put under early medical management with delayed surgical repair done which can improve the outcome. Aortic pseudoaneurysms following blunt trauma develop when the injury of the aorta is
Partial and the bleeding is contained by the intact adventitia or when the bleeding is contained by the surrounding tissues. These pseudoaneurysms remain asymptomatic for many years. However, these can enlarge progressively and compress the surrounding structures and present with pain and symptoms of compression. When the recurrent laryngeal nerve is compressed these cause hoarseness of voice. Compression of the oesophagus causes dysphagia, whereas compression of the left main bronchus leads to dyspnea and cough.

Chest x-ray of patients with pseudoaneurysms of thoracic aorta can show deformity of the contour of the aortic knob, soft tissue mass next to the aortic knob, a rim of calcification in the periphery of the mass, old rib fractures or any combination of these findings.

Although angiography is the traditional method for making the diagnosis, the use of non-invasive methods of imaging the aorta and the pseudoaneurysms such as computed tomography, magnetic resonance imaging, and echocardiography are used more in these days since they provide information about the wall of the aorta and the aneurysm, allowing visualization of the surroundings structures, help in localizing thromboses in addition to their ability to show the lumen.

Surgical intervention is needed when there is pain, symptoms of compression, or the size of the pseudoaneurysm increases to 1 cm or more during 1 year. Surgical options include aortic replacement, extra-anatomic aortic bypass and endovascular graft placement. There are no established guidelines about managing asymptomatic cases of chronic posttraumatic pseudoaneurysms of thoracic aorta. Some believe that medical management with beta blockers and follow up every 6 months or 1 year to assess the change in size is appropriate for cases with calcified aneurysms presenting...
more than 2 years after the trauma. Others think that the available evidence doesn’t justify immediate repair for all the cases. But the common practice is the immediate repair for all cases regardless of the calcification or absence of symptoms and that approach is supported by the available data regarding long term survival of patients who undergo the repair in comparison with those who don’t and by the relatively low incidence of postoperative complications in comparison with the high risk of rupture of the aneurysm and death at any time.

CONCLUSION:
Injuries of thoracic aorta have very high mortality rate and the small percentage that survive them and develop pseudoaneurysms of the aorta live with the risk of rupture of the pseudoaneurysms and death. Those survivors may present with symptoms and signs of the rupture of pseudoaneurysms or by compression of the surroundings or their aneurysms might be found accidently on routine imaging or imaging for an unrelated condition. Surgical intervention is the management of choice for symptomatic cases or those with rapidly growing aneurysms on follow up. Experts disagree about the best options to manage asymptomatic cases with slowly growing aneurysms, but the most common practice in managing these cases remains the immediate surgical repair for all the cases.

REFERENCES: