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CASE REPORT

An Unusual Presentation Of Aortic Dissection

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ABSTRACT

We report a case of painless dissecting aneurysm, mainly involving the descending aorta in a 46-year old Philippine patient, who was admitted to our hospital, presenting with cerebrovascular accident. His medical history was remarkable for hypertension for 9 years. The patient did not have chest or back pain. On clinical examination, it was observed that he had left sided hemiplegia. Computed tomography (CT) of the head showed multiple infarctions. Trans oesophageal echocardiography and Magnetic Resonance Imaging of the chest and abdomen showed dissecting aneurysm in the descending thoracic aorta with big thrombus. The patient was treated conservatively and was discharged in good condition.

Key Words: Cerebrovascular accident. dissecting aneurysm.

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Introduction

Aortic dissection is defined as the 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, [1] often presenting with tearing chest pain and acute haemodynamic compromise. Its prompt diagnosis remains essential for successful management [2]. Aortic dissection can be diagnosed premortem or postmortem because many patients die before presentation to the emergency department (ED) or before diagnosis is made in the ED. We report a 46-year-old Philippine male, who presented with painless Aortic dissection.

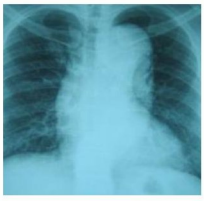
Case History

A 46-year-old Philippine gentleman, a known case of hypertension for 9 years, was admitted to the emergency department with sudden onset of slurred speech and weakness on the left side of the body that occurred early morning after waking from sleep. There was no history of any chest or abdominal pain.On examination, the patient was found to be fully conscious (Glasgow Coma Scale score of 15/15)Blood pressure was 170/100, the pulse rate was 61/minute regularly palpated over both femoral and radial arteries, body temperature was 36.7 and respiratory rate was 14/minute.

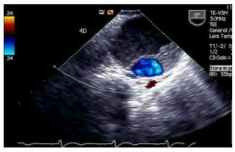
On Neurological exam, there was upper motor left facial palsy, dysarthria and left sided hemiplegia with power of grade 2/5 in the left upper and lower limbs with upgoing planter on the left side. There was no carotid bruit. Cardiac auscultation revealed normal first and second heart sound with no added sound or murmur. The lungs were clear to auscultation. There was no abnormal finding on his 12-lead electrocardiogram. His Chest X ray showed widened mediastinum with dilated ascending and descending part of aorta [Table/Fig 1]. Laboratory findings showed high serum creatinine- 175µmol/l, Urea- 9 mmol/l, Sodium -139mmol/l, Chloride-104mmol/l, Bicarbonate -23mmol/l, potassium -3.7mmol/l and sugar -6.53mmol/l. Non enhanced Computed tomography (CT) of the head showed multiple bilateral Centrum semi ovale, right frontal, right basal ganglia, and right cerebellar lacunar infarcts. Magnetic Resonance Imaging (MRI) of brain was performed, which showed the same results as in the CT finding. Moreover, Intra cranial Magnetic resonant angiography (MRA) was normal.

In the view of these findings (multiple sites of infarction and dilated mediastinum), trans oesophageal Echocardiography [Table/Fig 2] was done, which showed dissecting aneurysm in the descending thoracic aorta, with the dissecting flap seen in the aortic arch along the descending thoracic aorta, up to 40 cm from the incisors. The false lumen measured 2.2 cm and the true lumen measured 1.2 cm. There was an aortic arch thrombus occupying the most proximal part of the dissecting flap and filling the false lumen, measuring 1.6 cm.MRI of the chest and abdomen [Table/Fig -3] showed extensive aortic dissection starting from the aortic arch, with false lumen starting proximal to but not involving the origin of the subclavian artery, extending to the lower most part of the abdominal aorta with the false lumen larger than the true lumen.

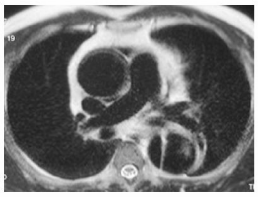
Kidney dimensions checked by ultrasound were within normal limits (right kidney 9.3 Cm and left kidney 9.4 cm), whereas MR Angiography of the renal arteries revealed extensive aortic dissection with perfusion of the right kidney from the true lumen and the left kidney from the false lumen, with the short stenotic segment at the origin of the left renal artery of less than fifty percent, which was regarded as hemodynamically non significant. As the dissecting process involves the descending aorta, the patient was treated conservatively with aggressive control of blood pressure and rehabilitation. After 4 weeks, his creatinine levels returned back to normal spontaneously and he was discharged from hospital in a good condition



(Table/Fig 1) widened mediastinum with dilated ascending and descending part of aorta



(Table/Fig 2) False lumen measure 2.2 cm and true lumen measures 1.2 cm and there is aortic arch thrombus occupying the most proximal part of dissecting flap and fills the false lumen and measure 1.6 cm.



(Table/Fig 3) Extensive aortic dissection starting from aortic arch with false lumen starting proximal but not involving the origin of subclavian artery

Discussion

Aortic dissection occurs most often because of a tear or damage on the inner wall of the artery. It is found that theincidence of acute aortic dissection in the general population ranges from 2.6 to 3.5 per 100,000 person-years [3],[4],[5] with males more frequently affected than females, with a ratio ranging from 2:1–5:1[6]. While its occurrence is relatively uncommon before the age of 40, when it does occur, it is usually in association with certain predisposing factors [7] and mainly affects the sixth and seventh decades.

There are two Classifications for Aortic dissection, the DeBakey and Daily (Stanford) systems, [18],[8],[9]. The Stanford system is more widely used. It classifies dissections that involve the ascending aorta as type A, regardless of the site of the primary intimal tear, and all other dissections as type B. In comparison, the DeBakey system is based upon the site of origin, with type 1 originating in the ascending aorta and propagating to at least the aortic arch, type 2 originating in and confined to the ascending aorta, and type 3 originating in the descending aorta and extending distally or proximally.

The main predisposing factor for acute aortic dissection is systemic hypertension, which presents itself in 72 percent of the patients, while the second is atherosclerosis, which is seen in 31 percent [10] of the patients. On the other hand, other predisposing factors include chromosomal aberrations, hereditary abnormal connective tissue, congenital aortic valve defects, coarctation of the aorta, inflammatory or infectious abnormalities, pregnancy (especially the third trimester), annuloaortic ectasia and aortic aneurysm [6]. In our case, the predisposing factor was hypertension.

The most common presentation is the acute onset of severe chest or back pain, although painless and/or atypical symptoms have been documented[11],[13]. In an analysis from the IRAD registry, painless dissection has been found to be reported in (6.4 percent)[14] of patients; mainly in the elderly and diabetic patients and in those with aortic aneurysm, or cardiovascular surgery.

In one study, up to ten percent of patients with painless AD presented with neurological symptoms [15]. Our case presented without chest, back, or abdominal pain.

In painless aortic dissection, the explanation for absence of pain is still a matter of debate [16]. More than one theory had been proposed [17],[18], like slow or gradual dissection with less wall stretching and sparing of the adventitial layer, the site of aortic innervations.

Involvement of the ascending aorta leads to neurological deficits including stroke or decreased consciousness due to direct extension of the dissection into the carotid arteries or due to diminished carotid blood flow [19]. On the other hand, involvement of the descending aorta leads to focal neurological deficits due to spinal artery involvement and spinal cord ischaemia. [20],[21]. In our case, the presentation was with multiple infarction and left sided hemiplegia.

Management of renal impairment due to dissecting aorta depends on its severity. Stents were used in more than one report, while others preformed surgical interventions; but our patient was managed conservatively and he regained his renal function. In general, for any hypertensive patient coming to the emergency department with CVA with multiple infarctions, renal failure with or without chest pain should raise the suspicion of aortic dissection.

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