A comprehensive review of diabetic foot complications

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Authors: A. Gimeno¹, M. T. Veintemillas², J. M. Escudero Fernandez¹, M. De Albert¹, C. Torrents Odin¹, L. Casas¹, R. Dominguez¹; ¹Barcelona/ES, ²Sabadell/ES
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Learning objectives

1. To illustrate the different imaging findings in diabetic foot complications.

2. To describe challenging and difficult scenarios, of the diabetic foot complications, in presurgical and postsurgical contexts.

3. To assess what the surgeon wants to know.
Background

Diabetic mellitus (DM) is a multisystemic disease with increasing prevalence, significant morbidity, premature mortality and impact on the healthcare costs.

Peripheral nerve dysfunction related to DM allows the possibility to suffer minor injuries that may remain undiscovered for a long while, causing deformity, altered weight bearing, subsequent osteoarthropaty, callus and ulceration. Peripheral artery disease that is frequently present in these patients causes difficulties to heal infections, leading to surgery treatments like debridement and amputations.(1)(2)

We review, from our experience, difficult and challenging scenarios of diabetic foot complications. We focus on what we consider the main difficulties for a radiologist, that help taking good management decisions, and try to answer our surgery department issues.
Findings and procedure details

What the surgeons wants to know in our center is defined below:

1. Existence and extension of osteomyelitis in presurgical, postsurgical contexts and
discrimination with Charcot neuro-osteoarthropathy (CN). Fig. 1 on page 30

2. Which kind of involvement is existing in the soft tissue (cellulitis, phlegmon, gangrene
or abscess), its extension and differences from inflammatory soft tissue affection.

3. Use of advanced MRI techniques to confirm and best delimitate of the features in
convetional MRI sequences.

4. To delimitate and assess the routes of spread and inform about the arterial and
venous blood-pool with a MRI vascularization map.

1. Osteomyelitis

Most common locations of osteomyelitis are the pressure points of the forefoot
(metatarsal heads, interphalangeal joints) and the plantar aspect of the posterior
calcaneus at the hindfoot. The diagnosis and difference from CN, is important to
avoid unnecessary amputations and to best delimitate the infection borders to avoid
recurrences after treatment.

Radiologist must know the different techniques to diagnose osteomyelitis and their
reliability. Fig. 2 on page 9

The choice of the imaging modality can be different in every treating center; the election
must be made depending on the patient's clinical presentation, availability of equipments
and expertise of physicians at the treating center.(3)(4)

MRI is the modality of choice because provides the best delimitation of the soft tissues
affection and allows differentiation between osteomyelitis and CN. We use this technique
when results of X-Ray are positive for osteomyelitis, when X-Ray is negative for
osteomyelitis with high clinical suspicion of infection or as a guide to perform a biopsy.

It is important to know that on conventional radiology and CT, bone infection may
not show up on the first 7-10 days(5) in the form of demineralization, destruction and
periosteal reaction. Fig. 3 on page 10
1.1 MRI features

The easiest method to determine if osteomyelitis is present, is following a skin defect or sinus tract to bone and check the bone marrow signal. Clear hypointensity on T1WI associated with avid enhancement in the bone marrow is a primary sign of osteomyelitis. T1 should be used to assess the spread of bone infection.\(6\)

Periosteal reaction and cortical interruption are another reliable signs of osteomyelitis. Periosteal reaction appears as a linear hypointensity and hyperintensity on T1WI/T2WI, around the cortical with avid contrast-enhancement.\(7\)(\(8\)\ Fig. 22 on page 28

Ghost sign is a specific sign of osteomyelitis.\(9\) It consists in a difficulty to identify the shape of the bone with an ill-defined margins on T1WI that reappears after contrast injection. \Fig. 14 on page 20 \Fig. 22 on page 28.

There are multiple secondary signs like ulcers, sinus tract, cellulitis, abscess, gangrene and septic arthritis that must be reviewed to make a correct diagnosis and not confuse with (CN).\(\text{Fig. 22 on page 28,Fig. 23 on page 29.}\\

Osteitis is a reactive change to soft-tissue or cortical infection, not to be confused with osteomyelitis. Bone marrow edema in osteitis shows hyperintensity on T2WI and fairly normal signal in T1, although contrast-enhancement may be present.\(\text{Fig. 4 on page 9,Fig. 5 on page 11,Fig. 6 on page 12,Fig. 7 on page 13.}\\

2. Soft tissue disease

2.1 Skin callus

Redistribution of fat and formation of skin callus appears as an area of low signal intensity on T1WI and T2WI with variable contrast-enhancement that may mimic soft-tissue infection. The absence of surrounding soft-tissue signal alteration is typical in calluses.

Typical locations are below the first and fifth metatarsal heads and at the tip of the great toe, in patients with rocker-bottom deformity appear under the cuboid bone.

2.2 Skin ulcer
Skin ulcer is almost mandatory in the diagnosis of osteomyelitis.

In general, location of skin ulcers are similar to those of calluses. In non ambulatory patients can be located in the calcaneus or lateral malleolus.

These skin ulcers facilitate direct inoculation of the bacterial agents in soft-tissue and bone. Skin ulcers appear as focal skin discontinuities with elevated margins, high signal intensity on T2WI, with rim-like contrast enhancement of the granulation tissue located at the periphery of the ulcer. Fig. 8 on page 14, Fig. 9 on page 15.

2.3 Sinus tract(7) Fig. 10 on page 16

2.4 Cellulitis

It is an acute non-necrotizing inflammatory process involving the skin and subcutaneous tissues.

Clinically, it presents with pain, erythema, edema and warmth. Nevertheless, similar symptoms are observed in diabetic patients without infection due to ischemia and venous insufficiency or in patients with early phases of neuropathic disease.

On MR, cellulitis appears as a skin thickening and reticulation of subcutaneous fat, with intermediate signal intensity on T1WI and high signal intensity on T2WI. Contrast-enhancement appears in cases of cellulitis but not in edema presented in diabetic patients without infection. Fig. 4 on page 9

2.5 Phlegmon

As the infection progresses, areas of cellulitis are replaced by areas of phlegmon. On MR, phlegmon appears as ill-defined areas with mass effect and without the characteristic trabeculation of the subcutaneous fat. They show low signal intensity on T1WI, variable signal intensity on T2WI depending of the grade of liquefaction and homogeneous contrast-enhancement. Fig. 11 on page 17

2.6 Abscess

Abscesses are uncommon in patients with diabetes and they are usually located close to the skin and sinus tracts.
On MR, abscesses appear as collections of fluid signal intensity (pus), surrounded by peripheral an irregular rim of contrast enhancement and T2WI hiperintensity. Caution must be taken when interpreting the peripheral rim enhancement of fluid collections, because it is constant in abscess but is also common in fluid collections next to non infected neuropathic joints. (10) Fig. 12 on page 18, Fig. 13 on page 19

2.7 Septic arthritis

It is usually located in interphalangeal and metatarsophalangeal joints in cases of lateral ulceration, or in ankle and subtalar joints in cases of malleolus or calcaneal ulceration.

On MR, it appears as a complex joint effusion with thickened and avid contrast-enhancement of the synovial. The most specific findings of septic arthritis are bony erosions; bone marrow edema and cartilage destruction. Presence of soft tissue defects also elevates the suspicion and allow distinction with joint effusion.

It is important to take in account that adjacent bony surfaces may demonstrate edematous changes, seen as increased T2WI signal, which can be seen in either septic arthritis or osteomyelitis. The bone marrow hipointensity in T1 will discriminate osteomyelitis from osteitis next to an infected joint. Fig. 14 on page 20

2.8 Septic tenosynovitis

It usually affects peroneal tendons when lateral malleolus ulcer is present, Achilles tendon from calcaneal ulcer and flexor tendons from plantar forefoot ulceration.

On MR, it appears as a thickened and avid contrast-enhancement around the tendon. Presence of complex fluid and even gas in the tendon sheath can be seen.

Partial or complete rupture of posterior tibial and Achilles tendon may be present in these patients because of deformities and angiopathy. Fig. 14 on page 20

2.9 Myositis

On MR, it appears as diffuse muscle enlargement, high signal intensity on T2WI and avid contrast-enhancement. Fig. 15 on page 21

2.10 Gangrene
Devitalized tissue appears as wedge-shaped and non-enhancing areas due to absence of vascularization, sometimes surrounded by reactive granulation tissue that enhances. Superimposed infection (wet gangrene) may show intralesional gas that may spread along the fascial planes, not to be confused to the gas entered in soft-tissues from the skin ulcer. Fig. 12 on page 18, Fig. 13 on page 19

3. Advanced MRI techniques

Diffusion and dynamic contrast enhancement MRI (DCE-MRI) can be used to differentiate osteomyelitis from CN. Described cut-off values are 0.98-1.04 x10^{-3} mm^2/s for diffusion. Ktrans value of 0.11 ml/min and Ve value of 0.19 ml/min are defined as possible cut-off values to discriminate CN from osteomyelitis in DCE-MRI. (11)(12) Fig. 16 on page 22, Fig. 17 on page 23, Fig. 18 on page 24

4. Routes of spread and vascular map

Involvement of different bone and soft tissue structures have demonstrated different patterns of spread, assessing possible routes of spread like septic arthritis, compartments involvement and presence of tenosynovitis is primordial.

Different compartments are partial barriers that prevent the spread of the infection, but spread can occur between them.

Joint spaces are poor obstructions to the spread of infections and periarticular osteomyelitis. Interestingly, tendons and their sheaths are not a common pathway for the spread of infections but they can be commonly local infected. (13)(6) Fig. 19 on page 25, Fig. 20 on page 26 Fig. 21 on page 27, Fig. 22 on page 28
<table>
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<th>Diagnostic method</th>
<th>Criteria for positivity</th>
<th>Sensitivity (S) and Specificity (E)</th>
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<tbody>
<tr>
<td>Histopathologic</td>
<td>Osteonecrosis and infiltration with leukocytes or chronic inflammatory cells, such as lymphocytes or plasma cells (debridement or amputation)</td>
<td>Gold standard</td>
</tr>
<tr>
<td>examination</td>
<td></td>
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<tr>
<td>Ulcer measurement</td>
<td>Area was determined by multiplying the longest and widest diameters; depth was graded as very deep (exposing bone), moderately deep (=&gt;$3 \text{ mm}$, but not exposing bone), or shallow (&lt;3 mm)</td>
<td>Exposed bone $S=0,32 \ E=1$.</td>
</tr>
<tr>
<td>Radiography</td>
<td>Focal or geographic areas of marrow radiolucency, loss of cortex with bony erosion, new bone formation, bone sclerosis with or without erosion, soft-tissue inflammation, sequestration, involucrum, cloaca, and periosteal elevation</td>
<td>$S=0,54, \ E=0,68$</td>
</tr>
<tr>
<td>MRI</td>
<td>Decreased signal intensity on T1-weighted images with focal enhancement after contrast and increased signal intensity on T2-weighted images</td>
<td>$S=0,9, \ E=0,79$</td>
</tr>
<tr>
<td>Bone scintigraphy</td>
<td>Increased blood flow and blood-pool activity and abnormally increased intensity localized to the bone</td>
<td>$S=0,81, \ E=0,28$</td>
</tr>
<tr>
<td>Leukocyte scan</td>
<td>Focal abnormal increased activity</td>
<td>$S=0,91, \ E=0,9$</td>
</tr>
<tr>
<td>CT/PET-FDG</td>
<td>Glucose hipermetabolism in an area suggestive of osteomyelitis at CT.</td>
<td>$S=0,89, \ E=0,92$</td>
</tr>
</tbody>
</table>

*Major diagnostic methods for diagnosing osteomyelitis associated with diabetic foot ulcer*[^1]
**Fig. 4:** Dorsoplantar and oblique radiographs in a and b. Sagittal T1WI in c, PD-FS in d and T1WI with fat suppression (T1-FS) and contrast administration in e. Enlargement of the soft tissue around the toe (yellow brackets) and skin defect (yellow arrow), subtle radiolucency at the head of the distal phalanx in b (yellow asterisk). Ulceration at the tip of the toe is associated with bony destruction of the distal phalanx due to osteomyelitis (yellow asterisk) and gangrenous tissue (red asterisk). Notice associated cellulitis (red arrow) and septic arthritis (blue arrow).

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Case of 69 year old woman, with high risk DM, with foot ulcer because of new shoes and clinical signs of inflammation.

Oblique prone radiography in a, sagittal and axial proton density with fat supresion (PD-FS) in b and c, axial T1-WI in d.

X-Ray shows demrialization and subtle radiolucency in the first metatarsal head (yellow asterisk) next to the ulcer (red arrow) that can not exclude osteomyelitis. Skin defext because of an ulcer (red arrow) in the medial aspect of metatarsal head, with regional contrast enhancement because of cellulitis (yellow star). Normal T1 hipointensity in the adjacent bone (yellow arrow) excludes osteomyelitis. Normal fluid in the interphalangeal joint can not be mistaken with arthritis (red asterisk). Radiolucency in the midfoot (blue circle) corresponding to subchondral cyst in the MRI(yellow circle), suggestive of early neuroostearthropaty.

Fig. 3

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**Fig. 5:** Axial and sagittal T1WI MRI of the forefoot (a and b), PD-FS in c and d, T1-FS and contrast administration e and f. Osteitis of the stump (yellow asterisk) next to the skin ulcer (red arrow). The absence of clear hipointensity in T1WI is suggestive of osteitis more than osteomyelitis (red arrowhead). Notice gangrenous area (yellow arrow), myositis and tenosynovitis in the adjacent soft-tissue (red asterisk).

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MR of the forefoot with sagittal PD-FS (a) and T1-FS postcontrast (b). Ulceration at the internal aspect of the forefoot (yellow arrow) is associated with periarticular osteomyelitis (yellow asterisk). Subluxation of the first metatarsophalangeal joint is also present. Notice severe tenosynovitis of the flexor hallucis longus (red arrow) and osteitis in the metatarsal body (red asterisk).

Fig. 6

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MR of the forefoot with axial and coronal T1WI (a and d) and T1-FS and contrast administration (c and b). Osteomyelitis of the distal phalanx of 1st finger (yellow asterisk) with skin ulcer at the tip of the toe (red asterisk).

Fig. 7

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Fig. 8: Forefoot MRI with axial and sagittal T1WI (a, b and d), T1-FS and contrast administration (c, e and f). Osteomyelitis of the medial and distal phalanx of 3th finger (yellow asterisk) with skin ulcer at the dorsal aspect of the distal phalanx (red asterisk) and multiple hypointense foci representing gas entered from the skin ulcer (yellow arrows).

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**Fig. 9:** Forefoot MRI with axial and sagittal T1WI (a, b and d), T1-FS and contrast administration (c, e and f). Osteomyelitis of the medial and distal phalanx of 3th finger (yellow asterisk) with skin ulcer at the dorsal aspect of the distal phalanx (red asterisk) and multiple hypointense foci representing gas entered from the skin ulcer (yellow arrows).

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Sinus tract: Skin ulcer extents to the bone through a tubular/pseudonodular path corresponding to a sinus tract. It shows high signal intensity on T2WI and contrast-enhancement in its periphery (“tram-track” pattern). (7)
Axial and coronal T1-FS with contrast administration.
There is a tubular tract with peripheral rim-enhancement in “tram-track” pattern (red bracket) that extends from the dorsolateral aspect of the midfoot to the fourth metatarsal head (yellow arrow), suggestive of osteomyelitis.

Fig. 10

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Fig. 11

MR of the forefoot with axial, coronal T1WI (a and b), T2-FSGE (e), and T1-FS with contrast administration (c, d and f). Skin defect in the surgical scar is present (yellow arrow) with a sinus tracts that extends through the bone (red arrow). Stump osteomyelitis (yellow asterisk) with an area of phlegmon (red asterisk) is present. Surgical scar is a typical place of recurrence. Assessment of soft tissue involvement is primordial in the presurgical MR to perform a correct debridement.

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Fig. 12

MR of the hindfoot with axial PD-FS (a and c) and T1-FS postcontrast (b and d). Calcaneus osteomyelitis (yellow asterisk) with skin ulcer at the lateral aspect (red asterisk). Notice the voluminous abscess (red arrow) and the extensive area of devitalized tissue (yellow arrows) without contrast enhancement.
Axial, T1-FS with contrast administration a, diffusion weighted image in c, ADC map in d.

Fluid collection with thick peripheral rim enhancement is present in the dorsal subcutaneous tissue. Restricted diffusion inside the dorsal collection discriminates very well abscess than other fluid collections (blue circle), gangrene shows absence of enhancement (yellow star).

Measurements of collections and their relationship with infection have been studied, larger fluid collections are more frequently found next to infected joints, but they can be smaller if a sinus tract is present(10).

Fig. 13

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**Fig. 14:** MR of the forefoot with sagittal T1WI (a, b and c), DP-FS (d, e and f), coronal and axial T1-FS with contrast administration (g and h). Ill defined margins that reappear after contrast injection are seen in the metatarsal head and proximal phalanx due to the ghost sign (yellow asterisk) suggesting osteomyelitis. Septic arthritis of the first metatarsophalangeal joint is seen as a DP-FS hiperintensity that enhances after contrast administration (blue asterisk). MR also shows skin ulcer next to the metatarsal bone with sinus tract (red asterisk). Notice the intraosseus abscesses (yellow arrow), septic fluid collection in the base of the first metatarsophalangeal joint (red arrow) and tenosynovitis of the flexor tendon (blue arrow) are also present.

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MR of the midfoot and forefoot. Axial and coronal T1WI (a and b), DP-FS (c and d) and T1-FS postcontrast (e and f) MR imaging.

T1 bone marrow hipointensity with contrast enhancement is present in the 2nd proximal phalanx (yellow asterisk), next to skin ulcer (red asterisk), corresponding to osteomyelitis. Tubular enhancement that extends in the dorsal aspect of the forefoot is suggestive of sinus tract (red arrow). Notice extensive inflammatory changes of soft-tissues because of cellulitis and myositis (yellow arrows).

Fig. 15

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Fig. 16: Axial T1WI (a), T1-FS postcontrast (b), ADC weighted imaging (c), coronal STIR (d), DCE-MRI (e) and its calculated parameters (f). Dorsoplantar radiography in g. 70 years old patient, with forefoot ulcer and signs of inflammation. Plain film shows heterogeneous density next to the ulcer in the first finger due to cellulitis and gauze (red arrow); MRI confirms foot ulcer and necrotic tissue without enhancement (yellow arrow). Head of first metatarsal shows radiolucency that corresponds to clear hypointensity in T1 with contrast enhancement due to osteomyelitis (blue circle). ADC shows values of 1.59 corresponding to the area of osteomyelitis, DCE-MRI parameters show the biggest slope and maintained enhancement (red arrowhead) in the most inflammatory active area. No signs of tenosynovitis were identified in the dorsal compartment (red brackets). DCE-MRI and diffusion have demonstrated to differentiate osteomyelitis from CN, in our treating-center we are evaluating the values of regional bone edema with DCE-MRI and diffusion to best asses spread of osteomyelitis in bone and try to predict recurrences (yellow star).

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Fig. 17: Dorsoplantar (a) and oblique (b) radiographs, DCE-MRI (c), sagittal T1WI (d), dorsoplantar radiograph after surgery (e), coronal T1-FS postcontrast (f), calculated parameters of MRI-DCE (g). 57 years old patient with four days of swelling, inflammation and warmth in the great toe next to an ulcer. Osteolysis, cortical defects and destruction of the head of the distal phalanx (yellow star) with permeative periosteal reaction in the rest of the phalanx (red asterisk), due to osteomyelitis. Demineralization and deformity in the proximal phalanx (yellow asterisk) is also seen. Ulceration and gangrene with T1 hipointensity and no enhancement (red arrowhead). Avid enhancement in the adjacent tissue due to hyperaemia next to necrotic tissue and cellulitis (red arrow). T1 hipointensity in the distal phalanx and no enhancement (red star) corresponds to osteomyelitis and wet gangrene. Extension to the proximal phalanx is also seen in MRI (yellow bracket). Amputation of the first finger until the base of the metatarsal was practiced (blue bracket).

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MR of the forefoot with coronal and axial T1WI (a and b), T1WI with fat supression and contrast adminsitration (c and d), diffusion (e) and ADC maps (f).

MRI shows osteomyelitis of the medial and distal phalanx of the 4th finger (yellow asterisk) with an area of phlegmon (red asterisk) and septic cellulitis (blue asterisk). Notice the capability of diffusion to delimitate the borders of the septic areas (green asterisks). This sequence is helpful to assess the limits of soft tissue infections.

Fig. 18

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The plantar soft tissue of the foot is constituted by five compartments divided by two intermuscular septa that arise from plantar aponeurosis and fascial planes. Intermuscular septa divide the medial, lateral and central compartments. The medial compartment contains the muscles of the great toe, the lateral one contains the muscles of the fifth toe and centrally there are the rest of the plantar muscles. The two lasts compartments are the interosseous compartment between the metatarsals and the dorsal compartment, which are divided by fascial planes. Infections tend to spread inside the compartments and along the path with least resistance. Fascial planes and intermuscular septa are barriers that prevent spread between compartments.

Fig. 19

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**Fig. 20:** 60 year old patient with bad control of DM and amputation of the 4th finger 5 years ago. T1 hipointensity of the fourth metatarsal head stump with avid enhancement suggestive of osteomyelitis (red arrows). There is a big area of wet gangrene(7) that spreads along the fascial compartments, showing absence of enhancement and gas bubbles in the plantar subcutaneous tissue with thin peripheral rim enhancement (yellow asterisks), following the central, medial compartment and plantar fascia (yellow arrows). Sparing of the lateral and interosseous compartment is present. Diffusion delimitate ischemic and septic affection in the soft tissue (red asterisks), with very similar shape to the contrast enhancement sequences (yellow asterisks). Vascular map shows absence of enhancement in the central foot and hind foot (yellow map). Notice area of myositis in the central compartment (red arrowhead).

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**Fig. 21**: 36 years old patient with previous surgeries, presents with 3 ulcers, gangrene and signs of infection. Gangrene next to the ulcers is clinically and radiologically seen (red arrows), with extension to the ankle and peroneal tendons (yellow bracket). Diffusion differentiate the extension of the most affected septic and ischemic areas (yellow star) and allows discrimination with abscess, notice the absence of restricted diffusion in the center of the collection (red asterisk). Osteomyelitis of the stump of the fifth metatarsal is present (yellow asterisk) next to the midfoot ulcer with no extension to the tarsal bones (absence of enhancement in the DCE-MRI parameters, red arrowhead). DCE-MRI is very useful to evaluate the areas of more inflammation, notice the highest slope next to the peroneal tendons and contrast enhancement around them (yellow arrow), due to septic tenosynovitis. Finally we perform vascularization map with a MIP that best delimitate areas of poor vascularization, very useful for planning the surgery, poor vascularization in the right forefoot is present (yellow rectangle).

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**Fig. 22:** 45 years old patient with amputation of the first finger presents inflammation next to the surgical scar without skin defect. Enhancement in the surgery scar that extends to the metatarsal head is present (yellow bracket). Ill defined T1 hipointensity that reappears with contrast enhancement is noticed in the second metatarsal (ghost sign), this could be consequence of osteomyelitis or fracture (red arrow). Fracture is more suitable in the X-Ray (red asterisk) but MRI is surrounded by an area of arthritis (yellow star) with the highest areas of inflammation in the vascular map (red arrowhead). Notice tenosynovitis of the abductor hallucis (yellow arrow). A biopsy was performed because of doubts in the radiological and clinical diagnosis. The microbiological results were negative to osteomyelitis. Afterwards we hypothesized that the theoretical clinical septic arthritis with peripheral bone destruction (yellow star) was periosteal reaction (red star) due to a fracture with inflammatory regional changes.

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<table>
<thead>
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<th>Feature</th>
<th>Neuropathic osteoarthropathy</th>
<th>Infected neuropathic osteoarthropathy</th>
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<tbody>
<tr>
<td>Acute local inflammation</td>
<td>X (acute)</td>
<td>X</td>
</tr>
<tr>
<td>Distribution</td>
<td>Intertarsal, tarso-metatarsal joints</td>
<td>Metatarsal heads, toes or calcaneus</td>
</tr>
<tr>
<td>Bony fragmentation, debris, fractures, subluxation/dislocation, sclerosis of bone ends, and osteophytes on radiography</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Deformity</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Low marrow MR signal intensity on T1-weighted images, high on fluid-sensitive sequences, post-contrast enhancement</td>
<td>X (Acute)</td>
<td>X</td>
</tr>
<tr>
<td>Low marrow MR signal on both T1-weighted and fluid-sensitive images</td>
<td>X (Chronic)</td>
<td></td>
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<td>Subchondral cysts and intra-articular bodies on MR imaging</td>
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<td>Focal periarticular bone marrow involvement</td>
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<td>“Ghost sign”</td>
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<tr>
<td>Sinus tract formation, replacement of soft-tissue fat, diffuse marrow abnormality, thick rim enhancement or diffuse joint fluid enhancement, and joint erosion on MR imaging</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>Progression of bone erosions, disappearance of subchondral cysts or intra-articular bodies, increased bone marrow changes, and contrast enhancement of the articular surface on follow-up MR imaging of neuropathic osteoarthropathy</td>
<td></td>
<td>X</td>
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**Fig. 23**

©
Fig. 1

Postsurgical diabetic foot is a challenging scenario for different reasons: difficulty to heal surgical scars provoke long term inflammation, which is also a possible entrance and place of recurrence of the infection, amputations facilitate the appearance of fractures due to instability. All these conditions can mimic clinical and radiological osteomyelitis.
Conclusion

Multiparametric MRI is the standard technique for the evaluation of diabetic foot complications. Knowing and assessing with precision the limits of the disease and differentiate osteomyelitis from CN is primordial to improve the outcome of the patient.
References


