Case Report

Takayasu’s Arteritis and Tuberculosis: a Case Report

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Abstract: The aetiology of Takayasu’s arteritis is unknown, but an association with tuberculosis has been reported. We report the case of a 12-year-old-boy with Takayasu’s arteritis: his blood pressure was 150/90 mmHg and fundoscopic examination showed grade I hypertensive changes. A tuberculin test was positive and acid-fast bacteria were seen in the urine. Angiography revealed involvement of the descending aorta, thoracic aorta and upper abdominal aorta, with fusiform enlargement and no filling of the left renal artery. He was started on prednisolone therapy, with cyclophosphamide being added subsequently. Despite vigorous treatment, including three courses of nitroprusside infusion, the severe hypertension persisted and his blood pressure became regulated only after left nephrectomy. Acid-fast bacteria were seen in the nephrectomy material. The exact role of Mycobacterium tuberculosis in the pathogenesis of Takayasu’s arteritis is still unknown. In this patient the tuberculin test was positive and acid-fast bacteria were seen in both the urine and nephrectomy material. This finding is suggestive of the association between tuberculosis and the disease process.

Keywords: Takayasu’s arteritis; Tuberculosis

Introduction

Takayasu’s arteritis (TA) is an idiopathic, chronic, systemic inflammatory disease that characteristically affects the aorta and its main branches. Its incidence varies between 1.2 and 2.6 cases per million people per year [1,2], and it is more common in Asians than in other racial groups. An exact epidemiological figure from our region is not available. The aetiology of TA is unknown, but an association with tuberculosis has been reported [3,4]. Cross-reactants between heat shock protein and mycobacterial antigens that trigger perforin expression by γ-δ cells has been suggested [5,6].

We report a 12-year-old-boy with TA who presented with hypertensive encephalopathy in whom tuberculosis bacillus was shown in the tissue specimen, implicating its role in the pathophysiology of the disease.

Case Report

A 12½-year-old boy was referred to Hacettepe University Department of Pediatrics for investigation of the cause of his hypertension, which had been noted 1 month previously when he had complained of headache. There was no consanguinity between his parents and no family history of rheumatic disease or hypertension. On physical examination his weight and height were within normal percentiles for his age, the peripheral pulses were palpable, blood pressure was 150/90 mmHg, and fundoscopic examination showed grade I hypertensive changes. Laboratory investigations revealed normal complete blood count, erythrocyte sedimentation rate 44 mm/h, C-reactive protein 0.50 mg/dl (<0.8), and normal liver and renal function tests, urinalysis and C3, C4 levels. A spot urine vanillylmandelic acid test was negative and renin level was 50 ng/ml/h (normal: 0.5–5.9). The tuberculin skin test was positive (an induration of 27 mm in diameter).

Irregularity of the descending aorta and widening of the mediastinum was noticed on a routine chest X-ray.
Echocardiography and abdominal ultrasound were normal. Renal scintigraphy with captopril was consistent with left renovascular hypertension and differential renal function was as follows: 22% left kidney, 78% right kidney. Angiography revealed involvement of the descending aorta, thoracic aorta and upper abdominal aorta, with fusiform enlargement and no filling of the left renal artery. There was also involvement of the innominate artery (Fig. 1). A CT scan showed diffuse wall thickening of the thoracic aorta and innominate artery (Fig. 2).

Initially the patient’s blood pressure was controlled with only oral amilodipine (10 mg/day). He was started on prednisolone (1.5 mg/kg/day) with a diagnosis of Takayasu’s arteritis. Because acid-fast bacteria were seen in the urine and his tuberculin skin test was positive, isoniazid (10 mg/kg/day) and rifampicin (10 mg/kg/day) were added to his treatment.

After the 7th day of the prednisolone therapy his blood pressure began to rise. Propranolol (1 mg/kg/day), furosemide (1 mg/kg/day) and prazosin (1 mg/kg/day) were added sequentially. The prednisolone was tapered to 1 mg/kg/day and cyclophosphamide (CP) was started (2 mg/kg/day) in the quest for more potent immunosuppression. On the 10th day the patient was in hypertensive encephalopathy (blood pressure 220/140 mmHg) and a nitroprusside infusion was given (up to 3 μg/kg/min) for 72 hours. His blood pressure was controlled with hydralazine (2 mg/kg/day), amilodipine (10 mg/day), prazosin (1 mg/kg/day), atenolol (1 mg/kg/day) and furosemide (2 mg/kg/day) in combination. Two additional courses of nitroprusside infusion were also given.

Renal scintigraphy repeated at the end of the 3rd week revealed a further decrease in left kidney function (12% left kidney, 88% right kidney); erythrocyte sedimentation rate and C-reactive protein levels rose to 124 mm/h and 9.60 mg/dl, respectively. He had fever and high blood pressure values (up to 170/120 mmHg, 2–4 times daily).