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Review Paper

Charcot Neuropathic Osteoarthropathy: An Updated Literature Review on Pathophysiology, Diagnosis, and Management

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ABSTRACT

Charcot Neuropathic Osteoarthropathy (CNO) is a progressive complication of peripheral neuropathy, most commonly associated with diabetes mellitus, and is characterized by inflammation, bone and joint fragmentation, and deformity of the foot and ankle. This literature review summarizes current knowledge regarding the anatomy relevant to CNO, its multifactorial pathophysiology, diagnostic approach, classification systems, management strategies, and prognosis. Evidence indicates that CNO arises from an interaction of neurotraumatic and neurotrophic mechanisms, inflammatory cytokine imbalance, osteoclast overactivity, microvascular alterations, metabolic disturbances in chronic hyperglycemia, and genetic susceptibility within the OPG-RANK-RANKL pathway. Clinically, CNO presents with a warm, swollen, erythematous neuropathic foot and progresses through well-defined stages described by the Eichenholtz system, while additional anatomical classifications by Trepman, Sanders Frykberg, and Brodsky, together with MRI based schemes, assist in staging and treatment planning. Diagnosis relies primarily on clinical suspicion supported by radiography and advanced imaging to document structural damage and to distinguish CNO from osteomyelitis and other inflammatory conditions. First line treatment is early immobilization and offloading using total contact casting or equivalent devices, with subsequent reconstructive procedures, such as osteotomy, arthrodesis, beaming constructs, and hybrid or external fixation, reserved for patients with fixed deformity, instability, or recurrent ulceration. Multidisciplinary care that optimizes glycemic control, vascular status, bone health, and infection management is essential. Infection and peripheral arterial disease substantially worsen outcomes and increase the risk of major amputation. Overall, timely recognition, accurate staging, and stage directed conservative and surgical interventions are crucial to preserving a plantigrade, shoeable, and infection free foot and to improving long term limb salvage and quality of life in patients with CNO.

Keywords: Charcot neuropathic osteoarthropathy, diabetic neuropathy, and total contact casting

INTRODUCTION

Charcot Neuropathic Osteoarthropathy (CNO), commonly referred to as Charcot joint, is a progressive and debilitating disorder that primarily affects the bones, joints, and soft tissues of the foot and ankle. It is most frequently observed in individuals with peripheral neuropathy, a condition in which nerve damage impairs protective sensation, most commonly due to diabetes mellitus. The condition is characterized by inflammation, joint dislocation, bone resorption, and deformity, often resulting in substantial functional impairment and an increased risk of ulceration or amputation.[1]

CNO is named after Jean-Martin Charcot, a 19th-century French neurologist who first described neuropathic arthropathy in patients with syphilis. Although initially associated with tabes dorsalis, contemporary cases occur predominantly in individuals with diabetic neuropathy. Less common etiologies include alcohol-related neuropathy, spinal cord injury, leprosy, and syringomyelia. The pathophysiology of Charcot joint involves a complex interaction of multiple factors. Peripheral neuropathy diminishes protective sensation, allowing minor injuries or repetitive trauma to go unnoticed, leading to sustained mechanical loading of bones and joints. In addition, autonomic neuropathy results in vascular dysregulation with increased blood flow to the affected region, which promotes osteoclastic bone resorption. The combination of mechanical overload and structurally weakened bone leads to fractures, joint dislocation, and ultimately foot deformity, with the cycle of destruction often exacerbated by continued weight-bearing in the absence of early intervention.[2]
Clinically, CNO often presents with a warm, swollen, and erythematous foot, mimicking cellulitis or deep vein thrombosis. Pain may be absent or mild because of the underlying neuropathy. If left untreated, the condition typically progresses through three phases: an acute inflammatory phase, a subacute reparative (coalescence) phase, and a chronic remodeling (consolidation) phase. The final stage frequently results in a rocker-bottom deformity, characterized by collapse of the medial longitudinal arch, which increases the risk of pressure ulceration. Diagnosis is challenging and requires a high index of clinical suspicion, particularly in high-risk populations such as patients with long-standing diabetes and established peripheral neuropathy. Imaging modalities including plain radiography, magnetic resonance imaging, and bone scintigraphy are commonly employed to confirm the diagnosis and to determine the extent of osseous and articular involvement. Distinguishing CNO from infection, particularly osteomyelitis, is critical for appropriate management and limb preservation.[3]
Management of CNO is aimed at arresting disease progression and preventing complications. Non-surgical measures, especially off-loading with total contact casting or customized orthotic devices, constitute the cornerstone of treatment during the acute phase. Surgical intervention may be indicated in advanced or unstable cases to correct deformity, restore alignment, or stabilize the foot and ankle. Charcot joint remains a major challenge in clinical practice, frequently leading to reduced quality of life and increased healthcare utilization. Early recognition, multidisciplinary care, and comprehensive patient education are essential to minimizing morbidity and optimizing long-term outcomes.

Anatomy of the Foot

The foot comprises 28 bones, including 14 phalanges, 7 tarsal bones (talus, calcaneus, cuboid, navicular, and three cuneiforms), 5 metatarsals, and 2 sesamoids. Structurally, it is divided into three functional regions-the hindfoot, midfoot, and forefoot—each defined by its proximal and distal articulations. The hindfoot consists of the talus and calcaneus, which articulate proximally with the tibia and fibula at the ankle mortise and distally with the midfoot at the midtarsal (Chopart) joint. The midfoot is composed of the navicular, cuboid, and cuneiform bones, which articulate with the metatarsals at the Lisfranc joint and may be associated with accessory ossicles such as the os naviculare and os peroneum. The forefoot, extending distal to the Lisfranc joint, includes the metatarsals, sesamoid bones, and phalanges, with the os vesalianum pedis located at the base of the fifth metatarsal.[4,5]

The foot and ankle complex contains five major joints: the ankle (mortise), subtalar, midtarsal (Chopart), midfoot (Lisfranc), and first metatarsophalangeal (MTP) joint. These joints enable a wide range of motions essential for mobility, including plantarflexion and dorsiflexion at the ankle, inversion and eversion at the subtalar joint, and stabilization across the midfoot and Lisfranc joint, while the first MTP joint plays a crucial role in push-off during gait. The ligamentous architecture comprises the lateral and medial ligament complexes of the ankle, the distal tibiofibular ligament complex, and the Lisfranc ligament complex, all of which contribute to joint stability and controlled mobility. The relatively weaker lateral ligament complex connects the talus to the fibula, whereas the stronger medial complex, consisting of the deltoid ligament and spring ligament, provides robust support to the medial ankle. [4,5]

The plantar fascia, originating from the calcaneus and extending to the metatarsal heads, is essential for reinforcing the medial longitudinal arch during the propulsive phase of gait. Muscles of the foot and ankle are categorized as extrinsic, originating proximal to the ankle, and intrinsic, arising within the foot itself. The extrinsic muscles are further grouped into four compartments, each with distinct functions, innervation, and clinical relevance, particularly in conditions such as compartment syndrome. The anterior compartment includes muscles such as tibialis anterior, extensor hallucis longus, and extensor digitorum longus, which are responsible for dorsiflexion and stabilization of the foot, whereas the posterior compartment contains powerful plantarflexors, including the gastrocnemius and soleus. [4,5]

The vascular supply of the foot is primarily derived from the dorsalis pedis and posterior tibial arteries, the latter dividing into the medial and lateral plantar arteries, which form the plantar arterial arch. Venous drainage is facilitated by structures such as the great saphenous vein and the dorsal venous arch. Neural innervation arises from branches of the sciatic nerve, including the common fibular and tibial nerves, which provide motor control and sensory input to the foot and ankle. Branches such as the sural nerve supply cutaneous sensation to defined regions of the ankle and foot. The intricate coordination of bones, joints, ligaments, muscles, vessels, and nerves underpins the functional roles of the foot in support, balance, and locomotion. [4,5]

Etiology and Classifications

Charcot neuropathic osteoarthropathy (CNO), which predominantly affects the lower extremities and is characterized by fragmentation of the bones and joints of the foot and ankle, most commonly occurs in patients with peripheral neuropathy. Conditions such as diabetes mellitus, peripheral neuropathy, trauma, and metabolic bone disorders can precipitate an acute local inflammatory response that may, in turn, lead to permanent structural damage of the foot. These changes result in abnormal plantar pressure distribution and substantially increase the risk of complications, including foot ulcers, osteomyelitis, and even amputation. In addition, a spectrum of other, less common diseases, ranging from relatively frequent to rare etiologies, may also predispose to the development of CNO.[6]

Several classification systems have been proposed to characterize CNO, reflecting its heterogeneous presentation. The Trepman, Sanders Frykberg, and Brodsky classifications all describe CNO according to the anatomical location of joint involvement, thereby facilitating communication and treatment planning. In contrast, the Eichenholtz classification is based on clinical and radiographic findings and stages the disease according to its temporal progression. More recently, MRI-based classification approaches have emerged, incorporating advanced imaging features to refine staging and improve early detection and management strategies.[7-11]

Table 1. Trepman Classification

Type	Localization	Joint
1	Plantar	Tarsometatarsal, naviculocuniform
2	Medio plantar	Subtalar, talonavicular
3A	Bassi ankle	Calcaneocuboid tibiotalar
3B	Calcaneus	Tuberosity fracture
4	Multi regions	Sequential, simultaneous
5	Forefoot	Metatarsophalangeal

Table 2. Classification based on MRI

Stage	Severity grade	
	Low severity: grade 0 (without cortical fracture)	**High severity: grade 1 (with cortical fracture)**
Active arthropathy (acute stage)	Mild inflammation/soft tissue oedema No skeletal deformity X-ray: normal MRI: abnormal (bone marrow oedema, microfractures, bone bruise)	Severe inflammation/soft tissue oedema Severe skeletal deformity X-ray: abnormal MRI: abnormal (bone marrow oedema, macrofractures, bone bruise)
Inactive arthropathy (becalmed stage)	No inflammation No skeletal deformity X-ray: normal MRI: no significant bone marrow oedema	No inflammation Severe skeletal deformity X-ray: abnormal (past macrofractures) MRI: no significant bone marrow oedema

Pathophysiology of Charcot Neuropathic Osteoarthropathy

The pathogenesis of Charcot neuropathic osteoarthropathy (CNO) is complex and involves multiple interrelated mechanisms. Historically, two principal theories, termed neurotropic and neurotraumatic, have been proposed to explain this condition. The neurotropic theory, first introduced by Charcot, focuses on autonomic neuropathy and posits that disturbed vasoregulation leads to increased blood flow and subsequent bone resorption. In contrast, the neurotraumatic theory emphasizes unrecognized repetitive trauma to an insensate foot, resulting in joint instability and structural destruction. These concepts are now regarded as complementary and are integrated by contemporary biochemical insights into inflammation, bone metabolism, and neurovascular dysregulation.[12,13]

1. Inflammation and cytokine dysregulation

Inflammation represents a central driver of CNO. An imbalance between proinflammatory and anti-inflammatory cytokines leads to an exaggerated and sustained inflammatory response. Elevated levels of mediators such as TNF alpha, IL 1 beta, and IL 6, together with reduced concentrations of anti-inflammatory cytokines including IL 4 and IL 10, disrupt physiological homeostasis. TNF alpha activates the RANK RANKL pathway, promoting osteoclast activity and excessive bone resorption. The ratio of RANKL to its antagonist osteoprotegerin is markedly increased in patients with Charcot changes, thereby amplifying both inflammation and osteolysis. [12,13]

2. Role of osteoclasts

Osteoclasts are key effector cells in CNO, driving disproportionate bone turnover and progressive osteolysis. Their precursors, circulating monocytes, exhibit heightened proinflammatory activity and resistance to apoptosis, which perpetuates the inflammatory cascade and accelerates skeletal damage. Additional factors such as macrophage colony stimulating factor and cytokines including IL 8 further enhance osteoclast differentiation and activity, reinforcing the cycle of bone destruction. [12,13]

3. Neuropeptides and bone metabolism

Neuropeptides and neural regulation also influence bone metabolism in CNO. Calcitonin gene related peptide supports osteoblast activity, collagen synthesis, and cytokine modulation, but its deficiency in Charcot patients diminishes anti-inflammatory capacity and contributes to bone fragility. Similarly, reduced nitric oxide availability disrupts the normal induction of osteoclast apoptosis, thereby increasing osteolysis and further compromising skeletal integrity. [12,13]

4. Microvascular alterations and bone resorption

Microvascular changes represent an additional pathogenic component. Sympathetic denervation in Charcot patients leads to increased blood flow, hyperemia, and augmented bone resorption. Individuals with CNO retain the ability to mount a pronounced inflammatory vascular response, unlike patients with neuropathy alone. Excessive vasodilation together with elevated venous pressure results in edema, impaired microcirculation, and tissue ischemia. These changes weaken the osseous and articular structures of the foot and ankle, ultimately predisposing them to collapse. [12,13]

5. Metabolic and biochemical factors

Chronic hyperglycemia in diabetes contributes to the development of Charcot neuro osteoarthropathy through several mechanisms. Advanced glycation end products modify collagen and impair the structural integrity of bone, thereby reducing its mechanical strength. Insulin deficiency disrupts anti-inflammatory signaling pathways, further promoting a proresorptive milieu in bone. Hyperglycemia induced alterations of tendons and ligaments also redistribute plantar pressures abnormally, increasing susceptibility to fractures and joint dislocations. Reduced bone mineral density, more frequently observed in type 1 diabetes, together with vitamin D deficiency, additionally contributes to skeletal fragility, while hypocalcemia and secondary hyperparathyroidism exacerbate osteopenia in affected individuals. [12,13]

6. Genetic factors

Genetic predisposition involving polymorphisms in genes regulating the OPG RANK RANKL axis has been implicated in Charcot neuro osteoarthropathy. Several studies indicate that specific single nucleotide polymorphisms within OPG, RANK, and RANKL may influence disease susceptibility, with distinct variants reported across different populations. These polymorphisms are thought to alter expression or function within the OPG RANK RANKL pathway, thereby modifying the balance between bone formation and resorption and contributing to the pathological bone destruction characteristic of this condition. [12,13]

CNO thus arises from a multifaceted interaction of inflammatory, neurovascular, metabolic, and genetic factors. Although diabetes remains a major precipitating condition, the rarity of CNO even among individuals with long standing diabetes underscores the need for further research to elucidate its specific triggers and underlying mechanisms. [12,13]

DIAGNOSIS

Clinical Manifestations

CNO frequently presents acutely, with sudden onset of erythema, swelling, and increased warmth of the foot or ankle, although the clinical onset may also be more insidious, with swelling that remains unrecognized for months or even years, and in some cases the condition is not diagnosed until substantial radiographic destruction has occurred. The typical signs and symptoms include unilateral warmth, redness, and edema of the affected foot, which is usually warmer than the contralateral side and may show partial resolution of erythema with limb elevation; pain is often minimal despite marked clinical and radiographic changes, and the condition frequently occurs in the absence of overt wounds or ulceration.[3,14]

Joint involvement most commonly affects the foot and ankle, particularly the tarsometatarsal, hindfoot, metatarsophalangeal, and ankle joints, whereas involvement of other joints is rare; diabetic neuroarthropathy can occasionally affect upper limb joints such as the hand and wrist, and knee involvement has also been described but remains uncommon. [3,14]

Recurrent attacks affecting the same foot are relatively uncommon, although subsequent episodes may occur in the contralateral foot, typically separated by a substantial interval between events. In chronic or untreated CNO, severe joint damage and disorganization can result in irreversible deformities, including rocker bottom foot with collapse of the midfoot arch and plantar bony prominences that markedly increase the risk of ulceration, medial convexity deformity due to medial displacement at the talonavicular joint, and tarsometatarsal dislocation; collectively, these deformities further impair weight bearing capacity and substantially heighten the risk of ulceration and infection. [3,14]

Diagnostic Criteria for Acute and Chronic CNO

CNO, often referred to as Eichenholtz stage 0, can be diagnosed clinically when several key features are present. These include established diabetic neuropathy together with a warm, edematous, and erythematous unilateral foot or ankle, in the absence of an alternative obvious cause. The condition should be suspected in any patient with diabetic neuropathy who presents with a unilateral red, hot, swollen foot, because early diagnosis is critical; delays are common and may permit rapid progression with serious complications. If left untreated, substantial and irreversible joint destruction may occur within approximately six months or less. A meticulous physical examination is essential and should be complemented by laboratory tests such as a complete blood count, erythrocyte sedimentation rate, and C reactive protein. When joint effusion is present, arthrocentesis with synovial fluid analysis is required to exclude alternative diagnoses, and image guided aspiration with ultrasonography or computed tomography is recommended for small or structurally distorted joints; analysis should include Gram staining, culture, total and differential cell counts, and crystal identification. Weight bearing radiographs of the affected joint are advised, and although plain radiography cannot definitively confirm acute CNO, it provides a baseline for monitoring disease evolution, and normal radiographs should not delay diagnosis or initiation of treatment.[15-17]

Acute CNO is primarily a clinical diagnosis, and assessment aims both to confirm the characteristic features of the disorder and to identify findings suggestive of alternative conditions. Bilateral or symmetric involvement may indicate systemic arthritides such as rheumatoid or crystal induced arthritis. Marked pain and tenderness are atypical for Charcot neuro osteoarthropathy and raise suspicion for gout, cellulitis, or complex regional pain syndrome. A large joint effusion is uncommon and warrants further evaluation, while substantially elevated acute phase reactants, such as ESR or CRP, more strongly suggest inflammatory or infectious etiologies, although mild elevation may occur in neuroarthropathy and is not diagnostic. Leukocytosis, particularly when accompanied by a left shift, is more consistent with infection than with CNO. If competing diagnoses cannot be excluded on clinical and laboratory grounds, magnetic resonance imaging or other advanced imaging modalities may be employed, although additional imaging is not required when clinical criteria clearly support the diagnosis. [15-17]

In chronic CNO, corresponding to Eichenholtz stages 1 through 3, the diagnosis is established in patients with a history of acute neuroarthropathy and radiographic evidence of joint damage, including subluxation, osseous fragmentation, osteolysis, or new bone formation. This diagnosis should be considered in individuals with diabetes who present with foot or ankle pain or ulceration, even in the absence of documented acute phase symptoms. For chronic disease, plain radiography is generally sufficient, as the radiological hallmarks of CNO in these stages are well characterized and reliably demonstrate the structural changes associated with the condition.[15-17]

MANAGEMENT

Overall management of patients with CNO begins with assessment for vascular impairment followed by systematic exclusion of infection, after which surgical strategies are guided by the Brodsky anatomical classification and the Eichenholtz stage. The overarching objective is to achieve a functional, shoeable foot that remains free from infection. Management of CNO is complex and continually evolving, largely because of the paucity of randomized controlled trials that define optimal surgical techniques and timing; a review of 30 studies including 860 patients failed to generate definitive recommendations, and a separate systematic review of 42 studies did not demonstrate superiority of either internal or external fixation. Treatment objectives change as the patient progresses through the Eichenholtz stages.[18,19]

- Early fragmentation stage: The principal goal is protection of the limb from microtrauma by immobilization, with total contact casting (TCC) and non-weight bearing (NWB) shown to shorten the fragmentation period.
- Coalescence stage: Treatment focuses on preventing or limiting deformity using continued immobilization and offloading, with patient adherence being critical.
- Resolution stage: The aim shifts to deformity correction in order to restore biomechanics, prevent ulceration, and allow normal ambulation; surgery is generally deferred until the disease is quiescent to reduce postoperative complications, except in cases involving the ankle or subtalar joint, and infection, which commonly arises in stage 3 in association with ulcers, requires prompt antibiotics and often surgical debridement. Throughout the course of disease, multidisciplinary team involvement, including psychosocial support, is essential for optimal outcomes.

Effective care for CNO requires coordinated input from multiple specialists, consistent with NICE guidance. An endocrinologist plays a central role in diabetes management, with a target glycated hemoglobin below 7 percent recommended before surgery, as values above 8 percent substantially increase perioperative complication risk. Vitamin D deficiency, which is common among patients with CNO, impairs bone quality and wound healing and should be corrected when present. Foot and orthopedic specialists are crucial, particularly for administering total contact casting and managing ulcers. Current NICE and IWGDF guidelines advise against the use of bisphosphonates and other medical agents such as calcitonin, methylprednisolone, parathyroid hormone, or denosumab for active CNO, based on contemporary evidence. [18,19]

Infection markedly worsens the prognosis of CNO, conferring up to a twelvefold increase in the risk of major lower limb amputation. Infection typically arises from foot ulcers at any disease stage. Acute infections, including sepsis or episodes of diabetic foot attack, must be treated as emergencies because of their high mortality risk. Management includes: [18-20]

- Urgent surgical debridement of infected tissue
- Microbiological sampling for culture and sensitivity

- Initiation of broad-spectrum antibiotic therapy, subsequently refined according to culture results Advanced wound care modalities, such as negative pressure wound therapy or larval debridement, may facilitate healing, and local antibiotic releasing calcium preparations can be used to fill bony defects in osteomyelitis. When infection control is achieved but ulceration persists, internal fixation can be considered provided there is no exposed bone, with appropriate casting and strict offloading remaining essential during this phase.

Peripheral arterial disease in patients with CNO further increases the likelihood of amputation and hospital admission. Vascular assessment with Doppler studies and imaging modalities such as CT or MR angiography is crucial to evaluate arterial sufficiency and to plan revascularization where indicated. In approximately 10 to 20 percent of cases, CNO involves the ankle or subtalar joint, leading to marked instability and severe deformity. In such cases:

- Early talocalcaneal or ankle arthrodesis using compression constructs is recommended to optimize stability.
- Tibiotalocalcaneal arthrodesis may be required for more extensive involvement. Although allograft reconstruction for height restoration has been described, it is associated with higher complication rates and is generally not recommended. [14,18]

Management within each Eichenholtz stage can be summarized as follows. [14,18]

- Development/fragmentation stage: Prompt diagnosis and immobilization are paramount. Total contact casting minimizes further trauma and unloads the limb. Cast changes every two to four weeks allow monitoring of ulcers and treatment response, and the fragmentation phase usually lasts two to four months.
- Coalescence stage: Ongoing protection, often via total contact casting or carefully supervised gradual weight bearing under radiographic surveillance, promotes fracture healing and limits deformity. Devices such as the Charcot Restraint Orthotic Walker (CROW) or similar offloading braces are useful alternatives depending on patient adherence.
- Resolution stage (reconstruction and deformity correction): Minor deformities may be managed with orthoses or limited osteotomies, whereas severe deformities require combined approaches, including osteotomy, arthrodesis, and internal, external, or hybrid fixation. Weight bearing computed tomography assists surgical planning by defining deformity patterns, and Achilles tendon lengthening can improve outcomes in both mild and severe deformity.

Delaying surgery in advanced cases is associated with higher rates of soft tissue complications. Internal fixation constructs frequently employ hydroxyapatite coated screws to improve purchase in osteopenic bone, while external fixation remains a key option when infection or persistent ulceration is present. Midfoot involvement, which accounts for about 60 percent of CNO cases, often requires dorsomedial and dorsolateral approaches to access and correct the affected joints. Surgical techniques in this context include:

- Beaming constructs, in which intramedullary screws restore stability of the medial and lateral columns, with hydroxyapatite coated screws reducing the risk of migration in weak bone.
- Hybrid fixation, which combines internal fixation with external frames in cases of severe deformity.
- Circular external ring fixation, used when ulcer healing is problematic or for temporary stabilization in infected or severely deficient segments.

Postoperative care typically involves approximately twelve weeks of non-weight bearing in a cast, followed by gradual progression to weight bearing, with adequate stabilization across all involved joints using high stiffness constructs being critical for durable outcomes.

A multidisciplinary, staged approach to CNO that integrates vascular, infectious, metabolic, and structural considerations remains fundamental to successful management, and tailoring treatment according to Eichenholtz stage, Brodsky location, vascular status, and presence of infection minimizes risk while improving long term functional results. [14,18]

PROGNOSIS

The prognosis of Charcot neuro osteoarthropathy (CNO) is highly dependent on early diagnosis and timely initiation of appropriate management. Rapid recognition of the condition and prompt offloading of the affected foot are critical determinants of favorable outcomes, whereas patients who present in advanced stages often exhibit severe, irreversible joint destruction that predisposes to foot ulceration, infection, and eventual amputation. CNO can profoundly affect quality of life, leading to pain, disability, loss of mobility, and work incapacity, and is associated with increased morbidity and premature mortality. Involvement of the ankle and hindfoot tends to be particularly severe and technically challenging to treat, frequently necessitating prolonged periods of immobilization and carrying a poorer overall prognosis compared with midfoot disease.[21]

CONCLUSION

Charcot neuro osteoarthropathy (CNO) is a disorder that affects the bones, joints, and soft tissues of the foot, most commonly in individuals with diabetes who have peripheral neuropathy. It typically presents with a warm, swollen, and erythematous foot which, if not promptly recognized and treated, can progress to severe deformity, ulceration, or even amputation. Diagnosis requires careful clinical evaluation supported by imaging modalities such as plain radiography or magnetic resonance imaging to assess structural damage, along with laboratory investigations to distinguish CNO from infectious processes. Initial management centers on immobilization with total contact casting and strict offloading in order to prevent further trauma and to stabilize the affected structures.

Optimal care of CNO demands a multidisciplinary approach that prioritizes glycemic control, sustained immobilization, and deformity correction tailored to the stage of disease. Surgical interventions, including internal fixation, osteotomy, or external fixation, are considered in the presence of severe deformity or when the ankle and subtalar joints are involved. Complications such as infection substantially increase the likelihood of amputation, underscoring the critical importance of early diagnosis and timely, coordinated treatment. With an appropriate, stage-directed strategy, patient outcomes and overall prognosis can be markedly improved.

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