The Foot 26 (2016) 7–14

Contents lists available at ScienceDirect

The Foot

journal homepage: www.elsevier.com/locate/foot

Review

Current concepts of Charcot foot in diabetic patients

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HIGHLIGHTS

- Charcot neuroarthropathy is uncommon but it leads to ulceration and amputation when is not detect early.
- A new classification system has been suggested to help in early diagnosis.
- New insights for pathological process exist and are covered in this article.
- Medical and surgical managements have been further studied and a better recommendation is suggested.

ARTICLE INFO

Article history:
Received 27 June 2014
Received in revised form 20 August 2014
Accepted 12 November 2015

Keywords:
Diabetes
Charcot
Neuroarthropathy
Foot
Ankle

ABSTRACT

The Charcot foot is an uncommon complication of neuropathy in diabetes. It is a disabling and devastating condition. The etiology of the Charcot foot is unknown, but it is characterized by acute inflammation with collapse of the foot and/or the ankle. Although the cause of this potentially debilitating condition is not known, it is generally accepted that the components of neuropathy that lead to foot complications must exist. When it is not detected early, a severe deformity will result in a secondary ulceration, infection, and amputation. Immobilization in the early stages is the key for success, but severe deformity may still develop. When severe deformity is present, bracing may be attempted but often patients will need surgical intervention. Good success has been shown with internal and external fixation. In patients with concomitant osteomyelitis, severe deformity, and/or soft tissue infection, a high amputation may be the best treatment of choice.

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1. Epidemiology

In 1868, Jean-Martin Charcot described neuroarthropathy in the foot in relation to tabes dorsalis [1]. The author proposed the first theory in how this process may occur. In 1936, Jordan was the first to describe Charcot in diabetes [2]. Charcot neuroarthropathy (CN) is a disabling and devastating condition. Although the cause of this potentially debilitating condition is not known, it is generally accepted that the components of diabetic neuropathy that lead to foot complications must exist. Untreated CN may lead to a rocker bottom foot which will lead to increase plantar pressure in the neuropathic foot. This cascade may lead to an ulceration and possible amputation. A recent study shows, however, that CN alone may not pose a risk for amputation, but CN along with ulceration has 12 times higher risk of having an amputation [3].

The incidence of CN is about 0.1 to 5% in diabetic neuropathy [4–6]. Since neuropathy is a common complication of diabetes, 80% of the cases occur in patients with diabetes for over 15 years, and 60% of the cases in patients with diabetes for over 10 years. The prevalence of CN ranges from 0.08% to 8.5% [6]. Lee et al. found a prevalence of 7.5% of CN in patients with diabetes, and 29% of those had neuropathy [7]. Frequently, this pathology occurs between the 5th and 6th decade with a mean age of 50.3, and no difference between genders [8]. Although it is more common unilaterally, it can involve both extremities in up to 39% of the cases and the incidence of ulceration is 17% per year [9].

2. Classification and staging

There are different types of classifications to describe CN. Most classifications are inconsistent since they have a wide spectrum of description using radiographical changes, clinical locations, and/or pattern of destruction. None of the classification suggests possible

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http://dx.doi.org/10.1016/j.foot.2015.11.001
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Table 1
Clinical and radiological description for Charcot neuroarthropathy.

Stage 0:	Clinically, there is joint edema, but radiographs are negative
Stage 1:	Osseous fragmentation with joint dislocation
Stage 2:	Decreased local edema, with coalescence of fragments
Stage 3:	No local edema, with consolidation and remodeling of fracture fragments. The foot is now stable and a rocker bottom type foot may be seen

treatments or prognostic factors. The first classification describe was by Eichenholtz in 1966 [10]. This classification uses three clinical and radiological stages of progression: the stage of development, stage of coalescence, and stage of reconstruction. The stage of development or stage 1 is defined by periarticular debris formation and fragmentation of subchondral bone, and joint subluxation and dislocation. Clinically, the foot is warm, erythematous, edematous, painful and with bounding pulses. In most occasions, the affected limb needs to be compared to the contralateral limb to appreciate the erythematous changes. During the coalescent stage, or stage 2, the large bone fragments become fused and unite with bone adjacent bones. Absorption of small debris is noticeable as well. Clinically, this foot will present with decreased warmth, and swelling, but instability of the joints may continue. Finally, the stage of reconstruction, or stage 3 is characterized by bone consolidation, and rounding of bone fragments. As revascularization occurs, decrease in sclerosis is appreciated. A stage 0 or pre-CN stage has been proposed where a sudden onset, warmth and erythema occurs in the neuropathic patient. Normal anatomy or joint distention may be observed radiologically [11,12] (Table 1).

The most common locations involved in the foot are midfoot joints (50%), hindfoot joints (28%), followed by ankle joint (19%), and forefoot joints (3%) [13]. Other classifications by location have been described, but resemble previous ones, and have been in small sample studies [5,16,17]. Most recently, Chanteleau and colleagues described an MRI classification of acute CN [14,15]. The best study describing pattern of destruction and proposing risk factors for poor outcomes is the prospective study done by Herbst et al. in 2004 [13]. They followed 55 patients prospectively and classified patients by injury (fracture, dislocation, or combination), and by location (ankle, hind foot, midfoot, and forefoot). They also measured bone mineral density with dual energy X-ray absorptiometry (DEXA). They found that poor outcomes noticed where associated with fracture pattern located at ankle and forefoot.

3. Clinical assessment and imaging

The general consensus is that in order to develop CN the patient has to be neuropathic. Currently, diabetes is the main cause for neuropathy in the lower extremity in industrialized countries [16]. Factors such as osteopenia, equinus, peripheral vascular disease, and nephropathy, have also been described as risk factors for CN [4].

Although the cause of this potentially debilitating condition is not known, a number of theories have been proposed:

- Following the development of autonomic neuropathy there is an increased blood flow to the extremity, resulting in increased bone reabsorption and osteopenia.
- Following sensori-motor neuropathy, the resulting sensory loss and muscle imbalance induces abnormal stress in the bones and joints of the affected limb, leading to bone destruction [17].
- Stretching of the ligaments due to joint effusion, may lead to joint subluxation [18].

The most common presentation is the neuropathic patient which sustains an unperceived, injury, continues to walk until a severe inflammatory process leads to osteopenia, distention of joint, and end stage foot and/or ankle dislocation [19]. In the CDUK audit of Charcot foot by Frame and colleagues, 12% of patients had surgery in the index limb prior to registration of the study. Also, 7% of patients presented with an ulcer were complicated with osteomyelitis [20]. There are two etiological theories to the development of CN. The Neurovascular Theory or French Theory was developed by Charcot in 1868 [1]. After studying more than 5000 chronically ill patients, the author concluded that the profound joint destruction and deformities were secondary to changes in the trophic centers of the spinal cord, specifically the diseased anterior horn cells which resulted in neurogenic and circulatory disruption may result in osteopenia.

In an attempt to support the French theory, Edelman et al. [21] described 3 cases in which CN develops after the lower extremity was revascularized. Subsequently, in a study by Shapiro et al. [22], blood flow was measured with laser Doppler flowmetry with local skin warming in patients with CN, neuropathy, and healthy patients. Increasing local skin temperature increased skin blood flow and vasomotion in healthy and CN patients, but not in diabetes mellitus (DM) with neuropathy alone. The authors concluded that blood flow in the controls and CN patients, despite the extent of neuropathy, is intact. The neurotraumatic theory or German Theory was described in 1870. The theory is based on unperceived trauma or injuries to an insensate joint will lead to stress fractures. This results in progressive and permanent damage to the bone and joints in the foot forming a problematic biomechanical foot. CN feet develop an increase in vertical pressures and shear stress due to deformity and equinus [23].

Most commonly, an acute presentation of the patient presents with erythematous, warm, edematous foot. The most common complaint is pain [19]. Usually, patients will not recall an injury. Clinically, patients demonstrate significant neuropathy with bounding pulses. If the patient does not seek treatment immediately, the foot will collapse giving the appearance of "rocker bottom". When presentation is chronic, a midfoot ulcer may become evident as well the patient continues to ambulate. Although, the classic description is rocker-bottom foot, other deformities such as ankle dislocation abduction of the forefoot, and swollen MPJ's can be evident (Fig. 1).

There are few diagnostic modalities that could be used clinically to assist in the diagnosis of this entity. The infrared cutaneous temperature monitor to detect skin temperature changes is the most accurate tool for diagnosis and monitor progression (Fig. 2). Patients with acute CN experience increased temperature in the affected foot when compared to the contralateral foot. Briefly, the monitor is closely placed to areas in the forefoot, midfoot, and hindfoot, to record the temperature and compare it to the contralateral side at the same location. Skin temperature differences of 4 °F (2 °C) when compared with the contralateral side indicate an active CN, and should be offloaded until normalization of temperature [24]. Laboratory studies are helpful when the diagnosis of CN is not clear. White blood cell count (WBC) with differential will help to distinguish between acute CN and an acute soft tissue or acute osteomyelitis. However, it is a nonspecific marker and may be elevated in CN. C-reactive protein (CRP) could be used to distinguish between acute CN and osteomyelitis and often elevated in infection, but it is a nonspecific marker for inflammation [25].

3.1. Imaging

Radiographs are the most useful in diagnosing the pathology, locate the area of involvement, evaluate quality of bone, and identify if the process is acute or chronic. If an infection is suspected, foreign body and soft tissue emphysema can be identified. Also, radiographs are helpful in correlating the ulcer location and the

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