Localized Polyarteritis Nodosa with Periostitis

LEONARDO M. ASTUDILLO, MD; FLORENCE RIGAL, MD; BERTRAND COURET, MD; ELISABETH ARLET-SUAU, MD, Professor of Internal Medicine; Department of Internal Medicine, University Hospital Purpan, Toulouse, France. Address reprint requests to Dr. L.M. Astudillo, Institut Claudius Regaud, Service de Radiothérapie, 20-24 rue du Pont Saint-Pierre, 31052 Toulouse cedex, France. E-mail: leoastu@club-internet.fr

A 45-year-old man presented in February 1999 with edema of the right ankle that became gradually inflammatory over a few months. Lower limb radiographs were normal. In November, treatment with nonsteroidal antiinflammatory drug and antibiotics for 8 days failed to improve the symptoms. In December, a swelling appeared in the lower third of the tibia. Bone radiography revealed periosteal new bone formation (Figure 1A, arrow). Technetium scintigraphy showed an uptake in regard to the swelling. Computed tomographic scan revealed new bone formation bordering the inferior and internal part of the tibial diaphysis (Figure 1B, 1C; arrows) with a normal cortical and associated with a thickening of the soft tissues. Biological assessment was normal: erythrocyte sedimentation rate 3 mm/h, C-reactive protein 13 mg/l, normal blood count, and normal protein electrophoresis. It was decided to carry out an excision of the swelling with cortical and periosteal biopsy. Histopathological study showed necrotizing vasculitis corresponding to polyarteritis nodosa associated with a perivascular inflammatory infiltrate and periosteal bone formation. In January 2000, antinuclear, antineutrophil cytoplasmic and anti-double-stranded DNA antibodies, rheumatoid factors, antiphospholipids, and cryoglobulinemia were negative. Chest radiograph was normal. There were no clinical signs of Crohn's disease. Oral steroid treatment was slowly tapered after 2 bolus infusions of 120 mg of methylprednisolone. This

allowed good clinical improvement of the swelling. The followup bone radiograph 1.5 years later revealed a moderate reduction of the periosteal new bone formation.

Localized vasculitis might be responsible for local production of bone growth factors associated with hypoxia, which could participate in the pathogenesis of periostitis¹⁻⁵.

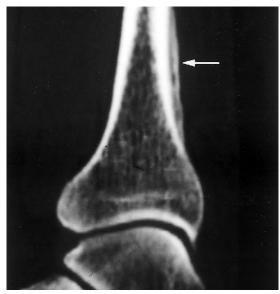


Figure 1B.

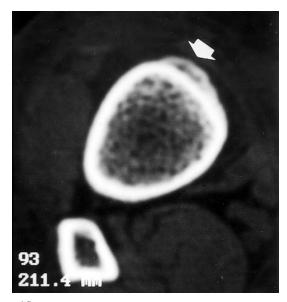


Figure 1C.



Figure 1A.

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REFERENCES

- Saville PD. Polyarteritis nodosa with new bone formation. J Bone Joint Surg 1956;38B:327-33.
- Woodward AH, Andreini PH. Periosteal new bone formation in polyarteritis nodosa: a syndrome involving the lower extremities. Arthritis Rheum 1974;17:1017-25.
- Brandrup F, Petersen EM, Hansen BF. Localized polyarteritis nodosa in the lower limb with new bone formation. Acta Dermatol Venereol 1980:60:182-4.
- Meijers KAE, Pare DM, Loose H, Eulderink F, Siewertsz van Reesema DR. Periarteritis nodosa and subperiosteal new bone formation. J Bone Joint Surg 1982;64:592-6.
- Short DJ, Webley M. Periosteal new bone formation complicating juvenile polyarteritis nodosa. J Roy Soc Med 1984;77:325-7.