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Reflex Sympathetic Dystrophy of the Lower Extremity

A Complication of Herpes Zoster With Dramatic Response to Propranolol

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REFLEX SYMPATHETIC DYSTROPHY (RSD) is a symptom complex consisting of pain, swelling, trophic skin changes and vasomotor instability in a distal extremity in response to an injury. The distal extremities, most often hands or feet, are affected, but involvement of sites such as knees, hips and zonal portions of the femoral head have also been reported.[1-7] The most common precipitating causes are trauma, either fracture or soft tissue injury, myocardial infarction, cervical spine or spinal cord disorders, osteoarthritis, cerebrovascular accident, malignancy, infection and drugs. However, as many as a third of cases have no antecedent event.[4,7]

First recognized as a distinct entity during the American Civil War, reflex sympathetic dystrophy was originally named causalgia (from the Greek words for heat and pain) because of its characteristic burning pain.[8,9] Since then, several different descriptive terms, such as shoulder-hand syndrome, Sudeck's atrophy, posttraumatic osteoporosis and postinfarction sclerodactyly, have been used synonymously with this rare disorder.

Herpes zoster infection has been associated with the development of RSD in an upper extremity. Ketz and Schliack[10] reported RSD of the shoulder and hand secondary to herpes zoster infection of the fifth and sixth cervical nerve roots. To our knowledge, however, this is the first published case of reflex sympathetic dystrophy of the ankle and foot of the same leg secondary to painful herpes zoster infection of the first sacral dermatome. Also, a dramatic response to propranolol was noted in this patient after failure of conventional RSD therapy.

Report of a Case

A 72-year-old man had a six-week history of painful swelling of the left ankle and foot. The

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pain was constant, dull and exacerbated by movement of the true ankle and metatarsal phalangeal joints and weight bearing. The patient had had herpes zoster infection, involving the first sacral dermatome on the left, which had resolved four weeks before onset of these symptoms.

The patient was treated with intramuscular injections of dexamethasone, acetaminophen with codeine, and nonsteroidal anti-inflammatory drugs, with no relief of symptoms. Roentgenograms of the foot at that time showed no abnormalities.

Past medical history included a myocardial infarction in 1976 and a cerebrovascular accident in 1978, with residual right hemiplegia.

On admission of the patient to this hospital, a physical examination showed no abnormalities of the head, ears, eyes, nose or throat. Chest was clear to auscultation and abdomen was soft and nontender. There was no hepatosplenomegaly. The skin over the left calf had many 3- to 6-mm hyperpigmented areas. Nonpitting edema involved the left foot and ankle to midcalf. The skin overlying the nonpitting edema was very tender to palpation and cool to the examiner's touch (Figure 1). Range of motion of the left ankle was normal but painful. There was mild muscle weakness on the right. Deep tendon reflexes were symmetric with no pathological reflexes. Dorsalis pedis pulses were absent bilaterally; all other pulses were palpable. Vibratory sensation and light touch were intact bilaterally.

Results of laboratory tests included the following: hemoglobin 14.8 grams per dl and hematocrit 44.3 percent, with a leukocyte count of 7,000 per cu mm. Serum alkaline phosphatase, albumin, calcium, phosphorus, uric acid, glucose and blood urea nitrogen values were within normal limits. Westergren sedimentation rate was 8 mm per hour. Tests for rheumatoid factor, antinuclear antibody, hepatitis B antigen and HLA-B27 were negative.

Roentgenograms of the left foot and ankle initially showed no bony abnormality (Figure 2). However, roentgenograms of the left foot taken four weeks later showed patchy osteopenia involv-

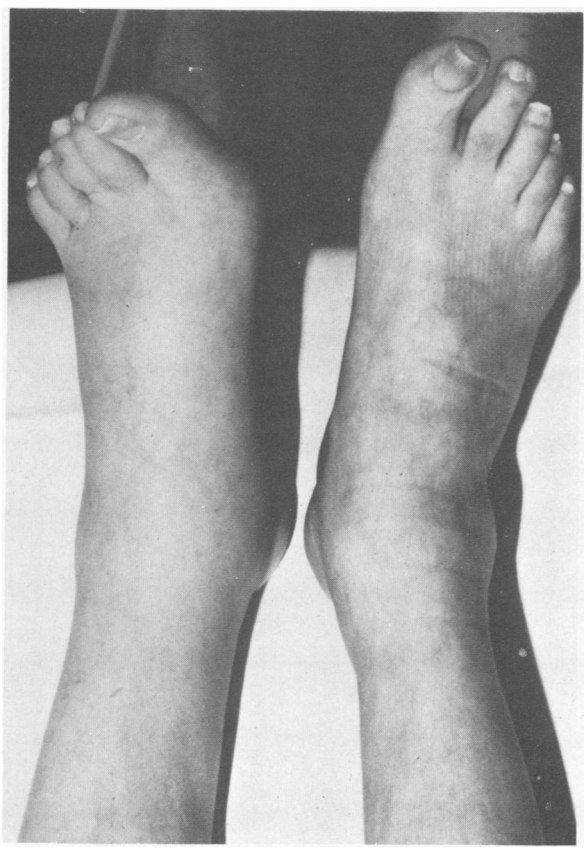

Figure 1.—Nonpitting edema of the left foot and ankle is contrasted with normal right foot.

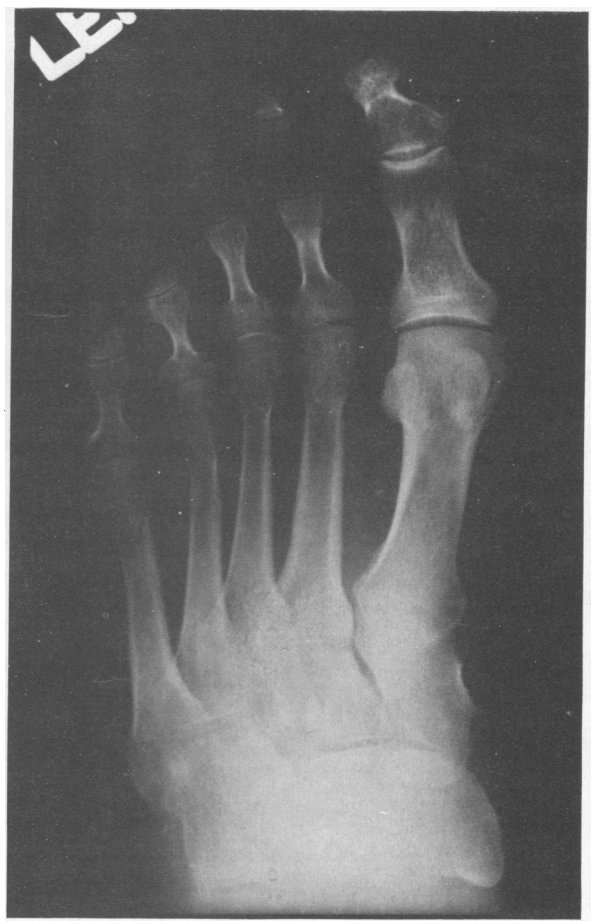

Figure 2.—Roentgenogram of left foot at onset of symptoms showing no bony abnormality.

ing the metatarsal phalangeal joints, consistent with the diagnosis of reflex sympathetic dystrophy (Figure 3). No erosive changes were noted. A bone scan with technetium 99m diphosphonate showed increased uptake in the left ankle and foot (Figure 4).

An aggressive physical therapy program was begun as well as a regimen of salicylate and nonsteroidal medications. After six weeks of intensive therapy there was no change in the patient's signs or symptoms.

Because there was no history of myocardial dysfunction, asthma or diabetes mellitus, oral propranolol therapy, 160 mg per day in four divided doses, was tried. Follow-up after a week showed a 50 percent decrease in swelling of the left lower extremity and the patient was able to bear weight. Within four weeks, the swelling, hyperesthesia and pain had completely resolved (Figure 5). Propranolol therapy was gradually tapered off during the next eight weeks. Repeat roentgenogram was

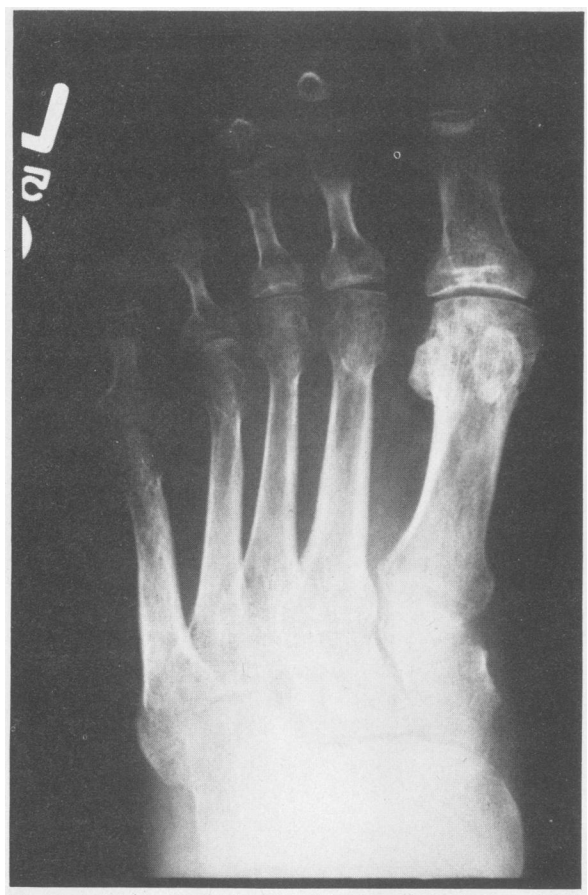

Figure 3.—Roentgenogram of left foot taken six weeks after symptoms appeared shows patchy osteopenia mainly involving distal metatarsal and interphalangeal bones.

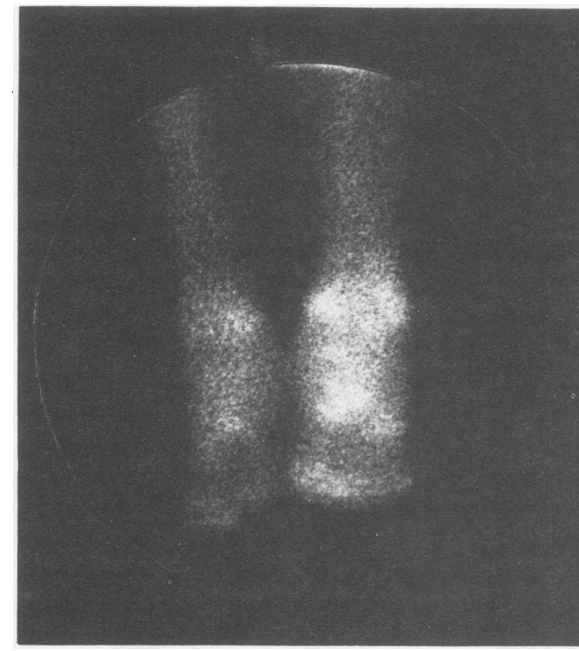

Figure 4.—Bone scintigram shows increased radioisotope uptake around the left ankle and metatarsal joints. Note the soft tissue swelling of left foot.

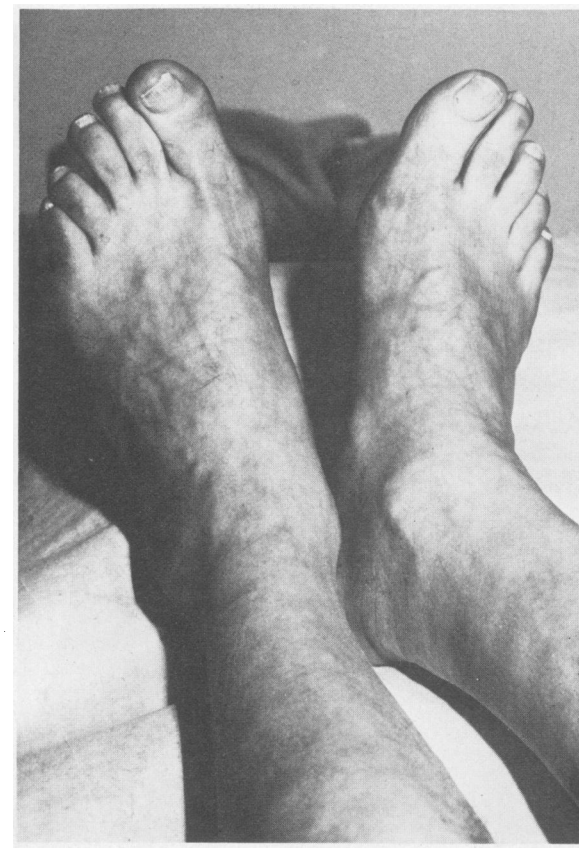

Figure 5.—View of left foot and ankle after four weeks of propranolol therapy, showing resolution of swelling.

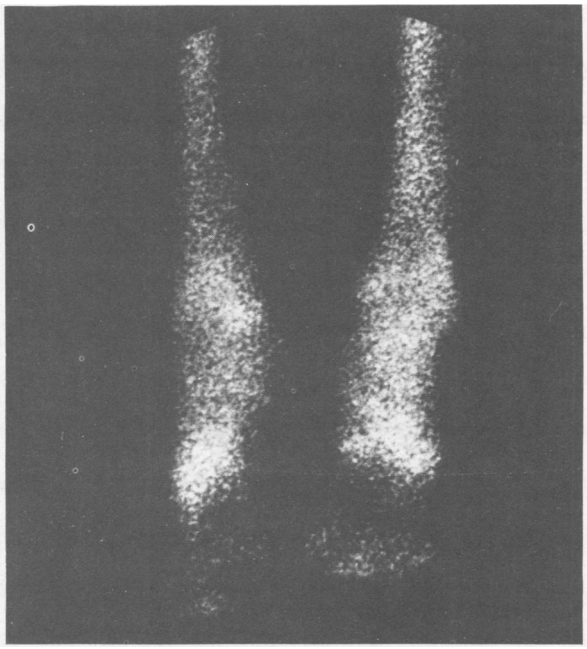

Figure 6.—Repeat bone scintigram after 10 weeks of propranolol therapy shows equal radioisotope uptake in both ankles and feet.

unchanged (patchy osteopenia still present); however, a repeat bone scan showed equal uptake of radioisotope in both ankles and feet (Figure 6). Eight months later, he was still asymptomatic.

Discussion

This case illustrates the salient clinical features of reflex sympathetic dystrophy. RSD is much more common in older adults, probably because of a predilection of the precipitating causes for this age group. Surprisingly, a substantial number of these patients have a history of psychiatric disturbance before the onset of illness, suggesting that a certain emotional profile may be a predisposing factor.[2,6]

The pain is described as burning in nature and most severe in the periarticular tissues. Swelling of the involved extremity can be pitting or nonpitting, as in this case, but localized to the painful periarticular area. Vasodilatation or vasoconstriction is noted. Sometimes Raynaud's phenomenon or hyperhidrosis is observed, but most often the involved extremity appears pale and cool to the examiner's touch.

Roentgenograms are helpful in making a diagnosis. Findings may be normal at onset but within a few weeks, characteristically, they show soft tissue swelling and patchy osteopenia that is juxtaarticular.[1,11,12] Figure 3 shows the mottled appearance of the bone that developed around the metatarsal joints in the patient's involved foot, contrasted with the earlier, normal appearing roentgenogram (Figure 2).

Other roentgenographic changes include diffuse osteopenia with a "ground glass" appearance, cortical bone resorption and juxta-articular subchondral erosions, usually appearing late in the course of the disease.[1,11] The erosive changes are believed to result from hypervascularity and increased osteoclastic bone resorption.[1]

Hypervascularity of the involved extremity accounts for the increased activity noted on both bone and joint scintigrams.[1,7,11] A bone scintigram in this patient showed increased uptake in the affected ankle (Figure 4).

The mechanism of RSD remains unknown. Turek[13] proposed that an irritative focus provokes continuous vasospasm of the terminal arteriole. Distention of capillaries and venules occurs and blood stasis results in the formation of edema. The local acidosis stimulates osteoclasts to resorb bone.

Melzak and Wall have suggested that sympathetic impulses feed the same receptor centers as the small fibers mediating pain transmission and large fibers that inhibit it, and are influenced by higher perceptual and cognitive processes.[14] Thus, RSD may result from stimulation of efferent sympathetic fibers by afferent pain impulses, cognitive or perceptual influences or direct irritation of the receptor centers.

Mobilization and aggressive physical therapy remain the mainstays of treatment. Anti-inflammatory drugs, high doses of corticosteroids given orally, sympathectomy and manipulation under general anesthesia all have been reported successful in selected patients.[2-6,15-17] Intravenous administration of guanethidine to the affected limb has been noted to achieve rapid reduction of pain and edema.[18,19]

Propranolol therapy was tried in this patient after conventional treatment failed, based on Simson's report[20] of dramatic improvement with propranolol in two cases of RSD. The prompt resolution of his signs and symptoms after the initiation of propranolol suggested that a spontaneous remission was unlikely.

The mechanism of action of propranolol in RSD is unknown. Propranolol is a β-adrenergic receptor blocker. It is capable of blocking the effects of catecholamines and sympathetic nerve stimulation on heart rate, cardiac output and contractility. Recent studies have shown administration of pro-

pranolol reduces the response to sympathetic nerve stimulation in human peripheral veins and arteries.[21,22] It is interesting to speculate that propranolol may act in RSD in part by diminishing the effects of increased sympathetic nerve activity on peripheral veins and arteries that occurs in RSD.

Herpes zoster infection can precipitate RSD in the lower extremities. This important history should be obtained in the differential diagnosis of a painful swollen extremity, especially in older adults. This case also supports the suggestion that propranolol is successful in the treatment of reflex sympathetic dystrophy and probably should be instituted early in the course of illness along with aggressive physical therapy.

Summary

Reflex sympathetic dystrophy is a syndrome characterized by painful swelling of an extremity. It is thought to result from increased sympathetic activity in the autonomic nervous system in response to an injury, but the exact cause remains unknown. A case of reflex sympathetic dystrophy of the left ankle and foot secondary to herpes zoster infection in a 72-year-old man is reported. Of particular interest was the dramatic response to propranolol therapy after conventional RSD treatment failed.

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Cardiac Arrhythmia at High Altitude

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IN PATIENTS with the sleep apnea syndrome, 24-hour electrocardiographic monitoring has shown a typical pattern of cyclic sinus arrhythmia in which the heart rate slows dramatically during apnea and increases when airway obstruction is relieved.[1,2] In this study a single subject was monitored at high altitude and this same pattern was found, suggesting that hypoxia, probably aggravated by periodic breathing, may be sufficient to produce pronounced sinus arrhythmia and sinus bradycardia in an otherwise healthy person.

Report of a Case

One of the authors (P.C.) was the subject of this study. At age 34, he had had 12 years of mountain climbing experience including successful ascents of two peaks higher than 6,000 meters and one summit higher than 7,000 meters. He had experienced the syndrome of acute mountain sickness[3-5] either when ascending in a single day from sea level to about 3,600 meters or on expeditions when first arriving at altitudes of about 5,000 meters.

The present study was undertaken as part of an expedition to Annapurna (8,078 meters) in Nepal. A 24-hour electrocardiographic (ECG) study was done in California on July 20, 1979, at an altitude of 100 meters. Two leads, CC5 and

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