


99Tc scintiscan. Since aortic graft infection has been described as a cause of secondary lower limb HOA, a growing number of cases of localized HOA revealing early cases of vascular graft infections have been reported [4]. In a review of 115 cases of HOA, deep infection was responsible for 2.6% of cases of periostitis. Patients usually suffer from progressive pain and swelling of one or both extremities, associated with radiographic periostosis; clubbing of the toes is rare [5, 6]. The pathogenesis of digital clubbing and HOA remains largely unknown. Current thinking suggests that localized activation of endothelial cells by an abnormal platelet population, with the ensuing release of fibroblast growth factors, plays a central role [2]. In the case of HOA associated with vascular infection, the substance that causes HOA could either be produced by vascular tissue or be a precursor activated by contact with abnormal vascular tissue. This theory is supported by the localization of HOA in the limb vascularized by the infected graft and clinical improvement after its removal [4, 5]. Diagnosis of HOA is made using physical examination and imaging studies, finding periostitis and ruling out osteonecrosis, although no international criteria exist [2]. Bone scintigraphy showing localized hyperfixation can help confirm the diagnosis.

BD is characterized by recurrent oral and/or genital ulcers, uveitis, skin and joint involvement. Vascular manifestations appear in 9–38% of patients, mostly as vein thrombophlebitis. Arterial involvement including thrombosis, stenosis and/or aneurysms is less frequent (10–15% of vascular involvement) [7]. Aneurysms, sometimes described as arterial atheroma, may be multifocal and involve mostly the abdominal aorta and the pulmonary arteries. Vascular surgery entails a specific risk of recurrent aneurysm on the anastomosis, aortic–intestinal fistulas, graft infection and thrombosis. A combination of corticosteroids with immunosuppressive therapy is necessary to prevent relapse and appears to be more effective than corticosteroids alone [8].

In our patient, scintigraphy with radiolabelled leucocytes helped localize aorta infection, as in other cases [9]. The diagnostic yield of scintigraphy with radiolabelled leucocytes for aortic graft infections may not be as specific in BD as in other settings because specific vascular inflammation may occur without infection and thus enhance vascular fixation.

Interestingly, high-dose steroids and azathioprine treatment did not appear to abate the HOA clinical features in our patient. The regression of HOA after treatment for graft infection is in favour of a causal link between vascular graft rejection and HOA, ruling out BD as a cause of HOA.

Our observation points out the importance of early diagnosis of HOA, even if it affects the lower limbs or if digital clubbing is lacking, as it can permit early diagnosis of life-threatening vascular graft infection. Particular attention should be paid to patients with BD as they are prone to infectious complications, because of both arterial Behcet-specific inflammation and a long course of immunosuppressive therapy.

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Gold, nitritoid reactions and angiotensin-converting enzyme inhibitors

SIR, Vasomotor or nitritoid reactions are well-recognized reactions occurring in approximately 5% of patients treated with gold [1]. Patients may experience facial flushing, nausea, vomiting, hypotension or syncope. Serious sequelae have been reported, including myocardial infarction (MI) [2]. The reactions are most commonly associated with gold sodium aurothiomalate (myocrisin) but have been reported with oral gold (auranofin) [3]. The reactions are


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