Charcot foot and Osteomyelitis in diabetes - Radiologist: Friend or Foe?

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Learning objectives

- To demonstrate the radiological spectrum of progressive destructive Charcot neuro-osteoarthropathy in the diabetic foot.

- To distinguish Charcot neuro-osteoarthropathy from osteomyelitis.

- To outline the imaging work-up to guide practitioners to the right diagnosis, and to prevent medico-legal problems.

- To clarify advances and limits of available imaging techniques.

Charcot foot is a significant problem with a rapid devastating nature. If not recognized it may lead to progressive deformity, ulceration, osteomyelitis and eventually to limb loss by amputation. The diagnosis is challenging and imaging plays a pivotal role. Charcot neuro-osteoarthropathy must be distinguished from other conditions, with efficiency and accuracy. Rapid diagnosis and early intervention is important to prevent further bone and joint destruction and safe the patient from limb loss. Since imaging plays a pivotal role in arriving at the definitive diagnosis, the radiologist 'makes the difference', in order to navigate to correct treatment strategy - plaster or surgery. This educational exhibit focuses on the value of several imaging modalities to guide the practitioners in arriving at an early diagnosis.
**Background**

Neuropathic osteoarthropathy is a rare phenomenon, making it difficult for health care providers to diagnose. It is estimated that the overall prevalence of the Charcot foot in the general diabetic population varies between 0.1%-7.5% \(^1\)\(^-\)\(^8\). However, diabetic patients with peripheral neuropathy develop Charcot neuro-osteoarthropathy at a rate of 29%-35% \(^6\).

Severe and irreversible Charcot related foot deformities will lead to foot ulceration which eventually may need amputation \(^9\). However, a rapid diagnosis and early intervention in the acute active stage of the Charcot neuro-osteoarthropathy may limit the destruction and prevent further deformation of the foot.

Clinical challenge emerges when a Charcot neuro-osteoarthropathy starts developing signs and symptoms of inflammation. To clinically differentiate Charcot neuro-osteoarthropathy from osteomyelitis is difficult, especially when an ulcer is present. Complicating the complex matter of the Charcot foot is the fact that osteomyelitis and Charcot's neuro-osteoarthropathy can coexist in the same extremity. Superimposed infection will pose a diagnostic dilemma. Despite the myriad of available diagnostic modalities, to differentiate between Charcot's and infected foot is a challenge.
Clinical presentation of Charcot neuro-osteoarthropathy in the foot:

**Acute stage.** Charcot neuro-osteoarthropathy of the foot has both an acute active and a chronic inactive phase. A hot swollen neuropathic foot, with unilateral erythema and edema, characterizes the acute active stage [Figure 1]. A (minor) trauma often precedes the clinical event. The skin temperature is at least 2 °C higher than the contralateral foot. The ankle and foot are the most common areas affected by neuropathic arthropathy, related almost exclusively to diabetic arthropathy. It commonly presents in the mid-foot but it may also occur in the forefoot and hind foot. Pain may or may not be present, depending on the nerve damage. Dry skin, sensory neuropathy, diminished vibratory sense and proprioception may also occur. The foot is unstable because of collapse of the longitudinal foot arch [Figure 2]. Differential diagnosis of the Charcot neuro-osteoarthropathy may be infection, (i.e. osteomyelitis, cellulitis, septic arthritis) or inflammation (i.e. gout), however also deep vein thrombosis is a potential mimicker.

In the acute active stage Charcot neuro-osteoarthropathy shows rapid and progressive bone and joint destruction within days [Figure 3]. These bony changes with mechanical deformities may yet be present and can be seen radiologically. In this stage prompt diagnosis and immediate treatment is necessary to prevent further bone and joint destruction. Immobility by total contact casting (with limited ambulation) should be the early intervention.

**Chronic stage.** The chronic inactive stage no longer shows a warm and red foot. The edema may persist but the difference in skin temperature between the feet is usually less than 2 °C. Crepitus, palpable loose bodies and large osteophytes are a result of extensive bone and cartilage destruction. Joint deformity and subluxation or even dislocation of the metatarsals may lead to a rocker-bottom-type deformity in which the cuboid becomes a weight-bearing structure [Figure 2,3]. The deformity of the foot with abnormal pressure distribution on the plantar surface from the diminished plantar arch, with reduced or loss of sensation and function, makes the foot vulnerable for increased pressure or (repeated) trauma, leading to excessive callus formation, blisters and foot ulceration [Figure 2D]. Direct inoculation of infection of the skin contribute further to callus formation. Foot ulceration can subsequently lead to infections, such as cellulitis and osteomyelitis, and this may eventually lead to (partial) amputation of the lower extremity.

**Infection.** Diagnostic problems start when a foot with Charcot neuro-osteoarthropathy begins to shows clinical signs of inflammation. If the patient has a red hot foot without any breakdown, no fever and a normal or slightly elevated serum C-reactive protein level or erythrocyte sedimentation rate, acute Charcot neuro-osteoarthropathy should be considered. Infection is a highly unlikely diagnosis, and an active phase of Charcot
process should primarily be considered. However, fever and elevated C-reactive protein or erythrocyte sedimentation rate may occur in the Charcot foot and an infection should always be considered.

In addition to the typical signs of infection (generalized redness, warmth, and swelling), osteomyelitis or cellulitis is more likely in the presence of a skin defect, such as a chronic ulcer or an iatrogenic wound. Although many ulcers are treated with antimicrobial therapy and debridement (if ischemic), many infected ulcers progress to more severe soft tissue infection, such as sinus tracks, severe cellulitis, and abscess formation [Figure 6-8]. Focal abscess formation is not infrequent in the diabetic population, with reports estimating that between 10% and 50% of all cases that have pedal osteomyelitis have concomitant soft tissue abscesses.\(^\text{16,17}\)

**Osteomyelitis.** Location is a guiding feature: Osteomyelitis is predisposed to occur at pressure points and areas of ulceration along bony protuberances.\(^\text{18,19}\) [Figure 2,6-10]. Consequently, the most common locations for osteomyelitis are at the metatarsal heads and at the interphalangeal joints in the forefoot [Figure 7], at the distal fibula, and at the plantar aspect of the posterior calcaneus of the hindfoot [Figure 9-10]. All these locations are not the side of predominance of neuro-osteoarthropathy, which is the midfoot [Figure 6,8]. The common pressure point in the midfoot, especially in a collapsed foot with rocker-bottom configuration is the cuboid bone: this side therefore is prone for infection [Figure 2]. These clinical manifestations often prompt referral for imaging evaluation.

Imaging of neuro-osteoarthropathy in the diabetic foot necessitates a good clinical history and often a quick examination by the performing radiological technician to locate and mark the ulcer (when present) and to optimize the quality of the study.

**Plain Radiography**

The initial screening modality is an X-ray of the foot in three directions (dorsoplantar, lateral, and pronated oblique). X-ray of the ankle should be performed in order to exclude ankle luxation. Both feet should be studied, in order to detect and compare metabolic disturbances and subtle changes. Although patients may present early in the acute active phase with normal X-ray, in Charcot neuro-osteoarthropathy an advanced early stage can be diagnosed on an X-ray film [Figure 3]. Evidence of radiological changes associated with the Charcot neuro-osteoarthropathy has been reported in up to 10% of patients with diabetes and neuropathy.\(^\text{20}\) Radiographs provide important anatomical information that is useful for the interpretation of many other studies performed. In addition, the X-ray can be used as a control exam for further follow-up.

The classic radiographic description of neuropathic arthropathy is that of the "five D's": normal bone density, joint distention, bony debris, joint disorganization, and dislocation [Figure 2,3]. The bone density is typically normal, except when the underlying density is decreased, as in elderly or type 1 diabetic patients. All patients have invariably large
joint effusions, showing joint distention on the X-ray. If debris is present, the effusions may decompress along fascial planes, carrying bony debris far from the joint. As part of joint disorganization, cartilage destruction occurs early, and erosive and productive changes coexist. Subchondral cysts may be present. Ligamentous laxity is a prominent finding, often associated with joint subluxation or dislocation. In the foot, the most frequent location for a diabetic Charcot's joint subluxation is the tarsometatarsal (Lisfranc's) joints. Other joints may be involved as well, including the talonavicular (Chopart), subtalar, and intertarsal joints [Figure 4].

On the lateral footview a detailed analysis of the angular relationships of the bones can be conducted. This is important for monitoring the progression of the deformity. Often, the calcaneal inclination decreases with equinus deformity at the ankle [Figure 2]. The tarsometatarsal joint is the most commonly involved joint. Subluxation of the first and second tarsometatarsal joints is efficiently evaluated on a dorsoplantar film of the foot, whereas subluxation at the third through fifth tarsometatarsal joints is best evaluated on a pronated oblique foot film [Figure 3 A-C]. The pronated oblique exam gives significant additional information about the plantar and dorsal borders of the forefoot and mid-foot, such as periostal reaction or cortical interruption.

**Osteomyelitis.** When reading plain radiographs, a radiologist is trained to assess certain features that discriminate normal from abnormal. In case of osteomyelitis however, the radiological changes may remain absent until 7-10 days. In case of the warm swollen neuropathic foot, the radiographic characteristics of neuro-osteoarthropathy and osteomyelitis, such as de-mineralization, destruction and periosteal reaction of the bones, almost always overlap, particularly when neuropathic osteoarthropathy presents in its later stages. Thus, to differentiate (diabetic) neuro-osteoarthropathy from osteomyelitis is difficult, using radiological characteristics.

**Magnetic Resonance Imaging**

MRI is known for its superb anatomical and pathological detail of soft tissue, ligaments and bone marrow, and it is useful for guiding surgical management. Thus the second step in analyzing the acute Charcot foot is (contrast enhanced) MRI. Coil choice is critical. A small surface coil is ideal for examining the forefoot and toes, yet the extremity coil should be used when the ankle, midfoot and hindfoot are imaged (i.e. chimney-type knee coil). Simultaneously imaging of both feet and ankles should be avoided; imaging quality will decrease dramatically. MRI protocols of the foot vary widely, however, Chatha et al presented clearly imaging protocols for the forefoot and ankle. The images are usually obtained with a small field of view (8-10 cm), FOV 12-16, with thin sections (3-4 mm) to optimize spatial resolution. Tailoring to the situation is mandatory.

The imaging protocol should include both T1 (defining anatomy) and STIR or T2 fatsat (detecting pathology) sequences. Scanning planes should be tailored for each
individual examination. As a rule of thumb, sagittal views are ideal for evaluation of midfoot neuropathic involvement, the plantar surface, and the posterior calcaneus. Axial and coronal planes are useful in the evaluation of the ankle and the surrounding tendons. Sagittal planes and a view parallel to the toes are adequate for imaging the metatarsophalangeal and interphalangeal joints.

Although there is no convincing evidence that contrast improves the diagnostic accuracy for osteomyelitis, the use of contrast (gadolinium 0.1 mmol/kg i.v.) is recommended. Its use better depicts devitalized regions, affected soft tissue with abscesses and sinus tracts, and joint or tendon involvement. Understanding of surgical changes and areas of magnetic susceptibility, which can obscure regions of interest, may be facilitated by contrast administration.

In the early phase of the acute stage, conventional radiology appears normal, but the MRI shows subchondral bone marrow edema with or without microfracture. The more advanced stage of the acute hyperemic Charcot neuro-osteoarthropathy is characterized by edema and swelling of soft tissues and muscles, and edema of bone marrow, without definite bone destruction. Especially the bone marrow edema typically is not restricted to one or two bones, yet is seen in the entire area of pathology, thus the entire midfoot. Bone marrow edema and its enhancement are typically centered in the subchondral bone suggesting the relation to joint disease. The joint capsule and adjacent soft tissues enhance on postcontrast images but the subcutaneous tissues typically show little enhancement. Joint effusion is frequently present. In patients with deformities of chronic osteoarthropathy, edema and enhancement of periarticular soft tissue and bone marrow may reflect more recent injury, or acute instability, suggesting superimposed acute form of neuropathic arthropathy.

Osteomyelitis. Primary imaging signs for osteomyelitis are focally decreased marrow signal intensity on T1, focally increased signal intensity on fat-suppressed T2 and STIR, and focal marrow enhancement on gadolinium-enhancement fat-suppressed T1 weighted images. Earlier described secondary MRI signs of osteomyelitis are the presence of a cutaneous ulcer, cellulitis, a soft tissue mass, a soft tissue abscess, a sinus tract, and cortical interruption. It remains a challenge to discriminate active Charcot neuro-osteoarthropathy from acute osteomyelitis. This is simply the case because MR imaging features are quite similar. Both entities will show subchondral bone marrow edema, joint effusions, soft tissue inflammatory changes (high signal intensity on T2 fatsat or STIR, somewhat low signal intensity on T1) and enhancement of bone and soft tissue after contrast administration. Therefore frequently an active Charcot foot is diagnosed as osteomyelitis. This is a potential disaster, since therapeutic approaches of both conditions are extremely different. An active phase of Charcot neuro-osteoarthropathy should be treated with plaster or total contact casting. However, when osteomyelitis is present, treatment consist of intravenous antibiotics and sometime surgical debribement.
of soft tissue abscesses, low to intermediate signal on T1-weighted images and high signal on T2-weighted images, with rim enhancement also needs addressing.

As the differentiation based on MR examinations of infected from noninfected neuro-osteoarthropathy is difficult, the presence of secondary signs is extremely helpful. A sinus tract, with tramtrack enhancement, disappearance of subchondral cysts, diffuse bone marrow abnormality, and bone erosions are in favor of infection. The important issue is to localize the skin or subcutaneous abnormality (e.g. ulcer, sinus track) and its relation to the area of signal abnormality. In order to maximize the detecting of this relation, the radiological technician should mark where the ulcer is located and tailors the imaging protocol accordingly. Close interaction with the MR-technician is advised. Once the ulcer is detected, the relation between ulcer and abnormal bone becomes clear and the diagnosis is readily made. Additionally, the "ghost sign" may be potentially useful, in which areas of osteomyelitis are more pronounced visible on contrast enhanced T1-weighted series, when compared to native T1-weighted series. Also the presence of cellulitis, defined as a loss of fat signal in subcutaneous tissue and high signal (although less than water) with diffuse inhomogeneous enhancement and poorly defined margins, is in favor of osteomyelitis.

A special area of evaluation in the rocker bottom foot is the cuboid bone, being an important location of osteomyelitis in the midfoot. If the T1-weighted image at that location shows low signal intensity, osteomyelitis is extremely likely.

**Computed Tomography**

The use of Multi Detector Computed Tomography (MDCT) in neuropathic osteoarthropathy is not well investigated, and literature is dated with only use of single slice CT. CT may show cortical destruction, periosteal new bone formation and small foci of gas within bone better than MRI, but it cannot distinguish between purulence, granulation tissue, inflammation or fibrosis. In our opinion the use of MDCT can be important, and is considered an additional work-up modality subsequently used following to the already performed MRI. The abnormal MR signals are not always particularly clear in delineating the frequently encountered anatomic deformities, structural bone damage, fractures, sequestra, (sub)luxations, or bone callus formation. Detecting the aforementioned osseous pathologies may need additional therapy. Also, in daily practice it enables clinicians easier assessment than MRI. Furthermore, MDCT with its reconstruction and even three-dimensional computer generated images is most useful in the preoperative planning.

The imaging CT-protocol for the fore- and midfoot should be coronal acquisition (0.6 mm), and sagittal and transversal reconstruction. When MDCT is performed, preferably both feet should be scanned, to determine bilateral deformities and right-left differences. On
the sagittal planes, as in plain radiography, a detailed analysis of the angular relationships of the bones can be conducted to diagnose the extent of the deformities. Subluxation of the tarsometatarsal joints is best evaluated on transverse and coronal planes of the foot. The descriptive “five D's” - normal bone density, joint distention, bony debris, joint disorganization, and dislocation - can be adapted from plain radiography description.

An indication of MDCT in neuro-osteoarthropathy in our daily practice is the follow up of immobilization therapy. When both clinical assessment as well as conventional radiography are not clear, the use of (follow up) MDCT enables analysis of the "five D's". Furthermore, bone healing and change of active periosteal reaction will proceed into inactive periosteal reaction, referred to as "cooling down of the bone", enables tailoring of the treatment [Figure 11].
Fig. 0

2. Charcot neuro-osteo arthropathy

A. Normal bone density is shown without joint distention, bony debris, joint disorganization, or dislocation in a patient with clinical complaints: no radiological characteristics of neuro-osteoarthropathy.

B-D. Rapid (within 4 months) progressive decrease of calcaneal inclination with equinus deformity at the ankle is shown, following tarsometatarsal joint destruction (B-D); the ‘rocker bottom’ deformity. The characteristic ulceration beneath the bony protuberance of the cuboid (D and inlay) is shown.

Fig. 0

Fig. 0

Fig. 0

5. Charcot neuro-osteo arthropathy

Two sagittal MRI footviews of a patient with advanced acute hyperemic Charcot neuro-osteoarthropathy shows edema and swelling of soft tissues and muscles, and edema of bone marrow. Especially the bone marrow edema typically is not restricted to one or two bones, yet is seen in the entire area of the midfoot. (STIR - short tau inversion recovery).

Advanced stage of active Charcot neuro-osteoarthropathy.

Bone marrow edema and its enhancement are typically centered in the subchondral bone suggesting the relation to joint disease. The joint capsule and adjacent soft tissues enhance on postcontrast images (T1 +) but the subcutaneous tissues typically show little enhancement. There is no ulcer present. (T1 turbo spin echo without contrast (T1 -), and T1 turbo spin echo with contrast (T1 +)).

Fig. 0

Charcot neuro-ostearthropathy with secondary signs – a plantar ulcer.

Sagittal MRI footviews of a patient with active Charcot neuro-ostearthropathy. The presence of secondary signs is extremely helpful in the differentiation of neuro-ostearthropathy with or without osteomyelitis. This patient with active Charcot neuro-ostearthropathy had a plantar ulcer at the bony protuberance of the cuboid and proven osteomyelitis. Similar to non-infected Charcot neuro-ostearthropathy, infected Charcot neuro-ostearthropathy shows edema and swelling of soft tissues and muscles, and edema of bone marrow in the cuboid. Enhancement of the cuboid bone and adjacent soft tissues on postcontrast images (T1+ and T1 FS+), together with the plantar ulcer makes osteomyelitis on MRI images very likely.

(STIR: short tau inversion recovery, T1 turbo spin echo without contrast, T1 turbo spin echo with contrast (T1+), and T1 turbo spin echo with fat sat and contrast (T1 FS+))

Fig. 0

Sagittal MRI footviews of a patient with Charcot neuro-ostearthropathy. The important issue is to localize the skin or subcutaneous abnormality: high signal intensity on STIR, low or intermediate signal on intensity T1-, and high signal intensity on T1+. This patient has a small cutaneous defect with subcutaneous edema at the metatarsals. An abscess is shown in the forefoot, with high signal intensity on STIR, low or intermediate signal on intensity T1-, and (ring) enhancement of the borders showing high signal intensity on T1+.

STIR (short tau inversion recovery), T1 turbo spin echo without contrast (T1-), and T1 turbo spin echo with contrast (T1+).

**Fig. 0**

8. Charcot neuro-osteopathic arthropathy

Charcot neuro-osteopathic arthropathy with secondary signs - a subcutaneous fistula tract.

Sagittal MRI footviews are presented in a patient with Charcot neuro-osteopathic arthropathy. This patient has a subcutaneous edema and swelling, a fistula tract (‘tramtrack’ sign) at the bony protuberances of the cuboid without any bone edema at the midfoot. This makes osteomyelitis unlikely. The use of contrast depicts quite well affected soft tissue with abscesses and sinus tracts.

STIR (short t2 inversion recovery), T1 turbo spin echo with fat saturation (T1 FS +)

Fig. 0

Fig. 0

10. Charcot neuro-osteoarthropathy

Lateral view in a diabetic patient with a hindfoot subcutaneous ulcer and sagittal and axial MRI footviews are presented in the same patient, with progressive osteomyelitis, which was proven by microbial culture, is shown at two different sections. MRI characteristics of osteomyelitis are similar to Charcot neuro-osteoarthropathy, with bone marrow edema and soft tissue inflammatory changes (high signal intensity on T2 fatsat or STIR, somewhat low signal intensity on T1) and enhancement of bone and soft tissue after contrast administration. The “ghost sign” may be potentially useful, in which areas of osteomyelitis are more pronounced visible on contrast enhanced T1-weighted series, when compared to native T1-weighted series.

STIR (short tau inversion recovery), T2 turbo spin echo with fat sat (T2 FS), T1 turbo spin echo without contrast (T1 -), T1 turbo spin echo with contrast (T1 +), and T1 turbo spin echo with fat sat and contrast (T1 FS +).

Fig. 0

Fig. 0

Conclusion

Imaging plays a pivotal role in arriving at the definitive diagnosis of Charcot neuro-osteoarthropathy. It remains a challenge to discriminate active Charcot neuro-osteoarthropathy from acute osteomyelitis, because imaging features are quite similar for both entities.

It is crucial to correct interpret the radiological characteristics of Charcot neuro-osteoarthropathy and osteomyelitis, but also to look beyond radiological features and integrate clinical history, anatomic location of pathology, and the presence of skin defects in the reading process.

This means that teamwork is essential, in which the reading radiologists actively interact with both referring clinicians as well as the MR technologists.

With the tools gained in this educational exhibit, the radiologist 'can make the difference' and guides the clinician to the correct diagnosis and treatment strategy - being a "friend" instead of a "foe".
References


