Spontaneous fractures in patients with diabetic neuropathy

A. J. KRENTZ, MB, MRCP(UK), Research Registrar
M. G. FITZGERALD, MD, FRCP, Consultant Physician
A. D. WRIGHT, MB, FRCP, Senior Lecturer in Medicine
M. NATTRASS, PhD, FRCP, Consultant Physician
Diabetic Clinic, Steelhouse Lane, Birmingham

Pathological fractures are a reported feature of diabetic polyneuropathy [1]. We describe four cases of spontaneous fractures in diabetic patients observed during a 12 month period.

Case 1

A 42 year old man with a 28 year history of insulin-dependent diabetes developed acute and painful swelling of the left foot. There was no history of trauma. His past medical history included proliferative retinopathy and ulcerative colitis. The foot was warm, tender and oedematous. Signs of a distal polyneuropathy were present with loss of knee and ankle reflexes and impairment of light touch, pinprick, proprioception and vibration sense. Pedal pulses were easily palpable. ECG investigations revealed impaired cardiac parasympathetic reflexes with a mean expiratory/inspiratory R-R (E/I) ratio on deep breathing of 1.08 (normal >1.10). Radiological examination revealed soft tissue swelling, vascular calcification and a fracture through the anterosuperior aspect of the calcaneum (Fig. 1). The acute episode resolved with a split plaster cast but 8 months later he presented again with an acutely swollen and erythematous left hallux. The entire foot had become grossly deformed and oedematous. Radiological examination confirmed a Charcot neuroarthropathy with degeneration of the calcaneum and destruction of the proximal phalanges of the hallux and second toe and of the base of the first metatarsal.

Case 2

A 63 year old man gave a 2 week history of pain in the right foot on weight bearing. No trauma had occurred. Diabetes had been diagnosed 15 years earlier and he had received laser therapy for maculopathy. Examination revealed absent knee and ankle jerks and diminished vibration and pinprick sensation. E/I ratio was reduced at 1.0. The right foot was deformed, warm and tender and a fracture through the shaft of the second metatarsal was evident on the X-ray (Fig. 2). Treatment with an elastic bandage was maintained for 2 months and the foot settled.

Fig. 1. Spontaneous fracture through the anterosuperior aspect of the calcaneum.

Case 3

A 64 year old woman with diabetes of 19 years duration had a 6 week history of painful swelling of the left foot exacerbated by weight bearing. There was no history of trauma. Symptoms suggestive of a distal polyneuropathy (spontaneous pains and paraesthesiae) had been present for 3 years and she had received treatment for proliferative retinopathy. Examination confirmed a marked distal sensori-motor polyneuropathy. There was no clinical evidence of peripheral vascular disease. Electrophysiological investigation demonstrated an unrecordable sural nerve action potential. Peroneal motor nerve conduction velocity was reduced at 35.8 m/s (reference range 38–62 m/s) and E/I ratio was reduced at 1.09. A healing fracture of the second metatarsal shaft was evident on the radiograph (Fig. 3). The acute pain and swelling resolved with
the application of a below-knee walking plaster for 6 weeks.

Case 4
A 59 year old woman with a 16 year history of diabetes gave a 4 week history of spontaneous swelling and discomfort around the left lateral malleolus. There had been no trauma. Background retinopathy and symptoms of a chronic distal neuropathy had been present for 5 years. Examination revealed signs of a distal polyneuropathy and a warm, swollen left ankle. No action potential was recordable from the peroneal nerve although E/I ratio was normal at 1.22. A healing transverse undisplaced fracture of the left fibula was diagnosed (Fig. 4) which healed satisfactorily within 3 months in a plaster cast.

Discussion
Radiological studies have demonstrated a number of non-infective conditions of the diabetic foot. Reported lesions range from asymptomatic osteoporosis to spontaneous dislocations and disabling Charcot osteoarthropathy [2,3]. Although perhaps not widely appreciated, pathological fractures are also a frequent radiological finding and are strongly associated with the subsequent development of osteoarthropathy [1,2].

Several features deserve comment. All patients had insulin-treated diabetes of long duration with evidence of retinopathy and peripheral neuropathy but pedal pulses were palpable in all. While all four patients experienced local discomfort, it is difficult to determine the degree to which pain may have been attenuated by the impairment of distal sensory modalities.

The pathogenesis of diabetic osteopathy remains unclear. A generalised decrease in bone mass (osteopenia) is a recognised chronic complication of diabetes [4]. Furthermore, patients with clinically evident diabetic neuropathy have a significantly reduced cortical bone mass compared with matched non-neuropathic controls [5]. Bone resorption has been linked to increased blood flow and arterio-venous shunting in the feet of subjects with diabetic neuropathy [6], and osteoarthropathy following revascularisation surgery has been reported [7]. Motor neuropathy involving the intrinsic muscles of the foot exposes the metatarsal heads to abnormally high mechanical pressures [8]. In addition, impairment of pain sensation and proprioception may allow repeated unrecognised trauma in patients with neuropathy [2,3].

Management of an acute neuropathic fracture generally consists of avoiding weight bearing and a cast of plaster or plaster substitute. Following union of the fracture, specialist footwear should be provided to support the foot and redistribute body weight [9]. In view of
the risk of subsequent osteoarthropathy, careful long-term follow-up of diabetic patients with spontaneous fractures is indicated. However, no form of therapy has yet been shown to retard the progression of diabetic osteoarthropathy [10].

Spontaneous pathological fractures are a potentially serious complication of diabetic neuropathy. Impairment of pain sensation in patients with peripheral neuropathy may cause the examining clinician to underestimate the possibility of a fracture. Prompt radiological investigation should therefore be performed in any diabetic patient presenting with a swollen or painful foot.

Acknowledgements

Dr Krentz is an ICI research registrar. We thank Dr L. Honigsberger for the nerve conduction studies.

References

1. El-Khoury, G. Y. and Kathol, M. H. (1980) Neuropathic fractures in patients with diabetes mellitus. Radiology, 134, 313.
2. Newman, J. H. (1979) Spontaneous dislocations in diabetic neuropathy. Journal of Bone and Joint Surgery, 61B, 484.
3. Newman, J. H. (1981) Non-infective disease of the diabetic foot. Journal of Bone and Joint Surgery, 63B, 593.
4. Hough, S. and Avioli, A. V. (1984) Alterations in bone and mineral metabolism in diabetes. In Recent advances in diabetes, 1st edn (ed. M. Nattrass and J. V. Santiago), p 223. Edinburgh: Churchill Livingstone.
5. Cundy, T. F., Edmonds, M. E. and Watkins, P. J. (1985) Osteopenia and metatarsal fractures in diabetic neuropathy. Diabetic Medicine, 2, 461.
6. Edmonds, M. E. and Watkins, P. J. (1982) Blood flow in the diabetic neuropathic foot. Diabetologia, 22, 9.
7. Edelman, S. V., Kosofsky, E. M., Paul, R. A. and Kozak, G. P. (1987) Neuroarthropathy (Charcot's joint) in diabetes mellitus following revascularisation surgery. Archives of Internal Medicine, 147, 1504.
8. Boulton, A. J. M., Betts, R. P., Franks, C. I. et al. (1987) Abnormalities of foot pressure in early diabetic neuropathy. Diabetic Medicine, 4, 225.
9. Mooney, V. and Wagner, F. W. Jr (1977) Neurocirculatory disorders of the foot. Clinical Orthopaedics and Related Research, 122, 53.
10. Edmonds, M. E. and Watkins, P. J. (1987) Management of the diabetic foot. In Diabetic neuropathy (ed. P. J. Dyck, P. K. Thomas, A. K. Asbury et al.), p 208. Philadelphia: Saunders.

Fig. 4. Healing spontaneous transverse fracture of the left fibula.