ng/dl (0.8–1.9). Anti-TG antibodies (Ab) and anti-TPO Ab were in the normal range, while the levels of anti-TSH receptor Ab were high: 85.3 U/L (< 1.5). Blood cell count, kidney and liver function tests, urate, acute phase reactants, auto-Ab (ANA, ACA, ENA, anti-dsDNA, anti-cardiolipin, RF), C3, C4 were in the normal range.

Plain radiographs of the hands revealed soft tissue swelling and a bilateral periosteal reaction that was more evident on the diaphyseal portion of several phalanges (Fig.1). On radiographs of the feet (not shown) a bilateral diaphyseal periosteal involvement of the first metatarsals was evident. No joint abnormalities were observed. The clinical history and radiological features were consistent with a diagnosis of TA associated with Graves'ophthalmopathy and pre-tibial mixedema.

TA is a rare extra-thyroidal manifestation of autoimmune thyroid disease, usually Graves' disease, although it has also been described in Hashimoto thyroiditis (2), together with ophthalmopathy and dermopathy. These peripheral manifestations of Graves' disease typically appear in chronological order, with exophthalmos first (30% of patients), followed by dermopathy (4% of patients) and then acropachy (0.1-1% of patients) (3,4). In these patients acropachy usually takes the form of clubbing of the fingernails and/or toenails with or without swelling and tightness of the skin; only 10% of patients with acropachy present the complete clinical picture with clubbing, soft tissue swelling, pain in the distal small joints and a periosteal reaction seen on plain radiographs (1). Typically, the periosteal reaction is solid, and localized on tubular bones (particularly in the hands and feet) with a symmetrical distribution. These radiological features and the past history of Graves' disease allow the differential diagnosis from other conditions associated with a periosteal reaction (5).

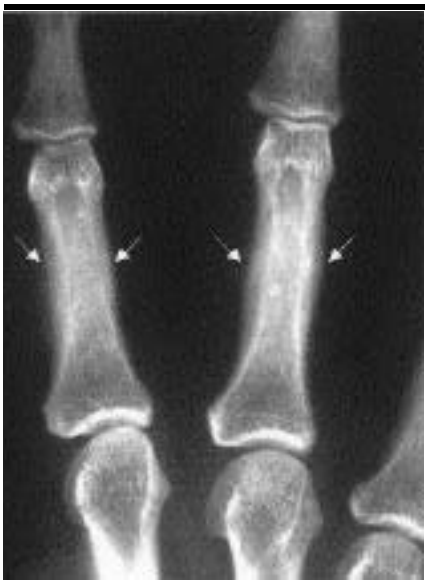

Fig. 1. Magnified view of right hand radiograph showing a periosteal reaction on both radial and ulnar side of the second and third proximal phalanges (**arrows**).

In most patients TA appears 1-2 years after the diagnosis of thyroid dysfunction (1), but its appearance up to 25 years after the onset of thyroid disease has been reported (6). In our patient the chronology of extra-thyroidal manifestations (ophtalmopathy, dermopathy and acropachy) was that usually described and they appeared in succession 17 years after the diagnosis of Graves'disease, despite her euthyroid status. Furthermore, she presented a complete clinical picture of TA that led her to consult our rheumatology unit whereas TA is usually observed by endocrinologists (1,7), dermatologists (4) and radiologists (8, 9).

Of particular relevance is the recent observation that TA and dermopathy are markers of severe Graves' ophtahlmopathy and identify patients in need of more frequent follow-up and more aggressive therapy for ophthalmopathy (7), such as our patient. No specific treatment for TA is available at present nor does its appearance seem to be inhibited by a good pharmacological euthyroid state (1). In our patient, physical therapy and a short course of therapy with a COX-2-specific inhibitor mitigated the articular pain without changing the soft tissue swelling of the fingers and toes; this clinical picture was stable at follow-up till May 2004.

F. CAPSONI, *Asso. Prof.*
F. MINONZIO, *MD*
P. SARZI-PUTTINI[1], *MD*
F. ATZENI[1], *MD*
B. AMBROSI[2], *Asso. Prof.*

Department of Internal Medicine, Ospedale Maggiore Policlinico, IRCCS; [1]Rheumatology Unit, Ospedale L. Sacco; [2]Endocrinology Unit, Istituto Policlinico S. Donato; University of Milan, Italy.
Address correspondence to: Prof. Franco Capsoni, Ospedale Policlinico, Medicina Interna 2, Via Pace no. 9, 20122 Milano, Italy.
E-mail: franco.capsoni@unimi.it

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Calcinosis universalis in systemic sclerosis with limited scleroderma

Sir,

Calcinosis universalis is a rare complication of dermatomyositis, psoriasis, uremia and systemic sclerosis of the limited cutaneous subset (lSSc) (1-5); it is characterized by intracutaneous, subcutaneous, fascial and intramuscular deposits of crystals of calcium phosphate (hydroxyapatite). We describe here a new case of calcinosis universalis complicating lSSc.

A 70-year-old Caucasian woman was recently admitted to the Department of Clinical and Experimental Medicine, because of severe difficulty in walking and rising from a chair and from the bed, and because of a worsening of dyspnea and dysphagia. She had a history of Raynaud's phenomenon and cutaneous ulcerations beginning at the age of 35. In 1973 (when she was 40 years old), the patient underwent surgical resection of numerous subcutaneous calcifications in her thighs and shoulders without any improvement in the ability to walk or move her arms. The diagnosis of lSSc was established 10 years later, in 1983. She was placed on low-dose warfarin for 1 year and subsequently on long-term treatment with colchicine 1 mg daily for more than 10 years without any beneficial effect on her calcinosis. She was permanently on kinesi- and masso-therapy and used to spend the winter on an African island. Her therapy for lSSc consisted of pentoxifylline, domperidone and methylprednisolone 4 mg/day.

On admission physical examination of the abdomen and chest was normal. She presented sclerodactylia, skin fibrosis of face, neck and forearms, and other typical sclerodermic features such as telangiectasias. The thighs and shoulders presented multiple calcifications which made her unable to walk and stand up.

The laboratory investigations showed an increased erythrocyte sedimentation rate (57 mm/Ih; normal value < 35 mmIh), C reactive protein (15.3 mg/Ln.v < 5), fibrinogen (632 mg/dl, n.v.: < 350), hyposidere-

mic microcytic anemia (Hb 10.2 gr/dl, MCV 81 fl, MCH 24 pg), and positive antinuclear antibodies (1:5120 anticentromere). Calcium, phosphate and parathormone serum levels were normal, as well as renal function and calciuria; creatine phosphokinase and aldolase were normal.

Chest X-ray was normal, a transthoracic echocardiogram revealed increased right ventricular systolic pressure (50 mmHg). Renal ultrasonography showed small hyper-echogenic spots (probably calcifications) on both renal pelvis. Esophagogram with barium revealed dilatation of thoracic esophagus.

Shoulders, hands and pelvis X-ray showed widespread calcifications in the superficial and deep tissues (Fig. 1 a-d). The patient was dismissed on therapy consisting of cyclic intravenous infusions of iloprost and oral omeprazole, acetylsalicylic acid 100 mg/day, methylprednisolone 4 mg/day, ACE-inhibitor and bosentan, which resulted in an improvement of dyspnea and dysphagia but no changes in her widespread calcifications.

This is an unusual case of lSSc with widespread periarticular and soft tissue calcifications. Indeed, in lSSc calcifications are usually small, subcutaneous and may become superficial, ulcerate the skin and lead to secondary infections (6). Subcutaneous calcification deposits at sites of trauma such as the forearms, elbows or fingers, occur in all subsets of scleroderma but are more prominent in lSSc and in patients with anticentromere antibodies (6). Calcinosis mimicking a tumoral process (7) and calcinosis universalis have been previously described in patients with lSSc (4, 5) and no medical therapy can modify the progression of calcinosis. Low-dose warfarin, reported to be efficacious in calcinosis universalis by Berger *et al*. (8), but not by Lassoued *et al*. (9), did not obtain any result in our patient.

Broad calcium deposition may also be observed in myositis ossificans progresiva which is inherited in an autosomal dominant pattern, and is first noted during childhood. Myositis ossificans can be localized or widespread but is associated with congenital defects including microdactyly of the large toe and thumb, exocitosis, absence of the two upper incisors, hypogenitalism, absence of the ear lobules and deafness (10).

Finally the differential diagnosis with calcinosis cutis universalis must be kept in mind when the deposition of calcium crystals is extensive but localized to the cutis (11).

The patient described here did not present clinical aspects of dermatomyositis, psoriasis, polymyositis or myositis ossificans and her renal function was normal. We consider her widespread periarticular and deep tissue calcifications to be a form of calcinosis universalis occurring as a complication of lSSc.

S. SIMEONI D. PETERLANA
E. TINAZZI S. MORUZZI
A. PUCCETTI[1] C. LUNARDI

Department of Clinical and Experimental Medicine, Section of Internal Medicine University of Verona and [1]Department of Experimental Medicine, University of Genova and Institute G. Gaslini, Genova, Italy.
Address correspondence to: Claudio Lunardi, MD, Dip. Medicina Clinica e Sperimentale, Policlinco G.B. Rossi, Piazzale Scuro 10, 37134 Verona, Italy.

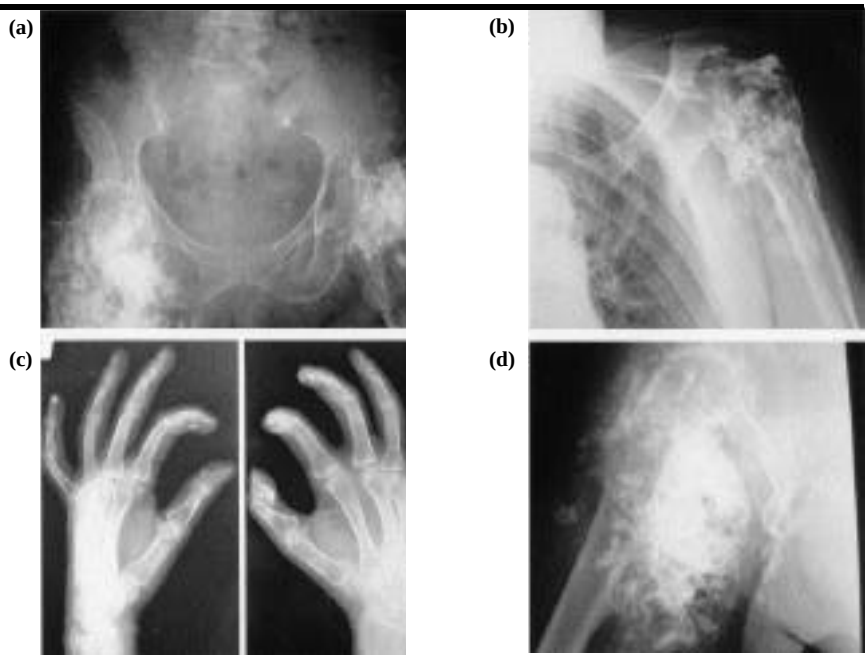

Fig. 1. X-ray images of ectopic calcifications at different levels: pelvis (**a**); left shoulder (**b**); hands (**c**); and right hip and thigh (**d**).

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"Long-term results of multiple synovectomy forpatients with refractory RA" by H. Nakamura *et al.*: Erratum corrige

Sirs,

We would like to publish the notification of an error in our paper "Long-term results of multiple synovectomy for patients with refractory rheumatoid arthritis. Effects on disease activity and radiological progression" by H. Nakamura *et al*. (*Clin Exp Rheumatol* 2004; 22: 151-7). It has been brought to the authors' attention that two earlier articles should have been cited in the paper, regarding the development of multiple synovectomy.

H. NAKAMURA, *MD*

Department of Bioregulation, Institute of Medical Science, St. Marianna University School of Medicine, Kawasaki, Japan.

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