NEUROPATHIC JOINTS IN DIABETES

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It is well recognised that neuropathy is common in diabetes mellitus, not only in the elderly, but also in younger patients with poorly controlled diabetes of long duration. It is not so clearly recognised that some patients with advanced lesions will develop neuropathic changes in the joints. Jordan (1936) seems to have been the first to have recognised this, and Bailey and Root (1947) found seventeen such patients in 20,000 diabetics. Out of 150 patients with diabetic neuropathy Martin (1953) discovered nine with "Charcot" joints.

Usually affecting the tarso-metatarsal joints, neuropathic joint changes in diabetes have been described in the ankle and knee (Shore 1947, Spear 1947). In 1958 Jacobs, reporting three new patients, estimated that only forty-five patients with this condition had been recorded in the world literature up to 1956. We are reporting here two further cases.

CASE REPORTS

Case 1—A man aged thirty-five, a post-office engineer, developed diabetes while in the Army in 1945. Although he had unstable periods with intercurrent infection, for many years his daily requirements had been 40 units of protamin zinc insulin and 20 units of soluble insulin, on a diet containing 160 grammes of carbohydrate. In 1957 the diabetes became unstable and on three occasions he was admitted to hospital with threatened coma. Insulin zinc suspension was tried but found to be of no help.

In April 1958 he injured the little toe of the right foot with a spade, with bruising but no pain. A few days later the skin broke, the toe became infected and he was admitted to hospital. A radiograph showed only subluxation of the middle phalanx of the little toe. Gangrene occurred and part of the toe was lost, but healing was satisfactory. The foot retained a good circulation and the blood pressure was 155/95 millimetres of mercury. He
was in hospital for two months and in bed for most of the time. Two weeks after his discharge a diffuse painless swelling of the right foot was first noticed (Fig. 1), with no inflammatory reaction; the radiographs showed destruction of the tarso-metatarsal joints of the right foot and new bone formation (Fig. 2). The knee and ankle jerks were absent on both sides and the plantar responses flexor. Vibration sense was absent on the right side below the knee, and diminished on the left. Position sense was normal but there was a patchy loss of pain and touch sensation. The fundi showed moderately advanced diabetic retinitis. The urine contained no albumin but usually one-quarter to one-half per cent of sugar; if the latter was not present he tended to have hypoglycaemic symptoms. The Wassermann reaction and Kahn test were negative.

Remaining fairly well controlled on 16 units of protamin zinc insulin and 44 units of soluble insulin, the patient has shown a gradual clinical and radiological improvement in the foot (Fig. 3).

Case 2—A married woman, aged seventy-five, and known to have had diabetes for nine years, was usually well controlled on a low carbohydrate diet only. She was admitted to hospital in 1958 for swelling of the left leg below the knee which had started in the foot over seven weeks before. A year previously she had had a feeling as if the arch of the foot had given way. The ankle and knee reflexes were normal; the plantar responses were flexor. The vibration sense was absent, and pain and touch were diminished from below the knee and position sense was normal. She had varicose veins in the left thigh. Radiographs showed some erosion of the tarso-metatarsal joints with loss of joint space in the left foot and extensive periosteal new bone formation around the shaft of the first metatarsal (Fig. 4). A biopsy of bone showed an irregular absorption and formation of bone and disorganisation of the articular cartilage, but no inflammatory changes were seen. The fasting blood sugar was 198 milligrams per cent. The Wassermann reaction and Kahn test were negative; the blood uric acid, blood urea, serum calcium and alkaline phosphatase were normal. The diabetes was controlled on a diet containing 160 grammes of carbohydrate and Tolbutamide 1 tablet twice daily, with a steady clinical and radiographic improvement of the foot.

DISCUSSION

These two patients are reported to draw attention to this rare complication of diabetes. Osteomyelitis, tuberculosis and tumour must be considered in the differential diagnosis; in 1951 Lister and Maudsley described a patient with this complication which was treated for some time as tuberculosis, and a biopsy was done before the correct diagnosis was made.

We believe that the prognosis of neuropathic changes in joints is better in patients with diabetes than in those with tabes dorsalis or syringomyelia, and that the earlier the diagnosis is made and the appropriate treatment given the better the chance of improvement in the affected joints.

Eloesser in 1917 showed that repeated small injuries are necessary in a desensitised joint to produce neuropathic changes. In the patients here reported there was definite diabetic neuropathy as shown by absent knee and ankle jerks, loss of vibration sense and diminution of pain and touch sensation.

Foster and Bassett (1947) placed the neurological lesion as "predominantly extramedullary, with the major involvement in the posterior root ganglia and the sensory components of the
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Peripheral nerves with relative sparing of the ventral roots and motor fibres of the peripheral nerves."

All reports agree that the small joints of the feet, especially the tarso-metatarsal and metatarso-phalangeal, are most commonly involved, and occasionally the ankle or knee. Perhaps this is because slight often-repeated injury is more likely to affect the small joints of the foot, with their comparatively weak capsule and ligaments, than the larger and stronger joints of the leg. Vascular insufficiency appears to play no part.

The first sign is a painless swelling of the foot, without heat or redness. This occurred in all seventeen patients recorded by Bailey and Root (1947). The radiographic changes include erosion at the joint surfaces, destruction of the joint spaces and extra-articular new bone formation. It is interesting that these joint changes were reversed in Case 1, with some improvement; Jacobs (1958) reported a similar finding in one of his patients. It is possible that in diabetes the neuropathy is reversible if nerve changes have not progressed too far. The most recent radiographs of our patients (May 1959) show improvement continuing, with absorption of extra-articular calcification and improved definition of the joint surfaces.

Treatment consists in stabilisation of the diabetes, physiotherapy—particularly faradism—and supporting bandages or stockings and surgical footwear.

SUMMARY

Two cases of neuropathic joints in diabetes mellitus are described. The condition, though rare, must be kept in mind in diabetes with neuropathy. With proper control of the diabetes and supportive measures to the joints the prognosis is relatively good.

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REFERENCES


