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Case Report

Aortic dissection with left supraclavicular pulsatile swelling: a rare presentation

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ABSTRACT

We are presenting a case of aortic dissection with rare presentation. Patient came with complaints of left sided supraclavicular pulsatile swelling and incidentally was recorded with high blood pressure. After series of investigations he was diagnosed as a case of aortic dissection.

Keywords: Aaortic dissection, Pulsatile swelling

INTRODUCTION

Aortic dissection occurs when an injury to the innermost layer of the aorta allows blood to flow between the layers of the aortic wall, forcing the layers apart.[1] In most cases this is associated with a sudden onset of severe chest or back pain, often described as "tearing" in character.[2,3] Also, vomiting, sweating, and lightheadedness may occur.[2] Other symptoms may result from decreased blood supply to other organs such as stroke or mesenteric ischemia.[2] Aortic dissection can quickly lead to death from not enough blood flow to the heart or rupture of the aorta.[2]

Aortic dissection is more common in those with a history of high blood pressure, a number of connective tissue diseases that affect blood vessel wall strength such as Marfan syndrome, a bicuspid aortic valve, and previous heart surgery.[2,1] Major trauma, smoking, cocaine use, pregnancy, a thoracic aortic aneurysm, inflammation of arteries, and abnormal lipid levels are also associated with an increased risk.[3,2] The diagnosis is suspected based on symptoms with medical imaging such as computed tomography, magnetic resonance imaging, or ultrasound used to confirm and further evaluate the dissection.[3] The two main types are Stanford type A which involves the first part of the aorta and type B which does not.[3]

Prevention is by blood pressure control and not smoking.[3] The treatment of aortic dissection depends on the part of the aorta involved.[3] Surgery is usually required for dissections that involve the first part of the aorta, while those that do not can typically be treated with blood pressure and heart-rate lowering unless complications result.[3,2] Surgery may be done either by an opening in the chest or by endovascular aneurysm repair (carried out from inside the blood vessels).[3]

Aortic dissection is relatively rare, occurring at an estimated rate of three per 100,000 people per year.[1,3] It is more common in males than females.[3] The typical age at diagnosis is 63, with about 10% of cases occurring before 40.[1,3] Without treatment, about half of people with type A die within three days and about 10% of people with type

B die within a month.[1] The first case of aortic dissection described was in the examination of King George II of Great Britainfollowing his death in 1760.[1] Surgery for aortic dissection was introduced in the 1950s by Michael E. DeBakey.[1]

CASE REPORT

40 years old male patient presented in OPD with chief complaint of Left side supraclavicular pulsatile swelling- 2 days. Patient was apparently well 2 days back when he noticed left sided supraclavicular pulsatile swelling early in the morning which was sudden in onset, with size of a marble which he could compress and was painless, over 2 days the swelling gradually increased to size of a lemon, He also had left sided dull aching chest pain. There was no other significant complaint. There was history of trauma to neck and no history of hypertension, diabetes, ischaemic heart disease. On examination; Pulse rate was 88/min. BP- 80/110mmhg. General physical examination: Left supraclavicular pulsatile, compressible, painless swelling was present. No other finding was present. ECG- Concentric LVH was present. Chest x ray- Cardiomegaly with prominent aortic arch and irregular aortic contour was noted. USG Swelling- Suggestive of vascular origin. CT Aortogram: A small intimal flap is noted involving the arch of aorta in post ductal region with sub intimal contrast enhancement. The intimal flap is seen extending approximately for 2.2cm. which was suggestive of dissection of aorta.

DISCUSSION

Aortic dissection (see the image below) is defined as separation of the layers within the aortic wall. Tears in the intimal layer result in the propagation of dissection (proximally or distally) secondary to blood entering the intima-media space. Mortality is still high despite advances in diagnostic and therapeutic modalities.

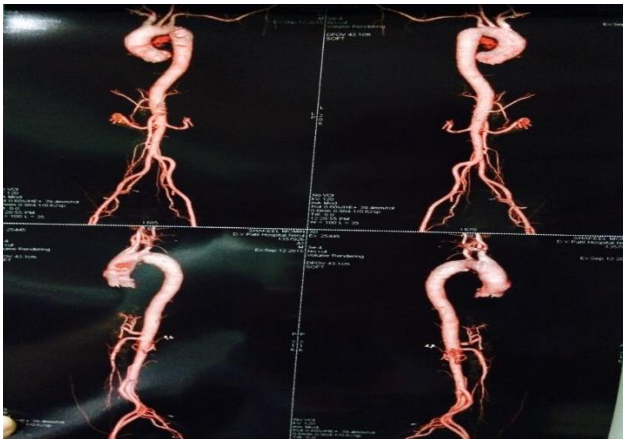

Figure 1: CT aortogram showing intimal flap.

Patients with acute aortic dissection typically present with the sudden onset of severe chest pain, although this description is not universal. Some patients present with only mild pain, often mistaken for a symptom of musculoskeletal conditions in the thorax, groin, or back. Consider thoracic aortic dissection in the differential diagnosis of all patients presenting with chest pain.

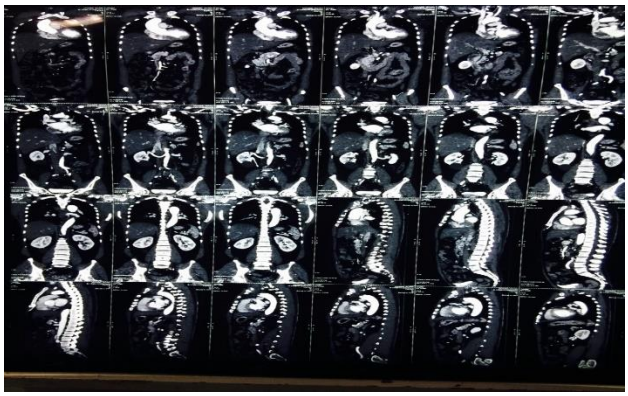

Figure 2: CT thorax showing dissection.

The location of the pain may indicate where the dissection arises. Anterior chest pain and chest pain that mimics acute myocardial infarction usually are associated with anterior arch or aortic root dissection. This is caused by the dissection interrupting flow to the coronary arteries, resulting in myocardial ischemia. Pain in the neck or jaw indicates that the dissection involves the aortic arch and extends into the great vessels.

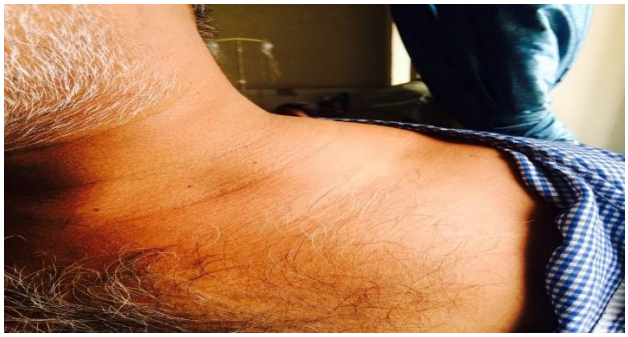

Figure 3: Left sided supraclavicular swelling.

Tearing or ripping pain in the intrascapular area may indicate that the dissection involves the descending aorta. The pain typically changes as the dissection evolves.

The pain of aortic dissection is typically distinguished from the pain of acute myocardial infarction by its abrupt onset and maximal severity at onset, though the presentations of the two conditions overlap to some degree and are easily confused. Aortic dissection can be presumed in patients with symptoms and signs suggestive of myocardial infarction but without classic electrocardiographic (ECG) findings.

Aortic dissection is painless in about 10% of patients.[4] Painless dissection is more common in those with neurologic complications from the dissection and those with Marfan syndrome.

Neurologic deficits are a presenting sign in as many as 20% of cases. Syncope is part of the early course of aortic dissection in approximately 5% of patients and may be the result of increased vagal tone, hypovolemia, or dysrhythmia.[4] Cerebrovascular accident (CVA) symptoms include hemianesthesia and hemiparesis or hemiplegia.[4] Altered mental status is also reported. Patients with peripheral nerve ischemia can present with numbness and tingling, pain, or weakness in the extremities.

Horner syndrome is caused by interruption in the cervical sympathetic ganglia and manifests as ptosis, miosis, and anhidrosis. Hoarseness from recurrent laryngeal nerve compression has also been described.

Cardiovascular manifestations involve symptoms suggestive of congestive heart failure secondary to acute severe aortic regurgitation. These include dyspnea and orthopnea.[4]

Respiratory symptoms can include dyspnea and hemoptysis if dissection ruptures into the pleura or if tracheal or bronchial obstruction has occurred. Physical findings of a hemothorax may be found if the dissection ruptures into the pleura.

Other manifestations include the following.[5]

- Dysphagia from compression of the esophagus,
- Flank pain if the renal artery is involved,
- Abdominal pain if the dissection involves the abdominal aorta,
- Fever,
- Anxiety and premonitions of death.

Medical management

Aortic dissection generally presents as a hypertensive emergency, and the prime consideration of medical management is strict blood pressure control. The target blood pressure should be a mean arterial pressure (MAP) of 60 to 75 mmHg, or the lowest blood pressure tolerated. Initial decreases should be by about 20%.[2]

Another factor is to reduce the shear-force dP/dt (force of ejection of blood from the left ventricle). Long-term management of physical, emotional, and psychological stresses are important to controlling blood pressure.

Beta blockers are the first-line treatment for patients with acute and chronic aortic dissection. In acute dissection, rapidly acting, titratable parenteral agents (such as esmolol, propranolol, or labetalol) are preferred. Vasodilators such as sodium nitroprusside can be considered for patients with ongoing hypertension, but they should never be used alone, as they generally cause reflex tachycardia. Calcium channel blockers can be used in the treatment of aortic dissection, particularly if a contraindication to the use of beta blockers exists. The calcium channel blockers typically used are verapamil and diltiazem, because of their combined vasodilator and negative inotropic effects.

If the individual has refractory hypertension (persistent hypertension on the maximum doses of three different classes of antihypertensive agents), an involvement of the renal arteries in the aortic dissection plane should be considered.

Surgical

Indications for the surgical treatment of aortic dissection include an acute proximal aortic dissection and an acute distal aortic dissection with one or more complications. Complications include compromise of a vital organ, rupture or impending rupture of the aorta, retrograde dissection into the ascending aorta, and a history of Marfan syndrome or Ehlers-Danlos syndrome.

The objective in the surgical management of aortic dissection is to resect (remove) the most severely damaged segments of the aorta and to obliterate the entry of blood into the false lumen (both at the initial intimal tear and any secondary tears along the vessel). While excision of the intimal tear may be performed, it does not significantly change mortality.

The particular treatment used depends on the segment or segments of aorta involved. Some treatments are

- Open aortic surgery with replacement of the damaged section of aorta with a tube graft (often made of Dacron) when no damage to the aortic valve is seen,
- Bentall procedure-replacement of the damaged section of aorta and replacement of the aortic valve,
- David procedure-replacement of the damaged section of aorta and reimplantation of the aortic valve,
- Thoracic endovascular aortic repair, a minimally invasive surgical procedure usually combined with on-going medical management,
- Replacement of the damaged section of aorta with a sutureless vascular ring connector-reinforced Dacron graft,
- Establishing the incidence of aortic dissection has been difficult because many cases are only diagnosed after death (which may have been attributed to another cause), and is often initially misdiagnosed. Aortic dissection affects an estimated 2.0-3.5 people per every 100,000 every year. Studies from Sweden suggest that the incidence of aortic dissection may be rising.[6] Men are more commonly affected than women: 65% of all people with aortic dissection are male. The mean age at diagnosis is 63 years.[7] In females before the age of 40, half of all aortic dissections occur during pregnancy (typically in the third trimester or early postpartum period).[8]
- Of all people with aortic dissection, 40% die immediately and do not reach a hospital in time. Of the remainder, 1% die every hour, making prompt

diagnosis and treatment a priority. Even after diagnosis, 5-20% die during surgery or in the immediate postoperative period.[7] In ascending aortic dissection, if surgery is decided to be not appropriate, 75% die within 2 weeks. With aggressive treatment, 30-day survival for thoracic dissections may be as high as 90%.[9]

Our case was a rare one with no significant complaint and incidentally was found to be hypertensive and after work up was diagnosed with aortic dissection.

CONCLUSION

AD, the most common subtype of acute aortic syndrome, is a rapidly lethal though infrequent clinical presentation in the emergency department. Although most patients present with typical aortic pain, many can present with atypical features or even remain asymptomatic, which can delay institution of appropriate therapy? Multiple imaging modalities that is available to confirm the diagnosis – though echocardiography and CT are most commonly utilized. The imaging preference should depend on the experience of the institution, availability, and hemodynamic stability of the patient. In general, acute AD involving the ascending aorta (Stanford type A or DeBakey type I-II) are considered surgical emergencies. Dissections confirmed to the descending aorta (Stanford type B or DeBakey type III) are treated medically, unless there is evidence of end organ ischemia from malperfusion or continued hemorrhage into the pleural or retroperitoneal space, for which surgical and endovascular options are available.

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