SPONTANEOUS DISLOCATION IN DIABETIC NEUROPATHY
A REPORT OF SIX CASES
JOHN H. NEWMAN
From Bristol Royal Infirmary

The clinical details of six patients who developed spontaneous dislocations in the foot or ankle are presented. All were shown to have diabetic neuropathy. This previously unreported condition can occur with a short history of diabetes. Some cases can be managed without operation, though arthrodesis probably offers the best chance of obtaining a stable foot of satisfactory shape.

Neuropathic joints in association with tabes dorsalis were first described by Charcot in 1868. It was not until 1936 that a similar abnormality was noted in diabetes mellitus (Jordan 1936) but since then bone and joint abnormalities have become well recognised. In diabetes typical neuropathic joints may be seen in any part including the spine (Feldman, Johnson and Walter 1974) and upper limb (Feldman et al. 1969) but the foot remains the area most frequently involved.

Various other patterns of bone and joint disease are recognisable in the feet of patients with diabetic neuropathy. In some patients osteopathy or thinning of the metatarsals occurs (Pogonowska, Collins and Dobson 1967); in others hyperostosis or osteoporosis may develop (Forgacs, Halmos and Salamon 1972). Heiple and Cammarn (1966) described two cases of spontaneous peritalar fracture-dislocations in which there was considerable bone destruction, and changes typical of neuropathic joints developed.

The purpose of this report is to draw attention to the occurrence of spontaneous dislocations in association with diabetic neuropathy. Five patients are described in whom the initial skeletal change has been confined to the soft tissues and dislocation has occurred without bone destruction; in a further case a remarkable degree of subluxation without dislocation occurred.

CASE REPORTS

Case 1. A sixty-seven-year-old retired train driver was first noted to be diabetic in 1973. He was controlled on 10 milligrams of glibenclamide daily and had no serious problems until 1975 when he presented with a deformed foot. Examination revealed a symmetrical peripheral neuritis with loss of sensation to pin-prick, position and vibration affecting both legs. His ankle jerks were absent but knee jerks were normal, as was neurological examination of his arms. His Wassermann reaction was negative and serum vitamin B12 level was normal. Radiographs showed a dislocation of the talonavicular joint but the bony architecture remained normal (Fig. 1). Two days later, open reduction was performed and the position stabilised with a Kirschner wire and plaster of Paris for six weeks (Fig. 2). A few days after removal of the temporary fixation redislocation occurred. Triple arthrodesis was performed two weeks later and despite delayed union produced a comfortable foot of satisfactory shape.

Case 2. A seventy-five-year-old woman developed a sudden pain in her left foot while ironing. Believing the cause to be an insect bite she bathed her foot in hot water and scalded herself. A week later she sought medical advice and was found to have a swollen foot with a partial-thickness scald on the dorsum. Deep pain sensation in her left foot was diminished and both ankle jerks were absent. Questioning revealed a two-week history of polydipsia and polyuria while her blood sugar on admission was found to be 23 millimoles per litre. Her diabetes was initially controlled with insulin but she was subsequently managed with oral hypoglycaemic agents. The initial radiographs of her left foot (Figs. 3 and 4) showed a complete tarsometatarsal dislocation. The medial cuneiform was dislocated but there was no...
bony damage. Once the scald on the left foot had healed, open reduction and tarsometatarsal fusion was performed. Skin healing was delayed but eventually a satisfactorily stable foot was obtained.

Case 3. A seventy-one-year-old woman presented in 1977 with an ulcer over the lateral aspect of her ankle. She was found to have diabetes mellitus with peripheral neuropathy. Treatment with insulin was started and when the ulcer was debrided communication with the ankle joint became apparent. The ulcer eventually healed after treatment in plaster but after six months the foot became swollen and another radiograph (Fig. 5) revealed that the ankle joint had now dislocated but there was relatively little evidence of bone destruction or infection. A Charnley compression arthrodesis of the ankle was performed but without success and the patient now has a typical neuropathic ankle joint (Fig. 6).

Case 4. A fifty-eight-year-old man who had been treated for diabetes mellitus with 25 milligrams of Diabinese daily for six months developed a swelling in his left foot six days before admission. Examination demonstrated a peripheral neuropathy and also an ulcer overlying the bony prominence in the swollen foot. There was some surrounding erythema but no clinical evidence of abscess formation. Radiographs of the foot showed a complete tarsometatarsal dislocation but there was no evidence of osteomyelitis (Fig. 7). Treatment with intravenous antibiotics was started but the cellulitis continued to spread and a below-knee amputation was performed. Six days later he died of a massive pulmonary embolus.

Case 5. A sixty-year-old man presented with a right foot that had been swollen for two months. There was no history of injury. Though asymptomatic he was found to be diabetic with a peripheral neuropathy. His diabetes was controlled with oral antidiabetic agents but he rapidly developed retinopathy and nephropathy with a general deterioration in his health. Radiography showed a complete tarsometatarsal dislocation similar to that seen in Case 4. There was some new bone formation which might be related to the fact that the patient had been walking on his deformed foot for several weeks and though much of the swelling subsided his foot remained flattened and stiff. He has now worn surgical shoes for five years and this has controlled his symptoms and prevented ulceration. Recent radiographs show slight further disorganisation of the foot with more new bone formation (Figs. 8 and 9).

Case 6. A sixty-three-year-old woman had been an insulin-controlled

Fig. 7
Case 4. Radiograph showing complete tarsometatarsal dislocation without destruction of bone or fracture.

Fig. 8
Case 5.

Fig. 9
Figures 8 and 9—Case 5. Radiographs five years after spontaneous tarsometatarsal dislocation. There was considerable new bone formation but relatively little bone destruction.
diabetic for thirty years and was known to have a peripheral neuropathy. She was seen because instability in her foot was beginning to cause pressure changes in the skin along the medial border. Stress radiographs (Figs. 10 and 11) demonstrated that the talonavicular joint could be subluxed so that much of the head of the talus became uncovered and on weight-bearing presented as a lump on the medial side of the foot threatening the integrity of the skin. Shoes to control the subluxation were provided but no other treatment was considered necessary.

The sixth patient did not have a complete dislocation but is included in this report because a grossly abnormal degree of movement could be demonstrated at the talonavicular joint. It is proposed that such instability represents a mild form of the same process.

DISCUSSION
In a foot with normal sensation dislocations around the tarsus only occur after major violence and even then they are infrequent. The dislocations reported here all occurred without any known injury although in Case 3 it was associated with infection of the ankle joint. However, I am unaware of a dislocation of the ankle occurring as a result of infection and it seems improbable that such an event would happen when the joint was draining freely, as in this case. It therefore seems likely that the neuropathic changes were responsible for allowing the dislocations in all cases. It has been stated that the earliest changes in the development of a neuropathic joint involve the soft tissues (Bruckner and Howell 1972). It is postulated that in these five cases the neuropathic changes in the ligaments were so gross as to allow the dislocations to occur in the absence of any readily detectable bony abnormality. Although it is recognised that spontaneous fractures occur in diabetic neuropathy (Johnson 1967), changes involving soft tissues to such an extent as to allow spontaneous dislocations have not been reported.

These cases of spontaneous dislocation of the tarsus are interesting for several reasons. Although the radiological changes are striking four of the patients were unaware of anything seriously amiss with the foot whilst such a derangement in a normal foot would require considerable force and would be all too apparent to the patient. All five patients with dislocations had only been known to have diabetes for a short time and none ultimately required insulin for its control. In contradistinction, most diabetic patients who develop the more common Charcot joints in the tarsus have had the disease for many years and require insulin.

Charcot joints are normally assumed to start with spontaneous fractures which when combined with ligamentous laxity lead to progressive destruction of the joint. Perhaps if these cases had been left untreated fractures would have subsequently occurred, followed by new bone formation and the development of the well-reorganised changes seen in neuropathic joints. It is possible that in some cases the ligamentous lesion may be of paramount importance and I would suggest that in
a few typical Charcot joints the first major derangement is a spontaneous dislocation which goes unrecongised allowing the other changes to develop secondarily. Some support for this idea is lent by the appearances in Cases 3 and 5. In Case 5 new bone formation was occurring after eight weeks and the appearance of the radiograph five years later could be mistaken for a Charcot joint, though there is still relatively little bone destruction. The final appearance seen in Case 3 is typical of a Charcot joint and although both infection and surgery had been factors it is likely that walking on an anaesthetic dislocated joint would have produced a similar appearance.

Spontaneous dislocations must be extremely uncommon and do not necessarily require surgical treatment. Special footwear can sometimes be provided to ensure that the skin is not threatened, but if the deformity is such that this can not be guaranteed then the dislocation must be reduced. Since the forces causing the initial dislocation are still acting, it seems logical to expect that redislocation will occur as in Case 1. Therefore, if reduction of the dislocation is required, fusion of the affected joint should be performed followed by prolonged immobilisation. Bony union can probably be achieved but if even this fails a stable foot of satisfactory shape should be produced. Only if gangrene is present or infection uncontrolled should amputation be necessary.

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REFERENCES


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