# NEW TRENDS IN MANAGEMENT OF CHARCOT FOOT AND ANKLE

*Essay*

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*By*

**Ahmed Nabil Abd-Elazem Ellithy**

(M.B., B.Ch.)

*Under the Supervision of*

**Prof. Dr. Mahmoud Fayed**
Professor of Orthopedic Surgery
Faculty of Medicine - Ain Shams University

**Dr. Mohammed Hassan Sobhy**
Lecturer of Orthopedic Surgery
Faculty of Medicine - Ain Shams University

*Faculty of Medicine*
*Ain Shams University*
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# LIST OF ABBREVIATIONS

| Abbrev. | Meaning |
|---|---|
| **ABPI** | Ankle brachial pressure index |
| **AFO** | Ankle fast orthosis |
| **CD** | Charcot deformity |
| **CN** | Charcot neuropathy |
| **CORA** | Center of rotation of angulation |
| **CROW** | Charcot restrain orthotic walker |
| **CT** | Computed tomography |
| **DM** | Diabetes mellitus |
| **HMPAO** | Hexamethyl propylene amine oxime |
| **IL** | Interleukin I |
| **In** | Indium |
| **MDP** | Methyl diphosphonate |
| **MRI** | Magnetic resonance imaging |
| **MTP** | Metatarsophalangeal |
| **NF-β** | Nuclear transcription factor beta |
| **OM** | Osteomyelitis |
| **PAD** | Peripheral arterial disease |
| **PPWB** | Prefabritcated pneumatic walking brace |
| **RANKL** | Receptor activator or nuclear transcription factor beta |
| **TC** | Technetium |
| **Tcc** | Total contact cast |
| **TCPO2** | Transcutaneous oxygen diffusion |
| **TNFα** | Tumour necrosis factor alpha |
| **TSF** | Taylor spatial frame |
| **WBC** | White blood cells |

# INTRODUCTION

When Jean-Marie Charcot described the entity that bears his name in 1868, little did he know the controversies he would create. Charcot joint disease (or Charcot neuroarthropathy) has been one of the most misdiagnosed conditions in patients with diabetes mellitus. Patients with this entity have been misdiagnosed and consequently mistreated for osteomyelitis, cellulitis, tendonitis and gout. [1]

Charcot neuropathy is a progressive deterioration of weight-bearing joints, usually in the foot or ankle. Historically, neuropathy of the knee was most frequently caused by syphilis, and neuropathy of the shoulder was usually caused by syringomyelia. Today, the Charcot foot occurs most often in patients with diabetic neuropathy, other predisposing conditions include alcoholic neuropathy, sensory loss caused by cerebral palsy or leprosy, and congenital insensitivity to pain.

The acute Charcot foot is characterized by erythema, edema and elevated temperature of the foot that can clinically mimic cellulitis or gout. Plain film radiographic findings can be normal in the acute phase of Charcot foot. A diagnosis of Charcot syndrome should be considered in any neuropathic patient, even those with a minor increase of heat and swelling of the foot or ankle, especially after any injury. [2]

Increasing understanding of the underlying pathogenic events provide strong support for an important role for

osteoclastic activity and proinflammatory cytokines. This has led to the successful use of bisphosphonates in patients with acute presentation. [3]

Unfortunately, a large number of patients will present with foot deformities, instability or ulcerations as their initial symptoms. For those patients, one can consider a variety of surgical options. [1]

The goals of Charcot reconstruction are threefold: first, correct the patient's equinus and reestablish the calcaneal inclination angle; second, maintain the rearfoot to leg relationship; and, finally, correct and stablize the degenerative joints. This can usually be performed percutaneously and without hemostasis. It is very important to allow patients to weight bear within 1 week postoperatively. This is possible with a unique combination of innovative external fixation techniques. Though this strategy is foreign and contrary to traditional thoughts and approaches to surgical intervention, external fixation allows for safe and percutaneous Charcot reconstruction. [4]

External fixation uses a system of devices that connect to the patient through the use of thin, smooth wires, threaded half-pins, or centrally threaded transfixion pins that are capable of immobilizing and rigidly fixating bone fragments. [5]

Use of fine wire external fixator frames in diabetic patients confers advantages including biomechanical stability, continuous access to soft tissues, early ability to bear weight, and

the ability to make adjustments postoperatively. Additionally, external fixation is the only option available for Charcot deformities associated with either osteomyelitis or in Eichenholtz stages I and II, in which internal fixation methods are contraindicated. [6]

External fixation allows the foot and ankle surgeon to approach Charcot foot and ankle deformities in a new way. Surgeons can now correct Charcot pathology with percutaneous techniques using a new generation of external fixation frames. These fixators allow the surgeon to adjust or manipulate the Charcot foot after surgery and the patient early weight bearing. External fixation provides the foot and ankle surgeon the opportunity to reconstruct and stabilize this destructive disease. [4]

# AIM OF THE STUDY

The aim of this study is to spot light the current thinking and surgical approaches in Charcot foot and ankle reconstruction.

# ANATOMY OF THE ANKLE AND FOOT

## A) Anatomy of the ankle

**Structure:** The ankle is a hinge joint with articulations between the tibia, fibula, and talus. The weight-bearing surface of the tibia articulates with the spool-shaped surface of the talus. The ankle has a mortise and tenon configuration, with the talus being the tenon articulation and the medial malleolus and distal fibula (lateral malleolus) forming the articulating sides of the mortise. [7]

**Ligaments of the ankle:** The ankle joint is reinforced laterally by the **lateral ligament** of the ankle, which consists of three separate ligaments:

**A)** ***Anterior talofibular ligament***, a flat, weak band that extends anteromedially from the lateral malleolus to the neck of the talus.

**B)** ***Posterior talofibular ligament***, a thick, fairly strong band that runs horizontally medially and slightly posteriorly from the malleolar fossa of the fibula to the lateral tubercle of the talus.

**C)** ***Calcaneofibular ligament***, a round cord that passes posteroinferiorly from the tip of the lateral malleolus to the lateral surface of the calcaneus.

The lateral ligament of the ankle resists inversion of the foot and may be torn during an ankle sprain.

The joint capsule of the ankle joint is reinforced medially by the large, strong **medial ligament** of the ankle (*deltoid ligament*) that attaches proximally to the medial malleolus and fans out from it to attach distally to the talus, calcaneus, and navicular via four adjacent and continuous parts; the tibionavicular part, the tibiocalcaneal part, and the anterior and posterior tibiotalar parts. The medial ligament stabilizes the ankle joint during eversion of the foot and prevents subluxation of the ankle joint. [8]

**Movements of the ankle:** The ankle joint is capable of being flexed and extended (*plantar- and dorsiflexion*). The body of the talus is slightly wider anteriorly and, in full extension, becomes firmly wedged between the malleoli. Conversely, in flexion, there is slight laxity at the joint and some degree of side to side tilting is possible.

The principal muscles acting on the ankle are:

- Dorsiflexors — tibialis anterior assisted by extensor digitorum longus, extensor hallucis longus, and peroneus tertius.
- Plantarflexors—gastrocnemius and soleus assisted by tibialis posterior, flexor hallucis longus, and flexor digitorum longus. [9]

**Stability of the ankle joint:** This is a hinge joint of great strength. Its stability is ensured by:

1- The powerful ligaments and tendons around it. (Fig. 1)

2- The insertion of the trochlea tail into the deep socket between the medial and lateral malleoli. The socket is deepened posteriorly by the inferior part of the posterior tibiofibular ligament (*transverse tibiofibular ligament*). [10]

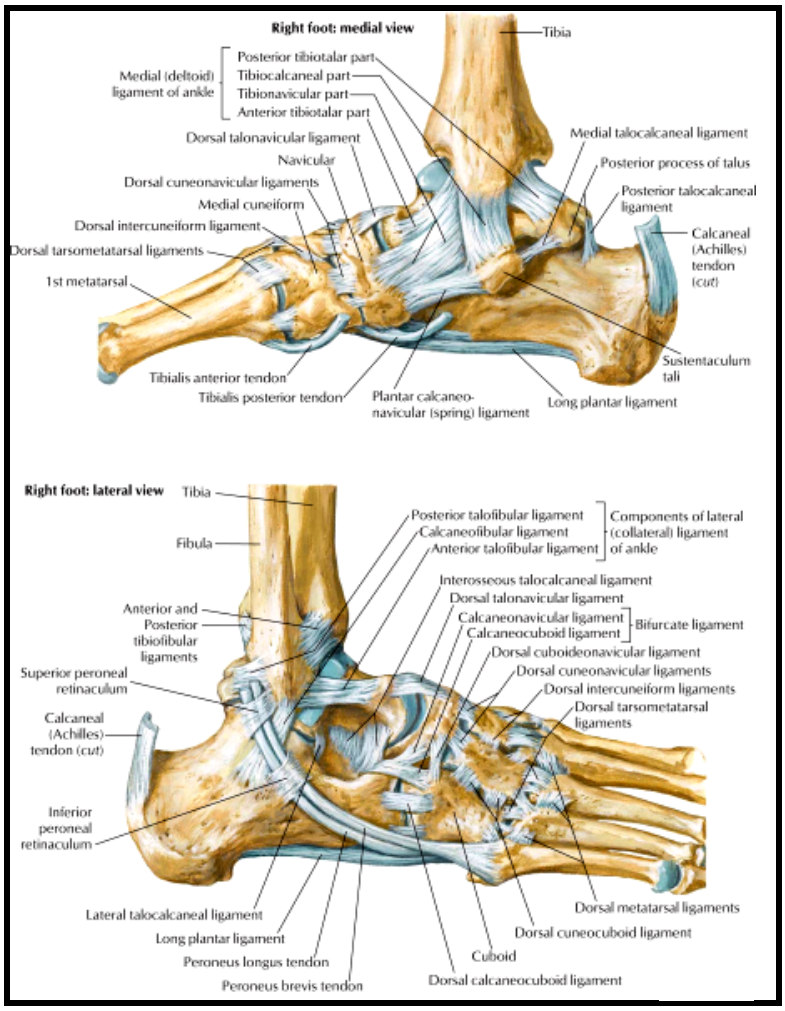


**Figure (1):** Ligaments of the foot and ankle. [7]