Periosteal reaction in systemic lupus erythematosus

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Abstract. The authors report three patients with systemic lupus erythematosus and periosteal reaction. Two of the three cases had systemic vasculitis and the third had local ischemia with ischemic necrosis.

Key words: Systemic lupus erythematosus (SLE) – Periostitis – Polyarteritis nodosa – Vasculitis

Although periosteal new bone formation has been reported in patients with polyarteritis nodosa, we are unaware of similar reports in patients with systemic lupus erythematosus (SLE). We have seen three patients with this association and they form the basis for this report.

Case reports

Case 1. A 24-year-old black male initially presented in November 1981 with night sweats, cough, pleuritic chest pain, fever, rash, and polyarthritis and was diagnosed as having SLE. Renal biopsy revealed vasculitis. He was treated with steroids and did well for six months when he developed an acute thrombotic-embolic occlusion of the left posterior tibial artery and subsequently required a below-the-knee amputation.

Laboratory data revealed increased double-stranded DNA, low C3 and C4, elevated erythrocyte sedimentation rate (ESR), and urinalysis with 2+ protein and 2+ heme.

Radiographs prior to the patient's amputation demonstrated changes of tibial ischemic necrosis (Fig. 1) with periostitis predominantly involving the distal tibia (Fig. 2).

Case 2. A 15-year-old black female initially presented with generalized rash, false positive serologic test for syphilis, and sterile joint effusions. She was diagnosed as having SLE and was started on systemic steroids. During the next year she developed multiple complications secondary to her SLE, including CNS vasculitis, seizures, membranoproliferative glomerulonephritis, myocarditis, arthritis, peripheral vasculitis, and hemolytic anemia. Superficial skin ulcerations developed over both distal extremities and were treated successfully with conservative management. The patient was continued on 60 mg per day of prednisone and subsequently required immunosuppressive therapy for management of her disease.

Laboratory evaluation demonstrated thrombocytopenia, elevated antinuclear antibody titer, decreased C3, and proteinuria. Because of left anterior tibial pain, a bone scan was performed which revealed slightly increased uptake in the left proximal tibia. X-rays at that time demonstrated diffuse periosteal involvement of the tibia and fibula (Fig. 3). The patient continued on steroid therapy and is currently followed at another institution.

Case 3. A 24-year-old white female had an 11-year-history of SLE without renal or CNS involvement. When steroids were tapered below maintenance doses, intermittent flares of disease manifested by fevers, arthralgias, and abdominal pain occurred. Her history was marked by Raynaud's phenomenon, skin rash, serositis, and arthritis. She had a history of intermittent sterile left knee effusions. Radiographs from an outside institution were reported as normal. She was maintained on steroids and non-steroidal anti-inflammatory drugs.

Laboratory evaluation demonstrated a low C4 and CH50, an elevated ESR, a positive ANA (Antinuclear antibody), and a normal complete blood count (CBC).

Six months prior to consultation she was seen elsewhere for severe knee pain. X-rays at that time were reported as showing ischemic necrosis with periostitis. Radiographs taken six months later revealed the changes of ischemic necrosis and periosteal reaction (Fig. 4). The patient is currently being followed at another institution.

Discussion

The musculoskeletal manifestations of SLE include myositis, polyarthritis, deforming erosive arthropathy, tendon weakening and rupture, osteonecrosis, soft tissue calcification, osteomyelitis, septic arthritis, acroserosclerosis, and distal tuft resorption [2, 3, 6–8]. Recent reviews [1, 4] fail to mention the association of periostitis.

Periostitis in association with polyarteritis nodosa was first described in 1956 [9]. One paper reviewed a total of 16 reported cases [5]. In another
series of 158 cases with polyarteritis nodosa, periostitis was reported in three patients.

In polyarteritis nodosa, periostitis is most common in the tibia and fibula. It is thought to be secondary to diffuse vasculitis giving rise to local ischemia and periosteal new bone formation [5, 10]. The clinical and pathologic similarities between SLE and polyarteritis nodosa make it tempting to attribute the changes in our cases to vasculitis. Periosteal new bone formation involved the tibia in all three patients; one patient had femoral and one had concomitant fibular involvement. Vasculitis was clinically evident in two of our patients, one of whom demonstrated classic changes on renal biopsy. Although clinically significant vasculitis was not present in our third patient she