Charcot Foot:
An Overview

By Robyn Evans, BSc MD CCFP FCFP and Mariam Botros, DCh DE

Case Presentation: Red, Hot Foot

Mr. R.T. is a 63-year-old who presents to his local walk-in clinic with a warm, red, swollen right foot. He had noticed for the previous couple of days that it was becoming more difficult to get his work boots on. He says it is not painful.

His past medical history is significant for type 2 diabetes for 14 years. He has hypertension. He is a non-smoker and drinks 12 beers per week. He does not test his blood sugars. His body mass index (BMI) is 28.

Medications include: metformin 1g bid, ramipril 10 mg qd, rosuvastatin 10 mg qd. He takes these prescriptions as indicated.

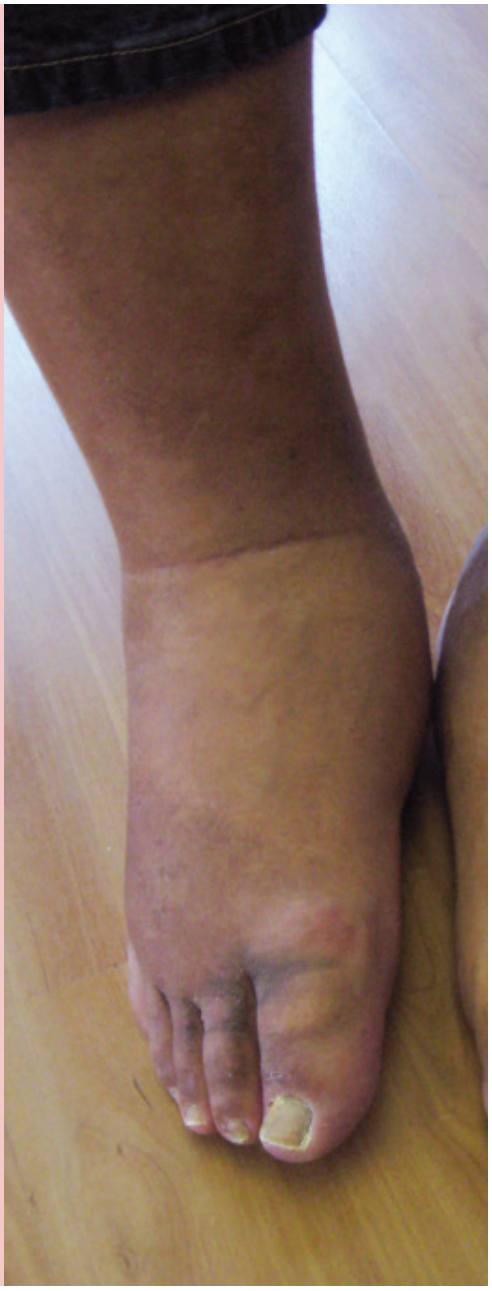
He works in a factory and wears steel-toed boots. His job requires a lot of walking.

The attending physician examines the right foot and notes:

- Pulses bounding at the right dorsalis pedis and posterior tibial
- No skin breakdown; specifically, web spaces are clear
- Right foot is swollen and warm to touch
- Homan's sign (the dorsiflexion sign) is negative
- No palpable tenderness anywhere in the right foot or calf
- Nail changes consistent with a fungal infection
- Patient's temperature: 37° C; heart rate: 76 beats per minute; BP: 136/87 mmHg
- Monofilament score 10 negatives/10 bilaterally
- Left foot shows no swelling or redness

Mr. R.T. is sent home with a prescription for cephalexin for 10 days and instructions to follow up with his own family doctor. Blood work is ordered to check complete blood count (CBC), C-reactive protein (CRP), uric acid, creatinine, blood sugar and HbA1c.

Four weeks later Mr. R.T. presents to his own family doctor concerned that his foot has a different shape at the arch and that there is a small open area.



Avoiding a Devastating Misdiagnosis

The scenario in this case study is not an unusual presentation or management for a red, swollen foot. However, the diagnosis of infection was incorrect, as the patient, in fact, had Charcot neuroarthropathy (CN). Charcot neuroarthropathy is often misdiagnosed.[1] The most common misdiagnoses for an acute CN are listed in Table 1: Differentiating infection/osteomyelitis from CN can be a particular challenge. This article outlines the basics of CN and highlights the need for a high index of suspicion when a patient with diabetes presents with a hot, swollen foot.

Table 1: Common Misdiagnoses of Acute Charcot Foot[2]

Infection	Inflammatory	Other
• Cellulitis • Osteomyelitis • Septic arthritis	• Acute arthritis • Gout • Pseudogout	• Deep vein thrombosis • Sprain/Strain • Fracture

What is Charcot neuroarthropathy?

Charcot neuroarthropathy (CN), also known as Charcot foot, is a rare inflammatory disease involving the musculoskeletal system of the foot and ankle.[3–4] The disease process ultimately results in deformity of the foot or ankle due to collapse, fracture and destruction of structures under significant pressure. Unfortunately, this can lead to increased risk of ulceration, amputation, use of financial resources for patient care, as well as expense to the patient for ongoing accommodative footwear. The risk of amputation with CN is 15% but increases to 35 to 67% in patients with an associated ulcer.[2] This condition has been classified based on clinical and radiologic findings (see Table 2).

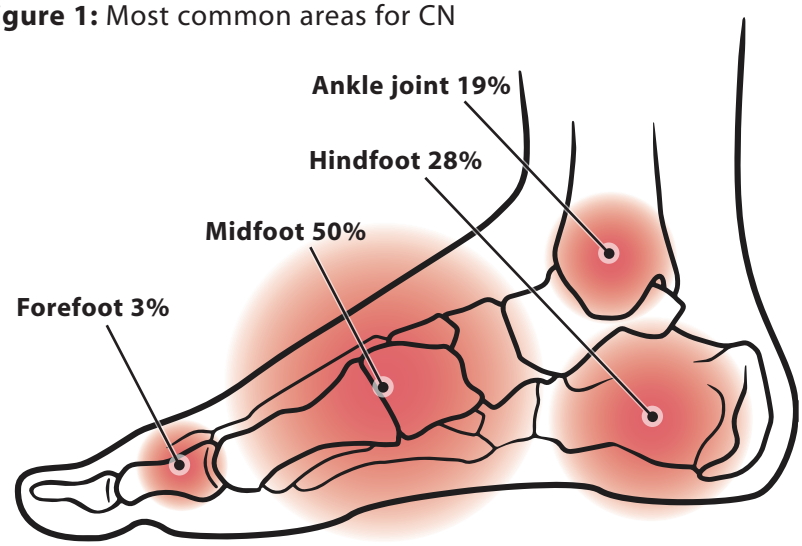
The Pathophysiology of Charcot Neuroarthropathy

The pathophysiology of CN is not entirely known. In 1868 Jean-Martin Charcot was the first to

Table 2: Classification of Charcot Neuroarthropathy[2, 5–6]

Eichenholtz Classification (plus Stage 0)	Description	Management
Stage 0	This is the beginning of the acute stage, characterized by erythema, edema and heat. X-ray evidence may not be seen.	• Immobilize (e.g., using a total contact cast [TCC], instant total contact cast [ITCC] or removable walking cast [RCW]). • Reduce weight-bearing activity. • Manage blood glucose levels.
Stage 1: Development	The actue stage is characterized by erythema, edema and heat. Bone resorption, bone fragmentation and joint dislocation may all be seen on X-ray.	• Immobilize (TCC/ITCC/RCW). • Reduce weight-bearing activity. • Manage blood glucose levels.
Stage 2: Coalescence	The subacute stage is characterized by decreasing warmth, edema and erythema, and by absorption of fine debris and fusion of large fragments and new periosteal bone formation on X-ray.	• Use patellar tendon-bearing brace (PTB). • Use Charcot restraint orthotic walker (CROW walker). • Manage blood glucose levels.
Stage 3: Reconstruction	The chronic stage is characterized by resolution of swelling and erythema. Consolidation of fractured bone and evidence of deformity may be seen on X-ray.	• Use patellar tendon-bearing brace (PTB). • Use Charcot restraint orthotic walker (CROW walker). • Use custom-made shoes with or without a brace. • Manage blood glucose levels.

Figure 1: Most common areas for CN



describe Charcot foot as a late sequela of tertiary syphilis,[6] but it was not described in diabetic patients until almost 70 years later.[7] The two basic theories of its etiology are neurotraumatic and neurovascular.[3,8] In the neurotraumatic theory, some form of trauma (acute, subacute or cumulative and repetitive) in the neuropathic foot initiates a cascade of inflammation. This then leads to intense osteoclastic activity and joint destruction. In the neurovascular theory, autonomic neuroarthropathy results in vasodilation and increased blood flow. This causes congestion in the venous system and ischemia to the ligaments and tendons, leading to joint instability. This increased blood flow also increases osteoclastic activity. If the patient continues to walk and the process goes unchecked, it results in destruction of the susceptible joint of the ankle or foot. Although diabetes is the major cause, any patients with peripheral neuroarthropathy can develop CN. Epidemiologic studies have identified other risk factors for CN (see sidebar, this page).[9]

What are the physical, historical and laboratory findings?

The diagnosis of CN should be based on a careful history and clinical examination of the skin and the neurologic, vascular and musculoskeletal systems (see Table 3). Though only a third of patients will report an inciting trauma, this cause should be considered.[3] Patient co-morbidities as well as gait and balance are important to consider when making management decisions. Unfortunately, 40% of patients will have an ulcer at the time of presentation with a Charcot foot.[3] If an ulcer is present, superimposed infection should be considered. Some patients have been treated for recurrent episodes of cellulitis with little response and no laboratory or systemic signs of infection. The most common

Risk Factors Associated with CN

- Peripheral neuroarthropathy
- Advanced age
- Male gender
- Caucasian
- Lower education level
- Increased body-mass index
- Decreased bone mineral density
- Pancreas and/or kidney transplant
- Elevated HbA1c
- Osteomyelitis
- Recent surgery

Table 3: Physical and Historical Features of CN[2–3]

Skin	Neurologic	Vascular	Musculoskeletal	Other
Varying amounts of swelling, erythema and warmth (3 – 5° C warmer than the contra-lateral, unaffected foot). Use infrared cutaneous temperature monitor. Ulceration may be present. Positive probe-to-bone test.	Sensory, motor and autonomic changes of diabetes. Test using the 10 g Semmes-Weinstein monofilament.	Pulses bounding in the foot.	Varies depending on the stage of CN. Early on, nothing will be seen. Later, joint deformity or instability will be present; classic "rocker bottom" deformity.	Complaint of pain in the foot.

Indications of Possible Infection or Cellulitis

- Proximal streaking of erythema, which is not a feature of CN
- Presence of constitutional symptoms
- Decrease of dependent rubor if the affected limb is elevated for several minutes. If there is infection, this erythema will remain.
- Laboratory evaluation indicating significant elevation of erythrocyte sedimentation rate (ESR) and CRP, which may be consistent with infection. (Unfortunately, patients with diabetes often have a muted response to infection, so these values may not increase as expected.)
- Presence of an ulcer, skin breakdown or other portal of entry.
- Presence of an ulcer with a positive probe-to-bone test.

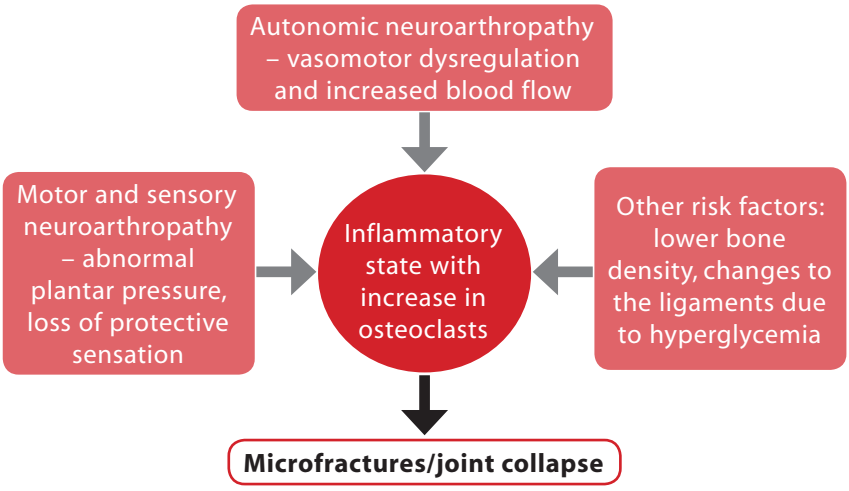
areas involved are the midfoot (50%) followed by the hindfoot (28%), the ankle joint (19%) and the forefoot (3%)[6] (see Figure 1 on facing page).

Imaging Considerations

Radiographs are the recommended initial imaging study to be done. The characteristic bony changes of CN can take weeks to see on plain X-rays and therefore are not useful for diagnosing CN in the early stages—when clinical intervention is critical. It is helpful to take bilateral X-rays to pick up subtle changes in the bone.[4]

For patients with diabetes and an ulcer, X-rays should look for bony abnormalities, soft tissue gas or the possibility of a foreign body.[10] Table 3 lists the musculoskeletal changes that can be expected at each stage of CN. A venous duplex ultrasound scan should be considered if deep vein thrombosis is suspected. Magnetic resonance imaging is able to detect bone marrow changes, soft tissue edema and joint effusion early in the disease.[8] Nuclear imaging techniques may be used when MRI is not available or contraindicated.

Figure 2: Factors Leading to Microfractures or Joint Collapse



Eichenholtz classified Charcot foot based on radiological findings in three stages,[11,12] and later Shibata proposed an additional Stage 0, which is characterized by erythema, edema and heat without X-ray confirmation.[11] Patients at this stage are often misdiagnosed with cellulitis, gout or deep vein thrombosis due to lack of radiographic evidence.[5]

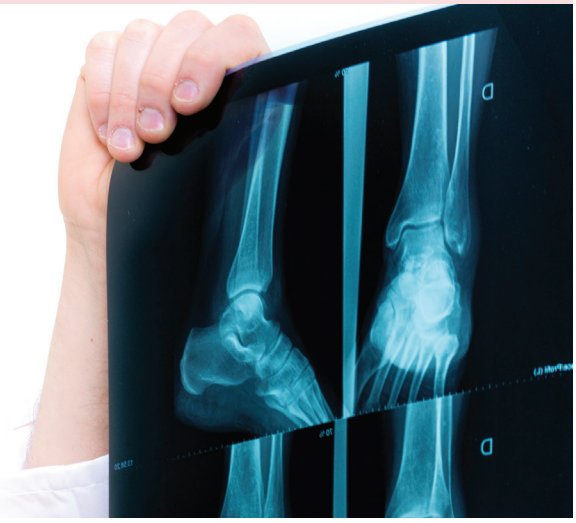
Management

Management of Charcot foot is based on the acuteness of symptoms, anatomic location and degree of joint destruction.[4] If a clinician is initially unsure about the diagnosis, it is recommended that they treat the condition as Charcot neuroarthropathy by offloading until diagnosis is confirmed or disproven. Early detection and protection are key to preventing further destruction of the foot.

In the acute stage, immobilization and reduction of weight-bearing activities for eight to 12 weeks is the mainstay of treatment. The gold standard for immobilization of Charcot foot is a total contact cast (TCC),

Important Facts about CN/Diabetes

- Patients with peripheral vascular disease are somewhat protected from CN as vasodilation is part of the pathogenesis.[3,8]
- Joints are the weak link in the structure of the foot, and therefore more susceptible.
- The midfoot is most often affected as it is subjected to more force during the phases of walking. This is the classic "rocker bottom" deformity. However, any joint of the foot can be affected.[9]
- Hyperglycemia causes increased risk of ligament and tendon weakening.[3,10]
- Patients with diabetes often have lower bone mineral densities, a factor for development of CN. This is more of an issue with type 1 than type 2 diabetes.[2]
- Only a third of patients will report trauma leading to their symptoms.[3]



An Alternative Scenario

The physician at the walk-in clinic was aware of a rare condition called Charcot foot. He was still concerned, however, that he would miss an infection in this patient with diabetes. He prescribed cephalexin but also advised the patient to remain non-weight bearing as if he had an acute fracture. He sent him home with an RCW, which was available in his pharmacy, instructions to stop working, and an urgent referral to a multidisciplinary clinic that deals with diabetic foot issues. The following day the blood report was obtained and indicated a normal CBC, creatinine, and CRP. His HbA1c was 9.6%.

but devices like a removable cast walker (RCW) are also commonly used to offload the foot. Continue immobilization until lower extremity edema and warmth resolve accompanied by evidence of fracture consolidation.[2,6]

In the subacute and chronic stages, recommend devices include the Charcot restraint orthotic walker (CROW) and the patellar tendon-bearing brace (PTB). In the chronic stage, custom-made shoes are indicated.[2,6]

Surgery may be considered if conservative treatment fails to establish a plantigrade foot.

There is currently no evidence for the use of bisphosphonates in managing CN.[2,6]

Conclusion

Charcot neuroarthropathy is a commonly missed diagnosis. It relies on an astute clinician, because early physical findings can be subtle with little help from imaging or laboratory. Early diagnosis is important for leading to early, appropriate management and prevention of further complications. CN should be suspected in any patient over 40 years old with peripheral neuropathy that presents with an acutely swollen foot with little or no known

Key Points

- ✔ A high index of suspicion is required to correctly diagnosis CN in a timely manner.
- ✔ Develop an approach to the red, hot, swollen foot with or without pain. Consider the possibility of infection, which can co-exist with Charcot changes.
- ✔ If Charcot neuroarthropathy is a concern, advise the patient to remain non-weight bearing while appropriate referrals are arranged.

trauma. It is unclear why not all patients with diabetic neuroarthropathy develop Charcot foot. Inflammation seems to be at the core of the process, and this may be related to risk factors and genetic predisposition.[13]

It can be difficult for healthcare providers on the front line to access the appropriate referrals in a timely manner. Enlist help from colleagues when referral to a multidisciplinary team is not possible. An orthopedist, podiatrist or chiropodist should be able to help with these difficult cases.

Consider CN in the differential diagnosis of a red, swollen foot to prevent the devastating consequence of a deformed foot and long-term effects on quality of life, morbidity and mortality.

References

1. Schmidt B, Holmes C. Updates on diabetic foot and Charcot osteopathic arthropathy. Curr Diabetes Rep. 2018;18(10):74.
2. Marmolejo V, Arnold J, Ponticello M, Andersen C. Charcot foot: Clinical clues, diagnostic strategies, and treatment principles. Am Fam Physician. 2018;97(9):594–99.
3. Rogers L, Frykberg R, Armstrong D, Boulton A, Edmonds M, Ha Van G, et al. The Charcot foot in diabetes. Diabetes Care. 2011;34:2123–29.
4. Dodd A, Daniels T. Charcot neuroarthropathy of the foot and ankle. J Bone Joint Surg Am. 2018;100:696–711.
5. Mautone M, Naidoo P. Charcot neuroarthropathy: An imaging review. J Med Imaging Radiat Oncol. 2015;59:395–402.
6. La Fontaine J, Lavery L, Jude E. Current concepts of Charcot foot in diabetic patients. The Foot. 2016;26:7–14.
7. Jordan W. Neuritic manifestations in diabetes mellitus. Arch Intern Med. 1936;57(2):307.
8. Strotman P, Reif T, Pinzur M. Charcot arthropathy of the foot and ankle. Foot Ankle Int. 2016;37(11):1255–63.
9. Holmes C, Schmidt B, Munson M, Wrobel J. Charcot stage 0: A review and considerations for making the correct diagnosis early. J Clin Endocrinol Diabetes. 2015;1(18).
10. Lipsky B, Berendt A, Cornia P, Pile J, Peters E, Armstrong D, et al. Infectious Diseases Society of America clinical practice guideline for the diagnosis and treatment of diabetic foot infections. Clin Infect Dis. 2012;54(12):e132–e173.
11. Holmes C, Schmidt B, Munson M, Wrobel JS. Charcot stage 0: A review and consideratons for making the correct diagnosis early. Clinicial Diabetes and Endocrinology. 2015;1(18):12 pp.
12. Schmidt MB, Holmes CM. Updates on diabetic foot and Charcot osteopathic arthropathy. Current Diabetes Reports. 2018;18(74):11 pp.
13. Gökhan K, Birsel O, Güven MF, Öğüt T. An overview of the Charcot foot pathophysiology. Diabet Foot Ankle. 2013;4(10).

SKIN SPECTRUM SUMMIT

The Canadian Conference on Ethnodermatology

A special invitation for members of Wounds Canada

The Skin Spectrum Summit is a landmark one-day event on ethnodermatology that takes place in three major Canadian cities.

Celebrating its fifth year, the Summit is an educational congress of General Practitioners, Family Physicians, nurses, nurse practitioners, and dermatologists committed to providing better dermatological care for Canada's diverse and growing ethnic communities, notably Fitzpatrick Scale Skin Types III through VI.

NEW LOCATION

Saturday April 6, 2019 | Toronto
Chestnut Conference Centre

Friday May 10, 2019 | Montreal
InterContinental Montreal

NEW LOCATION

Saturday June 1, 2019 Vancouver
Coast Coal Harbour Hotel

Toronto Chair:
Dr. Gary Sibbald,
University of Toronto

Montreal Chair:
Dr. Danielle Marcoux,
University of Montreal

Vancouver Chair:
Dr. Jason Rivers,
University of British Columbia

EDUCATIONAL OBJECTIVES

- Learn about the different skin conditions in Canada's ethnic
- population including the manifestations of common dermatological problems for persons with skin of colour
- Improve diagnostic practices of different skin conditions in the growing ethnic population
- Learn strategies and tools to better manage patients with skin of colour including potential unique challenges that they may face in their treatment

Members of Wounds Canada
can register for only $79 + HST, regular $179 (a savings of $100)
Please use the discount code: WC20

For more information and to register please visit
www.skinspectrum.ca
or email us at health@chronicle.org