

Takayasu's arteritis mimicking acute aortic disease.

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Introduction: Takayasu's arteritis (TA) is a rare idiopathic chronic inflammatory disease of unknown aetiology, resulting in a granulomatous panarteritis of the aorta and its major branches.

Case Report: A 56-year-old women was referred to our Hospital for a chest pain perceived as pressure-like in quality without dyspnea. The patient described multiple episodes of intermittent chest pain, fatigue, arthralgias, fever and weight loss of 5 Kg in the last two months. The physical examination and a chest and abdomen CT were unremarkable. She was in normal sinus rhythm without signs of ischemia or other ECG abnormalities. A routine laboratory workup was unremarkable apart from a platelet count of 500,000/Al, C-reactive protein of 15.9 and Hgb 7.4 g/dl. An echocardiographic study showed areas of crescentic thickening and diffuse echolucency of ascending aorta and arch

suggestive of diffuse inflammation (Panel A-top). A PET showed an inflammation of the wall of the aorta and subclavian, iliac and femoral arteries (Panel A-bottom). Therefore, diagnosis of early Takayasu's arteritis was performed. Oral prednisolone (1 mg/Kg/day) was administered and tapered

to 5-10 mg/day in two months; the patient had an excellent clinical response. After three months echocardiographic study (Panel B-top) and PET did not show an inflammation of the aorta and its branches (Panel B-bottom).

Discussion: Our case is about a non-typical chest pain considered as a pleuro-pericarditis by echocardiographic study, mainly an inflammation of aortic wall was found.

The use of echocardiographic study and high-resolution Doppler ultrasound was very important to discriminate a pleuro-pericarditis from a TA earlier.

B-mode ultrasonography clearly demonstrated the characteristic circumferential arterial wall thickening as a "macaroni-like", diffusely thickened intima-media complex. In our patient we found areas of crescent thickening and diffuse echolucency of ascending aorta and arch suggestive of a diffuse inflammation by an echocardiographic study only. However, to confirm a diagnosis, we performed ¹⁸F-FDG-PET to detect pre-stenotic disease in patients presenting with non-specific features commonly associated with TA. We did not perform CT angiography by this technique offers limited imaging of distal aortic branches. The principle advantage of ¹⁸F-FDG-PET is the detection of areas of high glucose metabolic activity in early TA. Indeed, we performed ¹⁸F-FDG-PET for the assessment of disease activity and response to treatment.

Our experience suggests that an accurate echocardiographic study and ¹⁸F-FDG-PET in second time, are important new clinical tools for the diagnosis of early TA and these may have a place in the monitoring of disease activity and response to treatment.

