



CASE REPORT CHARCOT,S FOOT IN TYPE 2 DIABETES MELLITUS

Waseem Raja*¹, MD. Rohey Jan² MD and M.H. Bhat³ MD, DM

Department of Medicine, Government Medical College Srinagar, Jammu & Kashmir- India.

*Corresponding Author: Waseem Raja

Department of Medicine, Government Medical College Srinagar, Jammu & Kashmir- India.

Article Received on 07/10/2016

Article Revised on 27/10/2016

Article Accepted on 17/11/2016

ABSTRACT

Charcot's foot, is an infrequent complication in patients with diabetes mellitus and severe neuropathy. We report a case of a 48-year-old diabetic female who presented with a painless, erythematous and swollen right foot. Plain radiographs of the right foot showed subluxations and bony fragments in the tarsal-metatarsal and metatarsal-phalangeal joints. The nerve conduction velocity test confirmed the presence of sensory neuropathy in both lower extremities. Magnetic resonance imaging of the right foot demonstrated destructive osseous debris and bony fragments with marked marrow edema. Charcot's foot was diagnosed and treated with a total-contact cast and bisphosphonate therapy. Charcot's foot should be suspected in any long-standing-neuropathic diabetic patient with a warm swollen foot without local or systemic signs of infection. Early recognition of Charcot's foot and immobilization with a total-contact cast can minimize potential foot deformity, ulceration and loss of function.

KEYWORDS: Charcot's foot, Diabetes mellitus, Neuropathic osteoarthropathy, Neuropathy Registrar, Resident, senior consultant.

INTRODUCTION

Neuropathic osteoarthropathy is defined as bony and joint defects that occur secondary to impaired sensation in a variety of disorders. In 1868, Jean-Martin Charcot described the neuropathic aspect of this disease for the first time; hence, the condition was named after him.^[1] At that time, syphilis was the most common cause of neuropathic osteoarthropathy. Since Jordan first reported a case of Charcot's foot which resulted from diabetes mellitus (DM) in 1936, DM has become the most common etiology of neuropathic osteoarthropathy today.^[2] Herein, we report a case of Charcot's foot in a diabetic patient with sensory neuropathy. The diagnosis and management of Charcot's foot are discussed herewith.

CASE REPORT

A 50 year old woman with a history of poorly controlled diabetes mellitus of 15 years duration presented to an orthopedic surgeon with swelling and pain of the right ankle of 1 year duration after a trivial trauma. She had no other joint involvement or any other systemic symptoms. A probable diagnosis of tuberculous synovitis was made and she received anti tuberculous therapy without any benefit. Later, she was referred to our hospital. A fine needle aspiration from the ankle joint revealed non-specific inflammation so anti tuberculous treatment was discontinued. On examination she had marked pallor and pedal edema. Her pulse was 90 per minute and BP 140/90 mmHg. All the peripheral pulses were well felt.

Neurologic examination revealed absent deep tendon reflexes, with impaired touch and vibration sense in both the lower limbs. The right ankle was swollen, painless on manipulation with presence of bony crepitus. Optic fundii showed proliferative diabetic retinopathy. Investigation revealed haemoglobin of 9.2 grams per dl, total leucocyte count of 7.6 per cumm and ESR was 21 mm in first hour(N=0 to 15). Serum biochemistry revealed urea of 44 mg/dl, creatinine of 2.1 mg/dl, mean plasma glucose of 146 mg/dl and HbA1c of 9.2 percent, calcium of 8.6 mg/dl and phosphorus 4.2 mg/dl. Nerve conduction studies showed sensorimotor axonal neuropathy in bilateral lower limbs. A plain radiograph and computed tomography scan of the ankle showed loss of joint space and sub-chondral sclerosis. 99mTc-MDP bone scan demonstrated increased tracer uptake in the region of right ankle and tarsal joints. MRI of the ankle showed fragmentation of the navicular, cuboid and uniform bones with significant debris around the inter-tarsal joints. Dislocation at the inter-tarsal and tarso-metatarsal joints, synovial thickening with effusion around the ankle mortise and mid-tarsal region. Tenosynovitis of tibialis posterior, flexor digitorum longus and flexor hallucis longus. Thickening of the planter fascia with edematous changes deep to it. Marked edematous changes noted in the skin and sub-cutaneous tissue in the distal leg, ankle and foot. The clinical presentation and sequence of events with characteristic imaging features in this case suggested a diagnosis of charcot neuroarthropathy of the right ankle. She received

2 milligrams of zoledronic acid and was planned for realignment arthrodesis on follow up.



Fig. 1: Plain radiograph demonstrating bony fragments in the navicular-cuneiform joint (black arrows), the cuneiform-second metatarsal joint (black arrow head) and the second metatarsal-phalangeal joint (white arrow) of the right foot

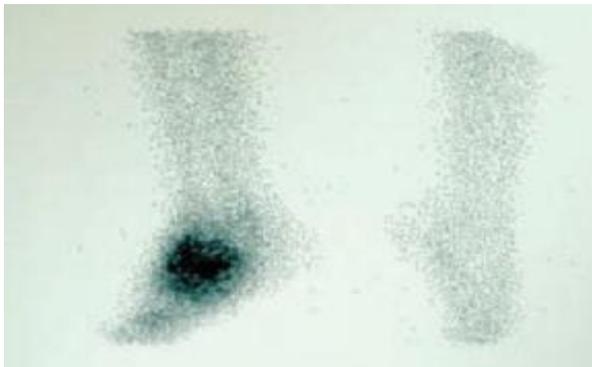


Fig. 2: Three phase bone scan (Tc-99m MDP) revealing focally intense tracer perfusion of the right foot

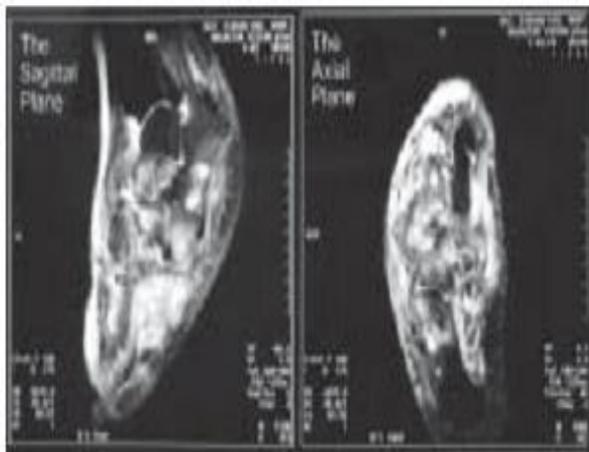


Fig 3: MRI of the right foot revealing fragmentation of navicular, cuboid and cuneiform bones with significant debris around intertarsal joints. Dislocation at intertarsal and tarso-metatarsal joints, synovial thickening with effusion also noted and tenosynovitis of tibialis posterior and FDL were also noted.

DISCUSSION

Neuropathic arthropathy should be considered in patients with neuropathy, even in those with a minor increase of heat and swelling of the foot or ankle, especially after an injury. Careful history-taking and simple physical examinations can help diagnose this condition early. Retinopathy, nephropathy and a previous foot ulcer in the history are predictive risk factors.^[3] Physical examinations, especially neurological findings of vibratory sensation, deep tendon reflexes and the 5.07 (10 g) Semmes-Weinstein monofilament test, are also correlative for the development of Charcot's foot.^[3] The prevalence of neuropathic arthropathy is 1%-2.5% of people with diabetes and up to 0.8%-9% of diabetic patients with neuropathy.^[4-7] It is most common in people with type 1 diabetes in the fifth and sixth decades of life, but can also occur in younger patients and in type 2 diabetic patients. Usually, the duration of diabetes is greater than 12 years.^[8] It attacks unilateral limbs in most cases, but bilateral involvement can occur in up to 25% of patients.^[9] Our patient was a young man with a history of type 1 diabetes for 16 years. Neuropathy appeared and his Charcot's foot was unilateral.

The lesion is often triggered by minor trauma. Approximately 50% of patients with Charcot's foot remember a minor precipitating traumatic event.^[10] An acute inflammatory phase is seen and the foot is erythematous, swollen, edematous and warm and can be painful, despite the sensory neuropathy. Pedal pulses are typically easily palpable. A temperature gradient of 2-5°C between the affected and contralateral foot may be noted.^[11] Patients who are afebrile have stable insulin requirements and normal white blood cell counts and often have no break in the skin integrity.^[12] These are all conditions that make infection unlikely.^[12]

Evidence of neuropathy is determined by a decreased or absent sensation to pin prick, light touch, or vibration. The NCV test can be done to confirm sensory neuropathy. Our patient had all of the described features. Imaging studies provide more information. The purpose of the investigation is to distinguish Charcot's foot from other conditions that cause pain and swelling of the foot, such as osteomyelitis, inflammatory arthritis, cellulitis, trauma, deep-vein thrombosis and gout.^[10,13] In addition, images are also necessary when surgical intervention is considered. Radiographs may reveal normal feet in the initial acute phase. The tarsometatarsal joint (Lisfranc's joint) is the most common site for arthropathy. It usually occurs on the medial column of the foot. The distribution of neuropathic arthropathy is 70% at the midfoot and 15% at the forefoot or rear foot.^[12] Isotopic uptake in the area of bony destruction is on average 2- to 3-fold higher compared with a normal condition.^[14] Three-phase bone scan (Tc-99m-MDP) has a high sensitivity but a low specificity for neuro-pathic arthritis. Gallium-67 scan has a high false-positive rate. Scanning using indium-111-labeled leukocytes has the highest sensitivity (87%) and specificity (81%) for detecting osteomyelitis in a

neuropathic foot.^[15] MRI can play a significant role in the differential diagnosis of complicated clinical situations.^[16] As the most-sensitive (100%) and specific (95%) test for osteomyelitis, MRI is able to help assess the extent of the disease and/or the presence of osteomyelitis or other infections. On MRI, high SI in the bone marrow on T2-weighted images indicates osteomyelitis. Soft-tissue abscess can be diagnosed by localized high SI in the soft tissues on T2-weighted images. Neuropathic osteoarthropathy, on the other hand, is suggested by low SI in the bone marrow on both T1- and T2-weighted images.^[17,18] In our patient, MRI showed destructive, osseous debris and bony fragments. There was low SI in the bone marrow on the T2-weighted image. Therefore, MRI can confirm Charcot's foot in difficult cases. The basic principles of nonsurgical management for Charcot's joint include early recognition, immediate and adequate immobilization with protected weight-bearing, and vigilant use of therapeutic footwear to prevent or limit permanent foot deformity, subsequent disability and potential lower extremity amputation.^[19] The most-important management during the acute phase is immobilization and off-loading with a total-contact cast and the treatment should be continued with signs of local inflammation until radiographic evidence of consolidation of the osteoarticular fractures and dislocations. And then, patients need partial weight-bearing in a walking brace to prevent a new attack.^[2,11,20,21] In a double-blind randomized controlled trial, the pamidronate was administered as a single dose of 90 mg intravenously. It reduced bone turnover and improved symptoms and disease activity in diabetic patients with active Charcot's foot.^[22] But the long-term benefits of this approach needs further study. Surgical intervention should be performed to manage chronic ulcers or correct the deformed bones.

CONCLUSION

Patients with Charcot's foot usually report a history of diabetes mellitus of over 12 years with neuropathy. It can be found by physical examination and the swelling and warmth of the involved joints. However, Charcot's foot may mimic or be superimposed on cellulitis or osteomyelitis. In such a situation, laboratory tests and imaging studies should be conducted to provide more information for the differential diagnosis. Scanning using indium-111-labeled leukocytes, can be used to detect the possible infectious focus, while MRI remains the most-valuable tool for confirming a diagnosis of Charcot's foot or the presence of infection. Since type 1 diabetes is rare among Oriental people, even diabetic specialists are likely to encounter both diagnosis and treatment challenges related to Charcot's foot and should bear in mind that earlier recognition helps facilitate less-expensive and more-effective treatment.

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