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Abstract

Diabetic Charcot neuro-osteoarthropathy is a severe, multifactorial limb-threatening entity with a neuropathic background. It evolves from bones, joints and soft tissue inflammation, to permanent deformity, recurrent ulcerations and amputation, if left untreated. This particular case emphasizes the ultrasound’s conclusiveness in diagnosing inflammatory recurrence and orthopedic hardware behavior after corrective arthrodesis, thus permitting the correct therapeutic decision to offload the foot. Ultrasonography proves its undeniable applicability whenever other imaging-techniques are unavailable, inconclusive or rejected by the patient.

Keywords: Charcot neuro-osteoarthropathy; ultrasonography; orthopedic hardware; therapeutic decision; offloading

Introduction

Diabetic Charcot neuro-osteoarthropathy (CN) defined by its neuropathic substrate, impacts bones, joints and soft tissues, being either unilateral, bilateral or multifocal but predominating anatomically at ankle and foot. Besides the multi-pathogenic mechanisms (chronic hyperglycemia, glycation-end-products (GEPs), microvasculopathy), the inflammatory response to foot trauma and pro-inflammatory diabetes-attributes, autoimmunity and genetics complete the CN’s pathogenic ensemble. The CN prevalence reaches 13% in the diabetic population, increasing to around 29% if the neuropathy coexists [1,2].

Despite its clinical and contextual features, the CN is often under-misdiagnosed, thus delaying the treatment. Certain imaging-techniques such as X-ray, magnetic resonance imaging (MRI), computed tomography (CT) or bone scintigraphy are not always easy-to-reach or conclusive. Few ultrasonographic (US) studies have been performed on the unoperated CN [3], but none on the operated CN, to our knowledge.

We report the case of a patient with internal-corrective arthrodesis for his deformed and unstable CN. The post-operative inflammatory episode and the orthopedic-hardware behavior benefited from the orthotically adapted treatment following US scanning. The results can be integrated in the context of tissue stress theory and foot pathomecanics’ conservative therapy [4].

Case report

A 61-year-old male with unilateral diabetic CN, hypertension and overweight was referred to the Podiatry Outpatient Clinic, after a previous successful orthopedic surgery. His history apparently started after a minor left leg injury when getting out of the car. The X-ray facilitated the diagnosis and decision to offload the foot with an Aircast XP Diabetic Walker for the next four months. A residual midfoot rocker-bottom deformity (stage 2/3 Eichenholtz, Brodsky-1) resulted. A trained orthopedist considered both the deformity pattern and the overall risk to be compatible with internal stabilization of the medial column procedure (fig 1). After wearing a plastered boot (non-weight-bearing) for three months, the patient
was advised to progressively load the foot over the next two months using the same offloading Walker. He was referred to be switched from gradual to full loading.

At presentation, a (sub)acute foot (moderately swollen, red and warm), probably related to the untimely wearing of the flexible-footwear despite the recommendations was found at clinical examination. The infrared cutaneous temperature (IRT) of the feet was intriguingly near-symmetrical. No lab exams, including systemic inflammatory-markers, were relevant. Previously recommended medication has been metabolically effective for the last two years (not including antiresorptive therapy).

US demonstrated synovial pannus of the lateral cuneonavicular joint and active bone erosions. It took six months to offload for the inflammatory activity (synovium and bone erosions) to recover. Ensuingly, a gradual reloading with an accommodative foot-orthosis and rigid rocker-bottom diabetic-boot was applied, thus preserving remission (fig 2).

One year later, the US depicted mild inflammatory reaction at the metallic-implant junctions with the navicular and the first metatarsal bone and synovitis of the native fifth metatarsalcuboid joint (fig 3).

**Fig 1.** Left foot X-ray-stage 2/3 CN, Brodsky dorso-plantar (a) and oblique (b) views-after internal stabilization of the medial column (first metatarsal-cuneiform-navicular joints’ arthrodesis).

**Fig 2.** Correlations between clinical aspect (left), ultrasound (middle) and orthotic devices (right) in the post-operative period: a) first presentation with a moderate warm, red and swollen left foot; color Doppler found in dorsal aspect, longitudinal (b) and midplantar views (c), hipervascularized synovitis (arrowheads) in the navicular-lateral cuneiform joint (curved arrow) and large (white arrows) or small (yellow arrows) ‘hot’ bone erosions; d) the aspect of the inappropriate flexible footwear of the patients. Stable remitted clinical edema after 6 (e) and 12 months (j); gray-scale and power Doppler ultrasound showed the remission of the vascualrization in synovium and bone erosions over navicular dorsal (f and k), midplantar longitudinal (g and l) and transverse (h) views (deep-echo suggests inactive erosion, green arrow), after wearing the Aircast-AFO (i) followed by the accommodative foot-orthosis/rigid rocker-bottom boot (m).
Discussion

The sensory protective loss will cause inaccurate nociception while the autonomic neuropathy will promote osteolysis via dysfunctional bony arterioles. Many other interrelated factors are causal, predisposing or precipitating in people susceptible to CN [2,5]. Any (non)surgical, repetitive events or minor cumulative trauma (gait), can generate local inflammatory and osteoclast hyperactivity [5-7].

If the peri-articular reactive marrow edema (ME) represents the earliest active-stage, the Charcot-joint’s synovium has an outstanding pathogenic role. The aggravation of local inflammation foreshadows the bone erosions location (future resorptive areas) and the growing damages [7,8].

The CN is classified on clinical, anatomical or imaging criteria but the first diagnostic imaging technique remains the X-ray [9-11].

In our patient, the diagnostic delay with unprotected-gait (high-internal stress) followed by insufficient offloading, led to residual, unstable rocker-bottom deformity; being clinically inactive and radiologically in stage 2/3 Eichenholz, it allowed arthrodesis to adapt to the destructive pattern. Five months post-op we noticed (sub) acute clinical appearance of his foot, but with near-symmetrical IRT. This is an important but debatable criterion, in terms of clinical activity [9,12].

Despite the stable radiological image and irrelevant IRT, US confirmed and localized the inflammatory processes. The premature and inadequate loading (flexible-footwear), residual but hyperactive synovium, and post-op activation of pro-inflammatory cytokines may explain the inflammatory recurrence in the operated area. The surgical event itself and certain biomechanical alterations partly explain the anatomical re-location [8,13] as was in the native fifth metatarsal-cuboid joint.

Overall, the type of surgery and host-attributes put at high-risk the conflict between the orthopedic-device and host-tissues [14,15]. The medial column stabilization arthrodesis displayed the mild inflammatory reactions as a conflict between the orthopedic-hardware, the navicular bone and the first-ray. However, the patient’s continuously evolving morbid background remains an important contributor to these various inflammatory processes.

The particularity of the case results from the exclusive US monitoring of the operated CN in view of the transition to the reloading phases. This technique, amongst other helpful attributes, is not fundamentally artificed by metal-implants and has a salutary long-distance utility considering the medical context. We could not find similar studies in the literature on this topic.

In conclusion, this case proves the reciprocal relationship between the US findings and the effectiveness of the offloading devices in modifying the balance between external forces and internal pathological forces activated by the affected structures.

References


