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Missed Charcot Neuroarthropathy: Case Report and Review

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In this case report, we review a case where an acute ankle fracture was the primary diagnosis for a diabetic patient, however Charcot neuroarthropathy was not a part of the differential diagnosis. This patient was subsequently mismanaged and misdiagnosed leading to a non-functional limb that required complete reconstructive surgery of the ankle. The purpose of this report is in an effort to further understanding of the potential complications associated with misdiagnosis and mistreatment of Charcot neuroarthropathy

Key words: charcot, neuroarthropathy, ankle fracture, diabetes, ankle arthrodesis

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One of the most challenging diagnoses in diabetic patients is early stages of Charcot neuroarthropathy. This is commonly mistakenly diagnosed as cellulitis, deep venous thrombosis or acute gout[1]. Literature reports that the diagnosis is missed from 25% to 79% of the time[2]. The diagnostic delay averages 29 weeks[3], allowing insensate patients to cause continued trauma to the foot, worsening the deformity. Another contributor to the delayed diagnosis is the incidence of Charcot neuroarthropathy is relatively rare and its estimated prevalence is approximately 1% of all neuropathic patients[4]. Nevertheless, early detection and treatment can minimize fractures and incapacitating deformities[5]. Some believe that acute Charcot joint disease is a "medical emergency," as there are therapies available that can alter its natural history[6].

Delay of the correct diagnosis of Charcot neuroarthropathy in the early stages is harmful because during the acute phase the foot and ankle bones are vulnerable to fragmentation and dislocation[7]. Stage 0 Charcot (inflammatory) arthopathy begins with initial perceived or non-perceived insult to the neuropathic foot-and-ankle complex, producing localized swelling, redness, and warmth[8]. Stage 1 Charcot neuroarthropathy (development) is distinguished by marked eythema and edema, and radiographic changes are evident in the bone-and-joint architecture[9]. The appearance of bony debris, fragmentation of subchondral bone,

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subluxation, dislocation, and periarticular fractures are common findings during Stage 1[10].

Overall, Charcot arthropathy of the foot and ankle is a deforming and destructive process that can lead to increased patient morbidity due to gross instability, recurrent ulcerations, and/or amputation[11]. Outcomes of early Charcot neuroarthropathy depend on proper recognition and early management. To reduce the rate of future complications for Charcot neuroarthropathy should be the goal of all treatment[12].

Diabetes mellitus is an indicator of poor prognosis for acute ankle fractures. Both clinical and basic science studies have revealed that the diabetic phenotype is associated with impaired fracture healing, reduced bone mass and bone mineral density, decreased collagen content and defects in cross-linking[16]. This contributes to diabetic patients having reduced bone formation, reduced cartilage formation, accelerated loss of cartilage and reduced vascularity/angiogenesis[17].

Case Report

This 54-year-old Caucasian male with past medical history of diabetes mellitus type II with neuropathy x 11 years, hypothyroidism and hyperlipidemia presented to Emergency Department after falling down the stairs. Plain film radiographs revealed a right ankle fracture. Patient did not follow up as instructed and remained weightbearing for 2 weeks. At inital follow up, patient was noted to have erythema of the extremity and was treated for cellulitis with PO antibiotics. On follow up, "cellulitis" had not resolved and he was referred for vascular workup. At 3 months he was told his "bones were disintegrating" and he was referred to our clinic for management.

Patient presented to the attendings clinic ambulating with aid crutches and was partial weightbearing on right foot in posterior splint. Pedal pulses were palpable and CFT was <3 seconds. Substantial edema and erythema were noted to the right ankle. Neurological exam revealed loss of protective sensation to right foot. Additionally, a subungual hematoma was noted to right 2nd digit and an abrasion was noted to ankle measuring 3.0 x 2.0 cm. Patient also displays visible valgus of right ankle.

Radiographs and a CT scan of the right ankle displayed diffuse soft tissue swelling, comminuted fractures of the distal tibia and fibula involving the articular surfaces and anterior displacement of the hindfoot relative to the tibial and fibular shafts.

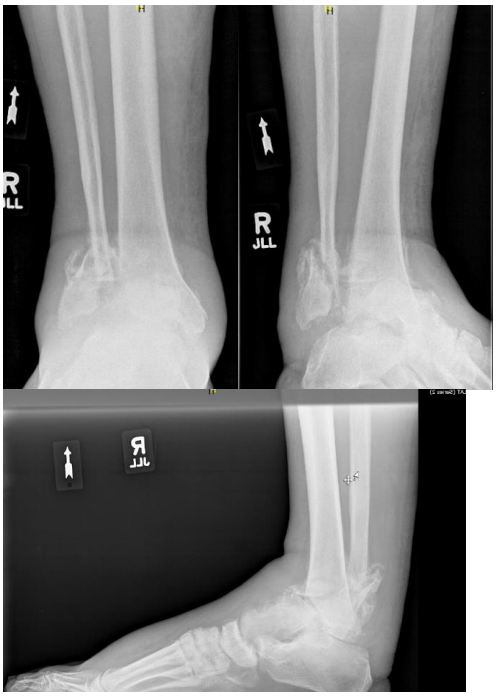

Figure 1. Initial radiographs AP, oblique and lateral of right ankle.

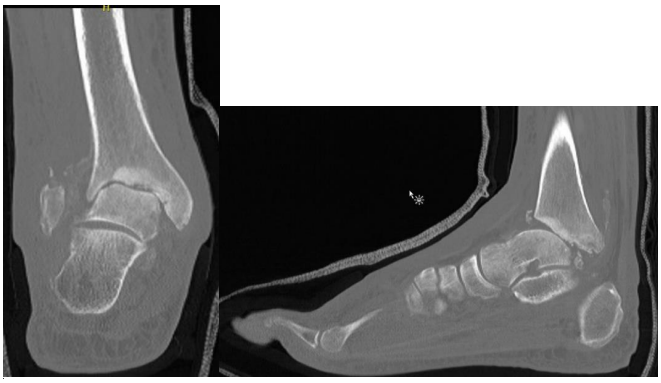

Figure 2. CT of right ankle.

Patient was initially managed with total contact casts and non-weight bearing in CAM boot for approximately 7 weeks. Once the erythema and edema had resolved, patient was consulted on surgical options in order to obtain a plantargrade, bracable foot. The patient was also informed that with or without surgery, he was at a high risk of limb loss. He was informed that due to the unstable nature of his deformity, the ankle could continue to deteriorate, further increasing risk of limb loss. He understood that this would be considered a limb salvage procedure and a plan was established for an ankle arthrodesis.

An anterior approach was taken for the procedure. The ankle joint was prepped manually and excess bone was removed. Tibia and talus were planed to ensure proper fit. Two Steinman pins were drive from plantar to dorsal through the calcaneus to the tibia under live fluoroscopy for temporary fixation. An anterior ankle joint fusion plate was placed over the anterior tibia and talus. Three locking screws were placed above the compression screw and four locking screws were placed distal to the compression screw. The Steinman pins were then removed and two 6.7 partially threaded screws were placed across the subtalar joint to span fixation into "normal" bone. The tibiotalar joint was then packed with autograft material. Final C-arm images were taken in multiple planes to ensure proper alignment and hardware placement. Patient was placed in a Jones compression posterior splint and instructed to be non-weightbearing.

Intraoperative inspection of the distal fibula revealed consolidation of the fracture. In order to spare the patient a second incision and risk further wound healing complications, it was felt that the fibula could be left intact.

Patient is currently 7 weeks post op. His anterior incision is healed. He has been non-weightbearing in a below knee cast and is being transitioned to a pneumatic walking boot with partial weightbearing. Complete consolidation of the ankle joint is not yet appreciated, and therefore a bone stimulator will be obtained at next visit. We will continue with close follow up and management of this patient to insure a stable, functional limb as the final outcome.

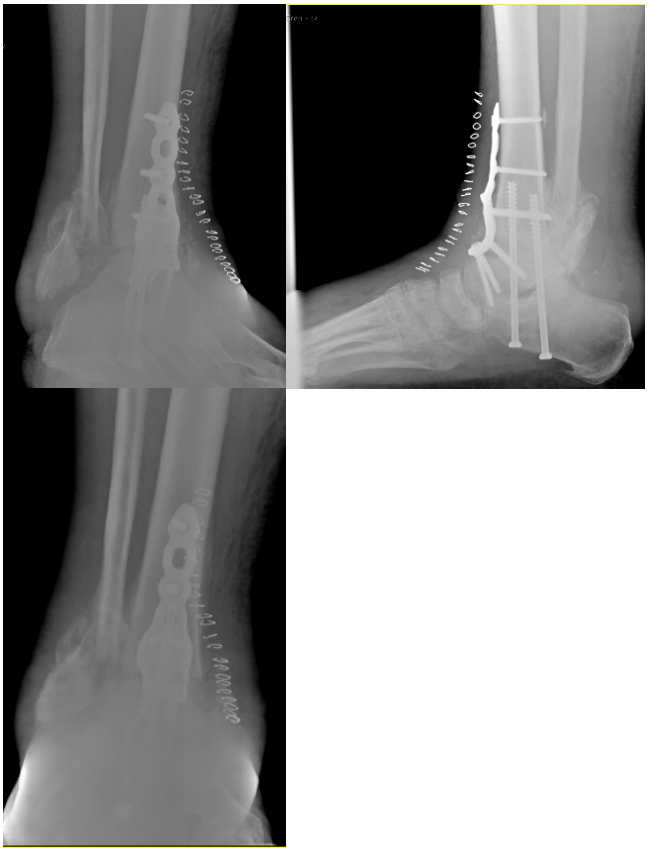

Figure 3. Radiographs AP, oblique and lateral postoperative 1 week.

Discussion

Early detection and treatment of Charcot is paramount. Clinicians must have a high index of suspicion in the setting of peripheral neuropathy +/- diabetes. Patient frequently present with a history of major/minor trauma in lower extremity displaying erythematous and edematous changes with calor compared to contralateral limb. A combination of clinical exam and imaging are necessary to correctly identify this disease as early as possible[15].

When appropriate intervention is undertaken in this high-risk patient population, the rate of ulceration decreases by 60% and lower extremity amputation by 85%[14].

This case demonstrates why it is paramount to make in early diagnosis Charcot neuroarthropathy. Surgical intervention might have been avoided had early detection been made and early offloading principles applied. This patient's terminal outcome is still in question as his fusion has not fully consolidated.. Even with heroic limb salvage endeavors such as the case above, limb loss is still a risk for this patient.

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