

Megan N. Severson and Michael D. Johnson

Background

- Definition: A destructive, non-infective process of bones and joints that occurs due to the loss of protective sensation that occurs with peripheral neuropathy
- Associations:
 - In the USA, most commonly associated with longstanding diabetes mellitus
 - Other associations include leprosy, tabes dorsalis, Charcot-Marie-Tooth, and alcohol abuse
- Patient burden: Commonly painless but associated with progressive deformity of the foot and joint instability leading to altered gait mechanics and load bearing
 - Increased risk for pressure ulcers, infections, and ultimately amputations
 - Increased patient morbidity and decreased quality of life

M.N. Severson, MD
Division of Orthopedic Surgery, Department of Surgery, University of Alabama – Birmingham Hospital, Birmingham, AL, USA

M.D. Johnson, MD (✉)
Department of Orthopaedics, University of Alabama – Birmingham, Birmingham, AL, USA
e-mail: michaeljohnson@uabmc.edu

Pathophysiology

It is not clearly understood but there are currently two theories which both hinge on the idea that the patient's continued weight bearing in the face of injury leads to progressive destruction of the involved bones and joints [2–4].

Neurotraumatic Destruction

- Joint destruction, fractures, and foot collapse occur due to cumulative mechanical trauma.
- Can be major, minor, or microtrauma that the patient does not recognize due to lack of protective sensation in the foot.
- Continued activity and weight bearing propagate the destruction and inhibit the healing process.

Neurovascular Destruction

- Trauma or another trigger causes a state of hyperemia due to vasomotor autoneuropathy.
- Dysregulation of blood flow leads to cytokine mediated bone resorption and ligamentous weakening.
- Leads to bone breakdown and joint dislocation with progressive deformity due to continued high levels of pro-inflammatory cytokines.

Presentation and Evaluation

Most present with a red, warm, swollen foot with a history of diabetes and no recollection of injury.

- Patients often present initially with a red, hot, swollen foot that is commonly misdiagnosed as cellulitis or osteomyelitis.
- Key finding in differentiating Charcot is that the erythema and swelling resolves with elevation of the limb. Osteomyelitis, cellulitis, or other infection will remain erythematous despite elevation.
- Will have evidence of peripheral neuropathy with decreased sensation with monofilament testing.
- May have concomitant vascular disease and workup with ankle-brachial indices (ABI's) and toe pressures may be warranted.
- Radiographs may show erosive changes and even new bone formation but lack the surrounding osteopenia associated with osteomyelitis. Joint subluxations and dislocations are commonly seen in advanced cases.
- Bone biopsy may be useful in differentiating between Charcot changes and osteomyelitis.

Staging/Classification

Modified Eichenholtz stages of Charcot arthropathy [5]

- Clinical/prefragmentation stage:
 - Red, hot, swollen foot with significant inflammation
 - Often confused with cellulitis and infection
 - Minimal changes on x-rays
- Dissolution/fragmentation stage:
 - Significant edema, warmth, erythema persists
 - Radiographs will show joint dislocation, bone fragmentation, and regional demineralization
- Coalescence:
 - Erythema, edema, and inflammation decreased
 - Bone debris absorbed, new periosteal bone formation, and early healing seen on x-rays

- Resolution:
 - Complete resolution of erythema, edema, and warmth
 - X-rays will show consolidation of healing with smooth bone edges and bony or fibrous ankyloses
 - Will likely have continued progression of deformity over time

Brodsky Anatomic Classification [1]

- Type 1 – involves the midfoot
 - Most common, about 60% of Charcot joints
 - Usually requires shorter treatment duration for healing
 - Progresses to classic “rocker bottom” foot deformity
 - High risk of development of pressure ulcers and associated infection
- Type 2 – involves the hindfoot joints: subtalar, talonavicular, calcaneocuboid
 - Significant instability but pressure sores and infection are less common
- Type 3 – tibiotalar joint involvement
 - Least common
 - Significant deformity, instability, and disability
 - Usually require significant bracing and/or surgical treatment

Treatment

- Goals are to preserve stability and alignment while the Charcot process evolves.
 - Initial treatment is total contact casting and protected weight bearing
 - Duration of casting depends on deformity and patient's ability to comply with non-weight bearing
 - Transition to AFO, Arizona brace, or CROW boot once initial warmth and swelling resolves
 - Shoe modification to unload pressure from bony prominences
 - If non-operative treatment fails, surgical treatment is considered. Options include

tendon Achilles lengthening, exostectomy, and fusion of affected joints

References

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