A 63-year-old man was hospitalized because of severe lesions in his right foot (Figure 1A). These evolved with minor pain and had first been observed approximately ten days before admission. The changes were initially limited to the plantar surface of the second toe, but the patient did not remember any recent trauma, because of a chronic loss of local sensibility. Remarkable antecedents were metabolic syndrome, arterial hypertension and type 2 diabetes, which were controlled by another service by diet in addition to metformin, gliclazide, and losartan. Two years previously, he had undergone a surgical procedure involving the distal area of the first right toe. His mother 83-years of age had had an amputation of part of the left extremity caused by similar conditions. Notwithstanding, he did not get enough knowledge about the self-management in this setting. He denied tobacco smoking, but confirmed social alcohol consumption. His evaluation on admission showed body mass index (BMI) 28.8 kg/m², waist circumference 110 cm, and arterial pulses in the lower limbs of decreased amplitude. The rest of the physical examination was unremarkable. Routine blood tests showed moderate anemia, mild neutrophil leukocytosis, elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) level, hyperglycemia, hypoalbuminemia, and high urea and creatinine levels. Cultures from samples of the foot wounds revealed *S. aureus* sensitive to clindamycin and ciprofloxacin, and these drugs were administered. The imaging studies of the affected foot disclosed bone and arterial changes (Figures 1B and 2). The patient underwent a surgical procedure on the day 4 of admission, and the blood control tests showed an improvement of hematological indices and normalization of renal function. After hospital discharge, he is under specialized surveillance at the orthopedic rehabilitation service.

What is your diagnosis?

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**Figure 1. A**: An extensive severe deep ulcer on the plantar surface of the right foot, after debridation of necrotic tissues, showing mummified features of the second toe, while the third toe appears with evidence of an ischemic disturbance. Surgical scars can be noticed over the big toe; **B**: Roentgenogram of the right foot showing destructive changes in the distal phalanges of the big and second toes, thinning and areas of destruction in some phalangeal and metatarsal metaphyses.

**Figure 2**: Phalangeal and metatarsal changes in detail, emphasizing the destructive changes observed on metatarsal heads and the increased density of periosteum along the metaphyses; worthy of note, the pedal arteries appear with a “rail tracking” aspect (arrows).
**ANSWER to PHOTO QUIZ**

**Diabetic foot (Grade 4 of Wagner and Meggitt’s classification)**

Diabetic foot is a severe complication of diabetes which is growing in incidence all over the world following the increasing number of people with this metabolic disease [1-5]. This is an anatomical and functional disorder due to peripheral occlusive arteriopathy and/or diabetic neuropathy. The expression “infected diabetic foot” has been utilized to characterize all inframalleolar infections that are diagnosed in individuals with diabetes mellitus [6]. Because non-invasive procedures have no efficacy, major amputations have increased in frequency in consequence of the lack of educational preventive measures and/or late medical treatment [1,7]. In the USA, 20.8 million people are diabetic and 2.9 million will have a foot ulcer during their lives; up to one quarter of individuals in this group will undergo some lower limb amputation [1,3]. Risk factors of diabetic foot are longstanding uncontrolled diabetes, local trauma, and smoking. The causes of amputation are trauma, ulcers, and difficult wound healing due to infection [2-5]. Up to 20% of the hospitalizations involving diabetic individuals are associated with foot ulcers, a major complication that has accounted for up to 70% of all the non-traumatic amputations [3,5,7]. It is worthy of note, that the group of diabetic individuals is up to 40 times more likely to undergo an amputation, and they present a higher mortality rate in comparison with non diabetic people [2,3,5,7]. The origin of diabetic foot ulcers is classified as neuropathic, ischemic and neuroischemic [5]. Studies have described prevalent neuroischemic foot in more than 80% of patients in this group [5]. Foot changes described here were initially detected on the plantar area of the toes, a phenomenon mainly observed in diabetic patients with neuropathic lesions in the lower extremities [4]. Peripheral diabetic neuropathies affecting the lower limbs predispose to repetitive minor trauma and ulcerations due to loss of local defensive sensation, in addition to uneven plantar pressure [4,6]. The lesions further evolve to the dorsal surface, suggesting the role of a vasculopathic factor [4]. As the vascular images of the affected foot showed extensive peripheral artery disease (Figure 2), one should consider that the association between neuropathic and ischemic mechanisms is consistent [3-5].

Pedal arteries with the “rail tracking” aspect of Monckeberg’s arteriosclerosis, and an increased density of metaphyses in the periosteum indicate a major circulatory impairment. The authors strongly believe that Monckeberg’s arteriosclerosis played a main role in this case. This condition is characterized by diffuse and circumferential calcification of the media layer of medium and muscular arteries usually without obstruction of the lumen [5]. However, the condition may also contribute to local thrombosis and distal artery obstruction. In addition to chronic renal failure, it is related to ageing, male gender, osteoporosis, diabetes and autonomic neuropathy; it occurs more often in the lower limbs of males, and has a “rail tracking” appearance in routine roentgenograms. Although education about self care to prevent foot lesions can reduce the amputation rates [4,7]. This cost-effective method has hardly been achieved in the vast majority of developing countries. Education about diabetic foot should be given by specialist doctors and nurses, or podiatrists [7]. Another major concern is the insufficiently updated knowledge of primary care physicians and of non specialist health care workers about the classification of risks associated with this condition [1]. Without a consensus, diverse classifications have been described.

Examples include those of Wagner and Meggitt’s, those from the University of Texas, Pedis, King’s, Kobe’s, and Amit Jain’s, among many others [1]. The Wagner-Meggits’ classification is often used in clinical settings and includes five grades: Grade 0 - only symptoms (pain); Grade 1 - superficial ulcers; Grade 2 - deep ulcers; Grade 3 - ulcer with bone involvement; Grade 4 – gangrene of the distal foot; and Grade 5 - total foot gangrene [1,2]. Kobe’s classification includes four types: Type 1 – mainly peripheral neuropathy; Type 2 – mainly peripheral arteriopathy; Type 3 - mainly infection; and Type 4 – a combination of these 3 types [1]. Amit Jain’s classification emphasizes the diabetic foot complications, and includes the following types: Type 1 – infective (cellulites, abscess, necrotizing fascitis, wet gangrene, etc.); Type 2 – non-infective (Charcot foot, peripheral arteriopathy, neuropathy, etc.); and Type 3 – mixed 1 and 2 [1].

This 63-year-old man described here had poorly controlled diabetes with a Grade 4 diabetic foot, a deep infected ulcer and ischemic necrosis, evolving to dry gangrene in the second right toe [1,2]. The diagnosis of osteomyelitis was further ruled out by respective complementary evaluation. Although this most affected toe become mummified, it did not develop into an autamputation [4]. Therefore, the patient underwent routine local care, insulin therapy, and antibiotics in the preoperative management for the tarsal and metatarsal amputation performed on his right foot [2,3]. Twelve sessions of hyperbaric oxygen therapy (on 2.5 atm abs for 60 minutes) were also utilized. The acronym TIME is used to describe the assessment and control of the foot wounds: Tissue management; Inflammation and infection control; Moisture balance; and Epithelial edge ulcer advancement [4]. Depending on the wound’s severity, the treatments of diabetic foot include prostaglandins, low-dose urokinase, hyperbaric oxygen and stem cells, in addition to diverse arterial surgical procedures [3,6]. The aim of the present account was to highlight an advanced stage of this severe diabetic complication, emphasizing the role of the patient’s education about prevention. The high medical and social burden associated with this disabling condition can be minimized by adoption of multidisciplinary preventive programs.

**References**