Mid Aortic Syndrome (MAS)

A 28-year-old female presented with hypertension and occasional non-specific abdominal pain for 1 year. On examination, her blood pressure was 180/120 mm of Hg and there was renal bruit. Routine investigations were normal. She then underwent CT angiogram of abdominal aorta which revealed an abrupt caliber change of the mid abdominal aorta. The coeliac, superior mesenteric arteries and both renal arteries originate from the stenotic portion of the aorta. Findings were consistent with mid aortic syndrome with bilateral renal artery, coeliac and superior mesenteric artery ostial narrowing. However, certain other conditions must be kept in mind in the differential diagnoses such as Takayasus arteritis and aortitis.

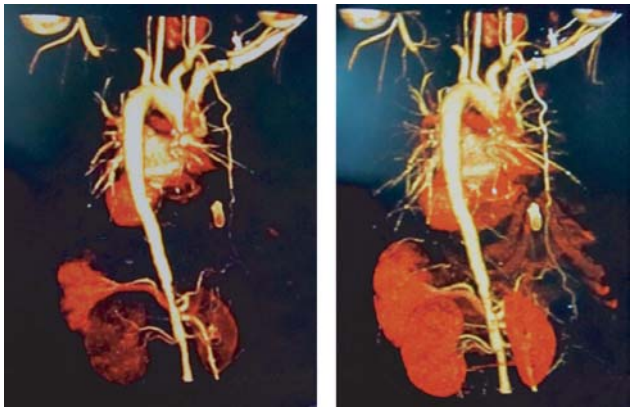

Fig 1. CT angiogram of abdominal aorta

Middle aortic syndrome is an uncommon condition that can cause severe hypertension with risk of life threatening complications. The disease should be suspected in any young severely hypertensive patient in whom renal and endocrine causes of hypertension have been excluded by routine investigations and especially if abdominal bruit is present. Poor volume peripheral pulses were not a clinical finding in this case.[1] The differential diagnoses include other important causes of renovascular hypertension. The ability to show the difference between MAS and Takayasu's arteritis may be very difficult.[2,3]

Middle Aortic Syndrome may be the result of inflammatory causes, embryonic/congenital origin, or relatively idiopathic. The exact mechanisms underlying the vasculitides are not known. Three pathogenic models of disease have been proposed to explain why the lesions of a particular syndrome are found only in specific vessels. It is likely that elements of each of these models contribute to the pathogenesis of vasculitic diseases.

The distribution of the antigen responsible for the vasculitis determines the pattern of vessel involvement. The recruitment and accumulation of the inflammatory infiltrate, including the expression of adhesion molecules and the secretion of peptides and hormones, are determined by the endothelial cell. Some endothelial cells are able to attract inflammatory cells, whereas others are not. Nonendothelial structures of the vessel wall are involved in controlling the inflammatory process. In addition to the endothelial cells which provide costimulatory function, other cellular components serve as antigen-presenting cells.[4]

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