Case Series

Charcot's osteoarthropathy – Lest we forget

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ABSTRACT

Charcot' osteoarthropathy (COA) is a rare, often misdiagnosed inflammatory debilitating complication of conditions most common being diabetes that needs to be discussed further to prevent associated morbidity. The natural history of the disease sees four stages and requires prompt diagnosis and treatment to ensure the desired outcome. We describe a case series of two cases of diabetes-associated osteoarthropathy and their management. A 72-year-old male with the early COA was managed with a total contact cast for both lower limbs and an 80-year-old male with the early COA with bone marrow edema in the tibia and talus, as well as, subtalar effusion, managed conservatively. The follow-up review documented clinical recovery in the form of a marked reduction in swelling of the lower limbs with the resolution of the functional status of lower limbs. It is the responsibility of every physician to ensure the goals of management which includes immediate offloading and good glycemic control.

Key words: Charcot's osteoarthropathy, Diabetes mellitus, Neuropathy, Offloading

INTRODUCTION

Charcot's osteoarthropathy (COA) of the foot was first described in 1883, in reference to tabes dorsalis [1]. Charcot's arthropathy is linked to multiple etiologies, with diabetes mellitus being the commonest. Other etiologies of COA include leprosy, poliomyelitis, trauma, syringomyelia, and heavy metal poisoning. Diagnosis is challenging and is often missed even by experienced physicians, leading to long-term sequelae [2]. The prevalence is increasing due to longer life spans and comprehensive diabetic screening [3]. The course of COA has been described by a modified Eichenholtz classification [4]. Here, we describe a case series of two cases of COA detected in the early phase (stage 1).

CASE SERIES

Case 1

A 72-year-male, with type 2 diabetes mellitus of 23 years of duration, presented with swelling and redness of both lower limbs and a fever of 7 days duration. The patient was hemodynamically stable. Local examination of the lower limbs revealed bilateral non-pitting edema with diffuse erythema until mid-shin. The temperature difference between the affected and unaffected limbs was 4°C using an infrared thermometer. Lower limbs became pale on raising followed by the return of color (Fig. 1). The spinothalamic sensations were preserved. The ankle joints had normal power with absent deep tendon reflexes. Plantar reflex was normal bilaterally. There was a loss of vibration and joint position sensations up to the knee joints involving knee joints (L4–S1 sensory dermatomes). The patient had a high stepping gait. The rest of the joints were normal. His ophthalmological examination was normal. Other systemic examinations were unremarkable.

Basic hematological and biochemical parameters were within normal range. The patient had an HbA1c of 7.5%, blood sugar fasting of 179 mg/dL, and post-prandial levels of 231 mg/dL. The patient was on tab metformin, vildagliptin, and gliclazide. Urine analysis revealed microalbuminuria. Ultrasound abdomen was also unremarkable. X-rays of bilateral feet and ankle joints were normal. Magnetic resonance imaging (MRI) of the feet and ankle showed mild effusion in the tibiotalar joints, confirming early COA (Fig. 2). The patient was managed as a case of the early COA with a total contact cast (TCC) for both lower limbs was applied for 3 months. The follow-up review documented clinical recovery in the form of a marked reduction in swelling of the lower limbs with the resolution of the functional status of lower limbs.

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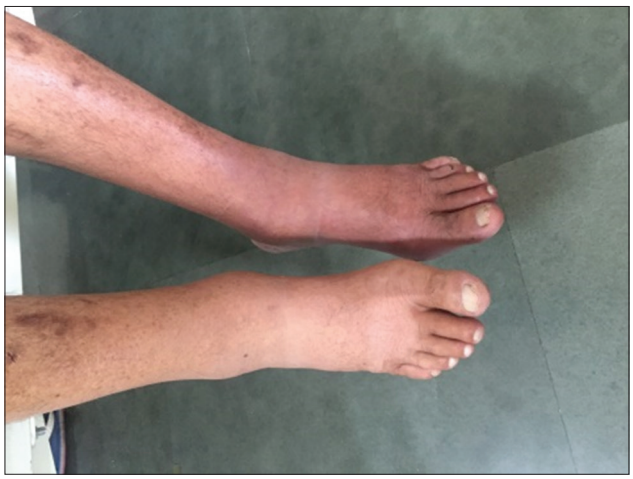

Figure 1: Comparative color changes between bilateral lower limbs. Left lower limb showing blanching after being elevated to 60° from horizontal for 60 s as compared to the normal right lower limb (Case 1)

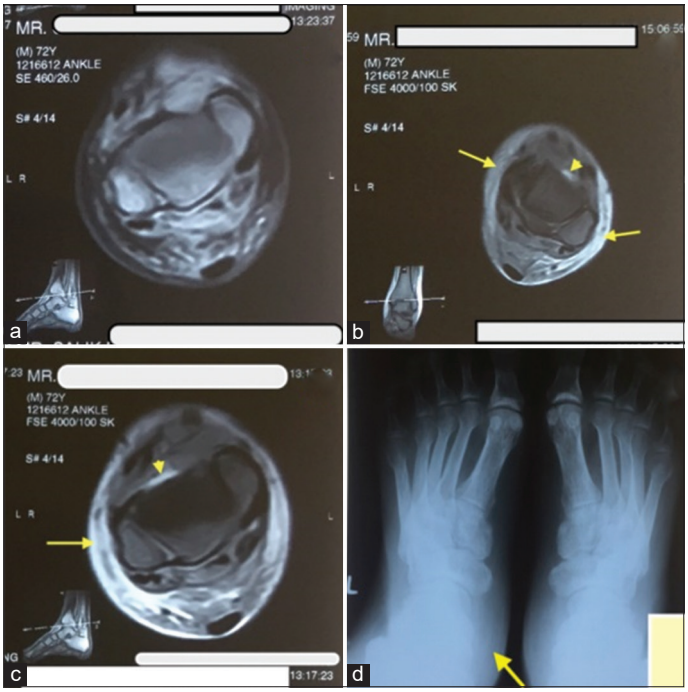

Figure 2: Magnetic resonance imaging feet and ankle. (a) T1 spin echo image; No bone destruction or marrow edema; (b and c) T2 Fast spin echo image; Yellow arrows showing soft-tissue edema; Yellow arrow heads showing tibiotalar effusion; (d) Yellow arrow shows soft-tissue swelling in plain X-rays of feet and ankle (Case 1)

Case 2

An 80-year-old male with type 2 diabetes and primary hypertension of a duration of 30 years with a history of coronary artery disease in the past was admitted with complaints of redness and swelling over the ankle joint of 2 weeks' duration. The patient was hemodynamically stable. Local examination of the lower limbs revealed bilateral non-pitting edema with diffuse erythema until mid-shin. Lower limbs became pale on raising followed by the return of color. The spinothalamic sensations were preserved. The temperature difference between the two limbs measured by the infrared thermometer was 3.8°C. The ankle joints had normal power with normal deep tendon reflexes. Plantar reflex was normal bilaterally. There was a loss of vibration and joint position sensations up to the knee joints involving knee joints (L4–S1 sensory dermatomes). The patient had a high stepping gait. The rest of the joints were normal. Other systemic examinations were unremarkable.

Basic hematological and biochemical parameters were within normal range. The patient had an HbA1c of 8.3% with blood sugar fasting of 201 mg/dL, and post-prandial levels of 280 mg/dL. The patient was on tab metformin, sitagliptin, and glimepiride. Urine analysis was normal. His ophthalmological examination was normal. Ultrasound abdomen was also unremarkable. X-rays of bilateral foot and ankle joints were normal. MRI of lower limbs showed bone marrow edema in the tibia and talus, as well as, subtalar effusion. He refused a TCC but agreed to be wheelchair-bound with restricted weight-bearing until recovery. The follow-up review showed a mild reduction in swelling of the lower limb with limited resolution of functional status. The patient was further counseled for TCC and was followed up after 4 weeks and had improved functional status of the lower limbs.

DISCUSSION

Charcot's arthropathy affects 0.8–8% of diabetics, without any sex predilection [5]. A complex interplay of diabetic control, neuropathy, trauma, and perturbations of bone metabolism results in acute inflammation of varying degrees with bone and joint disorganization, an altered foot structure, and abnormal loading. Other causes of COA include syphilis, leprosy, syringomyelia, chronic alcoholism, and meningomyelocele.

The underlying pathophysiology behind Charcot arthropathy is the increased pro-inflammatory mediator response to trauma which leads to activation of nuclear factor kB, which, in turn, increases the osteoclastic activity [6]. Underlying autonomic neuropathy is also a contributing factor toward the development of Charcot's arthropathy.

Clinically, the affected limb will have signs of inflammation and often misdiagnosed as a sprain, osteomyelitis, Sudeck's atrophy, deep vein thrombosis, cellulitis, or rheumatoid arthritis. The diagnosis and management of Charcot arthropathy are depicted in Table 1 [2,7]. A strong clinical suspicion with swift management is essential for better patient outcomes. Clinically, COA is of two types, acute and chronic. Acute COA presents with a red swollen limb which is often painless with a temperature difference of around 2–6°C from the contralateral limb. Chronic COA occurs after 6 months of untreated acute COA develops deformities, typically arch collapse with rocker bottom deformity and medial convexity. This leads to ulcerations, infections, and osteomyelitis subsequently [8].

The progression of COA is studied by Eichenholtz staging, as given in Table 2 [4]. Diagnosis of COA is clinicoradiological. The X-ray can be normal until 3 weeks of presentation; however, later can show atrophic or hypertrophic changes. Bone scintigraphy using technetium99m methylene diphosphonate bone scan is positive in all phases. Acute COA is characterized by reduced signal intensity on a T1 sequence and increased signal intensity on the T2 sequence [9]. Chronic COA is characterized by diffusely diminished marrow signal intensity and cyst formation.

Table 1: Diagnosis and treatment of Charcot's osteoarthropathy

	IMAGING		
	Plain radiographs	**MRI**	**Nuclear medicine scanning**
1.	Initial test recommended	Confirms clinical suspicions in the presence of normal appearing radiographs	If MRI is indeterminate then this is next investigation of choice
2.	Foot and ankle radiographs be taken in the weight-bearing position if possible	Investigation of choice	Labeled white blood cell scanning with 111In or 99 mTc, combined with a three-phase bone scan or sulfur colloid marrow examination; and PET scanning
3.	Low sensitivity; normal during early stages	Allows early detection	Can confirm diagnosis
4.	Later stages can show: Overt fractures, subluxations, reduction or breakage of bone angle and arterial calcification	Increased T1 and decreased T2 intensities in affected areas with decreased marrow intensity and cyst formation	Cost and availability is a challenge in many settings
TREATMENT			
1.	Offloading the foot and immobilization in a cast: Most important treatment that prevents further destruction.		
2.	Application of total contact cast for 3–6 months is treatment of choice.		
3.	Chronic patients are offered specialized footwear, total ankle foot orthosis with special attention to protect the unaffected limb also.		
4.	Protective weight-bearing is required post recovery from an active episode, involving weight-bearing devices such as prescription shoes, boots, or braces.		
5.	Little evidence for the use of available pharmacological therapies to promote the healing.		
6.	Holistic management of diabetes including strict glycemic control, lifetime surveillance to monitor for signs of recurrent or new COA episodes, as well as other diabetic foot complications.		

MRI: Magnetic resonance imaging

Table 2: The course of Charcot osteoarthropathy according to the clinical presentation of the foot: Modified Eichenholtz classification

Stage	Clinical presentation
0: Patients at risk for COA	Diabetic neuropathy and an acute sprain or fracture
I: Development-fragmentation	Erythema, edema and increased warmth, usually absence of pain
II: Coalescence	Diminution of erythema, edema, and warmth; Decreased joint mobility
III: Reconstruction-consolidation	Erythema, edema, and warmth are no longer present; Ulcers at sites of residual deformity

The mainstay for treatment of COA is offloading with the gold standard being TCC with immobilization of the affected limb for 3 months to 1 year. Other options are air casts, wheelchairs, crutches, and zimmer frames. NSAIDS and glucocorticoids can be used as adjuncts. Those with a poor outcome can undergo surgical management including exostectomy, arthrodesis, and amputation [10].

CONCLUSION

The incidence of COA has increased view increased awareness among physicians. It is crucial to suspect the diagnosis in the clinical setting and also to confirm with a suitable radiological investigation. Offloading of the affected limb and stringent glycemic control remains the gold standard of management. Surgical options must be considered promptly, especially in refractory cases and cases of chronic COA.

AUTHORS CONTRIBUTIONS

Amitabh Sagar: Concept design, conduct of study. Anmol Sharma: Conduct of study, writing, and proofing of manuscript.

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