Charcot arthropathy is an acute or subacute, often indolent, non-infectious or tumorous osteoarticular destruction of weightbearing skeletal structures in patients with reduced pain perception attributable to peripheral neuropathy mostly affecting the foot and ankle. The diagnosis is often missed at initial presentation, and it is well established that this kind of disease due to neuroarthopathy reduces overall quality of life and increases the morbidity of patients. Because of delayed treatment, nearly half of the patients develop new ulcer formations with an increased risk of amputation.

Case Report

A 43-year-old male presented to our orthopedic clinic with a hypertrophic left foot and an ulcer (2 × 2 cm) beneath the interphalangeal joint of the hallux resistant to conservative treatment (Figs. 1A-B). The patient also suffered from intermittent pain at the third metatarsophalangeal joint plan-
III with secondary overlength of digitus II due to destructive shortening of digitus I and pain to palpation at the plantar aspect of the first and third metatarsophalangeal joint. A stocking shaped hypoesthesia of both legs without motor dysfunction was present. Dorsalis pedis artery and tibialis posterior artery pulses were palpable.

Blood examination was unremarkable for C-reactive protein, white blood count, and uric acid.

Radiographic Findings

Native radiographs revealed soft-tissue swelling and massive destruction of the first metatarsophalangeal joint with areas of atrophic and hypertrophic bone changes and complete disorganization of the first metatarsophalangeal joint with nearly complete loss of joint space (Figs. 2A-B). Sclerotic bone and osteophytes surrounded the joint. Signs of an old fracture of the second metatarsal with cloudy callus between metatarsal II and III (B).
fracture of the second metatarsal were seen with cloudy callus between metatarsal II and III (Figs. 2A-B).

**Treatment**

The patient was taken to the operating theatre and an arthrodesis of the first metatarsophalangeal joint with resection of the hypertrophic bone and osteophytes using a locking plate were performed. We also conducted a condylectomy of the base of the proximal phalanx digitus II and III as well as a shortening osteotomy of the third metatarsal. The ulcer was debrided and primarily closed by suture. There was no evidence for bone infection intraoperatively. Microbiologic cultures were negative, the histopathological examination demonstrated bone changes characteristic for Charcot arthropathy (Fig. 3).

Two weeks after surgery the Kirschner wires were removed and mobilization without weight-bearing in a postoperative shoe was continued for another 4 weeks. After that time the ulcer was completely healed and the arthrodesis had fused. No pain was reported by the patient who has already been integrated in the working process again.

The last follow-up was conducted 1.5 years after surgery. The patient was still satisfied, there was no pain or new ulcer reported. He wears special orthopedic shoes and he is mobilized with full weightbearing without complications (Figs. 4A-B and 5).

**Discussion**

Peripheral neuropathy, foot deformities, external trauma, and peripheral edema are the most common causes for Charcot arthropathy and accompanying ulcer.4–6 The main localization of Charcot arthropathy are the Lisfranc and Chopart joints, the ankle, and the subtalar joint. Uncommon is the area of the

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**Figure 3.** Intraoperative situs showing the enormous dimensions of the bone and complete destruction of the joint.

**Figure 4.** Lateral (A) and anteroposterior (B) radiographs with weightbearing 18 months postoperatively demonstrate that arthrodesis had fused.
forefoot that is reported in this case. In the current case, Charcot arthropathy was diagnosed in the first metatarsophalangeal joint (Sanders and Frykberg Type I) with all typical clinical and radiological signs.

The cause for an active Charcot process is an insensate foot. Somatic neuropathy allows repeated insensate injury of the affected region and often results in delayed treatment.

Cumulative factors are additional deformities of the foot like claw toes, hallux valgus, pes cavus, or a long ray, like in this case.7

An important differential diagnosis is osteomyelitis, which sometimes is not easy to exclude, especially in patients with an ulcer.5 In our case there was no clinical or histopathologic evidence of infection or osteomyelitis.

Because of the complexity of Charcot arthropathy there is an increased risk for postoperative complications like delayed bony healing, wound infections, osteomyelitis, poor foot alignment and soft-tissue complications. Because of frequent clinical follow-ups, weightbearing restrictions, therapeutic footwear, and soft-tissue care is imperative to decrease complications and improve long-term results.8

Conclusions

The authors present a rare case of progressive Charcot arthropathy of the first metatarsophalangeal joint attributable to peripheral neuropathy. We demonstrate the progress of this destructive disease and the successful treatment performed.

Owing to the complexity of Charcot arthropathy, careful preoperative evaluation, timing, and dimension of surgery are important as well as treatment of associated comorbidities and sufficient postoperative care to reduce the complication rate and improve long-term results.

Figure 5. No signs of ulcer 1.5 years after surgery.

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References