The Charcot foot: What does the Radiologist need to know?

Poster No.: P-0018
Congress: ESSR 2018
Type: Educational Poster
Authors: C. Loupatatzis\textsuperscript{1}, M. C. Berli\textsuperscript{2}, C. W. A. Pfirrmann\textsuperscript{3}, A. Rosskopf\textsuperscript{2}; \textsuperscript{1}CH, \textsuperscript{2}Zurich/CH, \textsuperscript{3}Zürich/CH

Keywords: Musculoskeletal joint, Musculoskeletal bone, Musculoskeletal soft tissue, MR, Plain radiographic studies, Diagnostic procedure, Staging, Imaging sequences, Infection, Inflammation, Metabolic disorders

DOI: 10.1594/essr2018/P-0018

Any information contained in this pdf file is automatically generated from digital material submitted to EPOS by third parties in the form of scientific presentations. References to any names, marks, products, or services of third parties or hypertext links to third-party sites or information are provided solely as a convenience to you and do not in any way constitute or imply ECR's endorsement, sponsorship or recommendation of the third party, information, product or service. ECR is not responsible for the content of these pages and does not make any representations regarding the content or accuracy of material in this file.

As per copyright regulations, any unauthorised use of the material or parts thereof as well as commercial reproduction or multiple distribution by any traditional or electronically based reproduction/publication method ist strictly prohibited.

You agree to defend, indemnify, and hold ECR harmless from and against any and all claims, damages, costs, and expenses, including attorneys' fees, arising from or related to your use of these pages.

Please note: Links to movies, ppt slideshows and any other multimedia files are not available in the pdf version of presentations.

www.myESR.org
Learning objectives

- To review the etiology, course of disease and treatment of the Charcot foot
- To recapitulate the classifications of this disease and discuss the need for a new one
- To demonstrate the important role of imaging, especially MR-Imaging in early diagnosis, monitoring and evaluation of complications in Charcot foot
Background

Introduction

The Charcot foot or Charcot neuropathic osteoarthropathy (CN) has been first described 1868 by Jean-Martin Charcot, a French pathologist and neurologist, in patients with tabes dorsalis, a syphilitic myelopathy. Nowadays, the detailed pathomechanisms of this disease remain unclear, but there is consensus that polyneuropathy is the underlying basic condition of this disease. In industrialized countries diabetes mellitus is the main cause of neuropathy in the lower limb. The prevalence of CN in a general diabetic population is estimated between 0.1% and 7.5%, but regarding diabetic patients with apparent peripheral neuropathy, this prevalence is increasing up to 35%. The risk of getting a Charcot foot is not related to the type (I or II) of diabetes mellitus. Nevertheless, there are much more causes than diabetes for neuropathy and therefore for CN, e.g. alcohol abuse, heavy metal poisoning, traumatic injury, multiple sclerosis, malnutrition (Vit. B12 and folic acid deficiency) and many more. That's why the often-used term diabetic neuropathic osteoarthropathy (DNOAP) is only partially correct. The incidence of bilateral involvement of the feet in CN has been reported to be between 9% and 75%. The mean age of patients with CN present is in their fifties or sixties and most of them have had diabetes mellitus for at least 10 years.

Course of disease

Pedal CN has an active phase (inflammation and fragmentation) and a chronic inactive phase (coalescence and consolidation). The active phase is characterized by a hot, red and swollen foot (=inflammation), often without pain, due to the polyneuropathy (Fig. 1). A (minor) trauma is often reported prior to the onset of CN. The skin temperature on the affected foot is reported to be more than 2°C higher than on the contralateral foot. But this sign might not always be reliable in clinical practice because of its susceptibility to environmental influences. Furthermore, it can only be used reliably in unilateral Charcot foot involvement.

In the active phase the bone gets fragile (=fragmentation) due to temporary osteopenia leading to fractures and even to the collapse of the longitudinal arch of the foot, if loaded unprotected. This can occur in a very short period of time (Fig. 2). Differential diagnosis for an active Charcot foot may be infection (especially osteomyelitis), stress-fracture and physical overload in cases with fat pad atrophy.
The chronic inactive phase of CN shows no longer a warm, red foot, although some soft tissue edema may last. Prominent osteophytes and palpable loose bodies are the consequence of a substantial joint and bone destruction followed by bone proliferation (coalescence and consolidation). Due to the biomechanical roll-movement of the foot during the gait sequence, the foot often presents with a so called "rocker-bottom-deformity". This deformity is a result of deformity and subluxation/dislocation of the metatarsal bones in the Lisfranc-Joint: he cuboid bone becomes the most inferior bone in the foot (Fig. 3). The resulting changes of the pedal shape can lead to bony prominences (so called pseudoexostoses).Combined with the reduced loss of sensation in polyneuropathy, and a diminution of the elasticity of the soft tissue (due to alterations in the collagen structure), the foot is prone to extensive callus formation, blisters and ulcerations. This can lead to infections like cellulitis and osteomyelitis, which may result in amputation.

For a long time, there was the leading opinion, that once the Charcot foot activity has disappeared, recurrence of CN activity in the same foot was unlikely. However, newer studies show that CN recurrence rates are about 23% with a mean interval of 27 months (Fig.4).

Pathogenesis

The exact pathomechanism for the development of CN is not known yet and is thought to be multifactorial (Fig. 5). Both two main theories that have been discussed in the past are based on the presence of neuropathy. One is focusing on the neurovascular changes in blood supply (neurovascular theory) the other one on repetitive traumatic events due to the lack of sensory feedback (neurotraumatic theory). One current model of CN origin supports the hypothesis that once the disease is triggered in susceptible individuals e.g. due to a fractured bone, an unregulated inflammatory process is started, which leads to an abnormally intense stimulation and maturation of osteoclasts. Increased osteoclastic activity is responsible for the unrestrained bone turnover which causes more bone damage. This bidirectional relationship between inflammation and repetitive traumata has been shown to be essential.

Nevertheless, it still remains inexplicable why CN is a quite rare condition, mostly unilateral and usually a self-limiting process.

Treatment
Current state-of-the art treatment is the off-loading of the affected foot. The goal is to maintain a plantigrade foot with minimal deformity to avoid increased pressure points. There are several off-loading techniques, which depend on the availability of the infrastructure and the training of the treating staff. One commonly used method is the treatment of patients with custom-made removable total contact casts (rTCC) until the activity signs of the Charcot foot are significantly reduced or gone (Fig. 6). This might take up to 18 months, therefore compliance is a major issue. So far, there is no clinical value, which would allow to determine the completed healing process. Monitoring of the disease activity using MRI (e.g. in 3-month-intervals), gives the opportunity to decide when the time has come to end the off-loading treatment. After the off-loading treatment, the patients should be equipped with orthopedic shoes, which are professionally adapted to their feet [7].

The stabilization with the is considered as an alternative treatment option for the off-loading (Fig. 7 and Fig. 8). It is particularly used in feet with severe deformity or after the removal of osteomyelitic bone fragments. Surgical treatment options like reconstructive surgery in a stable, nonplantigrade foot or resection of bony prominences in a stable plantigrade foot are available. Major amputations need to be done in case of severe bone destruction including osteomyelitis or failed previous surgery. The main goal of all reconstructive procedures is to form a foot, which can be adequately equipped with an orthopedic shoe or an orthotic device.
Fig. 1: A typical case with a red, hot and swollen right foot in the active phase of Charcot neuropathic osteoarthropathy; same patient in two different views (a, b)

© Balgrist University Hospital

Fig. 2: Charcot foot with slight subluxation at the level of the Lisfranc's joint (yellow arrow) (a). Within 5 weeks dramatic collapse of the longitudinal foot arch with superior dislocation of the metatarsals and fragmentation of the tarsal bones (red arrow)(b)

© Balgrist University Hospital

Fig. 3: Rocker-bottom deformity of the foot. End-Stage of Charcot foot. a: clinical image, b: corresponding lateral radiograph
Natural Course of Disease

- Inflammation (normal x-ray)
- Fragmentation
- Coalescence
- Consolidation

Fig. 4: Charcot foot: natural Course of Disease with recurrence rates about 23%

© Balgrist University Hospital
Fig. 5: Multifactorial pathogenesis of Charcot foot (adapted from [8])

© Balgrist University Hospital
Fig. 6: Removable total contact cast (rTCC) used for off-loading treatment of active Charcot foot

© Balgrist University Hospital
Fig. 7: Ilizarov-Fixation in a patient with Charcot Foot. a: clinical image, b: corresponding lateral radiograph

© Balgrist University Hospital
Fig. 8: Lateral radiograph of the foot in a patient with CN before (a) and after treatment (b) with Ilizarov-Fixation. Note the significant improvement of longitudinal foot arch.

© Balgrist University Hospital
Classifications

The Charcot neuropathic osteoarthropathy has been classified in various systems using anatomical landmarks on x-rays and clinical symptoms.

Sanders and Frykberg Classification

Sanders and Frykberg identified 5 zones of disease distribution according to their anatomical location, as demonstrated in Fig. 9. Zone I: metatarsophalangeal and interphalangeal joints (Fig. 10), Zone II: tarsometatarsal joints (Fig. 11), Zone III: tarsal joints (Fig. 12), Zone IV: ankle and subtalar joints (Fig. 13), Zone V: calcaneus (Fig. 14). Most commonly involved are zone II (tarsometatarsal articulations) in about 45% and zone III (cuneonavicular, talonavicular and calcaneocuboid articulations) in about 35 % of CN cases.

Brodsky Classification

The Brodsky classification is another anatomy-based classification. Brodsky differentiated 4 anatomical areas mostly affected in CN as shown in Fig. 15. Type 1 (metatarsocuneiform and naviculocuneiform joints) is considered the most common (60%) and is often associated with symptomatic exostosis. Type 2 (subtalar, talonavicular or calcaneocuboid joints) is the second most common type (30-35%), symptomatic exostosis is less often observed here. Type 3 is divided into two subtypes: Type 3a (ankle) is seen in about 9% of CN cases, Type 3b (Calcaneus) is only seen in about 2% with the clinical manifestation being a pathologic fracture of the calcaneus tuberosity.

Eichenholtz Classification

Traditionally, the natural history of CN has been divided into three stages according to the Eichenholtz classification. However, the classification does not include the whole range of the disease, therefore the addition of the clinically important stage 0 has been proposed in the literature, the so called "pre-stage 1" or "Charcot in situ" (Table 1).
Fig. 16 - 18 show the radiographic appearance of left foot in the same patient through the stages 1, 2 and 3 according to the Eichenholtz classification. Stage 1 (Fig. 16), Stage 2 (Fig. 17), Stage 3 (Fig. 18).

Because of the missing radiographic findings, patients in Stage 0 are often misdiagnosed with other diseases, like gout, cellulitis or deep vein thrombosis. MRI allows an early diagnosis, which is crucial, because early off-loading in patients with CN Stage 0 can prevent the progression into higher debilitating stages.

Limitations of Eichenholtz Classification. Need for MRI-Classification?

Since medical imaging technologies rapidly advance, Chantelau and Gruetzner proposed to replace the classic Eichenholtz classification with a newer one, including especially MRI features.

Limitations of the Eichenholtz classifications are:

- Eichenholtz-Stages do not correlate well with the clinical symptoms of CN
- Eichenholtz-Stages do not cover the whole spectrum of CN and adding a Stage 0 as proposed by Mautone and Naidoo does not differentiate between an early active stage and a properly healed inactive stage
- Plain radiographs are not capable of diagnosing acute pathologies like cortical foot fractures, which often are only visible on CT or MRI scans. The bone marrow edema pattern can exclusively be detected on MRI.

Chantelau and Gruetzner proposed a new classification which is divided in two stages and two grades as seen in Table 2.

This MRI-classification offers a possibility to predict the outcome. Grade 1 stages usually take longer to heal than grade 0. Therefore, a longer duration of off-loading is needed. Once healed, grade 1 cases show a higher degree of deformity and foot function is much more reduced.

Because of the significance of MRI-Imaging in the management of Charcot neuropathic osteoarthropathy classification and grading systems are necessary and should be further developed for future use in daily routine.

Role of conventional radiographs
X-Rays of the Charcot foot are traditionally the standard imaging technique to establish the diagnosis, to stage and to monitor the disease. But the main value of plain radiographs is to assess the position of the bones to each other in general, and in particular under load (Fig. 19). Loading techniques in advanced imaging modalities like CT and MRI are still not sufficient to replace radiographs, because of their limited availability and the difficulty concerning standardization.

Typical measurements that help to determine the severity of deformation in Charcot foot, especially in follow up studies are: 1) Meary's angle or lateral talo-first metatarsal angle (angle between the line originating from the center of the body of the talus, bisecting the talar neck and head, and the line through the longitudinal axis of 1st metatarsal), which normally should be around 0°, 2) cuboid height (perpendicular distance from the plantar aspect of the cuboid to a line drawn from the plantar surface of the calcaneal tuberosity to the plantar aspect of the 5th metatarsal head) and 3) calcaneal pitch (angle between a line extending from the plantar aspect of the calcaneus to the plantar surface of the 5th metatarsal head and the line extending from the most plantar portion of the calcaneal tuberosity to the most plantar portion of the anterior calcaneus) (Fig. 20).

Dorsoplantar (dp) radiographs can reliably show the (sub)luxation in the Lisfranc's joint, especially the medial aspect of the joint (Fig 21). Oblique conventional radiographs are superior to dp-radiographs in visualizing the lateral aspect of the Lisfranc's joint (3rd to 5th tarsometatarsal joint) (Fig. 18). Dorsoplantar radiographs in follow-up studies show the increase in forefoot abduction relative to the hindfoot over time, the so-called hindfoot-forefoot angle (Fig. 21). As Hastings at al. showed there are 3 steps to get this measurement done: 1) quantify the talocalcaneal angle, which is the angle formed between the line that bisects the talar neck and head and the line parallel to the lateral cortex of the calcaneus, 2) bisect the talocalcaneal angle, 3) get the hindfoot-forefoot angle, which is the angle between a line parallel to the bisector of the talocalcaneal angle and a line through the longitudinal axis of the 2nd metatarsal (Fig. 21).

**Role of Magnetic Resonance Imaging**

MRI is the standard procedure in order to establish an early diagnosis in CN (when x-rays still appear normal). MRI also allows to determine the course of the healing process and the success of the off-loading treatment. Another very significant role of MRI is its ability to further evaluate complications of a Charcot foot, in particular soft tissue infections and osteomyelitis (Fig. 22).

As already mentioned above, there is no established MRI classification system. First attempts have been demonstrated by Chantelau and Gruetzner. The main benefit of MRI is to describe the course of the disease, since it is the only reliable qualitative method to
image changes inside of the bones, e.g. bone marrow edema. Most of the other options, like nuclear medicine techniques, are less sensitive.

**MRI-Protocol**

In order to assess all important information about the Charcot foot by MRI, the protocol doesn't need to be extensive. Essential is the use of large Field of View (FoV) to get a good impression of the entire foot. It is necessary to use a fluid sensitive sequence, like STIR (short tau inversion recovery) for assessing edema in the bone marrow and soft tissue. A classic T1 TSE (turbo spin-echo) sequence is irreplaceable to demonstrate the anatomy and the fat signal of the bone marrow. T2 weighted sequences can demonstrate the presence of subchondral cysts and help to identify fluid collections and sinus tracts. Especially sagittal large FoV images give a quick superior impression about the overall situation. Axial images are useful to assess the Lisfranc's joint disease. An MRI protocol proposal for Charcot foot evaluation is demonstrated in Fig 23.

**MRI-benefit 1: Diagnosis of early stage Charcot neuropathic osteoarthropathy**

Establishing an early diagnosis and therefore an early off-loading treatment is crucial for the prognosis and outcome of an acute Charcot foot (Fig. 24).

**MRI early stage Charcot neuropathic osteoarthropathy**

MRI is the best imaging modality to confirm diagnosis of suspected early active Charcot disease. Early signs of a Charcot foot in MRI are (subchondral) bone marrow edema, soft tissue edema, involvement of the Lisfranc's joint, joint effusion, and microfractures (subchondral). It is useful to directly assess the Lisfranc ligament (which runs from the medial cuneiform to the second metatarsal bone) which will be affected prior to Lisfranc's joint subluxation. No cortical fractures and no gross deformity is to be seen (Fig. 25).

**MRI-benefit Nr. 2: Monitoring of disease activity**

**MRI of late stage Charcot neuropathic osteoarthropathy**

MRI of late stage disease of Charcot neuropathic osteoarthropathy shows less or complete regression of bone marrow edema. Joint destruction and joint dislocations are
present. Especially the involvement of Lisfranc's joint leads to a typically superior and lateral dislocation of the metatarsals leading to a complete collapse of the longitudinal arch (Fig. 26). The talus head is tilted towards the sole of the foot (Fig. 27), the navicular bone dislocates into a medial position. The cuboid bone becomes the most inferior and therefore most prominent part of the foot on the plantar side, leading to an increased weight-bearing stress in the cuboid bone. Beneath the cuboid bone callus and ulcer formation is common. Prominent well-marginated subchondral cysts are a typical feature of the Charcot foot (Fig. 28b). Gross cortical fractures are present (Fig. 29), especially calcaneus fractures are common. Bone proliferation / sclerosis, debris and intraarticular bodies can occur.

**Monitoring of disease activity with MRI**

MRI is the best modality to monitor the disease activity of CN. As long as a significant amount of bone marrow edema is seen on MRI, consequent off-loading therapy with removable total contact casts (rTCC) has to be continued. After a significant decrease or complete disappearance of bone marrow edema, the next step in the therapy can be done, with installation of the orthopedic footwear (Fig. 30).

**MRI-benefit Nr. 3: Imaging of Complications: Infection/Osteomyelitis**

MRI has been shown to have a high diagnostic accuracy in diagnosing osteomyelitis of the foot, with a high sensitivity (77-100%) and high specificity (80-100%) rate. MRI has in particular a very high negative predictive value (98%): if there are no signs of osteomyelitis on MRI you can practically exclude osteomyelitis. Besides making the diagnosis, MRI is capable of defining the extent of the infection. Although in the presence of a Charcot foot discriminating active CN from acute osteomyelitis remains challenging. Both entities have similar image characteristics like bone marrow edema, soft tissue edema, joint effusions, fluid collections, and contrast-enhancement in bone marrow and soft tissues. Even the degree of signal drop in T1 sequences might be quite similar in both conditions. But making the correct diagnosis is crucial to prevent a potential disaster because the therapeutic approach is very different. Active CN needs off-loading therapy while osteomyelitis requires antibiotic therapy and possibly surgical debridement or amputation. There are some imaging features that help the radiologist to find the correct diagnosis - as listed in table 3 (Fig. 31, Fig. 32).

**Advanced MR-Imaging techniques**
The usefulness of MR-Imaging in differentiation between active phase of neuropathic arthropathy and osteomyelitis has been shown in the past. Unfortunately, there is still some overlap in the imaging appearance between both entities. Due to technical progress there are more and more advanced MR-Imaging techniques available with the possibility to add functional and quantitative information.

Diffusion-weighted imaging may contribute in the detection and extension of osteomyelitis: pure edema does not show diffusion restriction, whereas the presence of pus and inflammatory cells in infection leads to restricted diffusion with lower ADC-values than in pure edema. Dynamic Contrast Enhancement (DCE) Perfusion may help in the discrimination between viable tissue and necrosis. Furthermore, the enhancement pattern in DCE-Perfusion seems to be different between osteomyelitis and osteoarthropathic changes, increasing the potential of differencing lesions with bone marrow edema. Diffusion Tensor Imaging (DTI) may have the potential in evaluating peripheral nerve disease, although it is a very challenging technique, especially in the foot. MR Angiography (MRA) allows to non-invasively evaluate peripheral vascular impairment, which is frequently observed in diabetic patients. This can be done by contrast-enhanced 3D-MRA and even without contrast using nonenhanced MRA (gated 3D fast spin-echo MRA based on the acquisition of images during the diastolic and systolic phases of the cardiac cycle).

MRI-Limitations

There are some strict contraindications for MRI studies in general, e.g. implanted electronic devices like heart pacemakers (in particular older types), insulin pumps, neurostimulators and implanted hearing aids but also some types of intracranial metal clips and metallic foreign bodies close to the eye. Therefore, not all patients may undergo an MRI examination. Claustrophobia might be a limitation as well, but fortunately examinations of the foot are usually well tolerated, because the main part of the body and especially the head of the patient are positioned outside the scanner during the examination. Alternatively in those patients, CT and nuclear medicine imaging can be performed. In comparison to other radiological techniques the main disadvantage of the MRI is, that the images cannot be obtained under load. Therefore, there is still a need for conventional radiographs. In some cases, the MRI might not be able to distinguish severe charcot-activity from osteomyelitis with the need for additional biopsy performance or nuclear imaging methods.

Special Case 1
A 58-year-old male presented in the emergency department with a sudden shortening of the left leg. The history of the patient revealed a long-standing insulin-dependent diabetes mellitus (IDDM) type I for 26 years with polyneuropathy (Fig. 33, 34, 35).

Special Case 2

66-year-old male patient with known Charcot foot and insulin-dependent diabetes mellitus type II present with a new ulcer formation at the tip of the second and at the fifth toe (Fig. 36, 37).
Images for this section:

Fig. 9: Anatomical distribution in the Sanders and Frykberg classification. Zone II and III are most commonly involved in the Charcot foot (corresponding percentage distribution noted in the red fields)

© Balgrist University Hospital
Fig. 10: Radiographs of the left foot in dp (a) and oblique projection (b) in a CN patient involving Zone I according to Sanders and Frykberg classification mainly involving the metatarsophalangeal joints with multiple fractures (white arrows)

© Balgrist University Hospital
Fig. 11: Radiographs of the right foot in dp (a) and lateral projection (b) involving Zone II (same patient as in Fig. 10). Note the involvement of the tarsometatarsal articulations (white arrows) with lateral subluxation of the metatarsal bones in the Lisfranc's joint. This is the most common zone involved in CN

© Balgrist University Hospital
**Fig. 12:** Lateral radiograph of the left foot in a patient with CN involving Zone III according to Sanders and Frykberg classification (tarsal joints). The white arrow shows the typical inferior luxation of the talar head; the red arrow shows the cuboid, becoming the most inferior bone of the foot.

© Balgrist University Hospital

**Fig. 13:** Radiographs of the right foot in a CN patient in dp (a) and lateral (b) projection. According to Sanders and Frykberg classification there is an involvement of Zone IV (ankle joint destruction, white arrows) and Zone II (tarsometatarsal joints, red arrows) present.

© Balgrist University Hospital
Fig. 14: Lateral radiograph of the left foot (a) and corresponding sagittal T1 weighted MR image (b) showing the involvement of Zone V according to Sanders and Frykberg classification. The white arrow points to the calcaneal fracture.

© Balgrist University Hospital
Fig. 15: Anatomical distribution in the Brodsky classification: Type 1: metatarsocuneiform and naviculocuneiform joints, Type 2: subtalar, talonavicular or calcaneocuboid joints, Type 3a: ankle, Type 3b: calcaneus

© Balgrist University Hospital

<table>
<thead>
<tr>
<th>Stages</th>
<th>Clinical Findings</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 (Inflammatory)</td>
<td>localised warmth, edema, erythema</td>
<td>minimal, if any abnormalities (MRI would show subchondral bone marrow edema with or without non-displaced pathological fracture)</td>
</tr>
<tr>
<td>1 (Fragmentation)</td>
<td>localised warmth, marked edema, erythema</td>
<td>focal bone demineralisation (early), debris formation at the articular margins, fragmentation of subchondral bone, subluxation, dislocation and periarticular fractures</td>
</tr>
<tr>
<td>2 (Coalescence)</td>
<td>continued but decreased warmth, edema and erythema</td>
<td>absorption of fine debris, fusion of large fragments of adjacent bones and/or new periosteoal bone formation</td>
</tr>
<tr>
<td>3 (Consolidation)</td>
<td>decreased or absent warmth, edema and erythema</td>
<td>remodelled and new bone formation, decreased osteosclerosis and/or possible gross residual deformity</td>
</tr>
</tbody>
</table>

Table 1: Modified Eichenholtz classification from Mautone and Naidoo 2015 [14]

© Balgrist University Hospital
Fig. 16: Baseline: Radiographs of the left foot in dp (a), oblique (b) and lateral projection (c). Stage 1 Charcot foot according to the Eichenholtz classification. Note the subluxation, osteolytic appearance and debris formation at the articular margins of the Lisfranc's joint (white arrows)

© Balgrist University Hospital
**Fig. 17:** Radiographs of the left foot in dp (a) and lateral projection (b). Stage 2 Charcot foot according to the Eichenholtz classification. Note the absorption of the debris and the initial fusion of the fragment around the Lisfranc's joint (white arrows)

© Balgrist University Hospital

**Fig. 18:** Radiographs of the left foot in dp (a), oblique (b) and lateral projection (c). Stage 3 Charcot foot according to the Eichenholtz classification. Note the bone remodeling, ankylosis in the Lisfranc's joint (white arrows) and residual gross deformity. The lateral aspect of the Lisfranc's joint is better visualized in the pronated oblique projection, while the medial aspect is better seen in the d.p. projection

© Balgrist University Hospital
<table>
<thead>
<tr>
<th>Active Stage</th>
<th>MRI Findings</th>
<th>Inactive Stage</th>
<th>MRI Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical Symptoms</td>
<td>obligatory</td>
<td>no inflammation</td>
<td>no abnormal imaging or minimal residual bone marrow edema</td>
</tr>
<tr>
<td></td>
<td>- diffuse bone marrow edema</td>
<td>- no gross deformity</td>
<td>- subchondral sclerosis</td>
</tr>
<tr>
<td></td>
<td>- no cortical disruption</td>
<td></td>
<td>- bone cysts</td>
</tr>
<tr>
<td></td>
<td>- facultative</td>
<td></td>
<td>- osteoarthritis</td>
</tr>
<tr>
<td></td>
<td>- subchondral trabecular microfractures</td>
<td></td>
<td>- ligament damage</td>
</tr>
<tr>
<td></td>
<td>- ligament damage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>mild inflammation (swelling, warmth, pain?) increased by unprotected walking</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>- no gross deformity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>severe inflammation (swelling, warmth, pain?), increased by unprotected walking</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>- gross deformity, increased by unprotected walking</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>obligatory</td>
<td>residual bone marrow edema</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- fracture(s) with cortical disruption</td>
<td>- cortical callus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- bone marrow edema and soft tissue edema</td>
<td>- joint effusion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- osteochondrosis</td>
<td>- subchondral cysts</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- joint effusion</td>
<td>- joint destruction</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- fluid collection</td>
<td>- joint dislocation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- bone erosion/necrosis</td>
<td>- fibrosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- bone lysis</td>
<td>- osteophyte formation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- debris</td>
<td>- bone remodeling</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- bone destruction</td>
<td>- cartilage damage</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- joint luxation/subluxation</td>
<td>- ligament damage</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- ligament damage</td>
<td>- bone sclerosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- tenosynovitis</td>
<td>- ankylosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- bone dislocation</td>
<td>- pseudoarthrosis</td>
</tr>
</tbody>
</table>

Table 2: Proposed MRI-classification for CN modified from Chantelau and Grützner 2014 [15]

© Balgrist University Hospital
Fig. 19: Weight bearing radiograph in dorsoplantar projection (a: baseline, b: 5 months later). Notice the development of fractures (yellow arrow), subchondral cysts, erosions, joint distention, and luxation of the Lisfranc's joint (white arrows)

© Balgrist University Hospital
Fig. 20: Lateral weight bearing radiographs showing the typical course of Charcot foot disease over time (a: Baseline, b: 10 month later). Note the continuous increase of Meary’s angle or lateral talo-first metatarsal angle (yellow angle), the diminishment of cuboid height, which is becoming negative (blue distance) and the decrease of the calcaneal pitch (red angle) [18]

© Balgrist University Hospital
Fig. 21: Radiograph in dorsoplantar projection showing the changes in foot morphology in a typical Charcot foot patient over time (a: baseline, b: 10 months later). Note the increase in forefoot abduction relative to the hindfoot (hindfoot-forefoot angle) which is the angle between the longitudinal axis of the 2nd metatarsal bone (red arrow) and the bisection (yellow dotted line) of the angle which is formed by the following two lines: one through the talar neck and head and the other parallel to the lateral cortex of the calcaneus (green arrows) [18]

© Balgrist University Hospital
Fig. 22: Use of MRI for diabetic patients with neuropathy in the setting of Charcot foot. Three main MRI-benefits: confirmation of diagnosis in early Charcot, monitoring of disease activity, imaging of complications (infection/osteomyelitis)

© Balgrist University Hospital
**Fig. 23:** Proposed MRI-protocol for evaluation of the Charcot foot with four sequences; sagittal STIR (a), sagittal T1 (b), transverse T1 (c) and coronal T2 (d)

© Balgrist University Hospital
Fig. 24: Off-Loading therapy with total contact casts give the patient the chance of healing properly without debilitating deformities (courtesy of Thomas Böni, MD)

© Thomas Böni, Balgrist University Hospital
Fig. 25: Imaging of early active Charcot foot. a: lateral weight-bearing radiograph showing no abnormalities. b: sagittal STIR-Sequence in MRI showing classic bone marrow edema in the midfoot (white arrows). (Stage 0 according to the Eichenholtz classification)

© Balgrist University Hospital
**Fig. 26:** Late stage Charcot foot (all images from the same patient). Note the superior dislocation of the metatarsals at the level of Lisfranc's joint (red arrow in sagittal STIR image) (a) and the degree of bone destruction and fragmentation in the midfoot (blue arrow in sagittal T1 image) (b). There is still an extensive bone marrow and soft tissue edema present (yellow arrows in sagittal STIR image) (a). The coronal T2 shows a prominent subchondral cyst at the subtalar joint (green arrow) (c)

© Balgrist University Hospital
Fig. 27: Late stage Charcot foot (all images from the same patient). a: sagittal STIR, b: axial T2, c: sagittal T1, d: sagittal T1 fs after contrast administration. Note the inferior luxation of the talar head (blue arrows and green arrow) (a, c), the bone marrow edema (a) and the enhancement of the bone marrow after contrast medium administration (d). Joint effusion and synovitis of the ankle joint is present (red arrow) (d)

© Balgrist University Hospital

Fig. 28: Three sagittal images of different patients showing classic features of late stage Charcot foot. a: (sagittal T1) inferior dislocation of the talar head (red arrow), b: (sagittal STIR) prominent subchondral cysts at the Lisfranc's joint (white arrow), c: (sagittal STIR) bone proliferation, debris in the midfoot (green arrows)
Fig. 29: Late stage Charcot foot demonstrating gross cortical fractures of the second metatarsal bone (red arrows) (a: long axis STIR image of the forefoot, b: sagittal T1 image through the second metatarsal)
**Fig. 30:** Before off-loading therapy (a: sagittal STIR, b: sagittal T1): Active stage of Charcot disease with a significant amount of bone marrow and soft tissue edema (red arrows) (a). Also note the subluxation at the Chopard joint with downward tilt of the talar head (green arrow) (b). 7 months after a consequent off-loading therapy with a total contact cast (c: sagittal STIR, d: sagittal T1): note the almost complete disappearance of bone marrow edema

© Balgrist University Hospital

<table>
<thead>
<tr>
<th>Location of bone marrow abnormality (edema shown in fluid sensitive sequences, and reduction of fatty bone marrow shown in T1 sequences)</th>
<th>active Charcot foot</th>
<th>Osteomyelitis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>o pattern tends to be periarticular [14]</td>
<td>o tendency to involve a single bone with diffuse marrow involvement</td>
</tr>
<tr>
<td></td>
<td>o usually involves several joints and bones (mostly tarsometatarsal joints and metatarsophalangeal joints) [2]</td>
<td>o usually affecting weight-bearing surfaces of the toes, metatarsal heads, calcaneus, malleolus and a special area in Charcot: cuboid (in rocker-bottom deformity) [3]</td>
</tr>
<tr>
<td>sinus tracts</td>
<td>o usually not present</td>
<td>o often present</td>
</tr>
<tr>
<td>skin ulceration (radiological technician should mark the ulcer)</td>
<td>o can be present</td>
<td>o often present</td>
</tr>
<tr>
<td></td>
<td>o often relationship to sinus tract</td>
<td></td>
</tr>
<tr>
<td>fluid collections</td>
<td>o present</td>
<td>o present</td>
</tr>
<tr>
<td></td>
<td>o usually smaller than in case of infection, unless sinus tract is present</td>
<td>o usually larger than in active CN, unless a sinus tract exists over which the collection is drained (paradoxical decrease of size of fluid collection) [21]</td>
</tr>
<tr>
<td></td>
<td></td>
<td>o diffusion weighted imaging (DWI) might help in differentiation abscesses from non-infected fluid collections [22]</td>
</tr>
<tr>
<td>subcutaneous fat</td>
<td>o often present</td>
<td>o often disappear due to presence of cellulitis [23]</td>
</tr>
<tr>
<td>subchondral cysts</td>
<td>o typical image feature in CN</td>
<td>o tendency to disappear in case of infection/osteomyelitis</td>
</tr>
<tr>
<td></td>
<td>o the presence of subchondral cysts indicates the absence of infection</td>
<td>o best recognized if regular previous follow-up studies are present which demonstrated the disappearance of the cysts [23]</td>
</tr>
<tr>
<td>intraarticular bodies</td>
<td>o the presence of intraarticular bodies indicates the absence of infection</td>
<td>o often disappear in the setting of infection due to dissolution or obscuring by surrounding inflammation [23]</td>
</tr>
<tr>
<td>“The ghost sign” definition: bones that “disappear” on T1-weighted images and “reappear” (outline of the bones becomes distinct again) after contrast administration (or on T2-weighted images) likely have superimposed osteomyelitis [21]</td>
<td>o negative</td>
<td>o positive</td>
</tr>
</tbody>
</table>
**Fig. 31:** Clinical image (a) and MRI (sagittal STIR and sagittal T1) (b, c) from an ulceration at the sole of the foot directly beneath the cuboid bone as a typical complication of rocker-bottom deformity of the foot in Charcot neuropathic osteoarthropathy. MRI demonstrates contiguous spread of infection from the skin (yellow arrows) (b, c) ulceration to the cuboid bone, the subcutaneous fat in this location is missing and there is a focal replacement of fatty bone marrow within the cuboid (green arrow) (c); all signs of osteomyelitis

© Balgrist University Hospital

**Fig. 32:** MRI of a Charcot foot complicated with osteomyelitis. a: sagittal T1, b: sagittal STIR, c: sagittal T1 fat sat after contrast administration. Skin ulceration and sinus tract extending from the skin to the talar bone are present, showing a direct spread of infection (green arrows) (b, c). Diffuse bone marrow alteration is present within the talus. Note the disappearance of bony contours in the sagittal T1 weighted image (yellow arrow) (a)
and the reappearance of the bone structures after contrast administration or in the STIR image (red arrows) (b, c) demonstrating the "Ghost sign", which is pathognomonic for osteomyelitis

© Balgrist University Hospital

Fig. 33: Special case 1. Conventional radiographs (a: ap projection, b: lateral projection). Note the complete collapse of the hindfoot with impaction of the lower leg into the calcaneus due to an advanced Charcot foot, Sanders and Frykberg Zone IV, explaining the sudden shortening of the leg

© Balgrist University Hospital
Fig. 34: Special case 1: CT-scan in supine position in sagittal (a) and coronal (b) reconstruction. Note the grade of destruction and fragmentation of the bones in the hindfoot with complete dissolution of the talus and partially of the calcaneus, demonstrating the corrosiveness of the disease. CT-scan in standing position (c) in coronal reconstruction demonstrating the loading zone with medial dislocation of the lower leg in comparison to the CT image acquired in supine position (yellow arrow) (c)

© Balgrist University Hospital

Fig. 35: Special case 1: MRI study with sagittal STIR (a), axial T1 (b) and cor T1 (c) sequences. Note the huge amount of fluid and debris within the impacted zone of the hindfoot (green arrow) (a)

© Balgrist University Hospital
**Fig. 36:** Special case 2: Conventional radiographs in dorsoplantar (a), pronated oblique (b) and lateral projection (c). Typical Charcot foot signs are present with increase in forefoot abduction relative to the hindfoot due to subluxation in the Lisfranc’s joint (white arrows). Also note the collapse of the longitudinal arch with the cuboid becoming the lowest structure in the foot (yellow arrow) (c). Additional osteolysis is present in the tip of the 2nd toe (green arrows) (a, b) and the middle and distal phalanx of the 5th toe (red arrow) (b)

© Balgrist University Hospital
Fig. 37: Special case 2: long axis STIR-image (a), sagittal T1 image through 2nd toe and 5th toe (b, c) and short axis T1 fs after contrast administration (d). Note Charcot foot changes like the subchondral fracture of the 2nd metatarsal head (red arrows) (a, d), the deformities and subchondral cysts at the Lisfranc's joint (white arrows) (a, b). Additionally, signs of osteomyelitis at the distal 2nd and 5th toe are present with replacement of the normal bone fatty bone marrow signal in T1 (green arrows) (b, c). This is not a direct complication of the Charcot foot, because it is not the location where the Charcot pathology shows manifestations. Therefore, it's a case of Charcot foot concurrent to diabetes related pedal osteomyelitis.

© Balgrist University Hospital
Conclusion

The Charcot foot is a rare disease, associated with polyneuropathy, in industrialized countries most commonly seen in the long-term diabetic population. The Radiologist plays an important role in the management of this disease. Therefore, it is vital to be familiar with the typical imaging characteristics of the Charcot foot and to consider this diagnosis in a proper clinical setting. Recognizing this disease in early stages prevents a delayed onset of an appropriate therapy (generally off-loading therapy) and helps minimizing the disability of these patients.

Commonly used classification systems for Charcot foot are based on radiographic findings using either the location or the degree of bone changes to categorize this disease. Although radiographs are important to assess the position of the bones to each other in general, and in particular under load, MRI is the method of choice not only in establishing an early diagnosis, but also in monitoring the course of the disease activity and in diagnosing complications like osteomyelitis; therefore, a new widely accepted MRI-based classification system is needed.
References

1.

2.

3.

4.

5.

6.

7.

8.

9.

10.


13.

14.

15.

16.


27.
Personal Information

C. Loupatatzis
Department of Radiology, Spital Maennedorf, Asylstr. 10, 8708 Maennedorf, Switzerland, c.loupatatzis@spitalmaennedorf.ch

M.C. Berli
Department of Orthopedic Surgery, Balgrist University Hospital, Forchstrasse 340, 8008 Zurich, Switzerland

C.W.A. Pfirrmann, A.B. Rosskopf
Department of Radiology, Balgrist University Hospital, Forchstrasse 340, 8008 Zurich, Switzerland