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## Original Article

## Clinical features, radiological characteristics and offloading modalities in stage 0 Acute Charcot's neuroarthropathy - A single centre experience from South India



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## ABSTRACT

**Aims:** Stage 0 Acute Charcot's Neuroarthropathy (ACN) in Type 2 Diabetes patients is a challenging diagnosis with subtle clinical features and normal appearing plain radiographs of the affected foot. Delay in diagnosis can lead to progression of disease and irreversible deformities. There is a paucity of data on Stage 0 ACN from India. The aim of this study was to assess clinical and radiological characteristics and treatment outcomes in Indian Type 2 Diabetes patients with Stage 0 ACN.

**Materials and methods:** A comparative, case-control study was carried out amongst patients attending the Integrated Diabetes Foot Clinic at a tertiary care South Indian hospital. During the 3-year study period, a total of 1811 patients with Type 2 Diabetes Mellitus were screened. Of these, n = 10 patients with stage 0 ACN Charcot's arthropathy were identified based on clinical features and MRI imaging of the foot for confirmation of diagnosis. These were compared with an age and duration of diabetes-matched group of n = 50 patients without ACN as controls.

**Results:** Our study identified 10 patients (0.5%) with Stage 0 Acute charcot neuroarthropathy (ACN) in the study population. Those with ACN had higher BMI, poorer glycaemic control and greater degree of peripheral neuropathy ( $p < 0.05$ ). Clinically relative lack of pain and infrared thermometric temperature difference  $>2^{\circ}\text{C}$  in the affected foot were the most significant findings, while MRI foot was useful in early detection of active and severe stage 0 disease. Total contact cast was the preferred initial offloading modality, with delay in initiating complete immobilization leading to worse outcomes.

**Conclusions:** This is the first study to highlight the characteristic features of Stage 0 ACN in Indian Type 2 Diabetes patients. Thorough clinical evaluation, infrared thermometry and radiological findings on MRI foot leads to early disease detection. Complete offloading, preferably with total contact casts can prevent disease progression and chronic deformities.

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## 1. Introduction

Charcot's foot (or Charcot's neuroarthropathy) is an unique presentation of the diabetic foot, characterized by pain, insensitivity and significant bone and joint destruction leading to the typical 'rocker-bottom' deformity. The pathogenetic mechanisms

remain elusive, with evidence suggesting a role for motor, sensory and autonomic neuropathy leading to recurrent microtrauma and inflammatory cytokine mediated activation of the osteoclastic pathways [1]. The acute Charcot's foot is characterized by a swollen, hot and red foot, with variable pain sensation, requiring immediate offloading and immobilization [2]. If not tackled early, repetitive weight bearing can lead to chronic neuroarthropathy and fractures of bones or joints already weakened from inflammation [3]. In 1966, Eichenholtz divided the "natural" course of the Charcot's foot

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on the basis of X-ray findings into stage 1 (bone dissolution), stage 2 (coalescence) and stage 3 (remodelling) [4]. Stage 1 represents the damaging acute phase, stage 2 represents the repair phase and stage 3 represents the chronic, quiescent, healed phase. However, it had already been shown in 1976 by Classen et al. [5] that clinical symptoms (swelling, warmth, erythema and deep dull aching while walking) regularly precede the X-ray signs of Eichenholtz stage 1 by several weeks or months together with scintigraphic bone abnormalities. Edmonds and Watkins (1984) reported that immobilising and offloading a foot with these early changes may prevent fractures and deformity [6]. Subsequently, Shibata et al., in 1990 added a fourth stage, Charcot's foot stage 0 (clinical and scintigraphic signs without X-ray abnormalities) to the conventional classification [7]. The bone pathology of stage 0 could later be identified by magnetic resonance imaging (MRI) as reactive osseous oedema, similar to stress injury [8]. Early detection and treatment of acute Charcot's foot stage 0 has become imperative to prevent devastating skeletal destruction and permanent deformity [9]. However, most studies to date were X-ray based [10], consequently focusing on stage 1–3. We thus report the first series of stage 0 Acute Charcot's Neuroarthropathy (ACN) in an Asian Indian population with review of their clinical, radiological and therapeutic outcomes.

## 2. Materials and methods

A comparative, case-control study (Fig. 1) was carried out amongst patients attending the Integrated Diabetes Foot Clinic in the Department of Endocrinology, Diabetes & Metabolism at CMC Vellore in Tamil Nadu, India. As well as treating the local population, this centre serves as a national referral centre for patients from throughout India. The multidisciplinary diabetic foot clinic involves endocrinologists, physical medicine rehabilitation and orthotics experts and vascular surgeons. It provides specialized care to a wide diversity of foot problems in diabetes. Patients with a known diagnosis of type 2 diabetes, as defined by the criteria of the

American Diabetes Association (ADA) and attending the foot clinic were included in the study. Patients with clinical features of acute Charcot's neuroarthropathy (swelling of feet, redness, warmth, pain etc) and absence of radiological signs of Charcot's neuroarthropathy (on standing antero-posterior and lateral X-ray-foot) were considered as cases of stage 0 acute Charcot's neuroarthropathy (ACN) according to Shibata's classification [7]. Patients with clinical and radiological features of chronic Charcot's neuroarthropathy OR ACN with X-ray findings were excluded. Age matched people with diabetes attending the Foot Clinic for non-Charcot's complications of acute onset swollen foot due to other infective or inflammatory causes were included as controls. Demographic data and medical histories were taken from participants, including age, sex, weight, height, duration of diabetes, antidiabetic regimen, occupation, smoking habit and employment status. Biochemical investigations included the most recent HbA1c, fasting plasma glucose, lipid profile and creatinine levels. Clinical (presence of pain, purulent discharge, swelling, increased temperature, erythema) or biochemical (total and differential leucocyte count, ESR and CRP) evidence of infection were documented in all. Radiological evidence of Charcot's foot was based on findings of plain x-ray film, verified by two radiology consultants. Radiological evidence of osteomyelitis was also documented. A diagnosis of nephropathy was made in the presence of proteinuria of greater than 150mg/24 h s) or microalbuminuria of greater than 30 mg in 24 h. Retinopathy was considered to be present if any typical diabetes related changes were seen on fundoscopy. Peripheral vascular disease was diagnosed if peripheral pulses (dorsalis pedis and posterior tibial) were absent and ankle brachial pressure index was less than 0.9. Disease specific data including foot ulceration, location of ulcers and recommended off-loading treatment were also documented. Neurological assessment of the feet was sought to detect the loss of protective sensation (10 g monofilament) and vibratory sensation (128 hz tuning fork and more than 25 V on biothesiometry). The presence of painful neuropathy was determined by an interview. Further clinical assessment was conducted to detect the presence of callus, anhidrosis (dry skin), fissures, tinea pedis, active ulceration, corns, dermatopathy, cellulites, oedema reducible on limb elevation, infrared thermometry for detection of the inter-limb temperature differences and presence of amputation at enrolment. Treatment options included different forms of orthoses, non-weight bearing aids and custom made insoles for footwear.

During the 3-year period of July 2014 to August 2017, a total of 1811 patients with Type 2 Diabetes Mellitus attending the foot clinic were screened and 150 patients with Charcot's neuroarthropathy was identified. Of these, 140 of them were excluded as they had either chronic charcot's or ACN with X-ray finding. 10 patients with stage 0 ACN Charcot's arthropathy were identified. All 10 cases had X ray of the foot and MRI imaging of the foot for confirmation of diagnosis. Radiological findings in the MRI were classified according to Chantelau's and Grutznel's MRI Classification (Table 1). Out of the remaining patients with acute complication of the foot not due to ACN, an age and duration of diabetes-matched group of n = 50 patients who consented for the study were selected as controls. The study was approved by the Institutional Ethics Committee (IRB No: 8378) of Christian Medical College, Vellore. Appropriate informed consent was obtained from all study participants and confidentiality of data was maintained throughout the study.

Statistical analysis was carried out using SPSS (version 16; SPSS, Chicago, IL). The data was examined for data entry errors and outlier values. Bivariate analysis was carried out using the paired t-test for continuous variables and Chi-squared tests for categorical variables. A p-value of less than 0.05 was considered statistically significant.

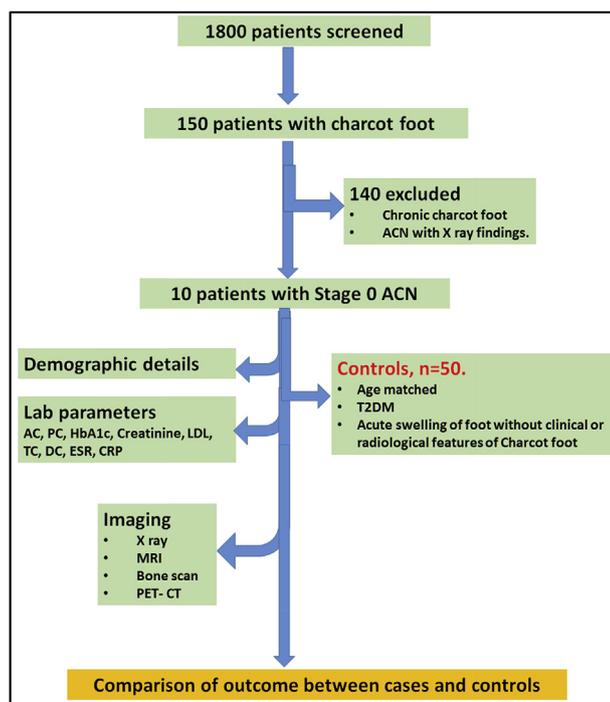


Fig. 1. Overview of the study design.

**Table 1**  
MRI classification of stage 0 Acute Charcot's neuroarthropathy.

Chantelau's and Grutznel's MRI Classification		
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, micro fractures, bone bruise)	Severe inflammation/soft tissue oedema. Severe skeletal deformity X-ray: abnormal (macro fractures) MRI: abnormal (bone marrow oedema, bone bruise, macro fractures)
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 macro fractures) MRI: no significant bone marrow oedema

### 2.1. Results and analysis

Our study identified 10 patients with Stage 0 Acute charcot neuroarthropathy (ACN) in the time period between July 2014 and September 2017 amongst 1811 patients who attended the Diabetes Foot clinic, thus accounting for 0.5% of the total population attending the foot clinic.

### 2.2. Baseline characteristics

The baseline characteristics of the cases and controls are summarised in the following Table 2. The cases and controls were matched for their age. There were no significant differences in both the groups with respect to sex, employment status, duration of diabetes, lipid profile, creatinine, or essential hypertension. Cases had a higher mean BMI than controls (29.2 kg/m<sup>2</sup> versus 26.1 kg/m<sup>2</sup>) which was statistically significant ( $p = 0.03$ ). They also had higher HbA1c (9.1% versus 8.1%,  $p = 0.04$ ), fasting plasma glucose levels (199 mg/dl versus 171 mg/dl,  $p = 0.04$ ), and peripheral neuropathy (100% versus 84%,  $p = 0.05$ ) when compared to controls which were statistically significant. There were no statistically significant differences between the cases and controls with respect to diabetic nephropathy and retinopathy. Controls had increased inflammatory markers (64% versus 20%) when compared to cases which was statistically significant ( $p = 0.02$ ).

### 2.3. Foot-associated pathologies

With respect to foot related characteristics (Table 3), pain was more common amongst the controls than cases (72% versus 50%) which was statistically significant ( $p = 0.04$ ). Swelling reducible on limb

elevation was more common amongst the cases (70% versus 42%) which was statistically significant with  $p = 0.03$ . Elevation of temperature by more than 2 °C on infrared thermometer was statistically higher amongst cases than controls (90% versus 30%,  $p = 0.01$ ). Features of peripheral neuropathy in the form of loss of vibration sense, loss of 10 g monofilament sensation was statistically higher in the cases than controls. Peripheral vascular disease, venous insufficiency, ulcerations were more prevalent amongst the controls than cases.

### 2.4. Characteristics of patients with Acute Charcot's neuroarthropathy

With respect to pattern of limb involvement based on Sanders and Frykberg classification, the most common area involved was the midfoot (40%). The right foot was affected in 60% of the cases and left foot was affected in 40% of the cases. Of the 10 patients who had MRI of the foot,  $n = 7$  had active, high severity disease (Grade I) on MRI and  $n = 3$  had active, low severity disease (Grade 0) on MRI. Progression to chronic destructive disease was seen in 4 of the 7 patients with Grade I disease but none in Grade 0 disease. Use of oral alendronate ( $n = 3$ ) was not associated with earlier disease stabilization ( $p = 0.15$ ), though resulted in faster resolution of pain and erythema ( $p = 0.04$ ). All the cases underwent offloading in the initial 6–8 weeks with total contact cast, which was followed by pneumatic boot in  $n = 4$ , ankle foot orthosis in  $n = 2$ , moulded insole in  $n = 2$  and Charcot Restraint orthotic walker (CROW) in  $n = 2$  patients. These findings have been summarised in Table 4. The typical MRI findings of one of the patients we identified with ACN have been depicted in Fig. 2a and b respectively.

**Table 2**  
Baseline characteristics of the study population.

Baseline Characteristics	Cases (n = 10)	Controls (n = 50)	p-value
Male % (n)	70% (7)	68% (34)	0.12
Mean (SD) age	58.9 (19)	60.3 (10)	0.18
Mean (SD) BMI (kg/m <sup>2</sup> )	29.2 (5.0)	26.1 (3.8)	0.03
Mean (SD) duration of diabetes (years)	14.1 (5.2)	16.6 (7.4)	0.31
Mean (SD) HbA1c (%)	9.1 (1.8)	8.1 (1.5)	0.04
Mean (SD) fasting plasma glucose (mg/dl)	199 (40.3)	171 (33.8)	0.04
Mean (SD) LDL-cholesterol (mg/dl)	123 (26.1)	132 (35.9)	0.16
Mean (SD) Triglyceride (mg/dl)	165 (42.2)	184 (59.5)	0.25
Mean (SD) creatinine (mg/dl)	1.1 (0.3)	1.3 (0.5)	0.12
% raised inflammatory markers (ESR/CRP)	20 (2)	64 (32)	0.02
% smokers (n)	60 (6)	44 (22)	0.03
% unemployed (n)	40 (4)	42 (21)	0.18
% hypertension (n)	70 (7)	72 (36)	0.33
% OAD only (n)	30 (3)	28 (14)	0.20
% insulin only (n)	40 (4)	28 (14)	0.15
% OAD with Insulin (n)	30 (3)	44 (22)	0.38
% retinopathy (n)	40 (4)	46 (23)	0.50
% nephropathy (n)	30 (3)	39	0.10
% neuropathy (n)	100 (10)	84 (42)	0.05

**Table 3**  
Foot-related characteristics in the study population.

Foot-related Characteristics	Cases(n = 10)	Controls(n = 50)	p-value
% Pain(n)	50(5)	72(36)	0.04
% Swelling reducible on limb elevation(n)	70(7)	42(21)	0.03
% Erythema(n)	50(5)	56(28)	0.16
% Infrared Temperature difference > 4 °C (n)	90(9)	30(15)	0.01
% Loss of vibration sensation(n)	80(8)	64(32)	0.04
% Loss of 10 g monofilament test(n)	90(9)	78(39)	0.05
% Peripheral vascular disease(n)	10(1)	22(11)	0.04
% Venous insufficiency(n)	10(1)	32(16)	0.04
% History of ulceration(n)	30(3)	56(28)	0.03
% Previous trauma (n)	60(6)	42(21)	0.02
% Callosities	20(2)	34(17)	0.08
% Anhidrosis	50(5)	64(32)	0.10
% Clawing of toes	40(4)	52(26)	0.06

### 3. Discussion

Our study which looked at Stage 0 Acute Charcot's Neuroarthropathy (ACN) showed that plain radiography did not pick up Stage 0 ACN and higher imaging modalities in the form of MRI of the affected foot and ankle were required. The prevalence of Stage 0 Acute Charcot's Neuroarthropathy in this study was 0.5%. The prevalence of Chronic Charcot's Neuroarthropathy in this population was 4.9% as depicted in our previous study in 2017 [11]. Lack of specific clinical and radiological markers in Acute Charcot's foot leads to delay in diagnosis in upto 25% of patients as shown in the study done by Korzon-Burakowska et al., in 2012 [12]. The higher prevalence of chronic Charcot's neuroarthropathy as compared to Acute Charcot's neuroarthropathy could be attributed to the delay in diagnosis of ACN due to lack of changes on plain radiographs which leads to progression of ACN to Chronic neuroarthropathy.

When comparing the symptoms between the cases and controls, it was found that pain was the predominant complaint amongst the controls (72%) as compared to the cases(50%). This is an important clinical clue in detection of ACN in an acutely swollen foot in a diabetic patient. Even previous studies in Acute Charcot's foot have documented the lack of pain in more than half of the patients, which is similar to our findings [13]. Our clinical examination findings showed significant loss of vibration sense and loss of 10 g monofilament sensation, suggesting the presence of

significant peripheral neuropathy amongst the cases as compared to the controls. This explains the decreased pain sensation in cases as compared to the controls. Further, the clinical finding of foot swelling which is reducible on limb elevation was significantly higher in those with ACN. This has been described as a sensitive early indicator of ACN in previous studies [14]. An infrared thermometry enabled temperature difference of >2 °C was significantly higher for cases than controls. Comparison of baseline characteristics showed that the cases had poorer glycemic control as compared to controls and were more obese. These findings are similar to those found in the studies done by Fabrin et al. [3] and Chisholm et al. [14]. When looking at other foot-related characteristics, cases had a significant increase in infrared thermometer readings as compared to the controls [15]. When looking at inflammatory markers such as CRP and ESR, it was found to be normal or minimally elevated in cases when compared to controls. This is in keeping with previous studies that have shown that the inflammatory response in ACN is largely localized and not systemic [16].

In the study done by Chantelau et al., in 2013, the authors showed that plain radiographs failed to pick up ACN and MRI should be preferred and immediate offloading should be done for Stage 0 ACN to prevent progression and deformities [17]. Our study has shown that progression to chronic destructive arthropathy was more common in Grade I disease as compared to grade 0 (n = 4 vs

**Table 4**  
Pattern of involvement, MRI grading and offloading modalities in the cases (n = 10).

Pattern of involvement(Acute Charcot's)	
Limb involvement in Acute Charcot's foot (n = 10)	
%Right foot (n)	60(6)
%Left foot (n)	40 (4)
%Bilateral (n)	0
Site of involvement	100%(N = 10)
%Forefoot(n)	30(3)
%Midfoot(n)	60(6)
%Hindfoot(n)	10 (1)
More than one region	30(3)
Chantelau's MRI classification of acute Charcot's foot (n = 7)	
% Active, high severity(n)	70(5)
% Active, low severity(n)	30(2)
Inactive, high severity(%)	0
Inactive, low severity (%)	0
Types of offloading –initial 6–8 weeks (n = 10)	
%Total contact cast (n)	60(6)
% Wheelchair	20(2)
% Walker	20(2)
Followup offloading (n = 10)	
% Pneumatic boot	40(4)
% Ankle-foot orthosis-AFO (n)	20(2)
% Moulded insole(n)	20(2)



**Fig. 2A.** T2 weighted MRI –axial images showing bone marrow edema suggestive of Acute Charcot's Neuroarthropathy.



**Fig. 2B.** T2 weighted MRI –sagittal images showing bone marrow edema of midfoot and hindfoot suggestive of Acute Charcot's Neuroarthropathy.

n = 0) which was similar to the findings by Chantelau et al. [17]. The advantages of doing an MRI imaging of the foot includes detecting occult fractures, marrow edema and other bone injuries that are invisible on plain radiographs [8]. It also helps in differentiating infected and non infected foot pathologies. In case of ACN, MRI shows bone marrow edema which is not restricted to one or two bones but is usually seen involving multiple bone like the entire midfoot [18]. Also, the marrow edema and enhancement are typically centered in the subchondral bone [18]. Relatively normal subcutaneous tissues with absence of ulcers and sinuses help in ruling out infections [19]. Apart from MRI, triple phase bone scan can detect ACN as they demonstrate increased uptake in early phase, blood phase and delayed phase of the scan. However, this can be seen in osteomyelitis also and hence can result in false positivity of ACN [20,21].

#### 4. Conclusions

Our study has shown that Stage 0 ACN is a medical emergency and is usually missed on plain radiography. High clinical suspicion with systematic examination helps diagnose Stage 0 ACN, with infrared thermometer being the most sensitive tool for picking up ACN with a temperature difference of  $>2^{\circ}\text{C}$  between the affected limbs being significant. Advanced imaging modalities like MRI of the foot and ankle helps in earlier detection of stage 0 ACN. Patients with Stage 0 ACN benefit from immediate immobilization and offloading to prevent further progression and deformities.

#### Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.dsx.2019.01.008>.

#### References

- [1] Aragon-Sanchez J, Lazaro-Martinez JL, Hernandez-Herrero MJ. Triggering mechanisms of neuroarthropathy following conservative surgery for osteomyelitis. *Diabet Med* 2010;27:844–7.
- [2] Johnson JTH. Neuropathic fractures and joint injuries. Pathogenesis and rationale for prevention and treatment. *J Bone Joint Surg (Am)* 1967;49-A: 1–30.
- [3] Mountziaris PM, Mikos AG. Modulation of the inflammatory response for enhanced bone tissue regeneration. *Tissue Eng B* 2008;14:179–86.
- [4] Eichenholtz SN. Charcot joints. With a foreword by Philip D. Wilson. Springfield, Illinois: Charles C. Thomas, Publisher; 1966.
- [5] Classen JN, Rolley RT, Carneiro R, Martire JR. Management of foot conditions of the diabetic patient. *Am Surg* 1976;42:81–8.
- [6] Edmonds ME, Watkins PJ. The Charcot joint: understanding its natural history leads to new treatment and prevention. *Abstract. Diabet Med* 1984;1:144A.
- [7] Shibata T, Tada K, Hashizume C. The results of arthrodesis of the ankle for leprotic neuroarthropathy. *J Bone Joint Surg (Am)* 1990;72-A: 749–56.
- [8] Moore TE, Yuh WTC, Kathol MH, El-Khoury GY, Corson JD. Abnormalities of the foot in patients with diabetes mellitus: findings on MR imaging. *AJR* 1991;157:813–6.
- [9] Sella EJ, Barrette C. Staging Charcot neuroarthropathy along the medial column of the foot in the diabetic patient. *J Foot Ankle Surg* 1999;38:34–40.
- [10] Chantelau E, Richter A, Ghassem-Zadeh N, Poll L. "Silent" stress injuries in the feet of diabetic patients with polyneuropathy—a report on 12 cases. *Arch Orthop Trauma Surg* 2007;127:171–7.
- [11] Dasgupta Riddhi, Cheema Damien, Cheema Anna, Mruthyunjaya Mahesh D, Mahata Koyeli Mary, Panwar Jyoti, Paul Thomas, Naik Dukhabandhu, Thomas Nihal. Clinical characteristics, foot-associated risk factors, offloading practices and radiological assessment in patients with type 2 diabetes mellitus and chronic charcot's neuroarthropathy: a CaseControl study from India. *Journal of Global Diabetes & Clinical Metabolism* 2017;2:13–5.
- [12] Korzon-Burakowska A, Jakóbkiewicz-Banecka J, Fiedosiuk A, et al. Osteoprotegerin gene polymorphism in diabetic Charcot neuroarthropathy. *Diabet Med* 2012;22:130–7.
- [13] Fabrin J, Larsen K, Holstein PE. Long-term followup in diabetic Charcot feet with spontaneous onset. *Diabetes Care* 2000;8:120–5.
- [14] Chisholm KA, Gilchrist JM. The Charcot joint: a modern neurologic perspective. *J Clin Neuromuscul Dis* 2011;19:214–8.
- [15] McCrory JL, Morag E, Norkitis AJ, et al. Healing of Charcot fractures: skin temperature and radiographic correlates. *Foot* 1998;8:158–65.
- [16] Petrova NL, Moniz C, Elias DA, Buxton-Thomas M, Bates M, Edmonds ME. Is there a systemic inflammatory response in the acute charcot foot? *Diabetes Care* 2007;30:997–8.
- [17] Chantelau Ernst A, Richter Andreas. The acute diabetic Charcot foot managed on the basis of magnetic resonance imaging – a review of 71 cases. *Swiss Med Wkly* 2013;143: 138–31.
- [18] Chantelau E, Grutzner G. Is the Eichenholtz classification still valid for the diabetic Charcot foot? *Swiss Med Wkly* 2014;12:139–48.
- [19] Ruotolo V, Di Pietro B, Giurato L, Masala S, Meloni M, Schillaci O, Bergamini A, Uccioli L. A new natural history of Charcot foot: clinical evolution and final outcome of stage 0 Charcot neuroarthropathy in a tertiary referral diabetic foot clinic. *Clin Nucl Med* 2013 Jul;38(7):506–9.
- [20] Tomas MB, Patel M, Marwin SE, Palestro CJ. The diabetic foot. *Br J Radiol* 2000;73:443–50.
- [21] Bilge Ergen Fatma, Eser Sanverdi Saziye, Oznur Ali. Charcot foot in diabetes and an update on imaging. *Diabet Foot Ankle* 2013;4:10–21.