The Diagnostic Dilemma of Charcot Foot in 73 Year-Old-Female

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ABSTRACT

Charcot neuroarthropathy (CN) is a progressive degenerative arthropathy of weight-bearing joints, which rarely complicates diabetes mellitus, usually in the foot or ankle. Commonly, when affecting the foot, it seems to be determined by the interaction of neuropathy, osteopenia, and proinflammatory cytokines on a calcified peripheral vasculature that maintains its ability to vasodilate despite widespread arteriosclerosis. Although often unrecalled, this arthropathy is probably triggered by trauma. Diagnosis is primarily clinical, given the lack and non-specificity of radiological and biochemical findings at the acute stage. CN should be considered in the differential diagnosis of any diabetic patient presenting with a warm swollen lower extremity. Offloading is essential and improves limb survival. Failure to institute corrective measures at an early stage results in a foot that is prone to deformity, ulceration, amputation, and loss of function. We report a case of Charcot foot in a 73 years old female and review of diagnostic modalities.

Keywords: charcot foot, charcot neuroarthropathy (CN)

INTRODUCTION

Charcot foot is a relatively uncommon complication of diabetes, but one which can lead to chronic ulceration marked deformity and amputation. Although its etiology is not fully understood, several advances have occurred in its management. Surgery has a definite role to play, but the mainstay of initial treatment is non-operative. Correct diagnosis can prevent progressive deformity. The timing and type of surgery are important as inappropriate surgery may worsen the problem and lead to premature amputation.

Charcot neuropathic osteoarthropathy (CN), commonly referred to as the Charcot foot, is a condition affecting the bones, joints, and soft tissues of the foot and ankle, characterized by inflammation in the earliest phase. The Charcot foot has been documented to occur as a consequence of various peripheral neuropathies; however, diabetic neuropathy has become the most common etiology. The interaction of several component factors (diabetes, sensory-motor neuropathy, autonomic neuropathy, trauma, and metabolic abnormalities of bone) results in an acute localized inflammatory condition that may lead to varying degrees and patterns of bone destruction, subluxation, dislocation, and deformity. Charcot foot or neuroarthropathy is estimated to affect between 1-2.5% of people with diabetes. It is most common in people with

Type 1 diabetes in the fifth and sixth decade of life but can occur in young patients and Type 2 diabetic patients as well. Usually, the duration of diabetes is longer than 12 years. In most cases, it is unilateral, but it may be bilateral in up to 25% of patients.

The tarsometatarsal (Lisfranc's) joint is the most common site for arthropathy, with initial involvement usually occurring on the medial column of the foot. The distribution of neuropathic arthropathy is 70% at the midfoot and 15 % at the forefoot or rearfoot. It is usually contained in one area. Nearly 50% of patients with neuropathy had an associated plantar ulcer.

Neuropathic arthropathy is either atrophic or hypertrophic. The atrophic form is

usually localized to the forefoot and causes osteolysis of the distal metatarsals. The metatarsal heads and shafts have a radiographic deformity that resembles a pencil point or "sucked candy cane" (**Figure 1**). The hypertrophic type usually occurs at the midfoot, rearfoot or ankle, and is traditionally defined according to the Eichenholtz classification system. The first stage is the developmental or fragmentation stage (acute Charcot) and is characterized by periarticular fracture and joint dislocation leading to an unstable, deformed foot.

The Charcot foot develops through three stages: *Stage I*: The Stage of Development, there is acute inflammation with hyperemia, bone softening and fragmentation and joint subluxation, dislocation, and destruction. During this stage, the longitudinal arch of the foot may collapse, giving rise to a rocker bottom foot or midfoot subluxation in the transverse plane, which causes this typical deformity. Subluxation at the ankle joint may lead to marked varus and valgus deformity such that the patient is no longer walking on a plantigrade foot. *Stage II*: The Stage of Coalescence, periosteal new bone formation is apparent along with the reduction of swelling. *Stage III*: The Stage of Reconstruction, bony consolidation takes place, and healing occurs. Stage I represents the acute Charcot foot and Stages II and III, the reparative process. Progress through all the three stages may take up to 2 – 3 years, although the acute phase may settle over months.

CASE REPORT

A 73-year-old-female patient, a housewife, presented with the painful left foot. She had a history of recurrent ulcers at the left foot for 10 years ago. Previously she had trauma at the left foot, which became ulceration, swelling, warmth, and she suffered from fever.

She suffered from knee pain at the left knee for 2 months before admission. The left knee was swelling, felt warm, redness, and there was 'clicking' sound while it was moving. She could not walk in the past 1,5 months before admission. She had never smoked cigarettes or consumed alcohol. She had no history of diabetes, hypertension, or

cerebrovascular attack.

Physical examination revealed an overweight female with weight 65 kg, height 162 cm/BMI 24.7. Pulse rate was 90 times per minute, blood pressure 130/60 mmHg, respiratory rate was 20 times per minute, the auxiliary temperature was 36°C, presented severe pain with VAS was 10/10. Her left knee was swelling, warmth, redness, with a positive patellar tap test. At the left foot, there was an ulcer and deformity. The ulceration was at the soles of the feet, size 3x2 cm, clear margin, base subcutaneous, with blood. Examination revealed a warm and swollen foot and a flat longitudinal arch.

ECG records showed sinus rhythm with heart rate 88 beats per minute. The result of the blood glucose level was 104 mg/dL, leucocyte 5.670/ μ L, uric acid 5.1 mg/dL. Left knee X-ray showed senile osteoporosis, spur formation, marked narrowing of joint space, osteophytes, subchondral sclerosis with the conclusion was left knee osteoarthritis Kellgren-Lawrence grade IV. Right knee X-ray showed senile osteoporosis, spur formation, narrowing of joint space, osteophytes with the conclusion was right knee osteoarthritis Kellgren-Lawrence grade III. Left foot X-ray showed deformity at metatarsal and phalanx os pedis sinistra (licked candy stick appearance), and soft tissue swelling with the conclusion was charcot disease (neuropathic osteopathy). The bacterioscopist test for Morbus Hansen was negative. An electromyogram of the lower limbs showed any evidence of peripheral neuropathy.

The patient was assessed as Charcot foot of left foot and bilateral knee osteoarthritis. The patient got NSAID injection intravenous, Ceftriaxone injection.

Figure 1. Anteroposterior radiographic view of a left foot showing metatarsal and phalanx deformity (licked candy stick appearance) of the Charcot neuroarthropathy foot.

Figure 2. Left foot photograph. Foot deformity of metatarsal digits I-V left foot with ulceration.

Figure 3. Left knee X-ray showing senile osteoporosis, spur formation, marked narrowing of joint space, osteophytes, subchondral sclerosis with the conclusion was left knee osteoarthritis Kellgren-Lawrence grade IV.

Figure 4. Right knee X-ray showing senile osteoporosis, spur formation, narrowing of joint space, osteophytes with the conclusion was right knee osteoarthritis Kellgren-Lawrence grade III.

Figure 5. Swollen left knee with effusion

Figure 6. The ulceration at the soles of the feet, size 3x2 cm, clear margin, base subcutaneous. Ulceration at the prominence bone

Figure 7. Portrait of the patient

DISCUSSION

Charcot's arthropathy is a progressive condition characterized by joint destruction, debilitating deformities, and pathological fractures. Though it can occur at any joint, it is most commonly seen in the lower extremity, at the foot and ankle. Historically the most common cause was syphilis, but diabetes is now the most common etiology. The major theories to explain Charcot joints are the Neurotraumatic and Neurovascular theory. The first suggests that the arthropathy is caused by unperceived trauma in an insensate foot and the latter, that there is an autonomic neuropathy causing a mismatch in bone destruction and synthesis. Amongst its other complications, Charcot arthropathy can lead to midfoot collapse and inversion of the plantar arch leading to a rocker-bottom foot deformity.

The precipitating factor in this patient was minor trauma that leads to ulceration. In patients with neuropathy, Charcot joint can develop very rapidly after minor trauma. Patients in the coalescence stage (subacute Charcot) present with resorption of bone debris. This patient

presents with neuropathy. Evidence of neuropathy is determined by decreased sensation and decreased protective impression of the foot that confirmed using Semmes-Weinstein 10-g monofilament wire. The 10-g monofilament correlates with the threshold of protective sensation. The patient cannot feel the monofilament when it is applied with enough pressure to bend the monofilament on 6 sites from 10 sites. It showed that the test was abnormal, and the patient is considered to be at risk for ulcer formation. This test is sensitive and specific for identifying the loss of protective sensation. Our patient has no evidence of diabetes mellitus and had an EMG that resulted a peripheral neuropathy.

Once the Charcot foot has reached a quiescent stage, the patient is usually left with a neuropathic, deformed foot, which is liable to recurrent ulceration over the bony prominences. Initial treatment will tend to be orthotic with footwear and insoles. However, recurrent ulceration in the presence of a fixed deformity will be an indication for surgery. Under these circumstances, surgery is indicated either to remove the bony prominences or correct deformities to produce a plantigrade

foot, which can be managed with orthotics. Before any surgery, it may be preferable to heal the ulceration, if practical, to minimize infection risk.

CONCLUSION

It has been reported a case of Charcot foot and bilateral osteoarthritis in 73 years old female. Physicians must consider the diagnosis of Charcot syndrome in any neuropathic patient with erythema, edema, and elevated temperature regardless of local or systemic signs of infection. In the patient with diabetes and lower extremity neuropathy, any minor injury requires careful observation because of the tendency of the limb to proceed to a Charcot process. Early identification and treatment of the Charcot process help prevent deformity and decreased function of the lower extremity, as well as possible subsequent amputation. Physicians should continually educate their patients about the proper care of a neuropathic foot and the use of orthotic

devices or custom footwear. The patient with a history of a Charcot process should be seen regularly, with close attention given to erythema, edema, or increased temperature in the foot or ankle.

Although Charcot's disease and its association with diabetes have been described many times in the literature, it is still often misdiagnosed and incorrectly treated as osteomyelitis, arthritis, or gout. The best safeguard is a high index of suspicion. A warm swollen foot in a diabetic patient with long-standing neuropathy without local or systemic signs of infection must be considered Charcot's foot until proven otherwise. The principle treatment is total abstinence from putting weight on the foot until warmth, swelling, and redness subside. Protective weight bearing methods may then be slowly instituted.

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