ABSTRACT

Sacroilitis as the initial presentation of subacute IE is rare. Arthritis associated with sub acute IE is asymmetrical and is limited to 1-3 joints, resembling joint changes in rheumatoid arthritis, Reiter syndrome or Lyme disease. Synovial fluid is usually sterile. We are reporting the case of a 16 year old male who presented with low grade fever of 2 months and sacroilitis which on evaluation was diagnosed as infective endocarditis of rheumatic mitral valve.

Key words: subacute infective endocarditis, sacroilitis, rheumatic mitral regurgitation

INTRODUCTION

Infective endocarditis is defined as an infection of the endocardial surface of the heart which may include one or more heart valves, the mural endocardium, or a septal defect. Its intracardiac effects include severe valvular insufficiency, which may lead to intractable congestive heart failure and myocardial abscesses. Musculoskeletal manifestations occur in 15 – 30% of all cases. It can occur as arthritis, musculoskeletal pain or as focal metastatic infections (e.g., spondylodiscitis).

CASE REPORT:

History

Our patient, a 16 year old 11th standard student, presented with low grade fever for 1 month without any other symptoms, followed by pain in
lower back on the right side for which he consulted a medical practitioner and was diagnosed with right sided sacroilitis, proven radiologically (X ray and MRI) and histologically. He was treated with NSAIDs for a period of 1 week, following which symptoms subsided. Symptoms recurred a week later when fever was associated with pain in Left hip joint, Left elbow, Left metacarpo-phalangeal joint and was referred to our department. He had no significant past or family history.

EXAMINATION

On general examination, he had clubbing, there was no pallor, icterus, lymphadenopathy or pedal edema. His pulse rate was 80 per minute and had a blood pressure of 110/70 mm Hg. The Left hip joint, Left elbow, Left metacarpo-phalangeal joints were swollen, tender and warm. Cardiovascular system examination revealed apex in 5th Left intercostal space in the midclavicular line, and a short systolic murmur in the mitral area. Spleen tip was palpable. Other systems were within normal limits. In view of the prolonged fever, clubbing, splenomegaly and systolic murmur at apex, clinically the possibility of infective endocarditis was considered and he was investigated further.

INVESTIGATIONS

On investigation he had an elevated total count with a rise in inflammatory markers like elevated ESR (70mm/hr) and CRP (22). His ECG showed normal sinus rhythm, Chest X-ray revealed cardiomegaly with straightening of left heart border (figure 1).

In view of the possibility of infective endocarditis, 3 blood culture samples were sent, and he was started on empirical antibiotic therapy with vancomycin plus gentamicin. We proceeded with a trans thoracic echocardiogram to look for evidence of endocarditis.

Trans thoracic echo showed Rheumatic mitral valve with anterior mitral leaflet doming, with thickness of 3mm, restricted mobility of posterior mitral leaflet, vegetation on anterior mitral leaflet of 6mm size and moderate mitral regurgitation.

The findings were confirmed by Transesophageal echocardiography.

On third day of hospitalization, on routine examination his radial artery pulse was found to be absent though the patient was asymptomatic. Suspecting an arterial embolism, an arterial Doppler was done which revealed an echogenic thrombus in proximal part of radial artery with ischemic flow. He was continued on antibiotics.

However his blood culture revealed no organisms after three days of incubation.

Our patient had one major criteria and 3 minor criteria as per modified Dukes criteria. Hence a diagnosis of sub-acute infective endocarditis on rheumatic mitral valve with emboli to right radial artery was made. He was continued on vancomycin plus gentamicin. He improved clinically, there were no...
further embolic events. He was discharged after the course of antibiotics and is currently under follow up.

**DISCUSSION**

Acute endocarditis is a febrile illness that rapidly damages cardiac structures, seeds extra cardiac sites, and, if untreated, progresses to death within weeks [6]. Subacute endocarditis follows an indolent course, causes structural cardiac damage only slowly, if at all rarely metastasizes and is gradually progressive unless complicated by a major embolic event or a ruptured mycotic aneurysm.

Rheumatic manifestations are known to be frequent complications of IE. Controversy, however, frequently exists about the actual incidence of these complications. This may be due to the small number of series describing the frequency and type of rheumatic manifestations, and the fact that some studies on rheumatic manifestations in IE have been described from tertiary referral centers, which implicates associated problems of referral bias.

In a study involving 108 patient by P Thomas et al [2] at the Poitiers CHU, it was found that patients with rheumatologic manifestations were generally younger than those without musculoskeletal involvement; diagnosis was made later, and prognosis was worse; streptococcus D was more often involved, and microscopic haematuria was more common. With the exception of vertebral osteomyelitis, the pathogenesis was not clear. Bacterial endocarditis may manifest as an acute arthritis, a febrile backache, tenosynovitis, and arthralgia/myalgia. These rheumatic manifestations may occur as isolated features or in combinations. They bear a temporal relationship to the infection and respond quickly once the endocarditis is treated. Such episodes of arthritis occurred up to 12 weeks before hospitalization[3]. A low-grade arthritis occurring as part of bacterial endocarditis, particularly when the rheumatoid factor is positive or unfamiliarity with the febrile backache symptom complex, may easily mislead the diagnosis.

Bontoux et al. (1967), Holler and Pecora, (1970) and Deshayes et al. (1974) described arthritis, febrile backache, and myalgia in bacterial endocarditis, which has helped to increase awareness of these problems.

There also remains the question of concomitant rheumatic fever especially when positive blood cultures are not obtained as is the case in our patient. But the lack of fleeting arthritis, and the presence of other features associated with bacterial endocarditis makes rheumatic fever less likely. It should be recognized that although rare, the two diseases are known to coexist.

The rheumatic manifestations of bacterial endocarditis deserve attention because of their variety and potential to mimic other rheumatic diseases, (for example Still's disease, which is a strong differential diagnosis in our case) drawing attention away from the serious nature of the underlying cause.

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