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Original Article

Diagnosis and treatment of Charcot foot in patients with diabetes mellitus

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Abstract

In recent years, we have seen a significant increase in the number of works devoted to the Charcot foot. The aim was to propose and evaluate the optimal approach to diagnosing and treating Charcot foot in patients with type 2 diabetes. We treated 115 patients with type 2 diabetes and Charcot foot. Patients were separated into two groups: the TCC group (65), where it was administrated the treatment with a total contact cast (TCC) and the ORTHOSIS group, where immobilization with rigid orthoses was carried out. The TCC group showed worse results (p=0.004). In the TCC group, the frequency of recurrence of Charcot arthropathy in the next 2 months after the end of treatment was significantly higher (p=0.04). The frequency of development of Charcot arthropathy on the contralateral limb during treatment or in the coming months after it did not differ significantly between patient groups (p=0.14). X-ray in patients with Charcot foot is ineffective in establishing a clinical diagnosis and choosing treatment tactics. MRI of the foot with or without intravenous contrast should be chosen. Staged conservative treatment of Charcot foot shows a positive effect in 80.9% of observations.

Keywords: Charcot foot, diabetes, MRI, total contact cast, orthosis.

Introduction

Jean-Martin Charcot's scientific work on "arthropathies of locomotor ataxia" was first published in 1868, when he was the chief physician at the Pitié-Salpêtrière Hospital in Paris and lecturer at the Faculty of Medicine [1]. In the description of the damage to the joints, Charcot noted severe deformations, crepitation and instability with a gradual decrease in the inflammatory process over a long period. He correctly considered peripheral or central polyneuropathy to be the main factor in the pathogenesis of such a lesion. William Jordan [2] first reported the association between neuropathic osteoarthropathy (Charcot foot) and diabetes in 1936. His clinical description of foot pathology in a 56-year-old female with diabetes is a typical presentation of

the symptoms according to which this pathology is now diagnosed.

In recent years, we have seen a significant increase in the number of works devoted to the Charcot foot. The prevalence of this complication of diabetic foot syndrome is very variable: from 0.15% to 29% among patients with diabetes – who have a diagnosis of diabetic neuropathy. The actual statistics of the occurrence of Charcot foot in the population of patients with diabetes are unknown due to the rare establishment of this diagnosis. In practical medicine, this pathology is often interpreted as an abscess, gangrene of the foot, dysentery, osteomyelitis, arthritis or pathological fractures of the foot [3].

The aim is to propose and evaluate the optimal approach to diagnosing and treating patients with Charcot foot and type 2 diabetes.

Material and methods

We evaluated the treatment of 115 patients with Charcot foot (Eichenholtz-I) and type 2 diabetes. All patients were treated in the surgical department of the Municipal Non-Profit enterprise City Clinical Hospital No.1 of the Ivano-Frankivsk City Council (Ivano-Frankivsk, Ukraine) during 2017–2021. The clinical examination of the patients included the search for non-specific signs of inflammation, structural deformities of the foot, assessment of sensory and motor neuropathy and sensitivity to vibration and pressure. Attention was drawn to local hyperthermia (the difference in local temperature between the diseased and unaffected limb often exceeded 3°C). All patients underwent an X-ray of the foot in the triple projection and in the position with load, as well as an MRI of the foot and ankle joint.

Treatment of patients with Charcot foot was conservative, lasting 5–7 months, and consisted of three stages (Table 1).

In all patients, the first stage lasted 5–7 days. We treated patients according to the PRICE concept (protection, rest, ice, compression, elevation). The second stage of treatment lasted from 2 to 4 weeks. It was a TCC (total contact cast) immobilization of the foot bones and ankle joint. At the third stage of treatment (lasting 4–6 months), patients were randomly divided into two groups. TCC immobilization was preserved in 65 patients (TCC group). In 50 patients, the lower limb was immobilized with rigid orthoses of the type Rigid Ankle-Foot Walker Orthosis (RAFWO). RAFWO is an individual folding system with a rocking sole that evenly distributes plantar pressure while immobilizing the foot and ankle joints. The baseline characteristics of the patients are shown in Table 2.

We processed the material statistically using the "Statistica 10" program. We calculated the mean, standard deviation (SD), the probability of differences in the research results (p) relative to the indicators of different groups (the results were considered probable when the reliability coefficient was less than or equal to 0.05), the median of the series, quartiles, interquartile range (IQR), the criterion Pearson consistency (χ^2), odd ratio (OR), we set the confidence interval (CI) at 95%, and we defined it as ±1.96 standard errors.

Table 1: Treatment of patients with Charcot foot (n=115).

The first stage (7–9 days)	**PRICE, n=115** **Protection** – limitation of the functioning of the affected limb and the use of crutches or a cart, if the patient urgently needs to move. **Rest** – limit the static and dynamic load on foot as much as possible **Ice** – hypothermia of the foot (insulated ice or cold batteries) was performed 24–48 hours after hospitalization. Duration 15–20 minutes every 4–6 hours. **Compression** – orthopedic splint, elastic bandage or Jones compression bandage (four layers: 1 and 3 – cotton fabric; 2 and 4 – elastic bandage) **Elevation** – raise the affected limb by 15–20° when the patient is sitting or lying down.
The second stage (2–4 weeks)	**TCC (total contact cast), n=115** TCC has immobilized the bones of the foot, prevented the spread of osteoarthritis, and reduced pain and edema. The term TCC was chosen according to Figure 1.
The third stage (4–6 months)	**TCC group, n=65** TCC immobilization was continued with bandage changes every 2 weeks and control examinations of the skin and soft tissues of the foot and ankle joint **ORTHOSIS group, n=50** TCC were replaced by RAFWO.

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Table 2: Baseline characteristics of patients with Charcot foot included in the study (n=115).

Demographics of the study population and baseline parameters	Total (n=115)	TCC (n=65)	ORTHOSIS (n=50)
Male/female	78/37	45/20	33/17
Age (years), mean (SD)	67.9 (12.0)	67.6 (12.3)	68.1 (11.5)
Height (cm), mean (SD)	174.1 (13.2)	174.8 (9.9)	173.4 (14.6)
Weight (kg), mean (SD)	93.4 (21.6)	92.7 (21.5)	93.8 (22.6)
DD (years), mean (SD)	18,21 (9.40)	18.91 (9.90)	17.71 (8.90)
SBP, mean (SD)	140.2 (24.1)	140.7 (23.8)	139.9 (25.6)
DBP, mean (SD)	82.2 (13.0)	81.2 (13.6)	83.4 (12.6)
RBG (mmol/l), mean (SD)	12.0 (7.1)	11.5 (7.8)	12.6 (6.9)
HbA1c, mean (SD)	7.390 (1.880)	7.740 (1.940)	7.738 (1.780)
CRP, mean (SD)	2.770 (3.9)	2.639 (3.90)	2.891 (3.95)
PCT, mean (SD)	0.150 (0.24)	0.159 (0.22)	0.151 (0.23)
SF-I	35 (30.4%)	19 of 65 (29.2%)	16 of 50 (32.0%)
SF-II	56 (48.7%)	32 of 65 (49.2%)	24 of 50 (48.0%)
SF-III	11 (9.6%)	6 of 65 (9.2%)	5 of 50 (10.0%)
SF-IV	8 (6.9%)	5 of 65 (7.7%)	3 of 50 (6.0%)
SF-V	5 (4.3%)	3 of 65 (4.6%)	2 of 50 (4.0%)
Foot edema	115 (100.0%)	65 of 65 (100.0%)	50 of 50 (100.0%)
Warm foot	115 (100.0%)	65 of 65 (100.0%)	50 of 50 (100.0%)
Red foot	72 (62.6%)	40 of 65 (61.5%)	32 of 50 (64.0%)
Pain	73 (63.5%)	38 of 65 (58.5%)	35 of 50 (70.0%)
Neuropathy	91 (79.1%)	50 of 65 (76.9%)	41 of 50 (82.0%)
PAD	30 (26.1%)	17of 65 (26.2%)	13 of 50 (26.0%)
CS	81 (70.4%)	46 of 65 (70.8%)	35 of 50 (70.0%)
NITU	35 (30.4%)	16 of 65 (24.6%)	19 of 50 (38.0%)

Note: SD – standard deviation; DD – diabetes duration; SBP – systolic blood pressure; DBP – diastolic blood pressure; RBG – random blood glucose; HbA1c – glycosylated hemoglobin; CRP – C-reactive protein; PCT – procalcitonin; SF – Sanders and Frykberg classification [4]; PAD – peripheral artery disease; CS – compartment syndrome; NITU – non-infected tension ulcers.

Results

We confirmed the diagnosis of SF-I (damage to the metatarsophalangeal or interphalangeal joints) based on the MRI images: atrophic changes or osteolysis of the metatarsophalangeal or interphalangeal joints with typical osteoporosis of the distal parts of the metatarsal bones. We diagnosed SF-II [damage to the tarsometatarsal joint (Lisfrancjoint)] in the presence of MRI images of instability of the Lisfranc joint (deviation of the base of the second metatarsal bone in the sphenoid joint). Clinically, dorsolateral subluxations of the foot were noted in these patients. In 70.2% of observations, there was a fracture of the base of the second metatarsal bone. In 22 patients, rupture of the plantar ligaments of the metatarsal and metatarsal bone was also diagnosed.

It should be noted that among patients with SF-II in 78.9% of observations, chronic deformations of the foot with zones of increased pressure and non-infected neuropathic ulcers were observed. SF-III (Chopart joint lesion) was established if subluxation of the Chopart joint with rupture of its ligamentous apparatus was detected on MRI images. In four patients, SF-III was combined with SF-II (subluxation in the Lisfranc joint). On

MR images, patients with SF-IV (ankle involvement, including the subtalar joint and the body of the talus) had lysis of the talus with concomitant subluxation of one of the two processes forming the ankle joint. In three of these patients, the talar lysis resembled the Harris-Brand central metatarsal breakdown seen in patients with leprosy. Patients with SF-V had extra-articular calcaneus fractures on MRI.

MRI has demonstrated high sensitivity and specificity for diagnosing Charcot foot and its SF type. We obtained excellent soft tissue contrast and sensitivity to musculoskeletal abnormalities, with high resolution in multiple anatomical planes. A normal bone marrow signal reliably ruled out osteomyelitis. However, an increase in the T2-weighted image of the bone marrow might indicate early osteomyelitis or be a predictor of the appearance of osteomyelitis, even with a normal T1-weighted image. We were good at detecting the earliest signs of neuropathic osteoarthropathy, such as bone marrow edema and trabecular microtears.

Within 7–9 days of using the PRICE concept, we were able to prepare the affected limb for TCC. It was very important to achieve a reduction in foot swelling. This was important for the stability of the foot and ankle joint during TCC immobilization. In a period of 6.9±1.33 days, we obtained a good result in 101 of 115 (87.8%). We corrected blood glucose and stabilized the changes that were associated with diabetes decompensation. The average terms of regression of the main clinical signs of Charcot foot are shown in Table 3.

In 81 patients with compartment syndrome, the use of the PRICE concept ensured its reduction in the first four days in 82.7% of observations. This made it possible to avoid a fasciotomy. A fasciotomy, in any case, can be a gateway for infection.

In the future, we recommended TCC immobilization to patients with follow-up visits after 2 and 4 weeks. Regression of clinical signs of Charcot arthropathy in immobilized patients with SF-I took 12.6±1.35 days (min 10.0; max 14.0). In patients with a lesion of the Lisfranc joint (SF-II), this period was 14.5±1.27 days (min 12.0; max 16.0), and in patients with a lesion of the Chopart joint (SF-III) – 13.1±1.45 days (min 11.0; max 15.0). The term of clinical regression was significantly longer in patients with SF-IV – 20.6±3.13 days (min 17.0; max 27.0) and in patients with SF-V – 28.1±2.23 days (min 25.0; max 32.0). Based on this, we considered that the key points for foot MRI to assess further TCC immobilization in patients with SF-I, SF-II, and SF-III were 14 days of immobilization, in patients with SF-IV – 21 days, and in patients with SF-V – 28 days (Figure 1). An MRI of the foot these days revealed thickening or thinning of the ligaments, reduction of soft tissue swelling, and stabilization of the foot bones.

Regression of clinical signs of Charcot arthropathy and a positive result of MRI imaging were the key indications for the third stage of treatment. Patients in the TCC group underwent dressing changes every 2 weeks and control examinations of the skin and soft tissues. Patients of the ORTHOSIS group performed a self-examination of the limb and consulted a surgeon only in case of changes in the skin of the lower limb. The results of the treatment are shown in Table 4.

We received a positive result in 80.9%. The TCC group showed worse results than the ORTHOSIS group (OR 0.15, 95%, CI 0.04–0.56, p=0.004). In the TCC group, there was a significantly higher frequency of recurrence of Charcot arthropathy in the next two months after the end of treatment (OR 5.43, 95%, CI 1.16–25.53, p=0.04). The appearance of Charcot arthropathy on the contralateral limb during immobilization or in the coming months after the end of treatment did not significantly differ (OR 5.91, 95%, CI 0.70–49.74, p=0.14), but was higher in the TCC group – seven cases *vs.* one in ORTHOSIS group.

Better healing of non-infected neuropathic ulcers was noted in the ORTHOSIS group – 94.7%. In the TCC group, this indicator was probably lower – 62.5% (OR 0.09, 95%, CI 0.01–0.88, p=0.05).

Table 3: Regression of clinical signs of Charcot's foot when using PRICE tactics (days).

Clinical sign	PR	Mean (SD)	Median (IQR)	Min–Max
FE, n=115	101 of 115 (87.8%)	6.9 (1.33)	7.0 (2.0)	5.0–9.0
WF, n=115	43 of 115 (37.4%)	7.9 (0.05)	8.0 (1.0)	5.0–9.0
RF, n=72	66 of 72 (91.7%)	6.6 (0.83)	7.0 (5.0)	4.0–9.0
P, n=73	73 of 73 (100.0%)	4.2 (0.51)	4.0 (3.0)	3.0–6.0
CS, n=81	67 of 81 (82.7%)	2.5 (0.70)	3.0 (3.0)	1.0–4.0

Note: PR – positive result; SD – standard deviation; IQR – interquartile range; FE – foot edema; WF – warm foot, RF – red foot; P – pain; CS – compartment syndrome.

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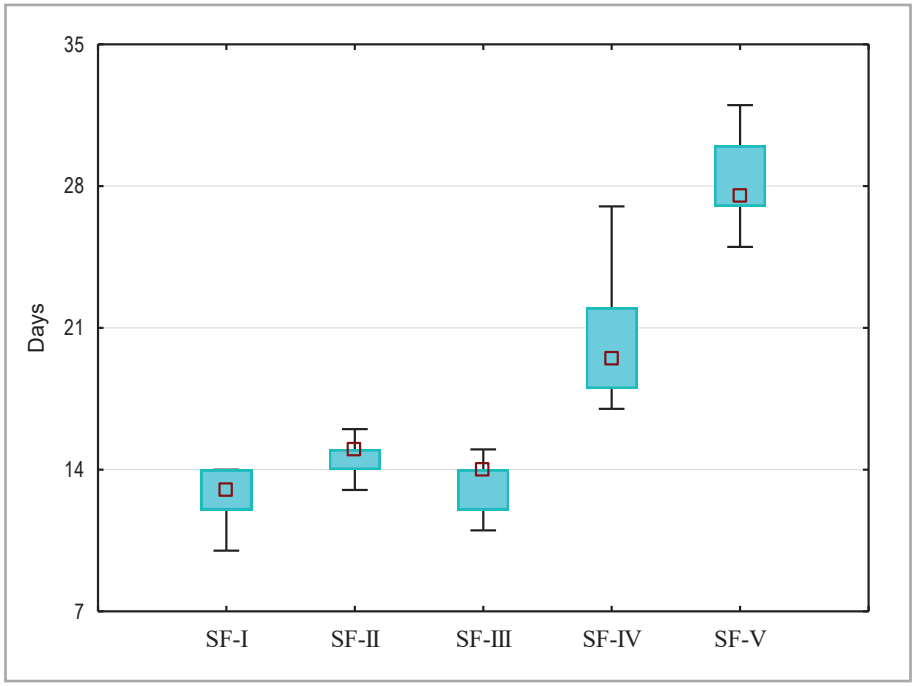

Figure 1: Terms of complete elimination of clinical signs during TCC immobilization in patients with Charcot foot (n=115) depending on Sanders and Frykberg (SF).

Discussion

Our current understanding of neuropathic arthropathy is based on a monograph published in 1966 by S.Eichenholtz. He summarized the available literature, clinical, radiographic and pathological data in 68 patients and proposed three stages of the course of Charcot foot: I – the stage of development or initiation of the process, II – the stage of deformation and III – the stage of reconstruction. The biggest debates about the effectiveness of diagnosis and the choice of a treatment option arise in relation to patients who have a stage of development (initiation of the process). Patients with Eichenholtz-II and Eichenholtz-III do not have such

Table 4: Results of patient treatment.

Result	Total (n=115)	TCC (n=65)	ORTHOSIS (n=50)	χ^2	OR (95%) (CI) p
PR	93 (80.9%)	46 of 65 (70.8%)	47 of 50 (94.0%)	8.41	0.15 (0.04–0.56) 0.004
RCA	14 (12.2%)	12 of 65 (18.5%)	2 of 50 (4.0%)	4.26	5.43 (1.16–25.53) 0.04
CACL	8 (6.9%)	7 of 65 (10.8%)	1 of 50 (2.0%)	2.14	5.91 (0.70–49.74) 0.14
RNITU (total)	28 (29.6%)	10 of 65 (38.5%)	18 of 50 (18.0%)	5.55	0.32 (0.13–0.79) 0.02
RNITU (n=35)	28 of 35 (80.0%)	10 of 16 (62.5%)	18 of 19 (94.7%)	3.81	0.09 (0.01–0.88) 0.05

Note: PR – positive result; RCA – recurrence of Charcot arthropathy; CACL – Charcot arthropathy on the contralateral limb; RNITU – regression of non-infected tension ulcers; χ^2 – Pearson's chi-squared test; OR – odds ratio; CI – confidence interval; p – p-level (significant level).

acute clinical symptoms. Their blood sugar is corrected, and they are recommended to visit an orthopedist in order to detect neuropathic non-infected ulcers in areas of increased pressure on the foot in time.

Typical clinical manifestations of acute Charcot foot (Eichenholtz-I) are asymmetric edema, hyperthermia and hyperemia of the foot, which are always accompanied by motor diabetic neuropathy. Patients do not always have a pronounced pain syndrome due to sensory neuropathy. Sensory and autonomic diabetic neuropathy are much less important in the development of Charcot foot, however, they certainly affect the clinical course of the pathology and the appearance of infectious complications [3, 4]. It should be noted that the acute form of Charcot's foot is an aseptic destruction of the bones and joints of the foot. A significant proportion of these patients do not have skin defects that could be a gateway for infection. The appearance of acute Charcot foot always occurs without any well-defined trigger factors. Violation of normal biomechanics of the foot and load distribution on the arch of the foot, even with a minor injury, activates the inflammatory cascade and increased expression of pro-inflammatory cytokines [5, 6].

X-rays of the foot in patients may reveal multiple areas of osteolysis, osteoporosis, osteosclerosis, hyperostosis, periosteal reaction, and articular lesions. This often leads to the fact that a specialist in radiation diagnostics formulates a false conclusion of osteomyelitis [7, 8]. Radiography should be considered a method with low diagnostic value for diagnosing Charcot foot. In patients, only MRI results should be taken into account. However, currently, no relevant scientific recommendations would confirm the use of MRI (with/without intravenous contrast) as an initial screening examination of patients with diabetes and suspected osteomyelitis of the foot or Charcot foot [6].

X-rays is usually appropriate as the initial imaging examination for suspected Charcot foot. This affordable method allows you to diagnose the appearance of problems with the joints and bones of the foot in patients with diabetes. However, this method is weakly effective for establishing the correct clinical diagnosis and choosing a treatment method [7, 8]. MRI with/without intravenous contrast should be the mandatory imaging modality in patients with foot edema and suspected osteomyelitis or Charcot foot. MRI differentiates these pathological conditions well and can be important for choosing surgical tactics. Our position does not contradict current clinical guidelines, which discuss the diagnosis and treatment of Charcot foot [8].

When establishing a diagnosis and treatment plan for Charcot foot in a patient with diabetes, the surgeon must solve many tasks, among which the most important is reducing the risk of infection and infectious osteomyelitis. Any invasive procedures with the foot should be treated very carefully. In most patients, the compartment syndrome regresses when the PRICE concept is applied. They do not require a fasciotomy. Early unloading of the affected foot has always been the main measure of treatment until the inflammation is eliminated and the fractures heal [9, 10].

The PRICE concept creates favorable conditions for further immobilization of TCC. It is recommended to keep such a bandage from 4 to 6 months, with a regular change of the bandage once every two weeks. Such terms of TCC changes are associated with a high risk of skin ulcers in the area of the foot, ankle joint, and lower third of the leg.

Long-term immobilization of the TCC increases the risk of Charcot foot on the contralateral limb due to redistribution of the load on it, and active rehabilitation after the termination of immobilization often leads to fresh injuries of the foot. Despite the fact that arthrodesis with/without external fixation can be recommended for patients with Charcot foot, which does not have the disadvantages of TCC immobilization, in practical medicine, surgical correction of the foot is used infrequently, due to contraindications: decompensation of diabetes, unsatisfactory skin condition, bone porosity, excessive body weight etc [11–15].

Staged non-surgical treatment of patients with Charcot foot [PRICE concept (5–7 days), TCC immobilization (14–28 days) and RAFWO immobilization (6–8 months)] is an alternative method that has a positive result in 80.9%. Certainly, immobilization by any method is effective in patients with Charcot foot. However, in the TCC group, patients noted inconveniences associated with regular visits (every 2 weeks) to the surgeon for clinical examination and dressing replacement. Patients in the ORTHOSIS group were mobile and independent of regular visits to the doctor. The use of RAFWO showed better results, especially in the regression of non-infected neuropathic foot ulcers that occurred in areas of increased pressure.

Conclusions

Charcot's foot is a dangerous complication of diabetic foot syndrome in patients with peripheral motor neuropathy. This is a progressive and destructive

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process, which is characterized by the destruction of the joints of the foot and leads to its deformation. Early diagnosis and early treatment allows achieving an effect, however, the process is irreversible, and modern diagnostic and treatment guidelines recommend variable treatment without specific and evidence-based recommendations for drug therapy. X-ray in patients with Charcot's foot is ineffective in establishing a clinical diagnosis and choosing treatment tactics. MRI of the foot with/without intravenous contrast should b e chosen. Staged conservative treatment of Charcot foot shows a positive effect in 80.9% of observations. The TCC group had worse results than the ORTHOSIS group (OR 0.15, 95%, CI 0.04–0.56, p=0.004), with a significantly higher probability of recurrence of Charcot arthropathy in the next two months after treatment (OR 5.43, 95%, CI 1.16–25.53, p=0.04) and a lower probability of healing of non-infected neuropathic ulcers (OR 0.09, 95%, CI 0.01–0.88, p=0.05).

Conflict of interest

The authors declare no conflict of interest.

Consent to participate

Written informed consent was obtained from the patients.

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