Charcot Joints in Diabetes Mellitus

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LESIONS of the feet in patients with diabetes mellitus are generally ascribed to occlusive vascular disease or sepsis and the importance of peripheral nerve disease as a causal factor is insufficiently recognized. Painless neuropathic ulcers and Charcot joints, although often stated to be common only in tabes dorsalis and syringomyelia, are by no means uncommon in diabetes.

Yet it was only recently (Jordan, 1936) that Charcot joints were recognized in connexion with diabetic peripheral nerve disease and as late as 1951 Parsons and Norton claimed that fewer than 22 cases of diabetic arthropathy had been recorded in the world literature.

In the course of a recent investigation at King's College Hospital 12 cases were discovered with bone and joint lesions closely resembling Charcot joints, which leads one to suspect that diabetic arthropathies may be more common than the rarity of reports would suggest.

Cases.—The age and sex incidence of the present cases are shown in Table I. In 4 cases there was

TABLE I.—THE AGE AND SEX DISTRIBUTION OF THE 12 PATIENTS WITH DIABETIC NEUROPATHIC JOINT LESIONS

	Sex		
Age group	M.	F.	Total
20–30			
30–40	2	1	3
40–50	3		3
50–60	1	1	2
60–70	1	3	4
Total	7	5	12

involvement of the interphalangeal or metatarsophalangeal joints, in one of them of both feet (Fig. 1). In 3 cases radiographs of the feet showed attenuation of the metatarsal shafts which tapered distally, absorption of the metatarsal heads and deformities of the foreparts of the feet (Fig. 2). In 5 cases the midtarsal joints were affected giving rise in each case to a deformity consisting of eversion and external rotation of the forepart of the foot and flattening of the longitudinal arches (Fig. 3). The radiological appearances were those of a hypertrophic arthropathy with considerable destruction of

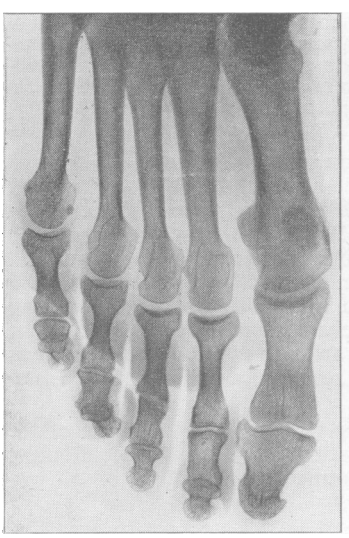

FIG. 1.—The proximal interphalangeal joint of the left 4th toe and the right 3rd toe shows an arthropathy with disorganization of the joint, absorption of subarticular bone, but without any evidence of infection or bone resorption.

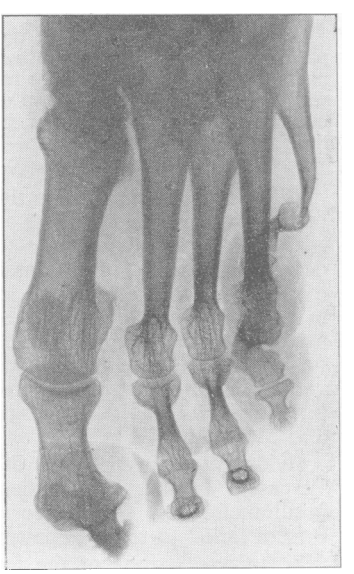

FIG. 2.—Radiograph showing attenuation of the shaft of the left 5th metatarsal bone with absorption of the head and resulting deformity of the little toe.

subarticular bone and excessive new bone formation in the surrounding soft tissues but without periosteal reaction or evidence of bone absorption (Fig. 4).

In the patients with lesions of the interphalangeal or metatarsophalangeal joints the arthropathies were often discovered only after X-ray examination. In the cases with arthropathies of the midtarsal articulation attention was usually drawn to the condition by the presence of deformity.

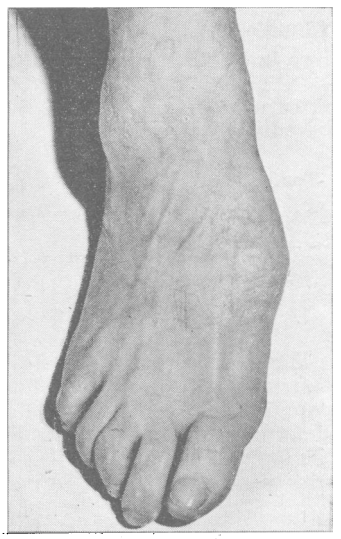

FIG. 3.—Photograph of the right foot of a patient with diabetic neuropathy and arthropathy. The prominence of the navicular bone with thickening in the midtarsal region and eversion and lateral rotation of the forepart of the foot are well shown.

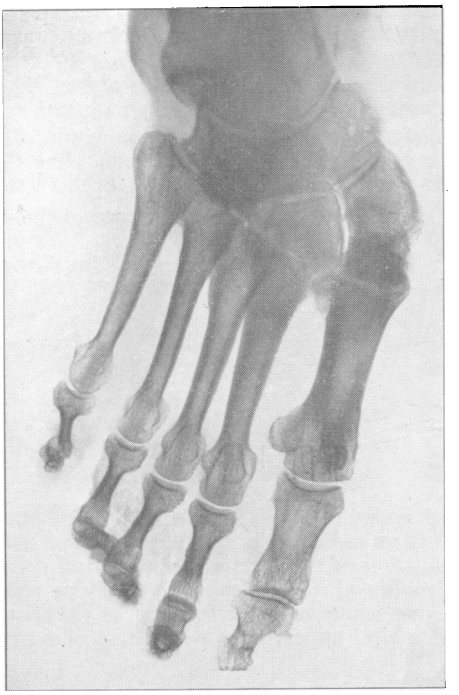

FIG. 4.—Radiograph of the right foot showing deformity of the tarsus with proximal displacement of the lateral metatarsals. The medial three tarsometatarsal joints show a severe arthropathy with considerable destruction of subarticular bone. There is no evidence of osteoporosis.

In most of the cases, as in those reported in the literature, the lesion was of slow onset and gradual progression over a period of some years. However, in one case the arthropathy developed rapidly with sudden swelling of the foot, local redness and a temperature in the absence of infection as occasionally occurs in the classical arthropathy of Charcot (Wilson, 1940). The gross disorganization of the joint with œdema and local heat raised the possibility of an infective condition being, at least in part, responsible for the extensive bone destruction. However, the erythrocyte sedimentation rate was normal and joint fluid obtained by aspiration proved sterile both on direct examination and on culture. As the patient had at no time been given antibiotic or chemotherapy it was felt that the above laboratory findings were strong evidence against an infective arthritis which, in any case, was not supported by the radiological appearances. A similar case has recently been reported by Lister and Maudsley (1951).

Evidence of the presence of occlusive peripheral vascular disease was found in only one of the 12 cases. In fact most patients had strong pedal pulses and vascular studies with the Boulitte oscillometer and skin-temperature measurements following the intravenous injection of 50 mg. Priscol failed to show impairment in the peripheral circulation.

Soft-tissue lesions of the feet were present in 6 patients at the time the arthropathy was first diagnosed, but were generally neither in close proximity to the involved joint nor associated with deep-seated infection. 5 patients gave a history of perforating ulcers or septic lesions about their feet, which had, however, preceded the development of the joint condition often by as much as five years. In 3 of these cases the affected joints were known to have been normal radiologically at the time the patients were treated for their ulceration. In 2 cases joint changes were discovered in both feet, although skin lesions were or had been present in only one foot.

Pathological evidence of the presence of nerve disease was obtained by means of nerve biopsies in 4 cases with bone lesions included in the present series. The histological sections showed varying degrees of myelin degeneration and of axis-cylinder degeneration without, however, any sign of endo- or peri-neural fibrosis or degenerative disease of the vasa nervorum.

Discussion

Bailey and Root (1942) collected 14 cases with degenerative bone lesions in diabetics and stated that the condition was not dependent on deficient blood supply. In 1947 the same authors reported painless destruction of the tarsus in 17 cases with chronic poorly controlled diabetes and thought that the bone destruction was the result of diabetic neuropathy and similar to the arthropathies of neurosyphilis, syringomyelia, nerve injuries and the neural form of leprosy.

In all cases in whom arthropathies were discovered in the present study, there was good evidence of neglected diabetic control and of the presence of a nerve disorder. The most characteristic feature was the absence of pain throughout the development of the joint changes. Diabetic neuropathy affects mainly the lower limbs at their periphery and with distal involvement of the nerves it was to be expected that the joint changes would be most common in the peripheral joints. A diabetic neuropathic arthropathy of the knee has been reported (De Takats, 1945; Spear, 1947; Shore, 1947), but appears to be very rare. Involvement of the ankle-joint seems to be more common (Foster and Bassett, 1947; Muri, 1949; Knuttson, 1951; and others) but neither was met with in the present series.

As regards the pathogenesis of the neuropathic joint lesions various theories have been proposed but most of them are open to criticism.

The frequent association of perforating ulcers and neuropathic joints has often led to sepsis being thought responsible for the bone changes. Hodgson, Pugh and Young (1948) expressed the opinion that infection leading to osteomyelitis was the direct cause of the bone change independent of any associated nervous disorder. The denial that a disturbance of nerve supply was responsible for the bone as well as the soft-tissue lesions does not seem well-founded particularly as there is abundant evidence that damage to the nervous system can and does produce such lesions. The soft tissue and bone lesions occur independently and are undoubtedly secondary to the peripheral nerve disorder of diabetes mellitus with loss of pain sensibility.

Foster and Bassett (1947) thought that they had obtained support for Charcot's original theory (Charcot, 1868) that neuropathic joints resulted from lesions of the autonomic nerves. They carried out investigations into the functional integrity of the autonomic nervous system in their two cases and found impairment or loss of sympathetic activity in both of them. They quoted Dreyfus and Zacharovitch (1937) and Wartemberg (1938) as having produced evidence of autonomic nerve damage in the cases of neuropathic joint lesions of syringomyelia and tabes dorsalis, and expressed the opinion that the association of loss of proprioceptive perception and autonomic nerve dysfunction predisposed to the development of a Charcot joint.

In the course of an investigation into diabetic neuropathy (Martin, 1952) evidence of autonomic dysfunction was discovered in all the cases of diabetic nerve disease with or without joint changes, which leaves little support for the hypothesis that such nerve damage *per se* predisposes to the development of the arthropathy. Furthermore evidence has been obtained that autonomic nerve disturbance is not essential to the development of a Charcot joint. Tests of vasomotor function in cases of tabes dorsalis have shown that joint lesions may be present in this condition with intact vasomotor nerves.

Eloesser (1917) produced what appeared to be Charcot joints experimentally in animals by subjecting joints to operative trauma after they had previously been rendered anæsthetic by resection of the appropriate posterior nerve roots. McMurray (1950) published the report of a case who from birth had shown marked insensitivity to pain, in whom a deformity was present of the left ankle suggesting a Charcot joint.

Neither autonomic nerve damage, ischæmia, nor sepsis can explain adequately the pathogenesis of a neuropathic arthropathy. Diabetic neuropathy shows a predilection for non-myelinated nerve fibres (Martin, 1952) which causes pain fibres to be involved early. It appears that the "mechanical theory", postulating that loss of afferent impulses allows minor trauma to damage the joints, has most to recommend it. The presence of histological evidence of nerve degeneration in all the 4 cases with diabetic arthropathy in whom nerve biopsies were obtained and the absence of Charcot joints in patients without diabetic neuropathy in the diabetic population attending King's College Hospital support the belief, that these bone changes are truly neuropathic and develop only in the presence of nerve disease and continued weightbearing.

Whilst the prognosis for the neuropathic soft-tissue lesion is usually good provided it is recognized early and treated, the prognosis of the neuropathic arthropathy appears to be bad and none of the cases has shown any tendency to improve. However, not all cases have continued to advance, in fact most of them have become arrested with no demonstrable change in the radiological appearances over the past few years.

Recently Parsons and Norton (1951) suggested that sympathectomy may be of benefit in treatment.

However, in view of the fact that the sympathetic fibres are already damaged in cases of diabetic neuropathy (Martin, 1952) it is difficult to see why surgical sympathectomy should prevent progress of the bony lesions which in any case tend to become arrested with treatment of the neuropathy.

As suggested by Marble (1948) the only treatment which proved helpful in progressive cases was the use of orthopædic appliances which prevented weightbearing and further deformity. Successful treatment depends on early diagnosis before gross deformity has developed and for that reason all cases of diabetic neuropathy and particularly those with soft tissue lesions should have their feet X-rayed routinely as otherwise only the late examples come to light and the early ones which are the most amenable to treatment will be missed.

Both neuropathic skin and joint lesions are the result of the underlying nerve disorder consequent upon poor diabetic control, and it is therefore most important that attention be directed towards treatment. Unfortunately there is, as yet, no specific cure for this complication of diabetes mellitus beyond rigid control of the metabolic disorder. With adequate diabetic treatment, however, recovery may be expected in about 60% of cases within six months to two years, unless the neuropathy has been of long standing. With recovery of the nerve disorder the progress of the bone changes appears to be halted.

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