Acroosteolysis in Diabetes Mellitus

To the Editor:

Acroosteolysis is a rare destructive process of the distal phalanges of the fingers and toes, leading to digital foreshortening. It can occur as a complication of systemic rheumatic diseases, thermal injury, metabolic disorders, and certain occupational exposures, but may also be familial in origin[1]. Acroosteolysis has not been recognized as a complication of diabetes mellitus. We describe a patient with diabetes mellitus who developed acroosteolysis of her fingers, most likely as a result of diabetic neuropathy.

A 45-year-old woman had poorly controlled type I diabetes mellitus for 16 years, complicated by retinopathy, nephropathy, and autonomic dysfunction including gastroparesis and orthostatic hypotension. She was deaf and communicated with sign language. She began to note dryness, induration, and superficial ulceration of the skin of her distal fingers at age 43 years. Painless foreshortening of her little fingers and numbness of her

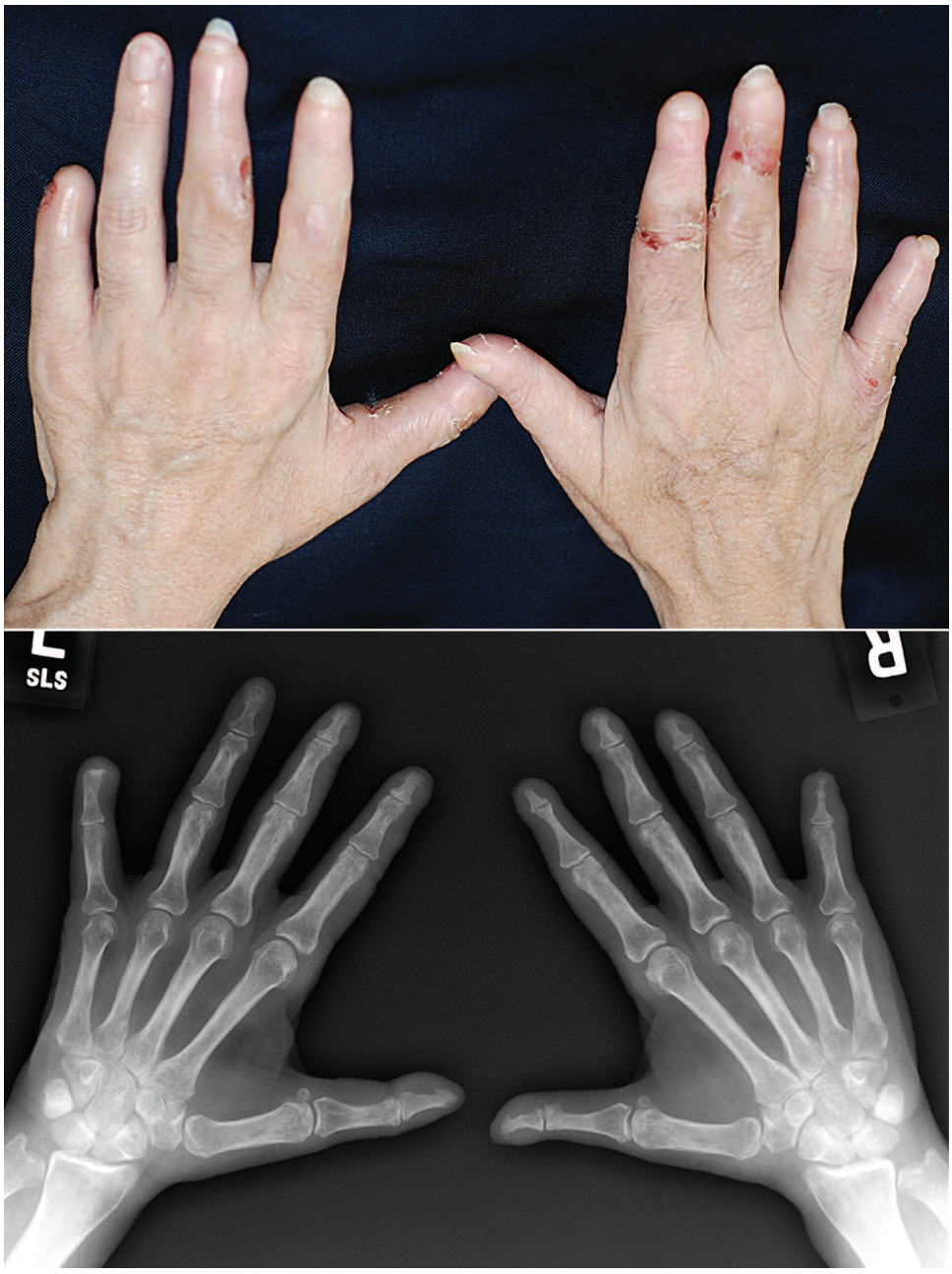

Figure 1. Clinical image (upper panel) and plain radiograph (lower panel) of patient's hands. There is sclerosis of the distal fingers. Areas of superficial ulceration are evident on the dorsal and volar aspects of most fingers. There is foreshortening of both little fingers and loss of the digital tufts in all but the thumbs and left ring finger. Radiographs show near-complete resorption of the distal phalanx of the left little finger and "pencil-sharpened" loss of bone in the distal phalanx of the right little finger. The tuft of the distal phalanx of the right index finger is absent. There is tapering of the mid portion of the middle phalanges (cola-bottle appearance) of the right index and both middle fingers.

hands and feet began shortly after. She denied Raynaud's phenomenon, psoriasis, or a history of thermal trauma or exposure to toxins. On examination, she had sclerosis, hyperkeratosis, superficial ulceration, and fissuring of her distal fingers. There was loss of the distal phalanx of both little fingers and tapering of the tips of both index and long fingers, as well as the right ring finger. Nailfold capillary telangiectasia, synovitis, and psoriasis were not evident. Sensation to light touch was intact in the upper and lower extremities. With a Rydel tuning fork, vibratory sense was 4 at the index fingers and normal at the wrists. It was absent at the toes, normal at the right ankle, and 4 at the left ankle and knee.

Photographs and radiographs of her hands are shown in Figure 1. Radiographs of the feet were normal. Erythrocyte sedimentation rate was 39 mm/h, C-reactive protein 1.7 mg/dl, and creatinine 1.4 mg/dl. Serum calcium, parathyroid hormone, thyroid-stimulating hormone, creatine kinase, and aldolase levels were normal. Antinuclear, anti-Scl-70, anticentromere, anti-SSA, anti-SSB, anti-Sm, anti-RNP, anti-dsDNA, and myositis-specific antibodies were negative. On a nerve conduction study, both sural and right median sensory nerve responses were absent and the tibial motor responses were reduced bilaterally with prolonged distal latencies. The peroneal motor responses were absent when recorded from the extensor digitorum brevis. These changes of a pronounced length-dependent sensory motor peripheral neuropathy with primarily axonal features were consistent with the history of poorly controlled diabetes. There was no evidence of focal nerve entrapment, such as carpal tunnel syndrome or cervical radiculopathy.

In a literature search using the Pubmed, Embase, and Scopus computerized databases, we were unable to identify a previous report of a similar form of acroosteolysis in the context of diabetes mellitus. Diabetic osteoarthropathy has a different clinical and radiographic presentation. The more common "hypertrophic" form is characterized by fragmentation of subchondral bone, bony debris, new bone formation, and sclerosis of bone ends[2]. It almost always involves the lower extremities, usually the forefeet or the hindfeet. A rare atrophic form is characterized by bone atrophy and destruction, with the frequent development of "mortar and pestle" or "pencil-in-cup" joint deformities[3]. There is a predilection for upper extremity joints.

Our patient's clinical findings of acroosteolysis, skin ulceration, sclerodactyly, and cold sensitivity are shared by other discrete clinico-radiographic entities, including vinyl chloride exposure[4], severe forms of carpal tunnel syndrome[5], and hereditary forms of neurogenic osteolysis[6]. This observation suggests a shared pathogenesis. The most instructive of these are rare cases of carpal tunnel syndrome, in which patients develop ulcerative digital skin lesions, sclerodactyly, and acroosteolysis in the sensory distribution of the median nerve[5,7,8,9]. Here the pathogenesis is almost certainly related to damage to the sensory fibers of the median nerve and impairment of autonomic innervation. The sensory neuropathy predisposes to physical or thermal trauma, while the autonomic neuropathy leads to vasomotor instability with color changes of the skin, edema and swelling of the fingers with subsequent fibrosis, and sclerodactyly. The combined injury of the sensory and autonomic nerve fibers thus results in loss of pain sensation and loss of vasoconstriction, leading to hyperemia[3]. The latter has been postulated to be an important mechanism in bone resorption[3]. Our patient's acroosteolysis might also have resulted from repeated needlesticks of her fingertips and her reliance on sign language to communicate. Acroosteolysis has been described in guitar players as a result of repetitive mechanical injury[10].

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