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Tuberculous Aortitis: An Extremely Rare Cause of Supra Aortic Trunks Stenosis

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Abstract

Aortitis are mainly described in inflammatory disorders such as Takayasu arteritis, giant cell arteritis or Behçet's disease. Thoracic infectious aortitis such as tuberculous aortitis are currently rare. They are always lethal without any treatment. The clinical manifestations are usually vague and nonspecific and may include pain, fever, vascular insufficiency, and elevated levels of acute phase reactants, as well as other systemic manifestations. As a result, aortitis is often overlooked during the initial work-up. A multimodality imaging approach is often required for assessment of both the aortic wall and aortic lumen, as well as for surveillance of disease activity and treatment planning.

Keywords

Aortitis; Tuberculosis; Imaging; Treatment

Introduction

Tuberculous Aortitis (TA) was first described by Weigert in 1882 and constitutes an extremely rare entity [1-3]. It generally develops at the distal aortic arch and the descending aorta that are close to specific groups of mediastinal lymph nodes, but exceptionally it develops in the ascending aorta [4]. Aneurysm formation is associated in about half of cases. Both abdominal and thoracic aorta are involved with equal frequency. We present a case of tuberculous supra aortic trunks stenosis.

Case Report

A 34-year-old man, with smoking habits, was hospitalized for left cervical lymphadenopathy. There was no cough, hemoptysis, evening pyrexia or night sweats and no history of limb claudication. Her past history was negative for connective tissue disorders, vasculitis or tuberculosis. The diagnosis of gonglionic tuberculosis was carried out by positive sputum and ganglion biopsy culture of *Mycobacterium tuberculosis*. Clinical examination revealed apyrexia, present and symmetrical pulses and vascular murmur at supra-aortic trunks. There was no fever, hypertension or difference in blood pressure between two arms. Laboratory findings showed a normal erythrocyte sedimentation rate, leucocytes $5.9 \times 10^9/L$; hemoglobin 12.6 g/L; C reactive protein 8 mg/dL; creatinine 0.6 mg/dL; GOT 21 U/L; GPT 36 U/L. A chest Computed Tomography (CT) scan showed a significant regular circumferential thickening of the right carotid artery wall from its origin extended about 39 mm and responsible for a narrowing of blood vessels up to 90% with a filiform path and of the wall of the left carotid artery left of its origin and extended over all its height and responsible for a narrowing of blood vessels estimated at 50% suggestive of Takayashu disease. A pressure gradient of about 100 mmHg was measured by Doppler echography (Fig.1).

The whole immunological investigation was negative eliminating an associated vasculitis. A control CT scan objectified regular thickening, stenosis and circumferential thickening of the right primary carotid artery from its origin, extended over 62 mm, reducing the light by about 70% of diameter associated with non-stenotic and circumferential irregular thickening of the left primary carotid artery from its origin extended over 57 mm and reducing light by around 15% (Fig. 2). This clear decrease in the size of the wall thickening of supra aortic trunks in favor of tubercular origin. In the absence of arguments in favor of takayashu disease, the patient was treated only with anti-tuberculosis treatment and had completed a 12 month course of standard regimen with a good clinico-radiological evolution (Fig. 3).

Figure 1: Pulsed Doppler recording of the right primitive carotid CPD: Perivascular thickening of CPD with hemodynamic repercussions as evidenced by the acceleration of velocities and filling of the dark window under systolic.

Figure 2: CT angiography: coronal reconstruction of the two carotid axes: peripheral thickening of the two primitive carotid axes.

Figure 3: CT angiography of supra aortic trunks: axial reconstruction persistence of right carotid thickening, responsible for stenosis. Regression of left peri-carotid thickening.

Discussion

Tuberculous aortitis is expected to increase in incidence throughout world because tuberculosis has been increasing in developing areas and is reemerging steadily in advanced countries [4]. Tuberculous infection may occur by direct extension from contagious lesions such as infected lymph nodes, empyema, and pericarditis and by hematogenous or lymphangitic spread from primary lesions. Most reported cases of the tuberculous aortic disease had other pulmonary or extra pulmonary tuberculous lesions [4]. Although aortic pseudoaneurysm is the most common vascular pattern of TA, isolated increased aortic wall thickness and arterial stenosis had observed [5]. TA usually involves the distal junction of the aortic arch along with the descending aorta, which is adjacent to the mediastinal structures. Involvement of the ascending aorta is rarely reported [3]. Our case is characterized by the existence of multiple stenosis of the supra aortic trunk. The initial antituberculous therapy regimen should last for no less than 9-12 months [1]. However, fatal outcomes are frequently reported even after anti-tuberculosis chemotherapy and surgical intervention [6].

Conclusion

Infectious aortitis like tuberculosis should be considered as an etiology for aortitis. Noninvasive imaging techniques like MRA are emerging investigation modalities to detect aortic wall inflammation and peri-aortitis. High degree of suspicion in an appropriate clinical setting in combination with imaging may justify the treatment. Development of diagnostic criteria for tuberculous aortitis based on the clinical findings and imaging may be a future research area.

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