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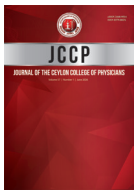

Case Report

A delayed diagnosis of Stanford non-A non-B aortic dissection presenting as Ortner syndrome: A case report

Pravinson M

Base Hospital, Kalmunai North, Sri Lanka

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ABSTRACT

The clinical presentation of aortic dissection is diverse and requires a high degree of suspicion to make a clinical diagnosis. Typical chest pain is present in about 80 to 90% of patients. Sometimes, acute aortic dissection may present with only a subtle intimal flap, potentially leading the lesion being overlooked on initial imaging studies. The lesion may evolve over time and later present as chronic aortic dissection with complications, mainly due to compression of adjacent structures. We describe a patient who initially presented with acute chest pain and hypertensive emergency later developing vocal cord palsy secondary to compression of the recurrent laryngeal nerve by an aortic dissection, consistent with Ortner syndrome. The dissection was classified as Stanford type non-A non-B. This subtype of aortic dissection is rarely reported in the literature, and its associated complications have only infrequently been described. Medical management is appropriate for stable chronic aortic dissections due to the high morbidity and mortality risk of surgery. This case also highlights the importance of careful evaluation of patients presenting with high blood pressure and acute chest pain.

*Corresponding Author: Pravinson M
Email address: pravinson.23@gmail.com
https://orcid.org/0000-0001-5527-6950

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Introduction

Ortner syndrome or cardio-vocal syndrome is a rare condition that causes hoarseness of voice due to compression or traction of the left recurrent laryngeal nerve by cardiovascular disorders such as enlarged left atrium or aortic arch aneurysm.[1] In this case, a Stanford type non-A non-B aortic dissection caused the palsy of left recurrent laryngeal nerve. According to the definition, the dissection flap starts anywhere between the origin of the brachiocephalic trunk and the origin of left subclavian artery. In this case, the origin of the tear has started between the left carotid and left subclavian artery. Stanford classification could not pinpoint the exact location of the proximal part of the dissection of this unusual type. A newer classification is now introduced by American Association of Thoracic Surgery to avoid confusion. The available literature mentions these types of dissections are few due to rare occurrence.[2] This case report also highlights the importance of high clinical suspicion so that an early diagnosis of acute aortic dissection may not be missed.

Case presentation

A 72-year-old man presented to the Emergency Unit, Base Hospital, Kaluwanchikudy, (Eastern Province of Sri Lanka) with sudden onset acute ischaemic type of chest pain. The pain was described as tightening and was associated with excessive sweating. He denied shortness of breath, nausea, vomiting or symptoms suggestive respiratory tract infection. His pulse rate was 96 beats per minute, and the blood pressure was markedly elevated at 260/160 mmHg. There was no evidence of focal neurological deficits. His ECG showed left ventricular hypertrophy pattern, with no acute ischemic changes. Troponin I was negative. The ultrasound abdomen revealed normal kidneys, with bilateral minimal pleural effusions. Chest X ray showed right lower lung consolidation. Urine full report showed mild albuminuria with red blood cells 20-40/high power field. Serum creatinine 1.26 mg/dl (0.6 – 1.20). Full blood count showed neutrophil leucocytosis and C-reactive protein was 20.3 mg/dl (< 5).

He was treated as having a hypertensive urgency and lower respiratory tract infection. An aortic aneurysm was not considered in the differential diagnosis during this presentation, and no focused clinical examination was performed. He was started on oral antihypertensive medication. Chest Xray performed one week later was documented as normal. His drug compliance was poor and few months later, he was lost to clinic follow up.

He returned to medical clinic about one year after the initial presentation with new- onset hoarseness of voice. He did not have a goitre or tracheal deviation. Chest X ray demonstrated a widened mediastinum (Figure 1) suggestive of an aortic aneurysm. However, the patient was lost to follow-up again. He appeared at the medical clinic again 6 months later because of the persistent hoarseness of voice. Contrast enhanced computed tomography of the chest revealed an aortic dissection involving both ascending and descending aorta extending from the distal part of left carotid origin to left common iliac artery. There was thrombus formation in the false lumen about 6 cm at proximal segment. Left vocal cord was in adducted position due to the mass effect by the aorta (Figure 2).

During this presentation his blood pressure was normal but there was significant radio-femoral pulse delay. He did not have focal neurological deficits, and abdominal examination did not reveal any bruits. Because there was no haemodynamic compromise at this current presentation, conservative management with blood pressure control was advised by vascular surgical team.

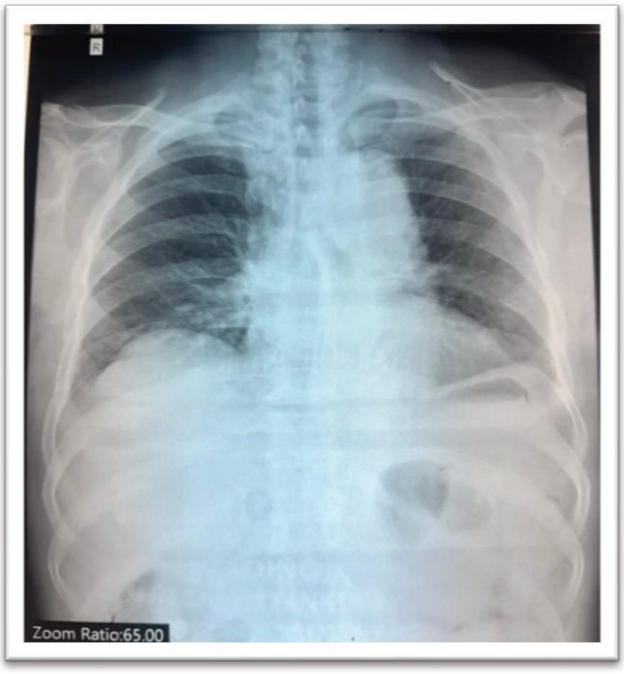

Figure 1. Chest X-Ray showing widening of the widen mediastinum.

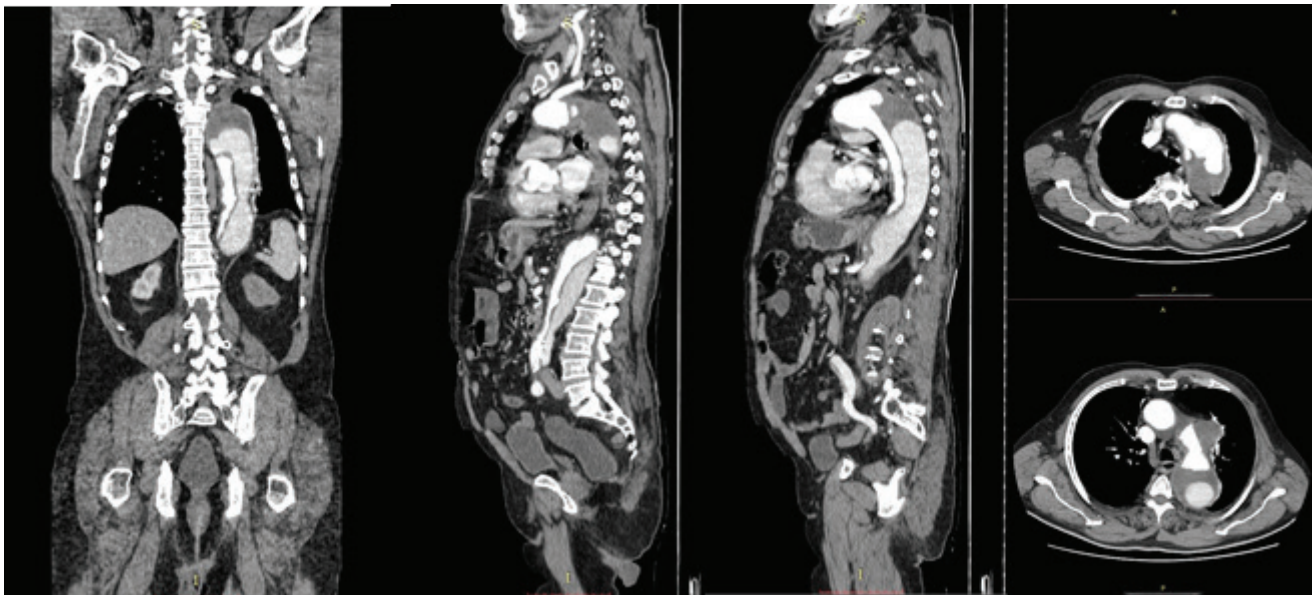

Figure 2. Contrast computed tomography images show the origin and extension of the dissection. The filling defect represents the large thrombus. Note the dilated proximal part of aorta which compresses the left recurrent laryngeal nerve.

Discussion

Acute aortic dissection should be considered when a patient presents with abrupt onset of sharp tearing chest pain radiating to posterior chest along with associated very high blood pressure.[3] According to the most widely used Stanford classification, Type A aortic dissection involves the ascending aorta, regardless of where the dissection originates and may extend to the aortic arch and descending aorta. Type B Involves only the descending aorta, distal to the origin of the left subclavian artery. Stanford non-A non-B aortic dissection usually involves the aortic arch, with or without extension into the descending aorta, while sparing the ascending aorta.

Type A dissection may present as acute inferior STEMI or syncope. A tearing type of pain radiating to posterior chest or back pain suggests type B aortic dissection which is also likely to have an intramural haematoma leading to back pain or abdominal pain.[4] Radiographic features of widen mediastinum and pleural effusions are also suggestive of aortic dissection.[5] Dissection begins with an intimal tear which extends into medial layer. Blood then cleaves the intimal laminal plain leading to dissection.[6] Hypertension is the most common cause of both acute and chronic dissection. Other causes include connective tissue disorders and large artery vasculitis.[7] Ambiguity of presentations, absence of early radiological features and lack of serology are among the common causes to miss the aortic dissection.[8,9]

In our patient, acute chest pain, high blood pressure with evidence of pleural effusion were enough evidence to suspect the dissection at first presentation. Pulse deficit and blood pressure are the easiest bedside tests for all suspected cases with aortic dissection. D-Dimer has a high negative predictive value when the value is less than 500 ng/dL. We suspect that the initial tear would have been small as the early chest x-ray did not reveal widened mediastinum.[10] Aortic dissection detection score along with D-Dimer levels is one tool to rule in or rule out acute aortic conditions.[11]

The anatomical location of the tear and the duration of disease determine the management of aortic dissection. Generally, acute disease is considered when patient present to hospital within in two weeks (hyperacute: < 24 hours, acute: 1-14 days, sub-acute: >14 to 90 days, chronic: > 90 days). Involvement of ascending aorta (Stanford type A) often warrant a surgical repair. Stanford type B dissection, in which the intimal tear originates in the aorta distal to the left subclavian artery, is usually managed medically unless presenting with organ hypoperfusion.[10,12] This strategy is applicable for both acute and chronic conditions. Our patient probably did not have any major complication in acute phase or chronic phase other than vocal cord palsy. There is no solid evidence for surgical procedures in chronic asymptomatic patients due to lack of clinical data.[13] Pain control and anti-hypertensive therapy to control blood pressure should be initiated to all type of acute dissections.[14]

The Stanford classification of aortic dissection does not clearly define the location of tear specially in the arch of aorta. These are collectively labelled as Stanford non-A non-B. The American Association of Thoracic Surgery (AATS) has proposed a newer classification to define the primary location of the tear. In this system, the aorta is divided into anatomical zones. Type A dissections involve zone 0 (the ascending aorta), whereas primary tear originating in zone 1 or beyond are classified as Type B dissections, regardless of retrograde or antegrade propagation.[15]

Conclusion

Acute aortic dissections may be missed due to their nonspecific clinical presentations and a lack of clinical suspicion. Some patients present later with complications arising from the dissection. Chronic Stanford type B and other non-A types of aortic dissections are generally managed medically as surgery can be associated with substantial morbidity and mortality.

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