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REVIEW ARTICLE

Acute Charcot Foot

Prof. Samar Banerjee

Professor, Dept. of Medicine, Specialist Diabetes Clinic, Vivekananda Institute of Medical Sciences, Kolkata

Introduction

Long back in 1703, William Musgrave for the first time described a neuropathic joint as an arthralgia caused by venereal disease.[1] In 1868, Jean-Martin Charcot gave the first detailed description of the neuropathic aspect of this disease as a complication of syphilis and named the condition after him.[2] In 1936, Jordan linked it to diabetes and now is considered to be the most common etiology of Charcot arthropathy.[3]

Charcot foot is usually seen in patients with peripheral neuropathy resulting from diabetes mellitus, leprosy, syphilis, poliomyelitis, chronic alcoholism or syringomyelia. Repetitive microtrauma exceeding the rate of healing may cause fractures and dislocations. Changes in circulation may result in resorption of bone, weakening of the bone and increasing susceptibility to fracture and dislocation.

Sohn et al. in a retrospective study found that Charcot arthropathy by itself does not pose a serious amputation risk, but amputation risk is multiplied in the presence of ulcer complications. In this study, Charcot patients and ulcer patients had 4.1 and 4.7 amputations per 100 person-years, respectively.[4]

Definition

Charcot arthropathy, also called as Charcot joint or neuropathic joint, is a progressive condition of the musculoskeletal system, characterised by joint dislocations, pathologic fractures, and debilitating deformities. This disorder results in progressive destruction of bone and soft tissues at weight-bearing joints. In its most severe form, it may cause significant disruption of the bony architecture. Charcot arthropathy can occur at any joint. But, it occurs most commonly in the lower extremity, at the foot and ankle.

Epidemiology

The prevalence rate of Charcot arthropathy is 0.1% to as high as 13% in specialised foot clinics. In patients with diabetes, the incidence of acute Charcot arthropathy ranges from 0.15% to 2.5%. Bilateral disease is less noted and is seen in below 10% of patients. Recurrence of disease occurs in less than 5% of patients. Usually men and women are equally affected, while others report a 3:1 predilection for males.[5]

A prospective study conducted in Singapore with 202 diabetic patients revealed that 42.1% of the patients had sensory neuropathy and 2% of them had Charcot arthropathy.[6] The incidences of Charcot foot in type 1 and type 2 diabetes do not differ, although osteopenia, as a predisposing factor, appears to be more prevalent in type 1.[7,8] However, Petrova et al. reported a difference in the presentation of Charcot arthropathy at type 1 and type 2 diabetic patients. In a recent study, the same authors emphasised a relative preponderance of type 1 diabetes compared with type 2.[7,9] Though the unilaterality of the condition is claimed in many clinical studies, acute Charcot arthropathy is reported as bilateral in 9% of the patients.[10] Moreover, after prospective computerised tomography examinations, bilateral neuroarthropathic changes are demonstrated in 75% of Charcot patients.[11] Chisholm et al. suggest that obesity is also a predisposing factor for Charcot arthropathy since at least two-thirds of Charcot patients are obese.[12–14]

The root cause of Charcot joint is sensory or autonomic

neuropathy and can occur as a complication of diabetes, syphilis, chronic alcoholism, leprosy, meningomyelocele, spinal cord injury, syringomyelia, renal dialysis and congenital insensitivity to pain. Diabetes mellitus happens to be the most common cause of Charcot arthropathy.[5]

Pathophysiology

The exact nature of pathogenesis of Charcot arthropathy is not yet properly understood. The following major explanation appears to be acceptable:

- Neurotraumatic theory
  A trivial or moderate unperceived trauma or injury to an insensitive foot due to sensory neuropathy renders the patient unaware of the osseous destruction that occurs with ambulation. This micro trauma occurring daily leads to progressive destruction and damage to bone and joints.
- Neurovascular theory
  The associated autonomic neuropathy may predispose the extremity to a brunt of an increased blood flow which, in turn, results in a mismatch in bone destruction and synthesis, leading to osteopenia.

Most probably, Charcot arthropathy results from a combination of the processes as described earlier. This development of abnormal bone that cannot protect the joint, results in gradual bone fracture and in the subluxation of the joint.

When the diagnosis is suspected as Charcot arthropathy, in addition to meticulous treatment of the affected joint, the cure of the primary cause, if possible, should be targeted. They are traumatic injuries (spinal cord injuries, peripheral nerve injuries), infections (syphilis, leprosy, yaws), disorders of neurological structures (myelomeningocele, syringomyelia, spina bifida), neurodegenerative diseases (amyloid neuropathy, neuropathies secondary to alcoholism and vitamin deficiency) or other neurological disorders such as congenital insensitivity to pain syndrome, steroid intake (post-renal transplant arthropathy, intra-articular steroid injections) and heavy-metal poisoning that belong to the same cluster of diseases leading to the destruction of afferent proprioceptive fibres.

This is the basic cause for subsequent repetitive traumas to remain unrecognised. After numerous minor or major traumatic injuries, progressive wear results in micro fractures, escalating into macro-fractures that eventually end up in massive joint destruction with characteristic clinical presentation.[15]

Trauma is considered as the most common etiological factor for Charcot arthropathy and was reported to be present in 22–53% of the cases.[7] Capillary leakage and subsequent formation of edema occurs as a physiological response to blunt trauma.[16] A higher energy trauma causes a disruption of marrow trabeculae leading to interstitial fluid and haemorrhage accumulation to marrow spaces. When this condition occurs in the foot of a non-diabetic patient, it is painful and quickly detected. But in a neuropathic patient, absence of pain leads to delayed detection and lack of required immobilisation flares up the inflammatory cycle.[17]

Local surgery of the foot is suggested as one of the triggering factors of Charcot arthropathy.[18] Armstrong et al. from 55 acute Charcot arthropathy patients reported that 4% of the patients had recent foot surgery as the only etiological factor.[10] Charcot arthropathy may also follow injudicious immobilisation after surgery, a long period of bed rest or casting.[7] Disobedience to a forbidden weight bearing after foot surgery is also underlined in a case report as a possible cause of Charcot arthropathy.[18]

Charcot arthropathy is also reported following a simultaneous pancreas-kidney transplantperation In a more recent study where data from 130 patients without any previous history of Charcot arthropathy were analysed retrospectively, six patients (4.6%) were diagnosed *de novo* Charcot arthropathy. Use of high glucocorticoid intake during the first year of transplantation was the main factor leading to bone resorption and myofibril proteolysis.[19]

Inflammation

Local inflammation is the important factor for a predisposing environment. The physiological balance between the pro and anti-inflammatory cytokines, that restrains the inflammatory response to a necessary extent, is altered in these patients. In a Charcot patient, the modulation of immune system is disturbed in countenance of pro-inflammatory cytokines.[20] The bone and soft tissues respond with an acute-phase release of pro-inflammatory cytokines, tumour necrosis factor-α (TNFα) and interleukin-1β (IL-1β).

There is an increase in the amount of TNFα, IL-1β and IL-6, whereas there is a decrease in the levels of IL-4 and IL-10 known as anti-inflammatory cytokines.[21] An abnormally intense and prolonged inflammatory response is inevitable under these circumstances.

Increased amounts of pro-inflammatory cytokines, especially TNFα appears to trigger another cytokine pathway that is centered on the polypeptide, the receptor activator of nuclear factor-κB (NF-κB) ligand (RANKL).

As a member of the TNF super family, RANKL as a ligand activates the receptor of NF-κB (RANK). The activation of RANK stimulates the intracellular pathways that end up by formation of nuclear transcription factor NF-κB. This NF-κB induces osteoclast precursor cells to differentiate into mature osteoclasts,[8] leading to the excessive osteoclastic activity in diabetic Charcot arthropathy.[22,23]

RANKL activity is antagonised by osteoprotegerin (OPG), whose expression is induced by NF-κB, as a self-limiting agent of its pro-inflammatory function. This elevated RANKL/OPG ratios fuel the progression of the inflammation.[23,24]

Osteoclasts work as executer cells, responsible for imbalanced bone turnover and eventually osteolysis. The first cell line to dysfunction seems to be the monocytes, the precursor cells of osteoclasts. In presence of high levels of pro-inflammatory cytokines, monocytes stimulate T lymphocytes in an exaggerated way. Moreover, monocytes in Charcot patients present reduced secretion of anti inflammatory cytokines, and increased resistance to apoptosis.[24,25] This resistance is mainly by IL-1β and TNFα causing the persistence of the abnormally intense and prolonged inflammatory response.[4] Ndip et al. reported that IL-8 and granulocyte-colony stimulating factor (GCSF) were inducing monocytes into an osteoclastic differentiation along with the RANKL/RANK pathway.[23]

Pitocco et al. reported a significant decrease in the circulating levels of IGF-1 in Charcot patients.[26] IGF-1 is a mediator of vasodilatation, and bisphosphonate's reducing effect over IGF-1 could have a beneficial contribution to restrain proceeding inflammation.

One report has shown that in the acute stage of Charcot osteoarthropathy, there is dissociation between the presence of local signs of inflammation, as demonstrated by increased skin temperature in the Charcot foot, and the lack of systemic response, as shown by a normal to slight increase in C-reactive protein (CRP) levels, normal white blood cell (WBC)count and mild increase in erythrocyte-sedimentation rate (ESR). CRP is one of the well established sensitive markers of inflammation widely used in clinical practice as a direct serological measure of acute phase response to injury and infection. Thus, there is dissociation between the local and systemic inflammatory response in acute Charcot osteoarthropathy.[27]

As such, when patients present with a hot red foot, with no obvious skin breakdown and a CRP level that is normal or only slightly raised, acute Charcot osteoarthropathy should be firmly suspected.

Neuropeptides

The central nervous system probably intervenes with the regulation and/or the modulation of the bone metabolism,[28] mediated through neuropeptides that are synthesised in unmyelinated sensory neurons and secreted from their peripheral terminals in the bone tissue. Offley et al.[29] reported that capsaicin-induced depletion of neuropeptides such as substance P (SP) and calcitonin gene-related peptide (CGRP) in unmyelinated sensory neurons of adult rats resulted in an increased bone loss and fragility and this effect could be reversed by daily injections of CGRP.[29]

CGRP binds to its own receptor and increases intracellular cyclic adenosine mono-phosphate (AMP)and calcium in osteoblastic cells,stimulates cell proliferation, synthesis of cytokines, synthesis of growth factors and synthesis of collagen.[22] CGRP also inhibits pro-inflammatory cytokine production and increases the release of IL-10 by monocytes. Denervated Charcot foot is deficient in CGRP release and an important source of anti-inflammatory impulse is compromised.[30]

Nitric oxide (NO) has a reciprocal effect on the modulation of bone metabolism.[31] NO's is able to induce apoptosis of pre-osteoclasts and decrease the resorption of the mature osteoclasts in mice .[32] There is diminished expression of eNOS in Charcot patients, which leads to a suppression of the osteoclast activity and contributes to a marked increase in the fragility of osteoporotic bone.[31] Insufficient amounts of NO production might induce osteocytes to apoptosis, indirectly enhancing the osteoclast function. Low concentrations of NO potentiate bone resorption while higher concentrations are inhibitory.[33]

Noradrenergic innervation of the bone tissue regulates the blood flow and act on the modulation of osteoblastic and osteoclastic cell metabolism. Osteoblasts express β-2 adrenergic receptors. Moreover, noradrenalin increases alkaline phosphatase activity and proliferation through α-1 receptors expressed on the osteoblasts.[34]

Microvascular structure and bone turnover

Baker et al. evaluated the rate of maximum micro-vascular hyperaemia (MMH) in patients with diabetic neuropathy and diabetic Charcot arthropathy. They observed that in Charcot patients, MMH is relatively preserved and significantly higher than patients with neuropathy alone.[35] Shapiro et al. found increased skin blood flow and vasomotion in both healthy control and Charcot subjects, compared to diabetic neuropathy patients.[36] Those findings suggest that Charcot patients preserve the ability to

vasodilate as opposed to patients with diabetic neuropathy alone, and it may be an explanation why all patients with diabetic neuropathy do not develop Charcot. However, peripheral arterial disease seems to have a protective effect on the development of Charcot arthropathy.[37] This is probably due to limited vasodilation capacity of the affected arteries.

If there is sympathetic vascular denervation, there will be an increase in local arterio-venous shunt flow, which functions in body thermoregulation in physiological conditions. The loss of regulation of the shunts increases venous pressure and fluid filtration through capillary leakage.[37] As a result, there is deep tissue edema which increases intra-compartmental pressure, compromises micro-circulation and causes a deep tissue ischaemia.[38] Moreover, extensive connective tissue edema impairs tensile strength and stability of tendons and ligaments, and the joints suffer from subluxations and dislocations.

In Charcot arthropathy patients, there is exaggerated osteoclast activity, as evidenced by an increase in alkaline phosphatase and collagen residues leading to an imbalance of constant remodelling processes.[39,40] La Fontaine et al. conducted histological examination of bone specimens obtained from diabetic patients and observed a distorted micro-structure with fewer trabeculae and fewer cells. They claim that the degenerative changes in the bone microarchitecture may not be a consequence, but a cause of Charcot arthropathy.[41]

Christensen et al. found significantly lower bone mineral density (BMD) values from the affected foot of chronic Charcot patients whereas no difference was in the calcaneal BMD between acute Charcot patients and the control group.[42] They also studied the biochemical indicators of bone turnover and statistically significant differences in osteocalcin concentrations reflecting increased bone turnover were demonstrated in acute Charcot foot.[40]

A recent study demonstrated the development of Charcot arthropathy after administration of high doses of glucocorticoids.[19] Glucocorticoids affect the bone turnover in countenance of resorption, and decreased bone formation may trigger or worsen Charcot arthropathy.

Vitamin D deficiency may also be a predisposition to the development of Charcot arthropathy.[19] Hypocalcaemia resulting from vitamin D deficiency stimulates parathyroid hormone (PTH) which in turn depletes calcium from the bone causing osteopenia. The level of 1,25 $(OH)_2D_3$ is significantly lower in diabetic patients and results in a less mineralised bone formation, a smaller growth plate and an inadequate turnover, all being reversed by insulin treatment.[32]

Hyperglycaemia

Hyperglycaemia stimulates free radical formation, hyperlipidaemia and advanced glycation end-products (AGEs), triggering the RANK/RANKL cytokine system.[20] There are reports of an *in vitro* inhibitory effect of physiological concentrations of insulin on the NF-κB and monocyte chemo-attractant protein 1 (MCP-1).[42] This non-RANKL inhibitory mechanism is impaired in diabetic patients who are deprived of insulin. But, those pathways may not be majorly effective since the Charcot arthropathy is rare even in diabetic neuropathy patients.[43]

Hyperglycaemia denaturates tendons and ligaments through a non-enzymatic collagen glycation and can cause tendon shortenings and thus, redistribution of the plantar pressures abnormally.[11]Because collagen is a structural component of the bone, AGE-related modifications of collagen, they also may impair the mechanical properties of bone itself, predisposing it to fractures and dislocations.[32,44]

Genetics

A correlation between diabetic Charcot arthropathy and OPG gene polymorphisms was suggested by Pitocco et al.[45] A strong association with Charcot arthropathy and the polymorphisms of those alleles were also demonstrated. Recently, Korzon-Burakowska et al. supported this association in their study conducted in the Polish population.[46]

Presentation

The clinical presentation of Charcot arthropathy is variable from mild swelling and no deformity to moderate deformity with significant swelling depending on the stage of the disease. The common presentations are:

1. profound unilateral swelling,
2. an increase in local skin temperature (generally, an increase of 3–7° above the non-affected foot's skin temperature),
3. erythema,
4. joint effusion,
5. bone resorption in an insensitive foot,
6. presence of intact skin and a loss of protective sensation,
7. pain is absent or significantly less, than would be expected based on the severity of the clinical and/or radiographic findings,
8. instability and loss of joint function,
9. passive movement of the joint may reveal a "loose bag of bones,"

10. concomitant ulceration complicates the diagnosis and points towards the presence of osteomyelitis.

Stages of Charcot Foot

The syndrome progresses through three general stages:

- **Stage 1** (acute, development-fragmentation): marked redness, swelling, warmth; early radiographs show soft tissue swelling, and bony fragmentation and joint dislocation may be noted several weeks after onset
- **Stage 2** (subacute, coalescence): decreased redness, swelling and warmth; radiographs show early bony healing
- **Stage 3** (chronic, reconstruction-consolidation): redness, swelling, warmth resolved; bony healing or non-union and residual deformity are frequently present.

Classification of Charcot Foot

Based on the location of the arthropathy, Charcot arthropathy is classified into five different patterns, as follows:[8]

- **Pattern** 1 involves the forefoot, which includes the interphalangeal joints, the phalanges and the metatarsophalangeal joint.
- **Pattern 2** involves the tarsometatarsal joint.
- **Pattern 3** involves the cuneonavicular, talonavicular and calcaneocuboid articulations.
- **Pattern 4** involves the talocrural, or ankle, joint, which is the articulation of the tibia, the fibula and the talus.
- **Pattern 5** involves the posterior calcaneus.

Usually patterns 2 and 3 are the most common, with approximately 45% of cases involving pattern 2 and 35% involving pattern 3.

Another commonly used classification system is the Brodsky and Rouse system. This system describes three anatomic Charcot joints (types 1, 2, and 3a and 3b):

- **Type 1** involves the mid foot.
- **Type 2** involves the hind foot.
- **Type 3a** involves the ankle
- **Type 3b** is a pathologic fracture of the OScalcis tubercle.

The multilevel Schön classification system is also used which comprises four types and characterises Charcot joints on the basis of sites and degree of involvement.[9] All four types have three subsets (e.g. type IA, IB, IC), which are based on the severity of involvement. The four types are as follows:

- **Type I** - The Lisfranc pattern
- **Type II** - The cuneonavicular pattern
- **Type III** - The perinavicular pattern
- **Type IV** - The transverse tarsal pattern

The Schön classification system allows the prediction of outcomes and the estimation of treatment duration. The diagnosis is often delayed because the initial signs of Charcot foot are non-specific and are more typically seen in other more common conditions such as infections and rheumatologic conditions. Many patients do not complain of the pain or have pain from neuropathy that was pre-existing.

Physicians who are not well expert in orthopaedic foot and ankle problems may see a Charcot foot very few times in their entire career, less frequently than other conditions such as septic arthritis, gout, rheumatoid arthritis and other inflammatory arthropathies. The American Orthopaedic Foot & Ankle Society (AOFAS) offers information on this site as an educational service.

Differential Diagnosis

The common differential diagnosis are as follows:

1. infection (osteomyelitis, cellulitis,abscess, deep tissue infection),
2. deep vein thrombosis,
3. acute gout,
4. neuropathic/traumatic fractures,
5. sprain,
6. inflammatory,
7. arthritis

Investigations

Laboratory Studies

1. The WBC count and ESR is done to distinguish between Charcot arthropathy and osteomyelitis. The WBC count is elevated when infection is present, and often with a left shift . Both WBC count and ESR are a non-specific marker for inflammation, and the results may be mildly elevated in patients with Charcot arthropathy.
2. Glycosylated haemoglobin (HbA1c) and blood sugar measurements to assess the level of glycaemic control.
3. Levels of alkaline phosphatase, calcium, phosphorus, Vitamin D3 and parathyroid hormone (PTH) to identify bone diseases, such as Paget disease.
4. Hypercalcaemia may be indicative of cancer or metastases.
5. Vitamn B12/folate deficiency could suggest peripheral neuropathy and chronic alcoholism.
6. Rapid plasma reagin (RPR)/fluorescent treponemal

antibody – absorption (FTA-ABS) tests aid in the diagnosis of syphilis.

Imaging Studies

1. Plain radiographs for staging the disease,to determine if active disease is present or if the joint is stable (monitor serial radiographs),to identify osteopenia, periarticular fragmentation of bone, subluxations, dislocations, fractures and generalised destruction.
2. Bone scanning can differentiate between Charcot arthropathy and osteomyelitis. An indium-111 WBC scan often is better than the technetium-99m scan.
3. Magnetic resonance imaging(MRI) distinguishes between osteomyelitis and Charcot arthropathy.

Diagnostic Procedures

1. Lumbar puncture is used if the RPR test is positive followed by an FTA-ABS test is ordered if tertiary syphilis/tabes dorsalis is suggested.
2. Bone probing is done with a blunt, sterile surgical probedown to the bone to rule out osteomyelitis.
3. Portable infrared dermal thermometry is used for skin temperature assessment. It can be used to monitor active inflammation. A 3–5° difference is generally seen in the acute stage.
4. Joint aspiration is used to help rule out a septic joint.
5. Synovial biopsy can be helpful. Small fragments of bone and cartilage debris are embedded in the synovium because of joint destruction. Some state that this is pathognomonic, whereas others state that it is highly suggestive of Charcot arthropathy.
6. Doppler ultrasonography is used to rule out deep vein thrombosis.

Treatment

Treatment consists of medical, surgical and rehabilitative methods.

Medical Therapy

Treatment of Charcot arthropathy is primarily non-surgical. Treatment depends upon the phase of the diseases This has two phases: an acute phase and a post-acute phase. Management of the acute phase includes immobilisation and reduction of stress.[11]

Immobilisation usually is done by casting as total contact casts, which allow patients to ambulate while preventing the progression of deformity. Casts must be checked weekly to evaluate for proper fitting, and they should be by every 1–2 weeks. But in cases with concomitant ulceration, their casts need to be changed weekly for ulcer evaluation and debridement. Serial plain radiographs taken approximately every month during the acute phase guide us to evaluate progress.

Casting usually is to be kept for 3–6 months and to be removed based on clinical, radiographic and dermal thermometric signs of quiescence. There are also other methods of immobilisation like metal braces and ankle-foot orthoses (AFOs), but they may prolong healing times.

Reduction of stress is necessary and can be done by decreasing the amount of weight bearing on the affected extremity. While total non-weight bearing (NWB) is ideal for treatment, patients are often not willing to accept this treatment. Studies have shown that partial weight bearing (PWB) with assistive devices (e.g. crutches, walkers) also is acceptable without compromising healing time. However, full weight bearing (FWB) in the acute phase tends to lengthen total time in the cast.

Healing time depends upon the location of the disease. Pattern 1, or forefoot pathology, heals in two-thirds the time of pattern 3 or pattern 4. One study revealed that the mean time in a cast is 18.5 weeks, while another study showed that the acute phase lasts 12.5 weeks.

After the stage of the removal of the cast, the patients' needs lifelong protection of the involved extremity by patient education and professional foot care on a regular basis for lifelong foot protection. After cast removal, patients should be advised to use a brace to protect the foot. Many types of braces can be used, like a patellar tendon-bearing brace, accommodative footwear with a modified AFO, a Charcot restraint orthotic walker (CROW) and a double metal upright AFO.[49]

Patients also should be advised to use custom footwear, which includes extra-depth shoes with rigid soles and a plastic or metal shank. If ulcers are present, a rocker-bottom sole can be used and for insensate feet use Plastazote inserts. Based on clinical, radiographic and dermal thermo graphic findings this regimen may be eliminated after 6–24 months. But continued use of custom footwear in the post-acute phase for foot protection and support is essential.

Antiresorptive therapy

Treatment by drugs which are antiresorptive in nature drugs has been thought of, because bone turnover in patients with active Charcot neuropathy (CN) is excessive. But, there is little evidence to support their use. Both oral and intravenous bisphosphonates[50] have been studied in the treatment of CN in small randomised, double-blind, controlled trials[51,52] or in retrospective controlled studies.[53] Those patients who cannot tolerate oral bisphosphonates but may benefit from intravenous therapy using pamidronate or zoledronic acid.[54]

Intranasal calcitonin as another antiresorptive agent has been studied. Calcitonin was associated with a significantly greater reduction in cross-linked carboxy-terminal telopeptide of type I collagen and bone-specific alkaline phosphatase than standard treatment in the control group that received only calcium supplementation and offloading. Calcitonin has a safer profile in renal failure when compared with bisphosphonate therapy.[55] But, a single dose of intravenous bisphosphonate generally does not require renal adjustment. There is no conclusive evidence for using bisphosphonates in active Charcot foot, and we should wait for evolving more trials.

The total healing process typically may be upto 1–2 years. The patient should be advised to prevent further injury, note temperature changes, check feet every day, report trauma and receive professional foot care.

Glycaemic Control

Glycaemic control is an integral part of treatment, and should be quickly targeted,preferably with insulin therapy.

Surgical Therapy

- The choice of surgical procedures and techniques depend upon the location of the disease and on surgeon preference and experience with Charcot arthropathy. Patients treated with surgery have longer healing times.[11]

Surgical procedures include

- exostosectomy of bony prominence,
- osteotomy, arthrodesis,
- screw and plate fixation,
- open reduction and internal fixation (ORIF),
- reconstructive surgery,
- fusion with Achilles tendon lengthening,
- autologous bone grafting
- and amputation.

Surgical methods can be based on Schön's classification system. The following recommendations may be made:[47-48]

- ORIF should be used for an ankle with displaced fractures
- Ankle arthrodesis is necessary in patients with tibiotalar destruction
- In cases in which the hindfoot has avascular necrosis of the talus, a talectomy with tibiocalcaneal fusion is necessary
- Arthrodesis may be necessary for patients with hindfoot involvement
- For a midfoot pattern, surgical correction of rocker-bottom deformity and osteotomies for bony prominences are used
- If there is an associated hindfoot/ankle equinus contracture, then a posterior release/Achilles tendon lengthening procedure is required
- For forefoot patterns, patients with bony prominences or recurrent ulcerations may need a resection arthroplasty or cheilectomy

Sohn et al. in a retrospective study compared the risks of lower-extremity amputation in patients with Charcot arthropathy alone and those with diabetic foot ulcers.[4] They observed that Charcot arthropathy by itself is not responsible for a serious amputation risk, but amputation risk is increased in the presence of ulcer complications. In patients younger than 65 years, amputation risk was 7 times higher for patients with ulcer alone than for those with Charcot arthropathy alone, and 12 times higher for those with Charcot and ulcer.

Della Paola et al. assessed in a cohort of 45 patients, as an alternative to amputation with Charcot arthropathy, surgical treatment of osteomyelitis of the mid-foot or the ankle and stabilisation with external fixation.[4] Thirty-nine patients healed when treated with emergency surgery to drain an acute infection with maintenance of fixation (average, 25.7 weeks); two were treated with intramedullary nails in follow-up surgery; and in four, infection could not be controlled and amputation was still necessary.[56]

Bone growth stimulation

There are limited trials for the use of external bone stimulation in Charcot arthropathy like ultrasonic bone stimulation for the ankle for the healing of fresh fractures.[57] Direct current electrical bone growth stimulators have been used specifically in patients to promote healing of fractures

in the acute phase in small case series.[58] These findings are promising, but there have been no subsequent studies to confirm this method, and its use has been practiced only as an adjunct therapy during the postsurgical period.

Complications

Charcot fractures if are not timely diagnosed or not treated properly, may progress to marked joint deformity and to skin ulceration over a bony prominence. Several infections can occur in the ulceration, requiring amputation of the extremity. Other complications of Charcot arthropathy are foot collapse leading to the formation of a clubfoot or a deformity termed as the rocker-bottom foot, in which collapse and inversion of the plantar arch occurs. Further complications seen in this condition are the ossification of ligamentous structures, the formation of intra-articular and extra-articular exostoses, the collapse of the plantar arch and the development of osteomyelitis.

Prognosis

Outcomes for Charcot arthropathy depends upon the occurrence of immediate diagnosis, location of the lesion and treatment. If joints are treated within 2 weeks of injury and when there is strict adherence to weight-bearing precautions more favourable outcome is elicited. Forefoot arthropathy heals earlier than midfoot, hind foot or ankle arthropathies, as the following list illustrates:

- Ankle - Mean healing time, 83 ± 22 days
- Hindfoot - Mean healing time, 97 ± 16 days
- Midfoot - Mean healing time, 96 ± 11 days
- Forefoot - Mean healing time, 55 ± 17 days

Surgical treatment prolongs healing time. The extent of the injury also affects healing time. The more severe the injury, the longer it takes to heal and the greater the likelihood of permanent deformity. It generally takes 1–2 years to completely heal a Charcot joint, from the active phase to quiescence.

Some Charcot joints, such as involving the ankle, may heal with fibrous tissue (non-union) to result in gross instability ("floppy foot"). This may predispose to ulcers and may be difficult to support with braces.

Conclusion

The Charcot foot syndrome is the result of a complex complication of diabetes and neuropathy. The destruction of the foot and ankle arises from a cycle of uncontrolled inflammation. When neglected the classic rocker-bottom foot deformity develops, but it can be avoided by early recognition and management. Initial treatment is off loading and surgery can be helpful in early stages involving acute fractures of the foot or ankle or in later stages when off loading is ineffective.

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"You educate a man; you educate a man.
You educate a woman; you educate a generation."
— Brigham Young