Current Trends in the Surgical Management of Diabetic Neuroarthropathy of the Foot and the Ankle

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By
Mohamed Ashraf Hammad
(M.B., B.Ch)

Under supervision of

Prof. Dr. Ali Ibrahim Hussein

Emeritus Professor of Orthopaedic Surgery
Faculty of Medicine - Ain Shams University

Dr. Mahmoud Mahran

Assistant Professor of Orthopaedic Surgery
Faculty of Medicine - Ain Shams University

Faculty of Medicine
Ain Shams university
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بسم الله الرحمن الرحيم

وقُلِ اعْمَلُوا فَسَيَرَى اللَّهُ عَمَلَكُمْ وَرَسُولُهُ والْمُؤْمِنُونَ

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List of abbreviations

ABPI	:	Ankle–brachial pressure index
CD	:	Charcot deformity
CGRP	:	Calcitonin gene-related peptide
CN	:	Charcot neuroarthropathy
CROW	:	Charcot Restraint Orthotic Walker
CT	:	Computerized tomography
EBG	:	Electric bone growth
HMPAO	:	Hexamethylpropyleneamine oxime
IL-1	:	Interleukin-1
In-WBC	:	Indium-111 WBC
NF	:	Nuclear transcription factor ß
NF-KB	:	Nuclear factor kB
OM	:	Osteomyelitis
OPG	:	Osteoprotegerin
PAD	:	Peripheral arterial disease
PPWB	:	Prefabricated pneumatic walking brace
RANKL	:	Receptor activator of NFk ß ligand
T1DM	:	Type 1 diabetes mellitus
T2DM	:	Type 2 diabetes mellitus
TCC	:	Total contact cast
Tc-MDP	:	Technetium 99m methylene di-phosphonate
TcPO2	:	Transcutaneous oxygen diffusion
TNF-α	:	Tumor necrosis factor–alpha
TSF	:	Taylor spatial frame

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Introduction

Neuropathic joints, often called Charcot joints, are caused by loss of sensation in the joint so that it is severely damaged and disrupted. The damage and disruption is often so gross that the diagnosis of a neuropathic joint is easily made, both on clinical examination and X-ray. (1)

However, the concept that the patient without sensation smashes the joint with impunity may be an over simplification as there may be problems related to autonomic neuropathy, poor blood supply and mismatch of bone destruction and synthesis.(2)

Any condition that causes sensory or autonomic neuropathy can lead to a Charcot joint. Charcot arthropathy occurs as a complication of diabetes, syphilis, chronic alcoholism, leprosy, meningomyelocele, spinal cord injury, syringomyelia, renal dialysis, and congenital insensitivity to pain(3). Diabetes is considered to be the most common cause of charcot arthropathy(1, 2).

It is often confused with osteomyelitis and massive infection of the foot necessitating early identification and management to prevent amputation of the lower extremity.(3, 4)

Ideally the goal in treating the Charcot deformity would be to prevent the initial breakdown within the foot. By each physician having a better understanding of the role of the autonomic and motor neuropathy in conjunction with sensory deficits, the Charcot process can be identified earlier and treatment begun sooner. The best treatment results for charcotart hropathy are achieved when treatment is initiated during the early stages of the disease. (5,6)

With the advent of advanced surgical techniques, the physician may be optimistic with the treatment of this condition. By thoroughly understanding the etiologic factors and deforming forces, treatment can be planned for each specific patient.(7, 8) Although the treatment prescribed is mainly conservative through immbolization & off-loading, Reconstructive surgery in acute Charcot may be considered if a deformity or instability exists that cannot effectively be controlled or accommodated by these methods (4, 9).

The goals of surgical intervention for the charcot foot & ankle are to restore alignment and stability, prevent ambutation, and allow the patient to be ambulatory (9, 10).

In this study, we are going to discuss & summarize the current trends in the surgical treatment options of the disease, with the incidence of possible complications.

Aim of the Study

The study aims at highlighting the current prospectives in the management of Charcot joint arthropathy of the foot & the ankle, it will mainly focus on the current & the newly used surgical trends in the treatment.

Pathogenesis of Charcot Joint

The main underlying cause of the Charcot foot in diabetes involves neuropathy, associated with a trivial trauma in many cases (11-14). The cardinal pathogenic mechanisms underlying diabetic neuropathy are chronic hyperglycemia and microvascular disease, leading to nerve injury via osmotic changes and ischemia, respectively (15) .

While neuropathy is certainly the common denominator, the type of neuropathy is a matter of discussio. Neuropathy may affect the peripheral nervous system leading to sensory loss or the autonomic system, impairing arterial perfusion and cellular turnover of foot and ankle bones (11, 12).

Pathogenesis :

Although Charcot believed that the joint destruction was secondary to unrecognized traumatic events as a result of sensory deficit, contemporaries of the time had observed that fractures of the metatarsals in insensate feet healed without complication with exuberant bone formation.

It has long been recognized that denervation is associated with distal hyperemia. Charcot wrote that the increase in distal limb blood flow he observed was most likely to be the result of

involvement of vasomotor nerves, and his observations were extended by Von Leyden and Brissaud, who concluded that the hyperemia was the result of loss of sympathetic innervation. (16) It was thus suggested that there is both a neurological and a neurovascular element in the pathogenesis of neuropathic osteo-arthropathy. Using scintigraphy, it has been shown that in patients with diagnosed neuropathy there is increased blood flow within bone thought to be due to an autonomic, neurally-mediated vascular reflex ultimately resulting in a hyperemia. (16)

Theories of Charcot's diabetic neuroarthropathy: (fig. 1)

Two competing theories have been proposed to explain the pathogenesis of diabetic neuroarthropathy.

1) ***The neurovascular theory*** views this condition as a neurologically mediated trophic defect resulting in increased osseous blood supply and osteoclastic activity in the absence of injury or repetitive microtrauma. This becomes clinically manifested as localized increased temperature with redness and dilated dorsal veins (15, 17).

Increased blood flow has also been noted in the foot bones and held responsible for increased bone resorption with reduced bone mineral density and, hence, predilection for fractures (18,19).

2) The neurotraumatic theory proposes that neuroarthropathy occurs when a bone or joint has lost its protective sensation and then enters a cycle of repetitive, excessive extension of ligaments and micro-fractures with increasing and often rapid disintegration of joints from continued weight-bearing. The feet become vulnerable with increased risk of unrecognized trauma (11, 12, 17). The latter can be a minor acute injury during normal daily activities such as walking, running or dancing, or it may be a chronic injury resulting from inappropriate footwear. Sensory deficits may involve light touch, temperature, and pain perception. The frequent findings of neuropathic ulcerations and increased pressures on the plantar aspect of the forefoot in acute diabetic neuroarthropathy support this theory.

Since neither of these theories can fully explain all cases of neuroarthropathy, such as its occurrence in paraplegic patients or its frequent onset following trauma, it is widely held today that both processes interact in varying degrees in the pathogenesis of this entity. (20,21,22)

Four factors are considered necessary for neuropathic arthropathy to develop:

(1) Peripheral neuropathy.

(2) Unrecognized injury.