

NEURO OSTEOARTHROPATHY (CHARCOT'S JOINT) IN A YOUNG DIABETIC

Pages with reference to book, From 225 To 227

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Neuro-osteoarthropathy (Charcot's Joint) in diabetic patients has been well described in literature¹⁻⁹. It has been defined as a chronic progressive degenerative process principally occurring in weight-bearing joints in the feet of diabetic patients who have underlying peripheral neuropathy and loss of sensation^{6,9,10}. Although considered as a clinical rarity^{3,6}, it has been reported more frequently in recent years^{7-9,11,12}. This observation can be attributed to the longevity of diabetic patients and dramatic decrease in mortality since the insulin era⁷.

The association between juvenile onset diabetes and skeletal lesions is relatively uncommon^{12,13}. We report a case of young woman who had juvenile onset diabetes and developed bilateral neuro-arthropathy involving feet.

Case Report

A 23 year old female, known diabetic for 8 years and on insulin therapy was admitted in January, 1990 with uncontrolled diabetes and severe hypertension. She complained of swelling of both feet for the past 2 years but denied having sustained any trauma to them. Clinical examination revealed a blood pressure of 210/130 mm Hg and fundal changes consistent with diabetes and hypertension. Both feet appeared swollen with slight tenderness. Neurological examination revealed decreased sensation to pinprick and bilaterally impaired sensation of position and vibration. Her ankle and knee jerks were absent on both sides. Pulses were palpable in both femoral and popliteal arteries. Dorsalis pedis and posttibial appeared normal. Investigations revealed an elevated blood urea (114 mg %) and creatinine (2.6 mg %). Radiographs of feet showed slight flattening of calcaneus and a crack in the bony bridge between calcaneus and navicular bone on the left side, while on the right side there was beginning of disorganization of the subtalar joint with loss of depth of the body of talus. (Figures 1,2).

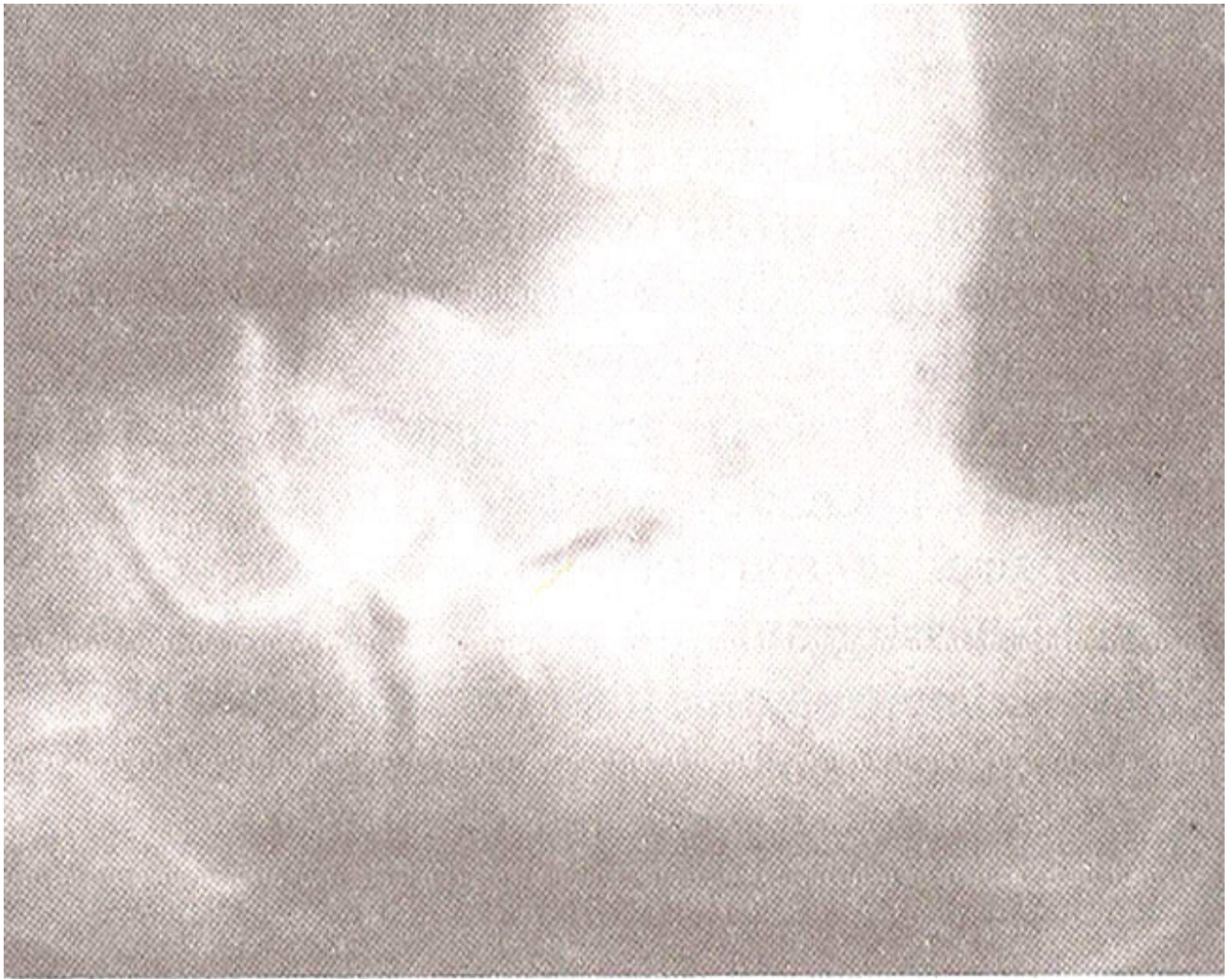


Figure 1. Right foot showing changes of charcot's joint.

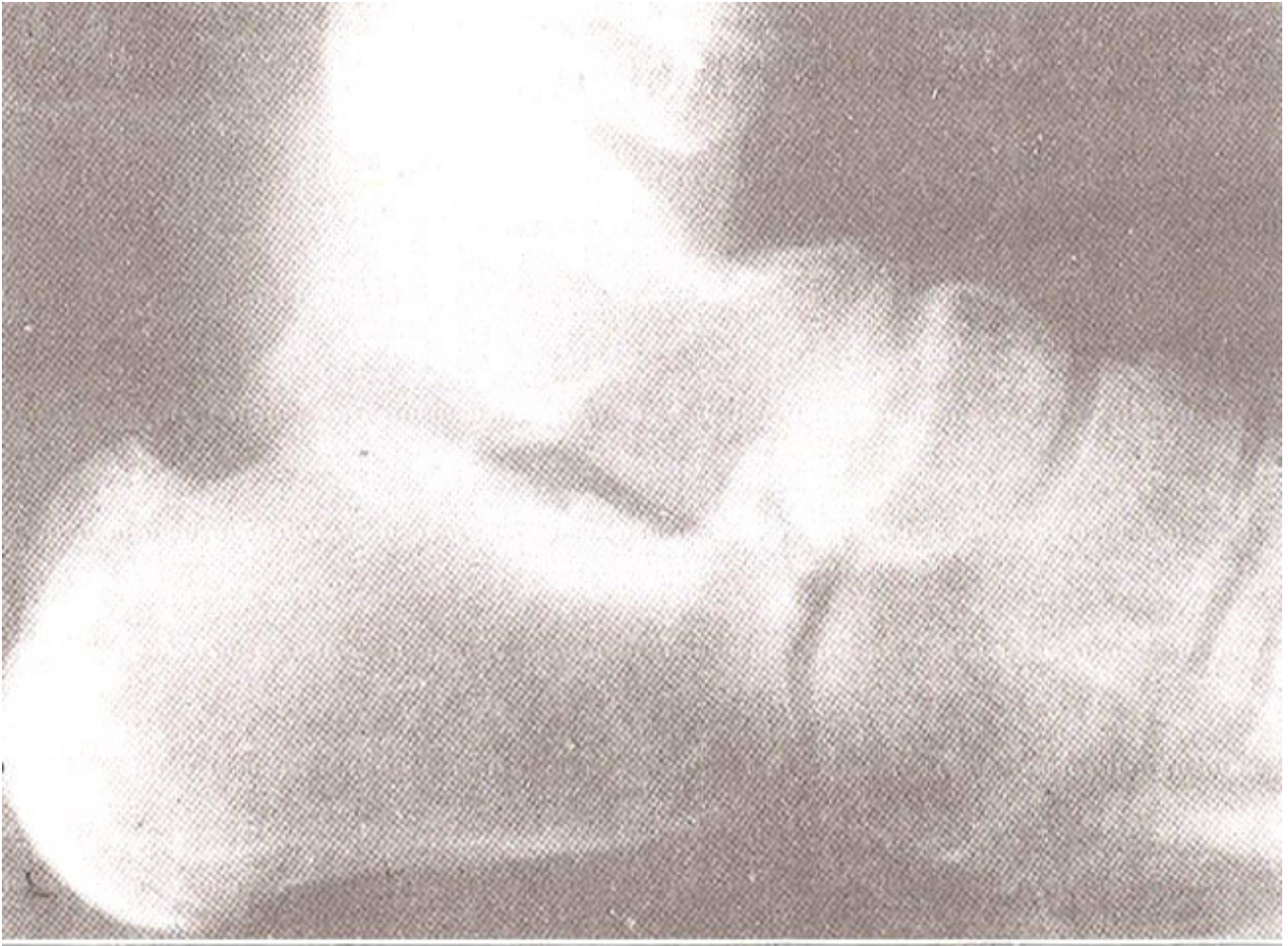


Figure 2. Left foot showing charcot's type changes.

Her diabetes was controlled on insulin and blood pressure reduced and maintained on medication. For her feet she was instructed complete bed rest, non-weight bearing and special protective shoes. She was discharged after control of her blood pressure and was advised periodic follow-up but she did not report back and died 3 months later of renal failure.

DISCUSSION

Since Charcot's initial report of neuropathic joints in association with tabes^{1,4,8,10,12,14}, a multitude of underlying causes have been ascribed to the development of this lesion, the predominant being syringomyelia, leprosy, spina bifida, cord trauma, multiple sclerosis and spinal dysraphism^{1,2,4,7-9,15-16}. Diabetes mellitus, however, remains the commonest cause^{5,16} and its associated bone and joint abnormalities are now well recognized^{2,3,5,7,10,12,13}.

The reported incidence varies between 0.1 to 6.8%^{5,8,18-20}. Majority of the cases are of adult onset diabetes often non-insulin dependent^{13,14,16} lesions occurring over the age of fifty^{5,6,13,14,16} after the patient has had diabetes for an average of 13 to 15 years^{6,13}. The blood sugar levels are often poorly controlled^{5,6,8} although osteopathy may even precede overt diabetes⁵. While the high risk group constitutes those with characteristic triopathy^{6,12}, the diabetics with renal transplants are at greatest risk^{12,21}. More recently an association between joint lesions and pulmonary changes in diabetics has

been described²²⁻²⁴. Therefore, patients with impaired respiratory function can constitute another high risk group.

Typical neuropathic joints in diabetics can be seen in any part^{16,25-27} but the foot remains the area most frequently involved¹⁴. The sites most often affected are the tarsometatarsal joints (60%) and to a lesser extent the joints of metatarso-phalangeal area (30%) and ankle (10%)^{6,9,18}. A clear distinction has been made between proximal foot destruction and distal foot erosion^{7,10}. On roentgenogram two forms exist - ATROPHIC and HYPERTROPHIC, the latter being described as osteophytic fragmentation, joint space narrowing, capsular distention and lipping that predominantly affects the lower extremities. The atrophic form commonly affects the upper extremities and the only change is resorption of bone ends^{1,2,6,9,16}. Transition from one form to the other does not occur^{6,16}. Extensive periarticular calcification is characteristic^{28,29} occasionally bone shards may be seen far from the joint suggesting their denovo origin from the joint capsule²⁹.

The pathophysiology of Charcot's joint remains controversial, In attempts to elucidate pathogenesis various mechanisms were described pertaining to trophic disturbances, trauma^{1,5,6,8} autonomic impairment³⁰, infection³¹, ischaemia³², bone atrophy³³, Osteoporosis^{5,6}, small vessel disease³⁴, and to an error of protein metabolism³. However no explanation was based on conclusive evidence that focussed on any single process. Current concepts suggest that osteolysis and bone resorption play a major role in the development of Charcot's joints³³⁻³⁶, and for this an adequate blood supply is a prerequisite^{37,38}.

The clinical finding of bounding pulses³⁹⁻⁴¹ and the development of Charcot's joint in ischaemic limbs following revascularization surgery⁹ strongly support this hypothesis. Edelman et al⁹ have suggested that while peripheral neuropathy is the initial primary defect, a neurally initiated vascular reflex leading to increased blood flow may play a pivotal role in the development of Charcot's joint. Mechanical trauma in an insensitive foot is contributory but a secondary mechanism⁹,

Whatever the mechanism, a marked disproportion between the radiologic severity of the disease process and the relatively slight complaint of discomfort is the most consistent clinical feature. Often there is no history of trauma and, when inflicted, may be trivial. This explains why the lesions remain unrecognized for long and the changes are far advanced when first presented¹⁸.

Our patient clinically had a classical Charcot's joint. She had severe clinical manifestations of diabetes and represented the high risk group. The lesion developed without known or obvious trauma and at the time of presentation her blood sugar levels were poorly controlled. Nevertheless the case is unusual in several aspects. The patient was rather young and had juvenile onset insulin dependent diabetes which is relatively uncommon in patients developing neuropathic joints. Moreover, the lesion appeared rather early. Harris & Brand¹⁰, observed five patterns of disintegration in the anaesthetic foot and our case resembled type II they described as central destruction which is infrequently encountered, as it is not common for talus to be the primary focus of disintegration. The pathogenetic factors in our case remain speculative. Where- as the destruction of talus seems to be entirely due to mechanical stress¹⁰, the presence of unimpaired peripheral circulation favours the hypothesis of osteolysis and bone resorption⁹. Probably both these factors in a neuropathic limb favour the development of Charcot's joint.

Diabetic neuro- osteoarthropathy has a characteristic intermittent course of activity⁵. Trauma unquestionably causes deterioration^{5,6}. Current treatment protocol, therefore, includes non- weight-bearing and immobilization of the involved extremity and prophylactic immobilization of contralateral extremity. Control of blood sugar levels is desirable and serial, roentgenograms are advised to monitor progress^{6,8,9,11,12}. In absence of marked bony deformities the long term prognosis is generally favourable⁶.

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