# Lisfranc Fracture Dislocation due to Charcot Joint in A Type 2 Diabetic Woman

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## Abstract

Charcot foot most often affects the metatarsals and the tarsals, and mostly occurs in diabetic patients with neuropathy. Clinical findings in patients with acute Charcot foot include erythema, swelling and heat, which may mimic cellulitis or osteomyelitis. We report a 62-year-old diabetic woman presented with painful erythematous swelling over left foot for 4 months. She had been treated with antibiotics for 3 weeks at another hospital under the diagnosis of left foot cellulitis but without improvement. Physical examination revealed erythema, swelling, and tender in left foot without temperature difference from right foot. Foot radiograph demonstrated one bony fragment at left metatarsal bone with erosion. Charcot joint with underlying diabetic foot, complicated with Lisfranc ligament tear and dislocation was confirmed by magnetic resonance imaging. The left foot was immobilized with a total contact cast and post-cast brace, followed by open reduction and internal fixation due to poor adherence of brace and poor stabilization of foot joints. Charcot foot should be suspected in any diabetic patient presenting with persisted painful erythematous swelling of foot not responded to antibiotic, especially foot radiograph showed bone fragments.(J Intern Med Taiwan 2011; 22: 199-205)

**Key Words: Charcot joint; Diabetes mellitus; Lisfranc fracture**

## Introduction

Charcot joint (neuropathic osteoarthropathy) is a progressive degenerative condition most often affects the metatarsals and the tarsals and could lead to ligament tears, small fractures, subluxation, dislocation, and deformity[1]. Diabetes mellitus is currently the leading cause of Charcot foot, with an incidence of 8.5/1,000 per year in patients with diabetes mellitus in a large population-based prospective study[2], and these patients usually have had diabetes mellitus for more than 10 years[3]. Clinical findings in patients with acute Charcot foot include erythema, swelling and heat, which

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may mimic cellulitis or osteomyelitis. Diagnosis of Charcot foot might be delayed if the clinicians were not familiar with this disease. If Charcot foot was managed inappropriately, complications such as slackness of the ligaments, joint dislocation, bone and cartilage damage, and foot deformity may develop[4-6]. We herein report a case of Lisfrans fracture dislocation due to Charcot joints and make a brief review of the literature.

## Case Report

A 62-year-old woman visited the hospital in May 2007 because of painful erythematous swelling over left foot for 4 months. The patient had not recently sustained any trauma or fever. Her past history included type 2 diabetes mellitus for 20 years and hypertension for 3 years, which were controlled by anti-hypertensive drugs, oral anti-diabetic drugs, and basal insulin. She had been treated with antibiotics for 3 weeks at another hospital under the diagnosis of left foot cellulitis, but without improvement while the swelling and tender exacerbated after discharge from that hospital.

On examination, she featured a weight of 69 kg and height of 1.61 m, with blood pressure 160/109 mmHg, pulse rate 70 beats/min, temperature 36.1 $^{o}$C, and respiration 18 breaths/min. Inspection of the left foot revealed swelling and erythema without ulceration. The swelling and erythema decreased after the patient elevated the left leg for 5 minutes in the supine position. Palpation of the left foot disclosed mild tenderness without temperature difference from right. The pulses of bilateral dorsal pedis arteries and popliteal arteries showed intact. Detail feet examination showed impaired bilateral plantar pressure sensation by monofilament test. Vibration perception test over bilateral first metatarsophalangeal joints by semiquantitative tuning fork (scale 0-8) showed abnormal proprioception (scale 0, bilateral). Bilateral ankle reflexes were absent. The remainder of the physical examination was unremarkable.

The white blood cell (WBC) count was 5,500/mm$^3$ without left shift, hemoglobin concentration was 11.7 g/dL, and platelet count was 257,000/mm$^3$. The biochemistry studies disclosed a fasting blood glucose level of 302 mg/dL, alkaline phosphatase 92 u/L, and glycated hemoglobin (HbA1C) 12.4 %, the other results lay within normal limits. Urine protein showed microalbuminuia and ophthalmologic examination showed preproliferative retinopathy. The C-reactive protein (CRP) concentration was 0.41 mg/dL (reference, 0-0.5 mg/dL), and the erythrocyte sediment rate (ESR) was 53 mm/hr (reference, 0-20 mm/hr).

Parenteral antibiotic with oxacillin 2 g every 6 hours was prescribed since admission but the foot condition still got no improvement. Lower limbs

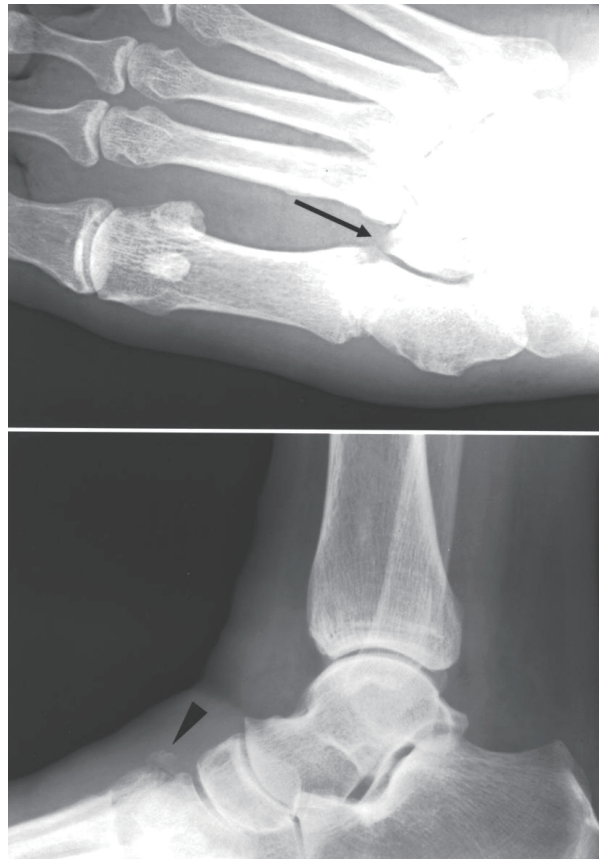

Fig. 1. Foot radiograph demonstrated one bony fragment (arrowhead) at left metatarsal bone with bone erosion (arrow).

compressive Doppler ultrasonography revealed no evidence of deep vein thrombosis, and the Ankle-Brachial Index was within normal limit (right 1.01, left 1.03, normal range 0.9-1.3). However, foot radiograph demonstrated one bony fragment at left metatarsal bone with erosion (Figure 1). Further imaging studies were arranged. Scintigraphy with Technetium (Tc)-99 and Galium (Ga)-67 showed increased and irregular uptake in the left midfoot, but active osteomyelitis in the bones of left midfoot or arthritis could not be differentiated (Figure 2). Magnetic resonance imaging (MRI) revealed subchondral bone marrow edema at tarsometatarsal (Lisfranc) joints and tarsal joints. The Lisfranc ligament showed swelling and edema, suggestive of wearing with subluxation of Lisfranc joint between base of 2nd metatarsal bone and medial cuneiform bone (Figure 3).

Charcot joint with underlying diabetic foot, complicated with Lisfranc ligament tear and subluxation was confirmed. The antibiotic was discontinued and total contact cast was used for immobilization of left foot. One month after total contact cast, the foot swelling relieved and the total contact cast was changed to a post-cast brace. Follow-up CRP concentration 1 month later was 0.128 mg/dL. Despite management with post-cast brace, follow-up foot radiograph in November 2007 showed destruction and subluxation in 1st-5th tarsometatarsal joints. By detail history taking, the patient did not wear the post-cast brace as the

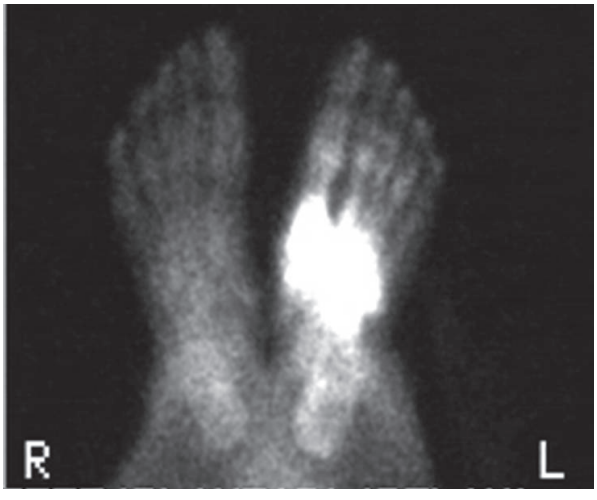


Fig. 2. Scintigraphy with 99Tc and 67Ga showed increased and irregular uptake in the left midfoot.

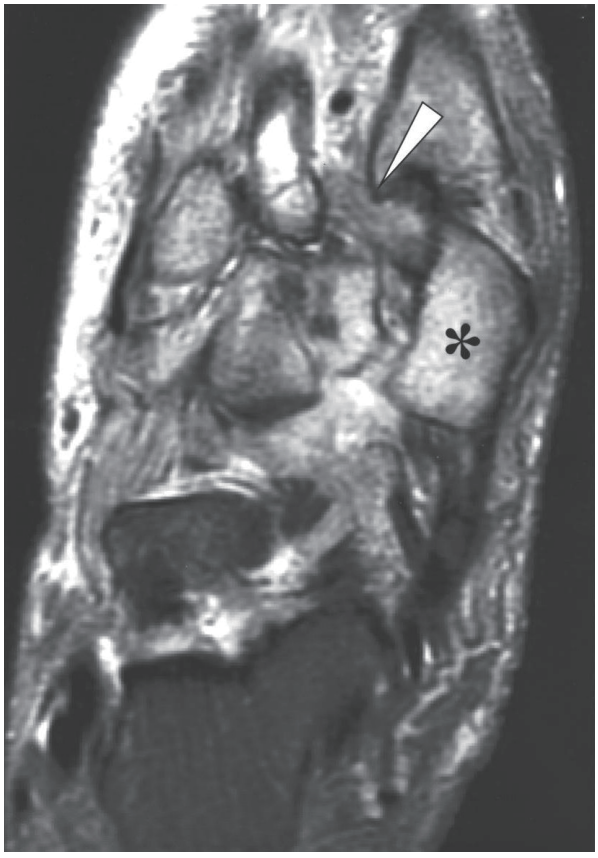

Fig. 3. Magnetic resonance imaging revealed subchondral bone marrow edema at the tarsometatarals and tarsals (asterisk). The Lisfranc ligament showed swelling and edema (arrowhead), suggestive of wearing with subluxation of Lisfranc joint between 2nd metatarsal bone base and medial cuneiform bone.

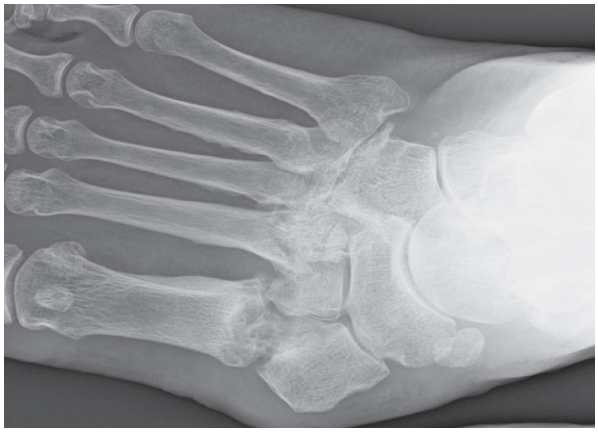

Fig. 4. Foot radiograph demonstrated bone erosion and dislocation of Lisfranc joints one month before surgical intervention.

physician's advice. The patient continued to follow up at the orthopedic and rehabilitation outpatient department and received conservative management and physical therapy. Follow-up foot radiograph in October 2008 showed bone erosion and dislocation of Charcot joints (Figure 4).Open reduction and internal fixation for left Lisfranc joints dislocation was performed in November 2008. Pathology of the removed bone revealed chronic inflammation. Her foot got stable in follow-up visits.

## Discussion

Minor trauma can initiate the process of Charcot foot but our case did not remember a precipitating event of fall or ankle sprain. It had been reported that 50% of patients with Charcot foot did not remember a precipitating event of minor trauma[1]. Both neurotraumatic and neurovascular theories contribute to the pathogenesis of Charcot joints. Neurotraumatic theory supposes that Charcot foot is caused by insensitive to pain caused by peripheral neuropathy, and repetitive mechanical stress of the foot leads to bony destruction. Neurovascular theory postulates that increasing blood flow leading to osteoclast activation and bone resorption.[7] Both processes may occur simultaneously, but neurotraumatic theory rather than neurovascular theory may contribute to the pathogenesis of Charcot joints in diabetes.[8,9] Four stages of Charcot foot based on clinical and radiographic changes are recognized. Stage 0 (inflammation) is characterized by erythema, edema, increasing temperature of foot without structure change. Stage 1 (development-fragmentation) is characterized by bone resorption, fragmentation, and joint dislocation. Stage 2 (coalescence) is characterized by bone consolidation, osteosclerosis and fusion after bone destruction. Stage 3 (reconstruction-consolidation) is characterized by bony remodeling, decreased osteosclerosis.[8,10] Foot radiograph of our patients, 4 months after her initial presentation, showed bone fragment, which is the radiological character of stage 1 of Charcot foot. However, poor adherence of post-cast brace resulted in progression to stage 2.

History of retinopathy, nephropathy, previous foot ulcer and abnormal neurological findings of vibratory sensation, deep tendon reflexes, and the 5.07 (10 g) Semmes-Weinstein monofilament test were correlative for the development of Charcot foot[11] and our patient featured most of such situations. Foot erythema, increasing temperature, and swelling in the acute phase of Charcot foot may resemble cellulitis, inflammatory arthritis, or osteomyelitis. Brosky described a test to differentiate neuropathic osteoarthritis from infection of plantar ulcer. The patient was told to elevate the involved leg for at least 5 minutes in the supine position and the swelling and redness should decrease in neuropathic osteoarthritis, while infection is considered if the swelling and redness persisted[12].

Absence of systemic symptoms and laboratory findings of infection and absence of skin ulceration may distinct Charcot foot from cellulitis or osteomyelitis[13], but the serum markers of inflammation have a low specificity[14]. The clinical presentations of our case mimic cellulitis or osteomyelitis, while the elevated ESR also challenged the diagnosis. It is reasonable to prescribe antibiotic in such a patient if there was doubt about the presence of infection. Little improvement after antibiotic treatment should be one of the clues to the diagnosis of Charcot foot.

Another clue to the diagnosis of Charcot foot is the radiographic findings. The radiological diagnosis of neuropathic osteoarthropathy is based on bone destruction, fragmentation, joint subluxation or dislocation, and bony remodeling, depending on the different clinical stages[5]. However, the foot radiographs may show normal results in the acute phase of Charcot foot[15]. Immobilization and serial follow-up foot radiographs in patients with initially normal foot radiographs and

persisted or deteriorated symptoms are necessary if Charcot foot is strongly suspected.

If there is any doubt of osteomyelitis such as ESR elevation of our case, MRI is a useful tool to detect early radiological events of neuropathic osteoarthropathy and to differentiate neuropathic osteoarthropathy from osteomyelitis by some characteristics. The presence of sinus tract, replacement of soft-tissue fat, fluid collection, and extensive bone marrow edema with low signal intensity on T1-weighted images and high signal intensity on T2-weighted images are MRI features of osteomyelitis[16,17]. Decreased both T1-weighted and T2-weighted signal intensity are characteristic MRI findings in subacute or chronic neuropathic osteoarthropathy[17]. Conversely, the increased T2-weighted signal intensity changes within the bone marrow of acute evolving neuropathic osteoarthropathy were similar to those of osteomyelitis, leading to diagnostic pitfalls[17].

The specificity of Indium(In)-100 WBC scan for osteomyelitis is more sensitive than 99Tc and 67Ga scan and can be as an additional imaging study to differentiate osteomyelitis from Charcot foot[18]. Needle biopsy for bacterial culture and tissue histology study to rule out osteomyelitis should be considered in indecisive clinical conditions of diabetes foot[19-21]. We suggest an approach to differentiate Charcot foot from cellulites/osteomyelitis as shown in figure 5.

Traditionally, Charcot foot was treated conservatively with casting until fractures healed or consolidated beyond stage 3. There is now increasing evidence that surgical intervention earlier in the course of the disease may be appropriate.[10] Our patient received total-contact cast and post-cast brace for immobilization and non-weight bearing, which is mandatory for management of

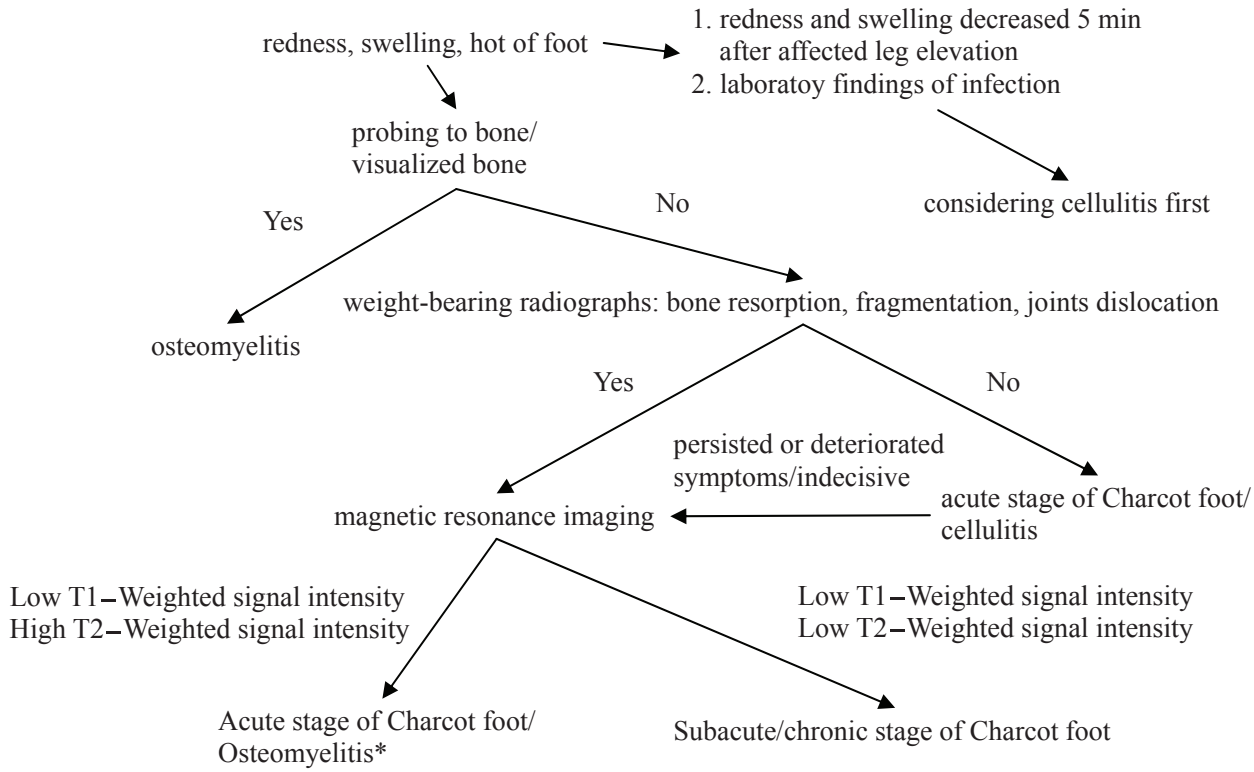


Fig. 5. A flow chart to differentiate Charcot foot from cellulites/osteomyelitis.

* Sinus tract, replacement of soft tissue, and fluid collection are suggestive of osteomyelitis. Needle bone biopsy and Indium -100 WBC scan can be as additional diagnostic tools in indecisive clinical conditions.

the fragmentation phase of Charcot foot[1]. However, more tarsometatarsal joints involvement followed later, which might be the result of poor adherence of post-cast brace. Surgical intervention with open reduction and internal fixation was performed finally to stabilize foot joints. Our case demonstrates that it's important to confirm the adherence of post-cast brace when treating such a patient.

Charcot foot should be suspected in any diabetic patient presenting with persisted erythema, swelling and tender of unilateral foot not responded to antibiotic, especially foot radiograph showed bone fragments. To avoid further joints destruction, education of pot-case brace use and closely follow up in such a patient is important.

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# 第 2 型糖尿病病患併發夏科氏足與 Lisfranc 骨折脫臼

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## 摘 要

夏科氏足是好發於跗骨與蹠骨的疾病，常見於糖尿病合併神經病變的病患。紅、腫、痛等急性夏科氏足的臨床表現與蜂窩組織炎或骨髓炎類似。我們報告一位 62 歲糖尿病女性，因爲左足發紅與腫痛 4 個月，接受抗生素治療 3 週病況未改善而至本院求診。理學檢查顯示左足紅、腫、壓痛但無溫度異常。左足 X 光片顯示蹠骨碎片與骨侵蝕。磁振照影確定爲糖尿病合併夏科氏足，Lisfranc 韌帶撕裂與 Lisfranc 關節脫位。病患接受石膏與支架固定左足，隨後因足部關節不穩定而接受手術治療。糖尿病病患出現足部紅腫疼痛使用抗生素治療無效，特別是足部 X 光片發現骨碎片時應排除是否爲夏科氏足。