Communications

Neuroarthropathy in Diabetes Mellitus

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Neuroarthropathy is a destructive joint process that occurs as a result of repeated daily "microtrauma" in patients with loss of protective pain sensation in their joints. It has been described in a variety of clinical settings, including tabes dorsalis, syringomyelia, peripheral nerve injuries, leprosy, congenital absence of pain sensation, and diabetic neuropathy. In diabetes mellitus, neuropathic changes may occur in any joint, but the foot is the area most frequently involved.[1-4]

The purpose of this report is to draw attention to the neuroarthropathy that may be seen in patients with peripheral neuropathy secondary to diabetes mellitus. Two diabetic patients with neuropathic joint disease are reported.

Case Reports

Case 1

A 55-year-old woman with a 17-year history of non-insulin-dependent, insulin-requiring diabetes mellitus presented with painless swelling in her left foot. She had peripheral neuropathy manifested by paresthesias and loss of vibratory sensation in both feet. There was no history of lower extremity trauma. Physical examination showed painless deformity with swelling in the metatarsal-phalangeal (MTP) joint of the left hallux. The clinical impression was that the findings were consistent with gout. Radiographs of the left foot demonstrated destruction of the distal MTP joint of her great toe consistent with a neuropathic joint. She continued to ambulate on her left foot. Repeat roentgenograms six months after presentation showed progressive destruction of the left MTP joint and new, healing fractures of the second and third right metatarsal (MT) joints. Five years after initial presentation the patient noted the insidious onset of bilateral ankle edema and difficulty with ambulation. Radiographic studies showed a new 1.5-cm avulsion fracture of the right calcaneus, deformity of the second and third MTP joints, and hypertrophic bone formation of the left first MTP joint. With joint immobilization and removal of weight bearing from the involved leg, the patient improved.

Case 2

A 57-year-old woman with a 15-year history of non-insulin-dependent, insulin-requiring diabetes mellitus presented with a four-week history of difficulty with ambulation. She had symptoms and signs of peripheral neuropathy but denied trauma or twisting injury to the legs. Physical examination showed painless joint deformity and prominence of the right navicular bone at the midfoot region. Roentgenographic studies showed a Lisfranc type of fracture-dislocation of the right second through fifth metatarsals (Figure 1). The involved extremity was made nonweight bearing and the patient has done well.

Discussion

The incidence of arthropathy in diabetes mellitus has been estimated to be 0.5 percent.[1] The main feature predisposing to arthropathy is the absence of or decrease in pain sensation. This leaves the joints without protection from the repeated microtrauma that occurs with normal activity. Unrecognized stress fractures may be the resulting initial lesion in many neuropathic joints.[4] In pa-

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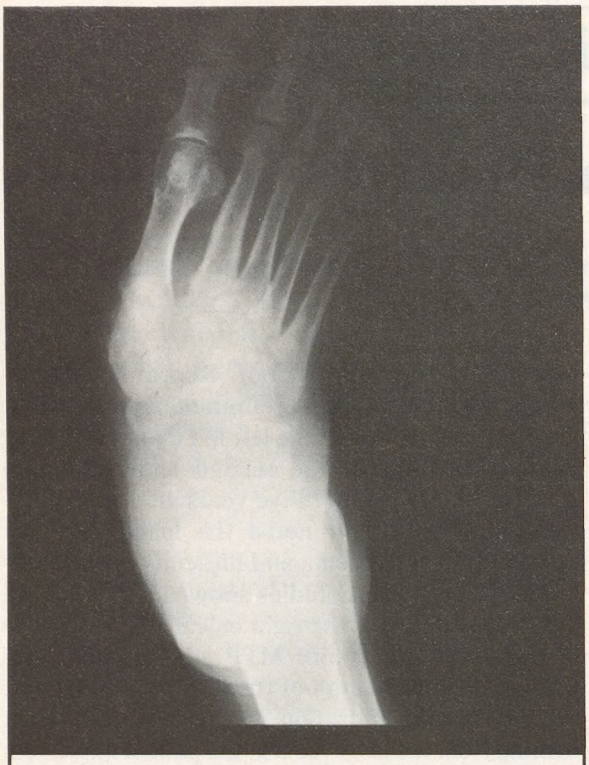

Figure 1: Case 2: Lisfranc fracture-dislocation of right second through fifth metatarsals

tients with normal sensation, once an injury occurs, pain is sufficient to prevent further damage to the joint. In neuroarthropathy, fractures are only one way in which joint stability can be disrupted and abnormal stresses applied to the joint.[3-7] Others include dislocation, laxity of ligaments, cartilage damage, bone erosion, and hypertrophic bone repair.[8]

The two patients reported here are noteworthy in the severity of their arthropathy, the first patient having had severe avulsion fracture amidst several smaller fractures, and the second patient having had a fracture-dislocation of which she was unaware. If pain sensation is decreased, use of the joint is not inhibited by pain; the result is swelling with local inflammation, stretching, and weakening of the joint capsule and ligaments and, finally, joint dislocation. It is the continuation of activity that is detrimental to healing and therefore a key element in the pathogenesis of neuroarthropathy.[3,4]

The role of osteopenia in fracture production is controversial. Reduced bone mass (defined by

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photon absorption densitometry) in affected diabetics has been shown to exist[2]; however, a number of investigators have found no role of osteopenia in diabetic fracture production.[2-4] Heath et al[2] compared with age matched controls the incidence of skeletal fractures in 1,000 diabetics. Although they did not identify those with peripheral neuropathy, their data did not support a role for osteopenia in skeletal fracture production. The only bone at increased risk for fracture in diabetics was found to be the medial malleolus.[2]

Sinha et al[3] studied 101 patients with diabetic neuropathic joints. The most common sites of involvement were the tarsal joints (47 percent), tarsal-metatarsal joints (34 percent), and ankle joints (11 percent).[3] Bilateral disease was present in 24 percent. The most frequent presenting complaints were bony deformity, ulceration, and soft tissue swelling. The most prominent signs of arthropathy were bony deformity, callus formation, ulceration, soft tissue swelling, and limp. All patients demonstrated anesthesia or hypoesthesia of the feet. The most common radiographic sign was disruption of articular surfaces as evidenced by irregular, narrowed, or obliterated joint spaces. Other radiographic signs included fragmentation, periosteal new bone formation, dislocation, vascular calcification, and bone resorption.[3]

Treatment of neuropathic joint disease includes immobilization and nonweight bearing of the extremity.[3,4,7] Continued trauma is a prerequisite for progression of joint destruction; thus, if repeated trauma is prevented, the joints will usually heal. Duration of protection of the joint must be based on clinical and radiological response. Pain is a poor guide of response, since these patients have diminished pain sensation. Premature resumption of activity may lead to further joint destruction.

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Pasteurella Multocida Meningitis in an Infant Following Occipital Dog Bite

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It has been estimated that approximately one million people are victims of dog bites each year in the United States, and children are bitten more frequently than adults.[1] Children under four years of age are at an increased risk of being bitten on the head, neck, or face.[2] This report describes the case of a ten-month-old boy who developed Pasteurella multocida meningitis 48 hours after being bitten in the occiput by the family dog.

Case Report

A ten-month-old white boy was admitted to the pediatric unit of Riverside Hospital, Toledo, Ohio, with the diagnosis of bacterial meningitis.

Forty-eight hours prior to admission the infant was attacked by the family's German shepherd dog and sustained three lacerations of the scalp: a 3-cm laceration over the left mastoid, a 3-cm laceration

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