

A Case of Type 2 Diabetes Presenting with Charcot's Joint at Diagnosis

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Neuropathic joints or Charcot joints are caused by loss of sensation in the joint to the extent that they are damaged and disrupted. The damage and disruption is often not grave and early diagnosis on clinical examination and X-ray is often missed since the joints, although swollen are painless. We present a case of a middle aged man presenting with Charcot joints caused by type 2 diabetes.

Introduction

Patients typically present insidiously or are identified incidentally or as a result of investigation for deformity. Unlike septic arthritis, Charcot joints although swollen are normal temperature without elevated inflammatory markers. Importantly they are painless.

The Case

History

A 51 year old male patient presented with gradual swelling and redness of the left foot for the last 3 months. The swelling was not posture dependent. There was no history of fever, pain or any major trauma on the left foot. He did not have a positive history of type 2 diabetes mellitus or tingling and numbness of both feet.

On Examination

On examination, his left ankle and foot was swollen with overlying skin erythematous but without any ulceration. There were multiple areas of bony swelling over the tarsometatarsal and metatarsophalangeal joints with features of synovial effusion. Painless bony crepitus was also felt on palpation over the metatarsophalangeal joints with maximum crepitus over the left 5th metatarsophalangeal joint. Most of the tarso- metatarsal and metatarsophalangeal joints were grossly disorganized, unstable but movement of the

left ankle joint was preserved in most directions with very little pain in passive movement.

Dorsalis pedis and posterior tibial artery pulses were normally palpable on the left side. On neurological examination, his joint position sense and vibration sense was reduced bilaterally in both feet with mildly decreased pain and light touch sensation. His power was normal (grade 5) in lower limbs and upper limbs both proximally and distally. His planter reflex was bilaterally normal and only his ankle jerks were diminished. He had no retinopathy or macular edema.

Investigations

Complete blood count including total and differential counts were normal with normal hemoglobin and ESR of 15. HbA1c was 8.6% with fasting plasma glucose of 178 mg/dL and post prandial plasma glucose of 280 mg/dL. His urea and creatinine were normal; blood tests for syphilis were normal as was vitamin B12. His urine MACR was raised to 54 µgm/gm.

The X-ray of the left ankle joint revealed grossly disorganized and misaligned tarsal bone, bony swelling of the phalangeal ends of the metatarsal bones (especially the 2nd and 3rd metatarsophalangeal joints), fracture of the 5th metatarsal joint and dislocation of the 5th metatarsophalangeal joint with distinct joint margins.



Photograph showing the foot (Left) and the Xray of the foot (Right)

A venous duplex scan excluded deep venous thrombosis. Nerve conduction test showed mixed sensorimotor (predominantly sensory) neuropathy- axonal and demyelinating.

Final Diagnosis

Based on the clinical features and investigations the patient was diagnosed as a case of Left Charcot's (neuropathic) Joints predominantly involving the tarsal and metatarsal bones, tarsometatarsal & metatarsophalangeal joints at the stage of initial diagnosis of Type 2 Diabetes.

Discussion

Diabetes mellitus is the commonest cause of neuropathic joint disease. The term Charcot's joint is used interchangeably with neuropathic joints. Other rarer causes of Charcot's joint are syringomyelia, leprosy, Charcot -Marie-Tooth disease, amyloidosis etc. In syringomyelia mostly the upper limb joints like glenohumeral, elbow or wrist are affected while in diabetes mellitus lower limb joints like tarsal or tarsometatarsal joints are affected. The Charcot foot commonly goes unrecognized, particularly in the acute phase, until severe complications occur.¹ The incidence of neuropathic joint in diabetes is around 0.5%, with nearly 10% of patients with diabetic neuropathy had Charcot foot.²

Charcot's foot is a condition of acute or gradual onset and, in its most severe form, causes significant disruption of the bony architecture of the foot. The acute Charcot foot is usually painless and may mimic cellulitis or deep venous thrombosis.¹ Marked swelling of the foot and lower leg is considered an isolated finding, mimicking deep venous thrombosis but prominent swelling of feet is more common in Charcot process than in cases of deep venous thrombosis.³

It often results in foot deformities and causes abnormal pressure distribution on the plantar surface, foot ulcers and, in some cases, requires amputation. The exact pathogenesis is unknown, but underlying sensory neuropathy and arterio-venous shunting due to autonomic neuropathy is

also thought to play a role. Repeated unrecognized micro-trauma or an identifiable injury may be the inciting factors of Charcot foot. In more than 50% of patients with Charcot foot there is no precipitating event such as a slip or a trip.

Clinical findings in patients with an acute Charcot process include warmth, erythema and swelling.³ Our patient had clinical findings of an acute Charcot foot with warmth, erythema and swelling with minimal pain or tenderness. Patients with Charcot foot may have some pain probably because the sensory loss is not complete, the presence of pain does not exclude the diagnosis. However such pain is always much less than would be expected for the severity of the clinical and/or radiographic findings. While cellulitis should be considered in any patient with diabetes, missing the diagnosis of Charcot foot can be disastrous since failure to initiate proper treatment of the Charcot foot exacerbates the problem. Inappropriate treatment with antimicrobial therapy and even incision and drainage can lead to unnecessary complications.

Radiographic study in neuropathic joints reveals reduced joint spaces, subchondral sclerosis, joint effusion, osteophyte, grossly destroyed joints and hypertrophic changes.² Fractures, extra-articular bone formations, bone resorption are noted in advanced cases. A close differential diagnosis is osteomyelitis but in osteomyelitis joint margins are indistinct and Indium¹¹¹ bone scan shows an increased uptake.

Although the initial radiograph may be normal, making diagnosis is difficult, immediate detection and immobilization of the foot are essential in the management of the Charcot foot. A lifelong program of patient education, protective footwear and routine foot care is required to prevent complications such as foot ulceration. Although initially described in patients with tertiary syphilis, the Charcot foot is now seen mostly in patients with diabetes mellitus. Treatment of Charcot's joint is mostly unsatisfactory. Primary focus of treatment is stabilization of the joint, off-loading and bisphosphonates. This patient was treated with off-loading and bisphosphonates.

Long standing diabetes mellitus is the commonest cause of neuropathic joint disease. Our patient presented with Charcot foot at diagnosis of diabetes probably because initial stages of type 2 diabetes are asymptomatic and may go unrecognized for long.

References

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