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Diabetic foot syndrome: Charcot arthropathy or osteomyelitis? Part I: Clinical picture and radiography

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Abstract

One of significant challenges faced by diabetologists, surgeons and orthopedists who care for patients with diabetic foot syndrome is early diagnosis and differentiation of bone structure abnormalities typical of these patients, i.e. osteitis and Charcot arthropathy. In addition to clinical examination, the patient's medical history and laboratory tests, imaging plays a significant role. The evaluation usually begins with conventional radiographs. In the case of osteomyelitis, radiography shows osteopenia, lytic lesions, cortical destruction, periosteal reactions as well as, in the chronic phase, osteosclerosis and sequestra. Neurogenic arthropathy, however, presents an image resembling rapidly progressing osteoarthritis combined with aseptic necrosis or inflammation. The image includes: bone destruction with subluxations and dislocations as well as pathological fractures that lead to the presence of bone debris, osteopenia and, in the later phase, osteosclerosis, joint space narrowing, periosteal reactions, grotesque osteophytes and bone ankylosis. In the case of an unfavorable course of the disease and improper or delayed treatment, progression of these changes may lead to significant foot deformity that might resemble a "bag of bones". Unfortunately, radiography is non-specific and frequently does not warrant an unambiguous diagnosis, particularly in the initial phase preceding bone destruction. For these reasons, alternative imaging methods, such as magnetic resonance tomography, scintigraphy, computed tomography and ultrasonography, are also indicated.

Introduction

Diabetes is sometimes called a global epidemic of the 21st century. Its prevalence has been alarmingly growing in the recent years; the number of patients with diabetes is projected to exceed 500 million by 2030[1]. In Poland, there are approximately 2.5 million people with diabetes with a considerable part of cases remaining undiagnosed[2]. Moreover, demographic changes, i.e. longer life expectancy and population ageing, mainly in developed countries, make treatment of diabetes and its complications a growing problem for clinicians and for the entire healthcare system.

Despite considerable progress in diabetes treatment, particularly in terms of its acute complications (hypo- and hyperglycemic coma), chronic consequences of this disease still constitute a serious clinical problem and lead to increased mortality, disability and lower quality of life. Pathological processes resulting from long-term glucose metabolism abnormalities affect the cardiovascular system, kidneys and eyes as well as the nervous system, musculoskeletal system and skin, which leads to severe abnormalities within the feet.

Diabetic foot syndrome

Diabetic foot syndrome, as defined by the World Health Organization, is infection, ulceration or deep tissue destruction associated with neurological disorders or peripheral vascular disease within the feet of patients with diabetes. These abnormalities may significantly affect the quality of life and, in most advanced cases, cause long-term hospitalizations. They are also the most common cause of non-traumatic extremity amputation[3].

Foot deformity resulting from motor nerve fiber abnormalities, e.g. in the form of mallet toes and high-arched foot, results in inappropriate weight bearing on the plantar surface of the foot and, in combination with impaired pain sensation, leads to the occurrence of poorly healing wounds on the feet of patients with diabetes. Wounds and ulcerations can also be caused by gait abnormalities occurring in the course of neuropathy as well as by frequently occurring edema (particularly in older patients) and visual acuity disorders caused by retinopathy which increases the risk of injury. Soft tissue ulceration in the feet is a very frequent complication; a lifetime risk is estimated at even 25%[4].

These ulcerations, frequently unnoticed by patients for a long time, are often a route of soft tissue infection, which in nearly 20% of cases also leads to bone involvement[5]. Unless intensive treatment is implemented instantaneously, amputation becomes necessary in many cases. The risk of amputation increases by 1.2-fold in cases with concomitant neuropathy, by 12-fold in cases with deformity and by 36-fold in patients previously treated for foot ulceration[6]. It is estimated that foot amputation due to diabetes is conducted every 20 seconds in the world. The risk is especially high if neuropathy is accompanied by lower extremity ischemia. Atherosclerotic changes develop earlier in diabetic patients than in individuals without diabetes. They involve many vascular segments and usually affect arteries distal to the knee joint[7].

Research indicates that, in almost 60% of cases, amputation is preceded by ulceration complicated with infection[8]. According to international guidelines, infection in the course of diabetic foot syndrome is diagnosed when there are at least two Galen's signs (increased temperature, edema, redness, pain), or if purulent discharge or general symptoms are present. Laboratory tests may also be useful in the diagnostic process. However, infection, even in severe cases, such as osteomyelitis, is manifested by increased leukocytosis in only 50% of diabetic patients[8]. ESR, and sometimes CRP, may be more helpful, although the level of CRP may increase also in the case of isolated soft tissue infection. Since the diagnosis of osteitis/osteomyelitis is frequently a significant clinical problem, it seems reasonable to combine evaluation of several parameters. In a meta-analysis conducted by Dinh *et al*., the most valuable clinical indicator of osteomyelitis was the positive probe-to-bone test[9], while Ertugrul *et al*. suggested a combination of the analysis of the size of ulcers with the ESR value with sensitivity and specificity for osteomyelitis (for cut-off points of 2 cm^2 and 65 mm/h) reaching 83% and 77%, respectively[10].

The gold standard in the diagnosis of osteomyelitis is bone biopsy with microbiological and histopathological evaluation. Nonetheless, it is an invasive procedure that requires considerable technical skill. That is why the diagnosis is usually based on combined evaluation of the patient's medical history, clinical picture and the results of additional tests.

In many cases, it is a significant problem for doctors tending to patients with diabetes to identify inflammatory changes within the bone structures of the feet early and to distinguish them from neurogenic arthropathy, which is not that common but also typical of diabetic foot syndrome.

Neurogenic arthropathy

Neurogenic arthropathy is also called Charcot arthropathy after the name of Jean-Martin Charcot, a French neurologist, who in 1868 described destructive chang-

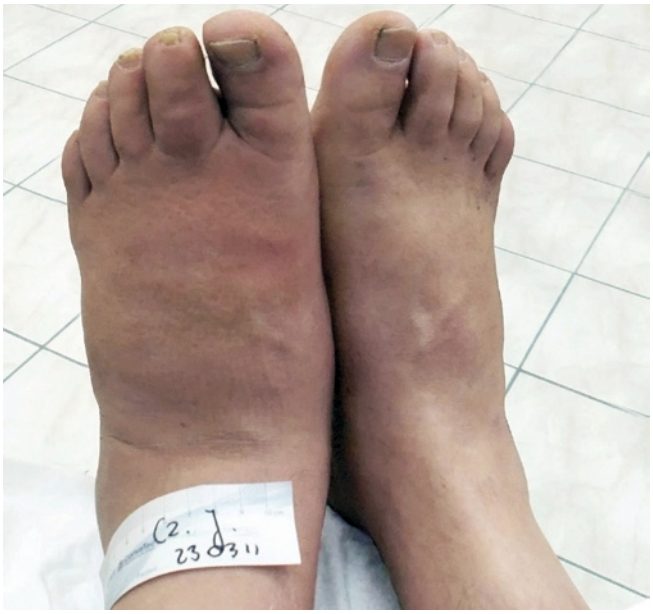

Fig. 1. *Patient with diabetic foot syndrome. Redness of the left foot and intensive edema in the active phase of Charcot arthropathy (courtesy of Dr. Anna Korzon-Burakowska, MD PhD)*

es within the musculoskeletal system in the course of central nervous system syphilis[11]. It was only in 1936 when an American doctor, William Riley Jordan, found an association between similar changes within the musculoskeletal system and diabetes[12]. In the 20th century, increasing use of antibiotics for syphilis and insulin for diabetes changed the natural history of these disease entities radically and resulted in the fact that, currently, the most common form of Charcot arthropathy is destruction of osteoarticular structures of the feet that develops on the background of diabetic neuropathy[13,14]. However, the literature also reports similar changes in the course of various peripheral and central nervous system conditions, such as syringomyelia, leprosy, alcoholism, heavy metal poisoning and secondary to injury[13]. They may affect the lower and upper extremities and the lower segment of the spine. In considerations about their not clearly explained pathogenesis, attention is paid to the neurotraumatic theory (pain sensation disorders result in repeated microinjuries that lead to bone and joint destruction) and the neurovascular theory (hyperemia resulting from autonomic neuropathy leads to osteopenia and susceptibility to injury). A combination of both these theories is probably the most accurate[15–19].

To date, there have been no population-based epidemiological studies, but diabetic patients are estimated to develop neurogenic arthropathy in approximately 0.1–0.5% of cases[20–22]. In patients with peripheral neuropathy, even 16% may suffer from arthropathy[23]. Recent clinical observations suggest that the frequency of this complication is increasing[24]. It mostly affects patients with a long history of poorly controlled diabetes, but cases of neurogenic arthropathy as the presenting sign of diabetes have also been reported[25]. In patients suffering from diabetes since childhood, these changes may develop as early as in the third or fourth decade of life.

Diabetic neuro-osteoarthropathy usually affects one extremity. Nevertheless, some authors report its bilateral occurrence in up to 75% of patients[24,26,27]. The course of Charcot arthropathy involves an active phase with non-specific symptoms that might suggest infection (edema, redness and increased temperature of the skin; the difference in temperature between both feet is usually >2°C) (Fig. 1). This clinical picture may lead to misdiagnosis as venous thrombosis, gout or pedal soft tissue and bone infection[28]. In some cases, patients report a minor injury (sprained ankle or tripping) or foot surgery, which are considered to be triggering factors of the destructive process[29]. In advanced diabetic neuropathy, patients tend to report pain of surprisingly low intensity, which is misleading for both patients and physicians who do not specialize in diagnosing diabetic foot syndrome. The lack of ischemia is also typical; pulse in the peripheral vessels is well-palpable.

The basic method to treat the acute phase of Charcot arthropathy is total contact cast (TCC) and offloading the affected limb as the lack of immobilization and continued weight-bearing may lead to irreversible bone destruction and significant foot deformity, frequently with the collapse of the longitudinal arch (rocker-bottom deformity) or formation of a medial convexity[14] (Fig. 2).

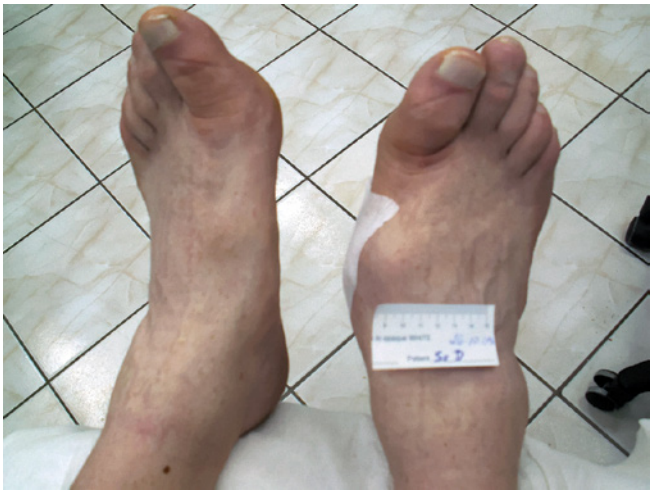

Fig. 2. *Patient with diabetic foot syndrome. Typical deformity of the right foot with medial convexity resulting from bone destruction and dislocations in the course of Charcot arthropathy (courtesy of Dr. Anna Korzon-Burakowska, MD PhD)*

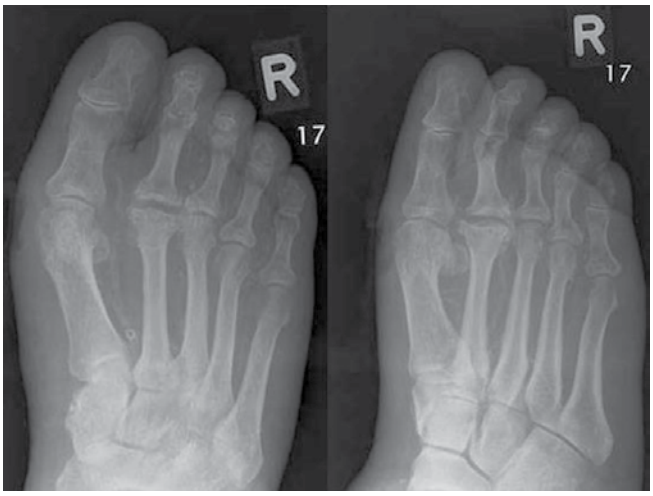

Fig. 3. *Radiographs in the dorsiplantar and oblique views. Soft tissue defect at the level of the 1st MTP joint. Osteopenia and blurred outline of the cortical layer of the head of the 1st metatarsal in the course of osteomyelitis*

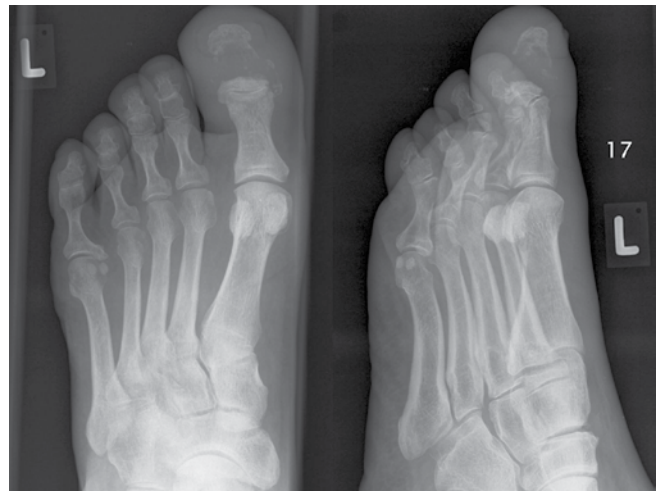

Fig. 4. *Radiographs in the dorsiplantar and oblique views. Significant soft tissue edema of the 1st toe. Osteolysis of the distal phalanx of the first ray. Radiography is consistent with infection within the soft tissues and bone structures*

Later in the course of the disease, edema, increased temperature and redness regress, and the patient may gradually start loading the foot. However, bone and articular destruction that has occurred in the course of improper treatment of the acute phase do not regress and can lead to a significant foot deformity, non-physiological distribution of weight-bearing forces and increased risk of ulceration with serious consequences. In this situation, the patient may require surgical intervention to correct the deformity. In extreme cases, foot amputation becomes necessary when the foot no longer performs its supportive function or is affected by a life-threatening infection.

The clinical picture of Charcot arthropathy, particularly in the acute phase, is non-specific, and results in a considerable delay of diagnosis in even 25% of cases, which significantly affects final treatment outcomes[30]. The differentiation of the nature of abnormalities requires combined assessment of the clinical picture and the results of additional tests. To date, no specific markers or laboratory parameters confirming the diagnosis of Charcot arthropathy have been discovered. It is usually recommended to test white blood cells, CRP, ESR and uric acid as well as to conduct a Doppler examination of the lower extremity veins in order to rule out a different etiology of symptoms. Imaging is indispensable to precisely assess the osteoarticular structures as, in many cases, it may play a very important role in the differentiation of changes within the feet of diabetic patients.

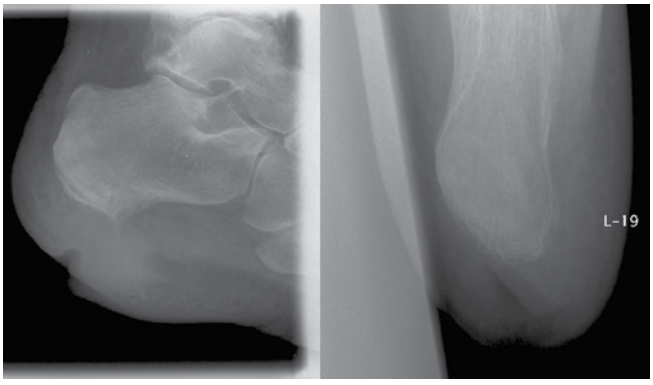

Fig. 5. *Radiographs of the calcaneus in lateral and axial views. Soft tissue defect as well as irregular and blurred outlines of the calcaneal tuberosity; abnormalities in the course of osteomyelitis*

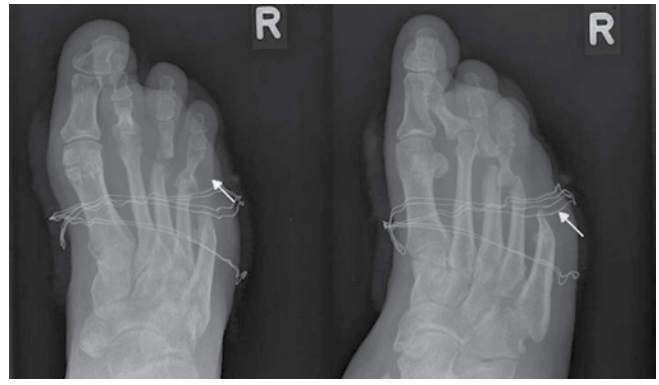

Fig. 6. *Radiographs in the dorsiplantar and oblique views. Status post surgical amputation of the 5th ray through the distal fragment of the 5th metatarsal and of the distal 3rd–4th metatarsals. 2nd MTP joint subluxation. Blurred outlines of the 3rd and 4th metatarsal stumps in the course of osteomyelitis*

Imaging in diabetic foot syndrome

Diagnostic methods used in imaging of bone abnormalities in the course of diabetic foot syndrome include plain radiography, magnetic resonance tomography, computed tomography, ultrasonography and isotope methods.

Plain radiography

Despite dynamic advancement in the field of medical imaging, plain radiography remains the first-choice examination in the case of suspicions of abnormalities within bone structures of the feet as it is broadly available and, despite limited sensitivity and specificity, it narrows the differential diagnosis in many cases.

In the case of osteomyelitis, radiography shows osteopenia, lytic lesions, cortical destruction, periosteal reactions as well as, in the chronic phase, osteosclerosis and sequestra[9,31]. However, signs of inflammation can be seen in radiography only with 30–50% reduction of bone mineral content, even 2–4 weeks after the onset of the process. Earlier signs include non-specific soft tissue edema and, in some cases, a defect at the site of ulceration or the presence of soft tissue gas. Unambiguous bone abnormalities, such as cortical fragmentation, marked osteopenia or periosteal reactions, observed in radiography in direct proximity to ulcerations are usually considered a sign of osteomyelitis, unless other etiology has been confirmed (Fig. 3, Fig. 4, Fig. 5, Fig. 6).

Unfortunately, the value of plain radiography is limited in the initial stage of osteomyelitis. In a meta-analysis conducted by Dinh *et al.*, the sensitivity and specificity of this method in the diagnosis of osteomyelitis were 54% and 68%, respectively[9]. This limited diagnostic value of radiography is mainly associated with late presentation of abnormalities. That is why, a repeated examination is recommended; only the absence of identifiable bone abnormalities over several weeks rules out osteomyelitis with a high probability.

Also in the case of Charcot arthropathy, the identification of bone changes in the early stage, when the most effective treatment is possible, frequently goes beyond the abilities of plain radiography. These abnormalities appear late, when the disease progresses, and they attest to progressive bone and articular destruction that was not prevented. Gradually, an image resembling rapidly progressing osteoarthritis combined with aseptic necrosis or inflammation develops.

The image includes: bone destruction with subluxations and dislocations as well as pathological fractures that lead to the presence of bone debris, osteopenia, and, in the later phase, osteosclerosis, joint space narrowing, periosteal reactions, grotesque osteophytes and bone ankylosis. In the case of an unfavorable course and improper treatment, progression of these changes may lead to significant foot deformity, that might resemble a "bag of bones"[32–35].

Type	Localization
Type 1	Involvement of the forefoot joints: interphalangeal and metatarsophalangeal joints
Type 2	Involvement of the tarsometatarsal joints
Type 3	Involvement of the cuneonavicular, talonavicular, and calcaneocuboid joints
Type 4	Involvement of the talocrural joint
Type 5	Involvement of the calcaneus

Tab. 1. *Charcot arthropathy classification in terms of localization according to Sanders and Mrdjenovich[36]*

The literature distinguishes two patterns of abnormalities in the course of neuroarthropathy: an atrophic pattern (with prevailing bone resorption, which frequently mimics septic arthritis or a proliferative process) and a hypertrophic pattern (with prevailing osteogenic processes that suggest severe osteoarthritis)[13]. However in some cases, atrophic and hypertrophic changes coexist with each other, or osteogenic processes may prevail after the initial resorption phase, which makes the division between these two types of patients clinically irrelevant.

Out of five localizations typical of Charcot arthropathy (Tab. 1), the most common are abnormalities in the

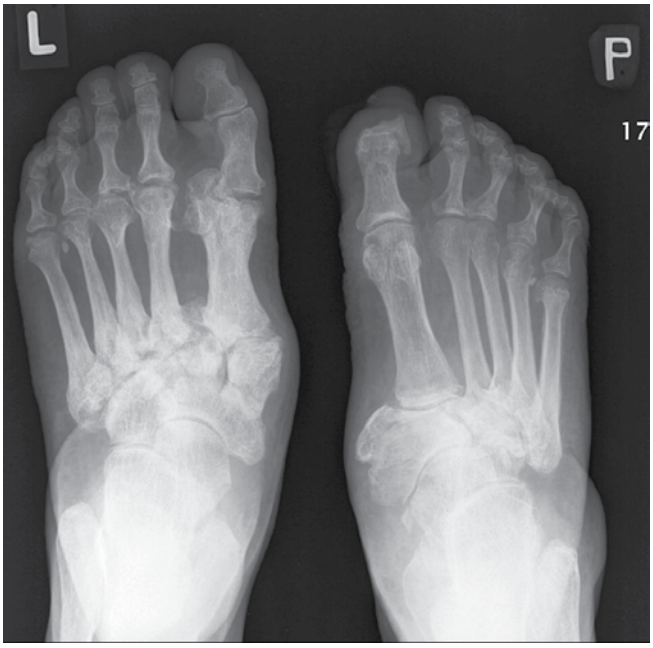

Fig. 7. *Radiograph in the dorsiplantar view. Bone destruction and numerous dislocations within the tarsal bones in the course of Charcot arthropathy*

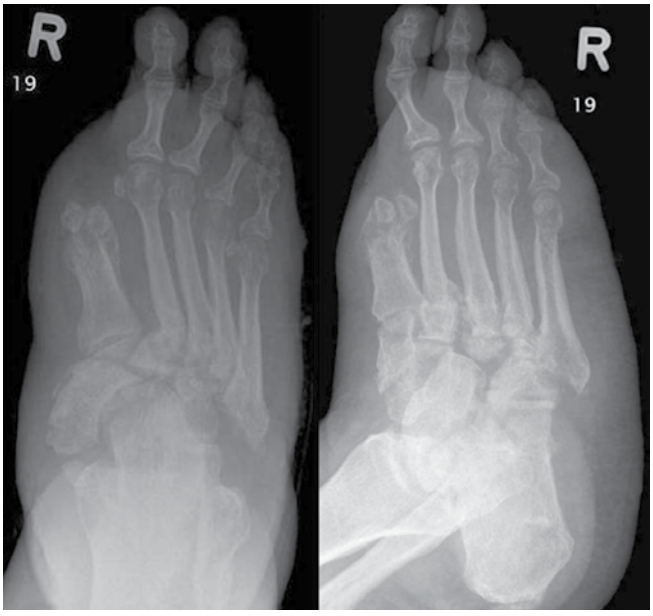

Fig. 8. *Radiographs of the right foot in the dorsiplantar and oblique views. Status post surgical amputation of the first ray through the distal fragment of the 1st metatarsal. Thickening of the soft tissue shadow. Bone destruction and numerous dislocations within the tarsal bones in the course of Charcot arthropathy*

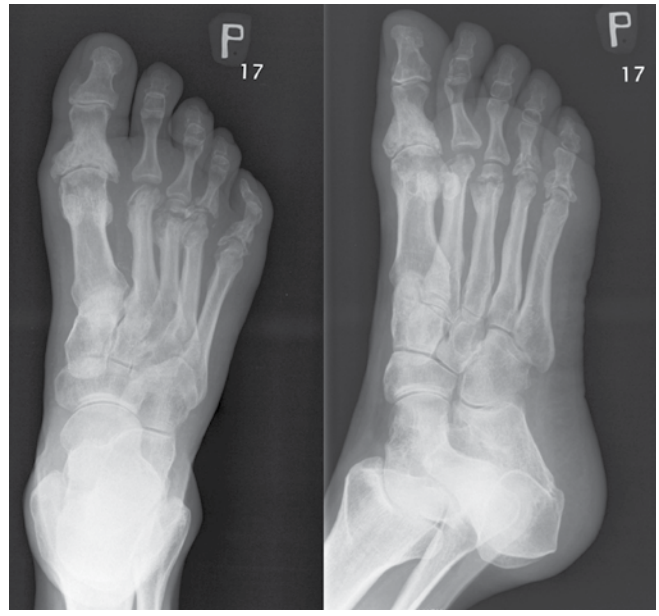

Fig. 9. *Radiographs in dorsiplantar and oblique views. Transverse osteolysis within the tarsal bones in the course of Charcot arthropathy*

tarsometatarsal joints and tarsal joints (type 2 and type 3); more rarely, the disease affects the forefoot and, in even fewer cases, the ankle joint and the calcaneus[29] (Fig. 7, Fig. 8, Fig. 9, Fig. 10, Fig. 11, Fig. 12).

There are a few types of abnormalities, particularly specific for neurogenic arthropathy, that can be identified on plain radiographs of the feet, i.e.:

- subluxation or dislocation in the tarsometatarsal joints, initially subtle but requiring detailed assessment of the distance between the proximal epiphyses of the first and second metatarsals and, in advanced cases, often causing medial convexity;
- tarsal osteoarticular destruction with the collapse of the longitudinal arch and rocker-bottom deformity of the foot;
- transverse osteolysis – detachment of the distal metatarsal epiphyses with preserved articular surface;
- pencil-like tapering of the phalanges;
- mortar-and-pestle deformity;
- avulsion fractures of the posterior calcaneal tuberosity[13].

Based on the analysis of radiographic appearance, neuro-osteoarthropathy has been traditionally divided into three stages:

1) development, when fractures, dislocations and bony debris develop;
2) coalescence, characterized by progressive fusion of bony fragments and sclerosis;
3) reconstitution, when the destruction process is inhibited, and fractures ultimately heal and stabilize[37].

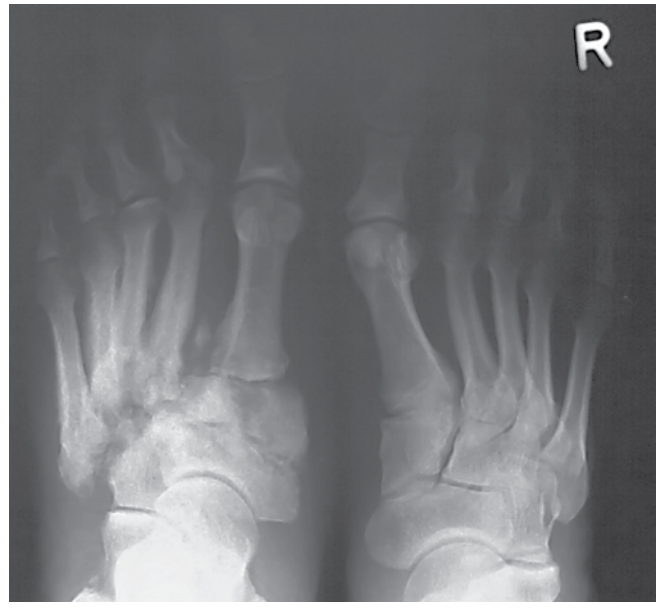

Fig. 10. *Radiograph in the dorsiplantar view. Bone destruction and dislocations within the tarsal joints and tarsometatarsal joints of the left foot in the course of Charcot arthropathy*

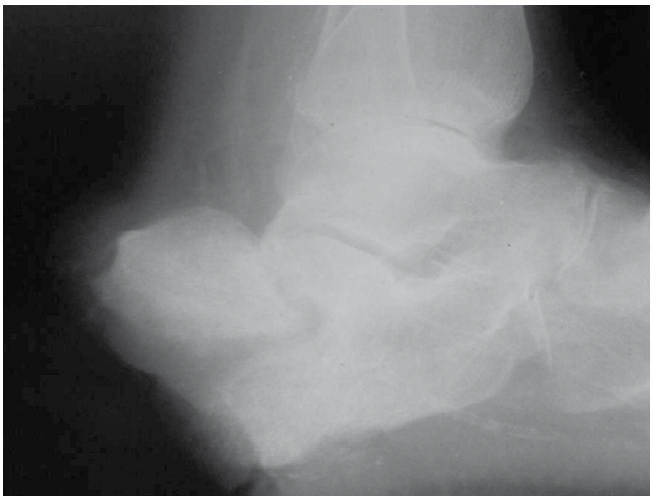

Fig. 11. *Radiograph of the calcaneus in the lateral view. Avulsion fracture of the calcaneal tuberosity. Soft tissue defect, blurred outlines of calcaneal bone fragments: Charcot arthropathy with concomitant osteomyelitis*

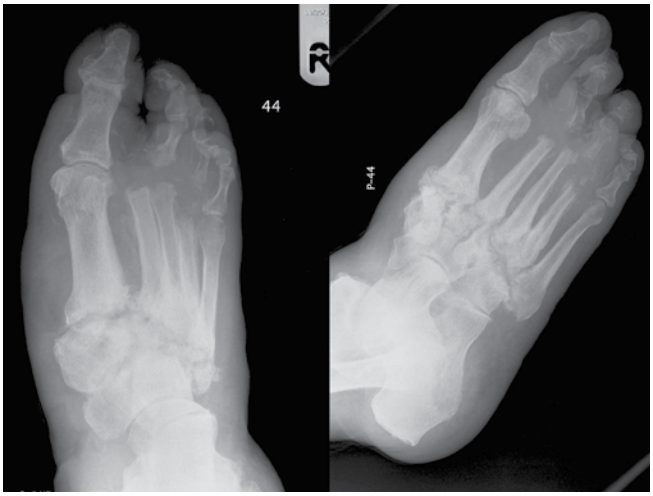

Fig. 12. *Radiographs of the right foot in the dorsiplantar and oblique views. Bone destruction within the tarsal bones in the course of Charcot arthropathy. Osteolysis of forefoot bone structures in the course of osteomyelitis. Thickening of the soft tissue shadow. An example of concomitant osteomyelitis within the forefoot and Charcot arthropathy within the tarsal bones*

Due to the advancement of diagnostic imaging methods that enable identification of the earliest signs of Charcot arthropathy, it was proposed to add stage 0 to the classification to account for the stage when no changes can be identified in radiography, but can be seen in more sensitive imaging methods, such as magnetic resonance tomography and scintigraphy[38].

Conclusion

Early diagnosis and differentiation between osteomyelitis and Charcot arthropathy is a significant diagnostic problem in patients presenting with bone abnormalities in the course of diabetic foot syndrome. Plain radiography remains a widely available and valuable first-choice method for imaging of bone abnormalities. Nevertheless, it is characterized by limited sensitivity, particularly in the early stage of both these processes. In many cases, other imaging modalities, including ultrasonography, are indicated, which is going to be discussed in the second part of this publication.

Conflict of interest

Authors do not report any financial or personal connections with other persons or organizations, which might negatively affect the contents of this publication and/or claim authorship rights to this publication.

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References

1. Whiting DR, Guariguata L, Weil C, Shaw J: IDF Diabetes Atlas: Global estimates of the prevalence of diabetes for 2011 and 2030. Diabetes Res Clin Pract 2011; 94: 311–321.
2. http://www.diabetesatlas.org/resources/2017-atlas.html.
3. Korzon-Burakowska A: Zespół stopy cukrzycowej – patogeneza i praktyczne aspekty postępowania. Choroby Serca i Naczyń 2007; 4: 93–98.
4. Singh N, Armstrong DG, Lipsky BA: Preventing foot ulcers in patients with diabetes. JAMA 2005; 293: 217–228.
5. Lavery LA, Armstrong DG, Wunderlich RP, Mohler MJ, Wendel CS, Lipsky BA: Risk factors for foot infections in individuals with diabetes. Diabetes Care 2006; 29: 1288–1293.
6. Armstrong DG, Lavery LA, Harkless LB: Validation of a diabetic wound classification system. The contribution of depth, infection, and ischemia to risk of amputation. Diabetes Care 1998; 21: 855–859.
7. Young MJ, Adams JE, Anderson GF, Boulton AJ, Cavanagh PR: Medial arterial calcification in the feet of diabetic patients and matched non-diabetic control subject. Diabetologia 1993; 36: 615–621.
8. Eneroth M, Apelqvist J, Stenström A: Clinical characteristics and outcome in 223 diabetic patients with deep foot infections. Foot Ankle Int 1997; 18: 716–722.
9. Dinh MT, Abad CL, Safdar N: Diagnostic accuracy of the physical examination and imaging tests for osteomyelitis underlying diabetic foot ulcers: meta-analysis. Clin Infect Dis 2008; 47: 519–527.
10. Ertugrul BM, Savk O, Ozturk B, Cobanoglu M, Oncu S, Sakarya S: The diagnosis of diabetic foot osteomyelitis: examination findings and laboratory values. Med Sci Monit 2009; 15: CR307–CR312.
11. Hoché G, Sanders LJ: On some arthropathies apparently related to a lesion of the brain or spinal cord, by Dr. J.-M. Charcot, January 1868. J Hist Neurosci 1992; 1: 75–87.

12. Jordan WR: Neuritic manifestation in diabetes mellitus. Arch Intern Med (Chic) 1936; 57: 307–302.
13. Jones EA, Manaster BJ, May DA, Disler DG: Neuropathic osteoarthropathy: diagnostic dillemas and differetial diagnosis. Radiographics 2000; 20: S279–S293.
14. Rogers LC, Frykberg RG, Armstrong DG, Boulton AJM, Edmonds M, Ha Van G *et al.*: The Charcot foot in diabetes. Diabetes Care 2011; 34: 2123–2129.
15. Brower AC, Allman RM: Pathogenesis of the neurotrophic joint: neurotraumatic vs. neurovascular. Radiology 1981; 139: 349–354.
16. Childs M, Armstrong DG, Edelson G: Is Charcot arthropathy a late sequela of osteoporosis in patients with diabetes mellitus? J Foot Ankle Surg 1998; 37: 437–439.
17. Cundy TF, Edmonds ME, Watkins PJ: Osteopenia and metatarsal fractures in diabetic neuropathy. Diabet Med 1985; 2: 461–464.
18. Forst T, Pfützner A, Kann P, Schehler B, Lobmann R, Schäfer H *et al.*: Peripheral osteopenia in adult patients with insulin-dependent diabetes mellitus. Diabet Med 1995; 12: 874–879.
19. Young MJ, Marshall A, Adams JE, Selby PL, Boulton AJ: Osteopenia, neurological dysfunction, and the development of Charcot neuroarthropathy. Diabetes Care 1995; 18: 34–38.
20. Fabrin J, Larsen K, Holstein PE: Long-term follow-up in diabetic Charcot feet with spontaneous onset. Diabetes Care 2000; 23: 796–800.
21. Klenerman L: The Charcot joint in diabetes. Diabet Med 1996; 13 (Suppl. 1): S52–S54.
22. Bailer CC, Root HF: Neuropathic foot lesions in diabetes mellitus. N Engl J Med 1947; 236: 397–401.
23. Cavanagh PR, Young MJ, Adams JE, Vickers KL, Boulton AJ: Radiographic abnormalities in the feet of patients with diabetic neuropathy. Diabetes Care 1994; 17; 201–209.
24. Rajbhandari SM, Jenkins RC, Davies C, Tesfaye S: Charcot neuroarthropathy in diabetes mellitus. Diabetologia 2002; 45: 1085–1096.
25. Ellenberg M: Diabetic complications without manifest diabetes – complications as presenting clinical symptoms. JAMA 1963; 183: 926–930.
26. Armstrong GD, Todd WF, Lavery FA, Harkless LB, Bushman TR: The natural history of acute Charcot's arthropathy in a diabetic foot specialty clinic. Diabet Med 1997; 14: 357–363.
27. Shaw JE, Boulton AJM: The Charcot Foot. Foot 1995; 5: 65–70.
28. Jude E: Charcot Foot: What's new in pathogenesis and medical management? In: Boulton AJM, Cavanagh PR, Rayman G (eds.): The Foot in Diabetes. John Wiley & Sons 2006: 265–273.
29. Sommer TC, Lee TH: Charcot Foot: The Diagnostic Dilemma. Am Fam Physician 2001; 64: 1591–1598.
30. Gill GV, Hayat H, Majid S: Diagnostic delays in diabetic Charcot arthropathy. Practical Diab Int 2004; 21: 261–262.
31. Lipsky BA, Peters EJ, Senneville E, Berendt AR, Embil JM, Lavery LA *et al.*: Expert opinion on the management of infections in the diabetic foot. Diabetes Metab Res Rev 2012; 28 (Suppl. 1): 163–178.
32. Frykberg RG: Charcot Foot: An update on pathogenesis and management. In: Boulton AJM, Connor H, Cavanagh PR (eds.): The Foot in Diabetes. John Wiley & Sons 2000: 235–260.
33. Dyet JF, Ettles DF, Nicholson AA: The role of radiology in the assessment and treatment of the diabetic foot. In: Boulton AJM, Connor H, Cavanagh PR (eds.): The Foot in Diabetes. John Wiley & Sons 2000: 193–213.
34. Świątkowski J: Osteoartropatie neurogenne. In: Leszczyński S: Radiologia. PZWL, Warszawa 1993: 206–212.
35. Borejko M, Dziak A: Badanie radiologiczne w ortopedii. PZWL, Warszawa 1988.
36. Sanders LJ, Mrdjenovich D: Anatomical patterns of bone and joint destruction in neuropathic diabetics. Diabetes 1991; 40 (Suppl. 1): 529A.
37. Eichenholtz SN: Charcot Joints. With a Foreword by P.D.Wilson. Charles C. Thomas, Springfield (Ill) 1966.
38. Shibata T, Tada K, Hashizume C: The results or arthrodesis of the ankle for leprotic neuroarthropathy. J Bone Joint Surg Am 1990; 72: 749–756.