Case Report

Acute Aortic Dissection — De Bakey Type I and Stanford A

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Abstract
We report a rare case of aortic dissection which fits into De Bakey Type I and Stanford A, presented with severe tearing chest pain, paraplegia, stupor, hypotension, and syncope. Echocardiography showed dissection involving aortic root. MRI showed aortic dissection involving ascending, arch and descending aorta. Patient was managed conservatively and he died within 12 hours after the admission.

INTRODUCTION

Acute aortic dissection is a sudden catastrophic event, success in salvaging the patient depends on the prompt diagnosis and initiation of early treatment. Aortic dissection is characterized by longitudinal cleavage of the aortic media by a dissecting column of blood. The term dissecting haematoma describes this condition more accurately.1

CASE REPORT

A 65 year male known hypertensive with ischemic heart disease since 3 years on irregular treatment, presented with sudden onset severe tearing chest pain from anterior to posterior aspect of the chest. Patient had syncope at the onset of tearing chest pain. Patient was non-smoker, non-diabetic and non-alcoholic.

On examination patient was stuporous, pulse and BP not recordable in the upper limbs but pulses were felt in the lower limbs. Early diastolic murmur of aortic incompetence was heard on auscultation. CNS examination revealed normal cranial nerves. Motor system revealed power of grade 5 in upper limbs and grade 1 in both the lower limbs. Deep tendon reflexes were diminished and extensor plantar response was elicited bilaterally.

On investigations, CBC, random blood sugar, blood urea, serum creatinine, were normal. ECG showed left ventricular hypertrophy (LVH) with ST depression in L-2, L-3 and aVF. Echocardiography showed ascending aortic dilatation with dissection involving aortic root extending to arch and also to the right subclavian artery. There was moderate aortic regurgitation, sclerotic aortic valve, moderate LVH, no regional wall motion abnormalities and no pericardial effusion was made out. Magnetic resonance imaging (MRI) in axial sagittal T1 showed dissection involving the ascending aorta, arch and descending aorta (Figs. 1,2) which was extending up to the level of kidneys. Both the false and true lumen were patent. There was no obvious extension into the carotids. There was no evidence of periaortic haematoma. He was diagnosed as a case of acute aortic dissection.

Patient's condition was deteriorating rapidly and went into shock. He was managed with inotrope (dopamine infusion 5 micrograms/kg/min) and other supportive measures to prevent further deterioration. But the patient died within 12 hours after the admission.

DISCUSSION

Acute aortic dissection is a rare entity. Incidence is 5-10 cases per million population per year.1 Commonest age group is 6-7th decade of life. It occurs more commonly in males than females. Aortic dissection involves a tear in the intima allowing blood to escape from the true lumen of the aorta, rapidly dissecting the inner from the outer layer of the media. Usually dissection proceeds distally; 38 percent proximally. Commonest underlying cause is hypertension in 72-80 per cent.2 Other causes are Marfan’s syndrome, Noonan Turner syndrome, Ehler-Danlos syndrome, age, atherosclerosis,
migratory pain is described in as few as 17 percent of cases.2 Dissection as it extends through the aorta. However, such origin to other sites, generally following the path of the dissection. In aortic dissection pain migrates from its point of origin and posterior chest pain denotes descending aortic dissection. Classically anterior chest pain denotes ascending aortic dissection (25 and 4 percent respectively).2 Neurological manifestations occur in 6-19 percent of cases.3 Less common manifestations are syncope with or without chest pain in 9 percent, paraplegia due to ischemic spinal cord damage, altered consciousness,3 cerebrovascular accidents, pulse loss with or without ischemic pain and congestive cardiac failure. Aortic regurgitation with musical quality murmur occurs in more than 50 percent cases in the proximal dissection. Lab investigations may show anemia due to haemorrhage or sequestration of blood in the false channel with mild to moderate polymorphonuclear leucocytosis.1 ECG may show LVH but no ischemic changes, and in proximal dissections it may reveal acute myocardial infarction when the dissection flap has involved a coronary artery. CXR may reveal widening of the aortic arch in 40-50 percent cases, and an increase in distance from intimal calcification to the outer edge of the aortic shadow of more than 1 cm (calcium sign).1 New biochemical method recently introduced is serial immunoassays of monoclonal antibodies to smooth muscle myosin heavy chains to detect acute aortic dissection. Four imaging modalities that help in diagnosis are aortogram, computed tomography (CT), magnetic resonance imaging (MRI), transesophageal echo (TEE). Aortogram may mislead the diagnosis by false negative aortogram by equal and simultaneous opacification of both true lumen and false lumen. CT require the use of an intravenous contrast agent for diagnosing two distinct aortic lumen. Transthoracic echo has poor sensitivity. TEE can be performed quickly at bedside, readily available, non-invasive, no contrast is necessary and cost-effective. The sensitivity and specificity of TEE were 99 per cent and 98 per cent respectively with positive and negative predictive values of 98 per cent and 99 per cent.4 MRI produces high-quality images and is the gold standard because of multiple views facilitating the diagnosis and determination of extent and in many cases reveals the presence of branch vessel involvement. MRI had a sensitivity of 88 per cent for identifying the site of intimal tear; 98 per cent sensitivity for the presence of thrombus and 100 per cent sensitivity for the presence of pericardial effusion. Cine-MRI technique showed 85 per cent sensitivity for detecting aortic regurgitation. MRI is relatively contraindicated for unstable patients, well suited for stable or chronic dissections. MRI has limitations due to its cost.

Early mortality is 1 per cent per hour in untreated cases. Twenty five per cent die within 24 hours, more than 50 per cent die within the first week, 75 per cent within one month, more than 90 per cent die within one year.1 Early emergency treatment is aimed at reduction of systolic blood pressure to 100-120 mm Hg with parenteral antihypertensives. Surgical results are superior to medical therapy in acute proximal dissection and conversely medical therapy offers relative advantage over surgery in uncomplicated cases of acute distal dissection. Long term medical therapy is aimed to control hypertension. The aim of reporting this case is the rarity of full blown clinical presentations with neurological features.

References