Review
DIABETIC NEUROPATHIC ARTHROPATHY
Ram Singh * Ashish Bhalla ** Atul Sachdev * S. S. Lehl *

ABSTRACT

Diabetic neuropathic arthropathy (Charcot's foot) is being increasingly encountered in diabetic patients following their prolonged survival. It remains the foremost predisposing factor for foot amputation in diabetics. Other conditions associated with Charcot's foot such as syphilis and syringomyelia are rarely encountered. Early diagnosis, staging, preventive measures and the availability and utility of newer imaging techniques is discussed. Conservative and early corrective surgical management techniques for stabilization of this condition and prevention of amputation are emphasized.

KEY WORDS: Diabetic neuropathic arthropathy; Charcot's foot; Staging; Imaging techniques; Management.

INTRODUCTION

Neuropathic arthropathy is a chronic, progressive degenerative disorder affecting one or more peripheral or vertebral articulations, which develops as the result of a disturbance in the normal sensory (pain or proprioceptive) innervations of joints (1). Charcot arthropathy is relatively rare, but an important and potentially devastating disorder (2).

Diabetes mellitus, syphilis, and syringomyelia are the most commonly associated clinical entities. It particularly affects patients with long-standing diabetes, often with severe peripheral neuropathy in both legs, together with features of autonomic neuropathy, especially postural hypotension and gastroparesis. It presents with massive osseous destruction and malposition of the articular constituents (3). Peripheral vascular disease is typically absent (2).

When neuropathic arthropathy is suspected, careful clinical evaluation should be performed to identify the underlying neurological disorder (1). Within the population of diabetic patients, it is widely accepted, that patients with neuropathic (Charcot) arthropathy of the foot and ankle have the highest likelihood of having to undergo lower extremity amputation. The current emphasis is on care of the foot, skin and nails along with proper selection of accommodative shoe wear (1). Table 1 shows the relationship between duration of diabetes and the development of neuropathic joint complications, as described by Forgacs, in a study of 372 cases (4). On an average, over a two year period, the process can result in a severely deformed foot, which is highly prone to ulcers, infection, and subsequent amputation (5). Table 2 describes the commonly involved joints in diabetic neuroarthropathy (4).

<table>
<thead>
<tr>
<th>Duration of diabetes (years)</th>
<th>Number of reported cases (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-5</td>
<td>27(9.4)</td>
</tr>
<tr>
<td>6-10</td>
<td>50(7.5)</td>
</tr>
<tr>
<td>11-20</td>
<td>153(53.5)</td>
</tr>
<tr>
<td>&gt;21</td>
<td>56(19.6)</td>
</tr>
<tr>
<td>Total</td>
<td>286(100)</td>
</tr>
</tbody>
</table>

**Table 2: Localization of Diabetic Osteoarthropathy (4).**

<table>
<thead>
<tr>
<th>Joints</th>
<th>Number of reported cases (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle</td>
<td>38 (10.2)</td>
</tr>
<tr>
<td>Tarsus</td>
<td>81 (21.8)</td>
</tr>
<tr>
<td>Tarsometatarsal joints</td>
<td>102 (27.4)</td>
</tr>
<tr>
<td>Metatarsophalangeal joints</td>
<td>117 (31.5)</td>
</tr>
<tr>
<td>Interphalangeal joints</td>
<td>34 (9.1)</td>
</tr>
<tr>
<td>Total</td>
<td>372 (100)</td>
</tr>
</tbody>
</table>

PATHOPHYSIOLOGY

Etiopathogenesis of foot disease in diabetes mellitus is multifactorial and results from a combination of peripheral neuropathy, vascular compromise and superimposed infection. Foot complications in diabetic patients are common and account for more hospital days, than any other aspect of their disease.

Charcot process is poorly understood. Diabetic neuropathy with reduced sensitivity to pain and mechanical overloading of the joints, associated with frequently disturbed circulation in the lower extremities are the etiopathogenetic factors involved in diabetic osteoarthropathy (synonyms: neuroarthropathia diabetica, Charcot joint)(6). It may be initiated by injury, often apparently trivial, which may cause a minor periarticular fracture and occasionally a major fracture. Charcot changes are more likely to develop in bones that have pre-
existing diabetic osteopenia, which in turn may be related to increased blood flow in the neuropathic foot. Following the injury, osteoplastic activity is stimulated, presumably in an attempt to remodel the fractured bone. This persists, resulting in destruction, fragmentation and finally remodelling. Acute Charcot arthropathy may be confused with local infection or inflammatory arthritis. Chronic Charcot foot is classically described as a bag of bones (2).

Three stages in development of Charcot neuroarthropathy have been described. In the first stage there is osteonecrotic destruction of the bone and cartilage. Clinically it has an insidious onset, is painless and a non-inflammatory hydroarthrosis, with massive swelling of the soft tissues, is typical. In the second stage, there is irregular transformation of the bone due to multiple resorptive processes. In the third stage this transformation process stabilizes and the bone ends become more pointed and more sclerotic (resembling a licked candy stick). For mechanical reasons, the tarsometatarsal, metatarsophalangeal joints and the metatarsals are primarily involved (6).

CLINICAL FEATURES

Neuropathic arthritis is a destructive arthropathy frequently associated with loss of proprioception. A third of the patients, however, may have no demonstrable neurological deficit. Patients with diabetes mellitus, syphilis, syringomyelia and other neuropathies are particularly prone to develop this joint disease. The diagnosis of Charcot's joints should be considered in anyone who develops what appears to be a severe osteoarthritis or a transverse fracture of the tibia or fibula after a minor trauma. Scoliosis with particularly destructive changes on radiography, should prompt a search for syringomyelia or syphilis (7).

Diabetes mellitus is the leading cause of Charcot neuroarthropathy. The most common location is along the medial column of the foot. Over a two year period, the process can result in a severely deformed foot, which is highly, prone to ulcers, infection, and subsequent amputation (5).

Initial presentation is akin to an acutely inflamed joint, which is usually associated with pain at the affected site. Later, warm, swollen joints, with a grossly disorganized radiographic appearance develop, inspite of which, the patient is often pain-free. Neglect of this condition results in progressive deformity or instability, often complicated by ulceration and infection, which can ultimately result in loss of independent mobility, loss of the affected limb, and even death (8). A third of patients may however, have no demonstrable neurological deficit (7).

INVESTIGATIONS

Diabetic patients with neuropathy had significantly more radiographic abnormalities of the bones and joints than non-neuropathic and age-matched non diabetic control subjects (9). Therefore, familiarity with the spectrum of findings in the different imaging modalities appears essential.

Early changes may be subtle and the characteristic changes are seen only in advanced cases. In problems concerning the diabetic foot, magnetic resonance imaging, bone scans and leukocyte scintigraphy appear to be the most effective tools for detection of osteomyelitis, and a negative study makes osteomyelitis unlikely. However, the osteoarthropathy may be indistinguishable from that of acute osteomyelitis by these imaging modalities.

Three-phase bone scans, particularly Technetium scans (Tc99), demonstrate an early increase in bone uptake, which is due to the increase in blood flow through the bone that accompanies the active Charcot process. It is a very sensitive test, but it also detects infective and other inflammatory changes, and thus is not specific. It is important to differentiate Charcot arthropathy from infection, particularly osteomyelitis. Indium labelled white cells can be used to distinguish the two: a negative white-cell scan generally excludes infection, although occasional false-negative results may be seen in immunosuppressed patients, who have low-grade osteomyelitis (2).

Magnetic resonance imaging shows bone destruction clearly. Recently, an open trial of the bisphosphonate, pamidronate (which inhibits osteoclast activity), was found to reduce swelling, local temperature and markers of bone turnover.

STAGING

To help identify the early stages of the disease process, history, physical examination and radiographs of the patients need to be evaluated. In a recent study, the authors were able to identify five stages of Charcot deformities (5). This can be a useful guide for screening and managing patients of Charcot's joint.
Stage 0 is a clinical stage in which the patient presents with locally swollen, warm, and often painful foot. Radiographs are negative and Tc\textsuperscript{99} bone scan is markedly positive. Indium and gallium scans are normal.

Stage 1 in addition to the clinical finding of a painful swelling, X-rays of the affected joint demonstrates periarticular cysts, erosions, localized osteopenia, and sometimes diastases.

Stage 2 is marked by painless joint swelling with X-rays showing joint subluxations, usually starting between the second cuneiform and the base of the second metatarsal and spreading laterally.

Stage 3 is identified by joint deformity, which is clinically evident and X-rays show dislocation and arch collapse.

Stage 4 is evident by significant joint deformity, which is painless and shows the healed and stable end result of the process. Radiographically, there is bony trabeculation across joint spaces, indicative of mature fusion.

Clinically, there is no temperature gradient between the two feet except in stage 0 and 1, when the red and warm joint is noted due to an excessive blood flow.

CONSERVATIVE MANAGEMENT

The neuropathic foot is a common complication of diabetes mellitus and is associated with development of chronic ulcers and Charcot joints. Treatment of these complications presents a complex management task (10). The best approach is to treat cases on individual merit. The clinical staging suggested by Sella EJ and Barette C can serve as guideline for management of such cases (5).

Treatment of stage 0 consists of limited weight bearing and close observation, while the diagnosis becomes clear. Stage 1 can be treated with casting followed by a University of California Biomechanics Lab orthosis (UCBL), to maintain the arch, while allowing limited weight bearing. In Stage 2, a partial weight bearing total contact cast, followed by a Charcot restraint orthotic walker (CROW), can be used. Surgery may be needed at this stage, while the joints are still reducible (5). Ulcers, if present, can be treated with weekly local debridement, antibiotics, and total contact casting.

In a retrospective, uncontrolled study by Boninger and Leonard, "Total contact, laminated, bivalved ankle foot orthosis" (TCAFO) proved to be a safe, functional, and cost-effective therapy for complications of the neuropathic foot (10). Morgan and colleagues used CROW with success in 18 patients and found it to be a better method for prolonged immobilization and protection, necessary for healing in neuropathic arthropathy (11).

ROLE OF SURGERY

Surgery may be needed at a stage while the joints are still reducible (5). Arthrodesis with rigid fixation is recommended. Reconstructive foot and ankle surgery is a salvage procedure for the deformed neuropathic foot. Surgical arthrodesis of the deformed neuropathic foot, performed as a salvage procedure, can preserve the limb as a stable functional unit and create an acceptable alignment of the ankle-foot complex, that will promote viability of the overlying soft-tissue structures (12).

Stage 3 can be treated with casting for the acute phase and then followed with patellar-tendon-bearing ankle-foot orthosis (CROW), or caliper orthosis. If ulcers are present, they are treated with weekly local debridement, antibiotics, and total contact casting. Occasionally decompressive osteotomy may be required. Stage 4 may need surgical removal of the bony prominences causing the non-healing ulcers (5). Surgery to prevent deformity is recommended early, before the destructive stage (stage 3). Close follow-up, especially in a non-compliant population is necessary.

In most cases, a plantigrade, stable and functional foot can be achieved with simple non-operative techniques such as the use of a total-contact cast or shoe modification. A few patients, in whom there is uncontrolled instability or major osseous prominences causing recurrent ulceration, will require reconstructive surgery (either exostectomy or osteotomy/arthritis). Although some patients will have an improvement in function, ongoing vigilance is necessary (8).

In a study by Schon and colleagues, 50 patients were studied. Non displaced neuropathic ankle fractures, typically healed uneventfully with casting and bracing. For displaced ankle fractures, closed reduction and casting generally resulted in loss of reduction and progressive deterioration. Better results were obtained with open reduction and
internal fixation, using supplemental Kirschner wires and screws. Ankles with Charcot neuroarthropathy and pre-existing arthritis, typically required arthrodesis. Of the ankles with neuropathic avascular talar necrosis, approximately one third did well with non-operative interventions and two thirds required surgery. Chronic, unstable, malaligned Charcot ankles often required arthrodesis. Neuropathic calcaneal fractures were managed successfully nonoperatively. For feet with transverse tarsal joint involvement (Schon Type IV), management was more complex. Non operative treatment was successful for less than half. Two thirds of the feet with ditarsal involvement (Schon Types I, II, III) were managed successfully nonoperatively; one third required surgery for recurrent ulceration, instability or osteomyelitis. Half of the feet with forefoot neuroarthropathy required surgery for malalignment, ulceration and/or difficulty with shoe wear or braces. This study illustrates the pattern of Charcot involvement of the foot and ankle with corresponding methods of treatment and subsequent therapy (13).

**CONCLUSION**

The authors' primary purpose of writing this article is to alert physicians and orthopedic surgeons of the often unanticipated but significant complications of seemingly mild injuries in diabetic patients. With the rising population of diabetics in our country and prolongation of life expectancy, these complications are more likely to be encountered in day to day practice. A careful examination of the foot for injuries, neuropathy and early deformity, along with timely intervention, will save many limbs from getting amputated.

**REFERENCES**


