be added to Sweet's syndrome and pyoderma gangrenosum as a further neutrophilic dermatosis that can be induced by G-CSF.

Acknowledgments

The authors thank Dr Philippe Chemaly for performing the histopathological examination and taking the photomicrographs.

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Leuconychia in reflex sympathetic dystrophy: a chance association?

SIR, Reflex sympathetic dystrophy (RSD) is a primary polymorphic condition of persistent pain and swelling, which frequently occurs secondary to a trauma associated with vasomotor disorders affecting the local microcirculation of an extremity and its autonomic control. This condition affects all or part of an articular region from the skin to the bone and conventionally exhibits three clinical phases. The description of these phases is not essential for a diagnosis of RSD. Although the nail abnormalities associated with RSD are typical, to our knowledge leuconychia has never been reported.

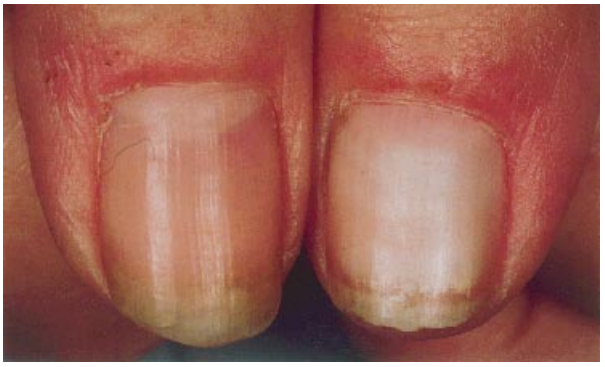

Figure 1. Leuconychia of the nail plate of the left thumb is seen on the hand affected by reflex sympathetic algodystrophy.

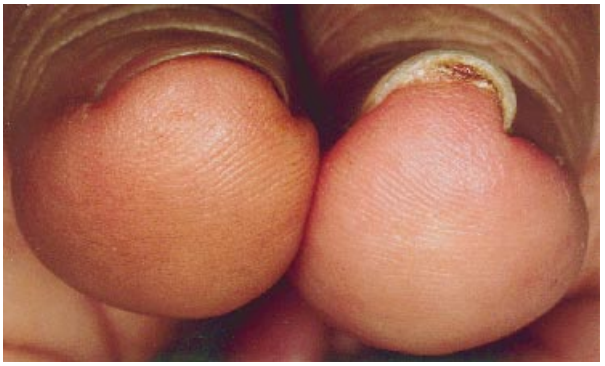

Figure 2. Overcurvature of the affected nail and distal hyperkeratosis are evident.

A 46-year-old woman with no previous medical history showed up with an undisplaced fracture of the radial styloid process following a fall on the left forearm. This was confirmed by X-ray and bone scintigraphy. The treatment consisted of the application of a plaster which the patient maintained for 1·5 months. Upon its removal, a follow-up X-ray showed the persistence of the fracture line and the presence of a callus. Clinical examination revealed no spontaneous pain or tenderness. During the following days, the patient complained of a resurgence of nocturnal and diurnal pain in the arm and the left hand and the appearance of oedema and redness of the skin of the hand, limiting its movement. Scintigraphy revealed increased uptake in the carpus, a sign of the presence of RSD. Treatment with calcitonin and physiotherapy were successful in modulating the episodes of pain despite the persistence of pronounced oedema in the wrist and fingers. After 7 months throughout which the skin signs persisted, isolated leuconychia of the left thumb occurred at the same time as overcurvature of the nail plate (Figs 1 and 2). Electromyography eliminated carpal tunnel syndrome, while an arterial Doppler and capillaroscopy excluded any arterial disorders. Scintigraphy still showed increased uptake in the carpus. Culture and light

microscopy (LM) of the nail plate eliminated microbial infection.

A punch biopsy was performed in the middle of the nail. LM of the nail bed was unremarkable. LM of the nail plate showed the presence of moderate parakeratosis, and electron microscopy (EM) demonstrated a marked alteration in the pattern of corneocytes dissociated by clear spaces and aggregates of filaments. There were numerous large intercellular dilatations in the hyponychium. These features confirmed the diagnosis of leuconychia. After 18 months' follow-up, the clinical signs have diminished, the oedema is in the process of disappearing and the pain is less persistent. The nail plate has progressively lost its whitish colour and resumed a normal curvature. Scintigraphy has confirmed regression of the RSD.

RSD is a primary polymorphic condition or one which is secondary to a trauma associated with vasomotor disorders affecting the local microcirculation and its autonomic control. This condition affects all or part of an articular region from the skin to the bone and conventionally exhibits three clinical phases. The first stage corresponds to the acute phase, and is characterized by a pseudoinflammatory picture involving skin hypersensitivity with dysaesthesia and hypo- or hyperhidrosis of the affected extremities, combined with periarticular oedema. These symptoms, which are not accompanied by any laboratory findings of inflammation, are associated with a reduction in sympathetic activity and may last from several days to a month and be accompanied by an increase in skin temperature, increased hair growth and either acceleration of nail growth with overcurvature or retarded growth.

The second or dystrophic stage lasts 3–6 months: the cutaneous hypersensitivity and periarticular oedema persist, but are accompanied by ischaemic features with incipient subcutaneous atrophy, reduction in skin temperature, pallor, cyanosis and hyperhidrosis. Hair growth is thought to increase with the appearance of local hypertrichosis, and the nails are brittle and fragile. The third or atrophic stage may progress indefinitely over time with features of retraction, predominantly of the soft parts, associated with anhidrosis, skin pallor and cooling of skin temperature. The pain is variable and associated with diffuse osteoporosis, hypertrichosis and brittle and fragile nails. The description of these phases is not essential for a diagnosis of RSD.

Although the nail abnormalities associated with RSD are typical, Tosti *et al.*[1] nevertheless describe an acute inflammation resembling bacterial paronychia associated with proximal leuconychia, Saunders and Hanna[2] report the appearance of unilateral clubbing in 'causalgia', a clinical variant of RSD, and O'Toole *et al.*[3] describe the appearance of Beau's lines in childhood RSD. In our patient, leuconychia of the thumb with overcurvature occurred during the second phase of the disease and corresponds to the lesion of the radial region. Although the occurrence of nail overcurvature is described in the literature in the context of RSD, to our knowledge leuconychia has never been reported. As RSD corresponds to a neurovascular disorder, its association with leuconychia is probably not arbitrary.

All neurovascular disorders may cause leuconychia depending on the context: peripheral neuropathy is a commonly cited cause of total leuconychia,[4] and Arnold[5] describes the appearance of isolated cases of leuconychia adjacent or contralateral to a nail trauma and suggests a neurological mechanism, a variant of the axonal reflex, to be responsible for the leuconychial nail punctuations. Harris *et al.*[6] describe a patient with unilateral transverse total leuconychia of the finger nails which followed a spinal cord injury. The persistent oedema of the fingers and terminal phalanges may also be responsible for slow and progressive trauma to the matrix, a potential origin of 'traumatic' leuconychia.

Leuconychia secondary to trauma localized at the base of the nail plate is well known. Cryotherapy for the treatment of periungual warts or over-zealous manicure are common causes. Repeated trauma to the matrix by pressure on the free edge of the nail plate when the nail is not cut short is a cause of transverse leuconychia.[7] The leuconychia occurring in our patient would therefore be secondary to the combination of two phenomena: neurovascular deterioration and chronic trauma to the nail matrix. In our opinion, leuconychia is unlikely to be caused by pressure on the vascular bed from the pinching effect of increased transverse overcurvature of the nail. Classically, on LM, leuconychia is seen to be due to moderate parakeratosis. On EM, the corneocyte cytoplasm shows clear spaces and aggregates of filaments. This picture is typical of true leuconychia and is not found in leuconychia due to pressure on the vascular nail bed.[8]

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