angioplasty and stenting. Percutaneous intervention has recently gained popularity because it is less invasive. A review by Ribichini et al. (10) summarized the success and safety of percutaneous treatment of these lesions.

In our case, there was no steal, but there was severe myocardial ischemia due to diminished blood flow to LIMA in which there was distal subclavian stenosis. A careful evaluation before the operation for preventing of this syndrome is essential.

Conclusion

Although SAS is very rare and the patients are usually asymptomatic, it has clinical importance in patients with CABG, especially those with composite T-grafting technique. Patients with angina undergoing CABG using LIMA grafting should be considered in terms of left SAS as a possible cause of myocardial ischemia.

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Available Online Date/Çevrimiçi Yayın Tarihi: 27.05.2013

Dilated cardiomyopathy due to miliary tuberculosis

Miliyer tüberkıloza bağlı dilate kardiyomiyopat

Introduction

Miliary tuberculosis (TB) is a potentially lethal form of tuberculosis resulting from massive lymphohaematogenous dissemination of Mycobacterium tuberculosis bacilli. Involvement of heart in tuberculosis occurs in one to two percent of patients with tuberculosis (1-6). We as well introduced a case with dilated cardiomyopathy (DCM) secondary to miliary TB, which was improved with antituberculosis therapy.

Case Report

A 15-year-old girl was presented with weakness, fatigue, weight loss, and swelling on the neck. Productive cough, night fever and night sweat particularly for the last three week. Her personal and family medical history was remarkable revealing a history of TB in the children of her aunt and uncle. She had a single bacille Calmette-Guerin (BCG) scar. Auscultation of the lungs revealed bilateral rales. On cardiac auscultation, she had tachycardia and grade 2–3/6 regurgitant systolic murmur on the mesocardiac focus. Purified protein derivative was negative, gastric aspirates was negative, and TB screening performed via polymerase chain reaction (PCR) method was negative as well.

The neck and abdominal ultrasonographies revealed multiple lymphadenopaties (LAP) on cervical chains and submandibular region. Postero-anterior chest radiograph showed miliar appearance (Fig. 1A). Her electrocardiogram was considered unremarkable. Thoracoscopic echocardiography (Fig. 2A) showed enlarged left ventricle end-diastolic dimension of the left ventricle was 6.21 cm, end-systolic dimension of the left ventricle was 5.6 cm and decreased systolic function. Systolic ejection fraction and shortening fraction were found 21% and 10% respectively. There was grade 1-2 mitral regurgitation secondary to the dilated annulus. Other causes of DCM were excluded. Cervical lymph node biopsy showed signs consistent with caseified granulomatous lymphadenitis. On thoracic computed tomography (Fig. 2B), calcified noncalcified lymph nodes of pathological size were detected in bilateral axillary, paratracheal, perivascular, carinal, subcarinal, bilateral hilar and paracardiac regions. Cavitation was observed in a nodule in the left lung. It was thought that these findings might be consistent with miliary TB.

After making the diagnosis of miliary TB, four-drug combination antituberculosis therapy with anticongestive medications was commenced. The patient, whose symptoms relieved on the 2nd-3rd week of antituberculosis therapy, was discharged to be followed in the outpatient clinic. Control echocardiography exam performed on the 2nd month of poliiclinic follow-up (Fig. 2C), end-diastolic dimension of the left ventricle was 5.06 cm and the systolic ejection has been increased up to 66%. Within two months, 6 kg increase in body weight and the improvement in anemia of chronic disease and disappearance of LAP’s in the neck was determined. Control chest radiograph (Fig. 1B) and tomography (Fig. 2D) was considered unremarkable.

Discussion

Tuberculosis myocarditis is extremely rare and may occur usually secondary to hematogenous or direct spread, as well as retrograde spread from the lymph nodes, of TB of another focus to the myocardium (1-5). TB myocarditis may present itself with rhythm disorders, as well as different pictures such as sudden cardiac death (1, 4, 6).
Antituberculosis therapy is the mainstay of the treatment of TB myocarditis (3). The present patient case as well had miliar TB-related DCM changes and clinical signs of congestive heart failure. On the 2nd-3rd week of the antituberculosis therapy, DCM changes and congestive heart failure relieved. After treatment with antituberculosis drugs, improvement of symptoms of tuberculosis and myocarditis, and disappearance of LAP’s and weight gain was thought that the patient may be tuberculosis myocarditis.

Conclusion

Tuberculous myocarditis is a disease likely to be improved completely with appropriate and prompt diagnosis and treatment, as was in the present patient case.

References


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Available Online Date/Çevrimiçi Yayın Tarihi: 27.05.2013
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doi:10.5152/akd.2013.152