

Charcot Neuroarthropathy: A case study of Foot Presentation and Management

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SUMMARY

Charcot neuroarthropathy (CN), also referred to as Charcot arthropathy represents a rare yet critical complication of diabetic peripheral neuropathy. Misdiagnosis or inadequate treatment of this condition can lead to severe consequences, including the need for major amputations and loss of limb functionality. While the precise pathogenesis of Charcot arthropathy remains elusive, two prevailing theories have been proposed. The neurotraumatic theory and neurovascular theory. Clinical presentation typically involves unilateral swelling, erythema, and localized warmth to the foot, often progressing to a “rocker bottom” deformity over time. We present a case of a 45-year-old male with long-standing type 1 diabetes mellitus (T1DM) who presented with a painless, swollen, and warm right foot, initially misdiagnosed as a diabetic foot infection. Further examination revealed a collapse of the longitudinal arch and a clinical diagnosis of Charcot’s neuroarthropathy was established. Imaging studies confirmed destructive changes consistent with Charcot neuropathy, leading to subsequent management with total contact casts and referral for reconstructive surgery. Prompt detection and appropriate treatment are imperative given the potential ramifications of Charcot foot, with current management strategies emphasizing prolonged immobilization and physical off-loading.

Key Learning Points:

- Misdiagnosis or inadequate treatment of CN can lead to devastating consequences, including major amputations and loss of limb functionality.
- Clinical presentation typically includes unilateral swelling, erythema, and warmth to the foot, often progressing to a “rocker bottom” deformity over time.
- We present a case study of a 45-year-old male with long-standing type 1 diabetes mellitus who was initially misdiagnosed with a diabetic foot infection but ultimately diagnosed with Charcot’s neuroarthropathy.
- Prompt detection and appropriate treatment are crucial to mitigate the potential consequences of Charcot foot, with current management strategies emphasizing prolonged immobilization and physical off-loading.

INTRODUCTION

Charcot neuroarthropathy (also known as Charcot arthropathy/Charcot foot) is a rare but serious

complication of diabetic peripheral neuropathy affecting 0.15-2.5% Diabetic patients. The consequences of misdiagnosis or of failing to treat the condition can be devastating leading to major amputations and limb loss.

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While exact pathogenesis of Charcot arthropathy is not known, researchers have postulated two theories. The neurotraumatic theory states the occurrence of a “stress” fracture in the presence of sensory peripheral neuropathy, where the patient, lacking protective sensation, continues to bear weight, thus resulting in a deformed hypertrophic “nonunion.” On the other hand, the neurovascular theory suggests bony resorption due to increased vascular inflow, leading to bony weakness and subsequent mechanical failure resulting in deformity.^{1,2}

Recent evidence indicates that the actual pathophysiological process likely involves a combination of both mechanisms. Trauma triggers the upregulation of osteoclasts, which in turn contribute to the destruction of poor-quality bone, either facilitating or hindering the healing process of acute or subacute fractures. Consequently, the observed acquired deformities manifest in patients whose initial trauma remains unresolved.¹

Cases typically present with a history of a trauma demonstrating considerable unilateral swelling erythema and localized increase in temperature to the foot, with intact skin. In advance case with considerable mid foot destruction, a typical “rocker bottom” appearance is seen. In most cases, progression of deformity takes a more chronic form and is seen over a period of months to years.³

CASE PRESENTATION

A 45 years old male patient with T1DM of over 25 years duration (on basal-bolus regimen) presented to the foot clinic of Baqai institute of diabetology &

Endocrinology (BIDE), with two months history of painless, swollen, and warm right foot. During this 2-month period, he took multiple courses of Antibiotics prescribed by GPs suspected as a case of diabetic foot infection. He also had a past history of treated Diabetic foot infection in the past prior to this event which healed completely. The patient had a history of poor glycemic control despite the basal Bolus regimen with an average HbA1c of 10% over several years.

On physical examination he had a red, hot, swollen right foot with no open wound or signs of trauma/break but collapse of longitudinal arch with a Rocker bottom deformity was seen. All pedal pulses were readily palpable with ABI of 1.1, his touch and vibration sense were intact. Skin temperature was recorded with a calibrated infra-red skin thermometer demonstrating a significantly raised temperature of right foot as compared to left foot. Right foot: 34.7-36c. Left foot 30.9-31c with no signs of any systemic infection. Physical Examination and left foot examination was unremarkable. A clinical diagnosis of Charcot's neuroarthropathy was provisionally made. X ray of Right foot and lab work up was sent. The results are shown in Table-I.

X-rays showed a destructive change in the mid-foot consistent with the diagnosis of Charcot Neuropathy (CN) with Rocker bottom deformity (Fig.1).

The patient was then managed for CN and over a 6 months period total contact casts (TTC) were applied. At this stage his CN had become quiescent. Subsequently he has been provided with bespoke foot wear and orthoses to prevent ulceration over the planter rocker sole deformity. Patient was referred for

Table-I: Laboratory investigations of the patient presenting with Charcots foot.

Date	Tests name	Results	Reference range
5/10/22	Complete blood count:		
	Hb:	12.2 g/dl	11.1-13.8 g/dl
	MCV:	87fl	76-96 fl
	WBC:	8.0*1000/ul	4.0-11.0*1000/ul
	PLT:	290*1000/ul	150-400*1000/ul
5/10/22	Serum Electrolytes & creatinine		
	Creatinine:	0.73mg/dl	0.6-1.1mg/dl
	Sodium:	140 mEq/L	136-145 mEq/L
	Potassium:	4.2 mEq/L	3.8-5.2 mEq/L
	Chloride:	99 mEq/L	96-107 mEq/L
	Bicarbonate:	26 mEq/L	22-29 mEq/L
5/10/22	U. Microalbuminuria	15	<30
5/10/22	HBA1C:	10.6%	
5/10/22	TSH:	2.2mIU/ml	0.4-4.2mIU/ml



Fig 1: X ray of right foot showing Charcot's foot.

osteotomy with realignment arthrodesis to reconstruct the longitudinal arch.

DISCUSSION

Diabetic Charcot neuroarthropathy (CN) is an uncommon but limb threatening condition. It is an acute localized protracted inflammatory condition resulting from the interaction of several component factors (diabetes, polyneuropathy, trauma and metabolic abnormalities of bone) which, in turn, may lead to varying degrees and patterns of bone destruction, subluxation, dislocation and deformity. Its pathogenesis is not fully understood but peripheral sensory and sympathetic neuropathy is considered to be

predisposing factors.^{2,3} Various stages of development of Charcot's arthropathy is shown in Fig.2.

The classical clinical presentation of an acute CN is a red/pink/dark (depending on skin colour), swollen, warm/hot foot with mild to moderate pain, if any at all. This clinical presentation closely resembles that of infection with cellulitis, deep vein thrombosis, or even acute gout, thus it is possible to initially misdiagnose acute CN for any of the former. The temperature difference between the affected and contralateral limb can differ by several degrees, not uncommonly by 4–7°C. This difference in skin temperature is used as a marker of resolving of the acute phase, thus when the skin temperatures of the two limbs are $\leq 1^\circ\text{C}$, immobilization of the affected limb can gradually be removed.³

Another clinical feature is the presence of very good and possibly exaggerated arterial flow. Characteristically, the pedal pulses when palpated are very strong and often 'bounding'. Ankle pressures are frequently abnormally high, demonstrating the presence of medial wall calcification, which is a common feature of CV. In the early onset of CN, X-ray examination may appear normal but later, bony destruction becomes evident and the bones have a 'fluffy' appearance. The most frequent site for diabetic CN is the mid-foot area but it can occur in the ankle, rear or forefoot. In those with foot deformity, approximately 60% are in the tarsometatarsal joints (medial joints affected more than lateral), 30% metatarsophalangeal joints and 10% have ankle disease.

Many patients who present with the acute active phase of the Charcot foot disease are incorrectly diagnosed as having cellulitis or an abscess that requires surgical treatment. There exist several clinical insights that aid in distinguishing acute Charcot foot

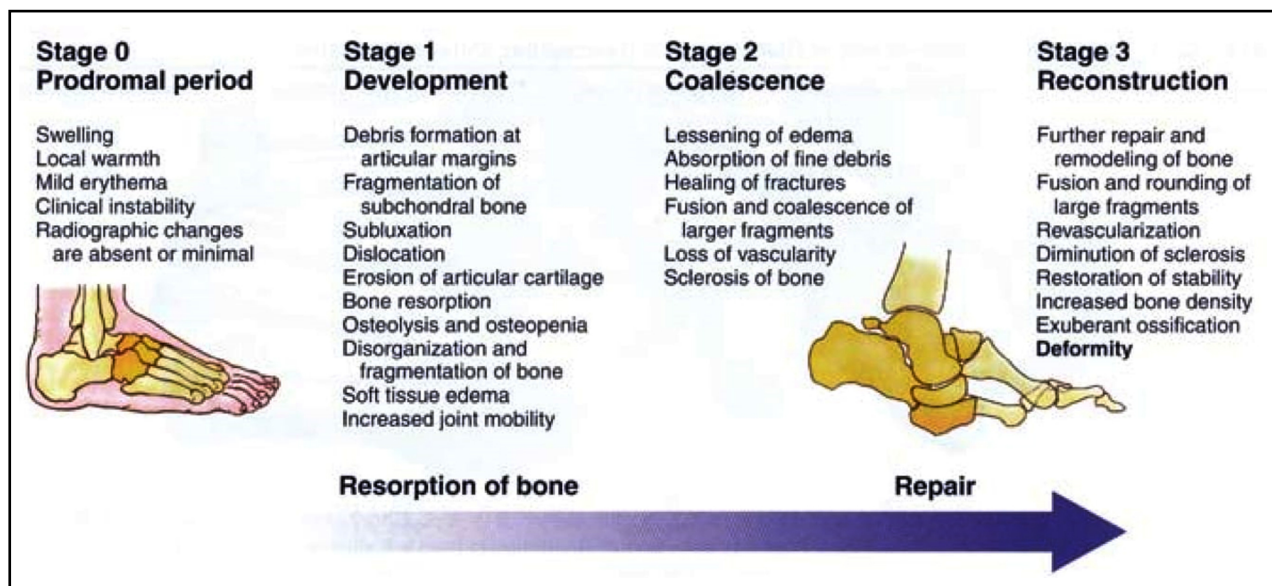


Fig 2: Stages of Charcot foot.

arthropathy from infection. Patients experiencing infection typically exhibit symptoms of malaise and may report escalating blood sugar levels or an elevated insulin requirement during the prodromal phase—features absent in Charcot foot cases. Furthermore, elevating the foot with the patient in a supine position often results in the dissipation of erythema in Charcot foot, contrasting with the persistent erythema observed in infection cases.

Studies have shown Charcot neuroarthropathy is common in female (63.5%), compared to male (36.5%), more on Right side (0.2%), then left side (0.15%) and bilateral (0.05%). There is significant association ($p < 0.05$) of CN with duration of diabetes, HBA1C and Neuropathy. However no significant association ($p > 0.05$) was found with Age, BMI, height.⁵ Prompt detection and treatment are essential due to the potentially devastating consequences of a Charcot foot. The current treatment of Charcot foot consists of prolonged immobilization and total physical off-loading, either with a total contact cast or a removable

walker cast.⁶ Generally speaking, the off-loading process works best when it begins as soon as possible. Off-loading allows the injured foot to heal as well as stops the underlying deteriorating tissue damage.⁷

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