

Charcot's Neuroarthropathy in Type 2 Diabetes Mellitus Patient

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Abstract: Charcot neuroarthropathy (CN), also known as Charcot foot, is a devastating complication to diabetes. It manifests as an acute aseptic inflammation of bones and joints in the feet. If not diagnosed and treated in time, it may lead to collapse of bones in the foot. Management of Charcot foot is based on the acuteness of symptoms, anatomic location and degree of joint destruction. The management include pharmacologic and surgical treatment. The best treatment strategy for CN should be its identification and our focus should be on the prevention of its progression to an unstable foot deformity predisposing patients to ulcerations, infections, and amputations. As providers, our lack of education surrounding this subject is causing us to miss the best opportunity to have a profound impact on our patient's lives. We report a female, 50 years old with Charcot foot with secondary infection and type 2 DM presented with wound of his right foot since 2 weeks ago. History of DM since 8 years ago and was being treated with insulin. Education and our own self assessment should guide us to improve our awareness of the condition, our diagnostic accuracy and knowledge. As a result of this paradigmatic shift, we believe that placing a greater emphasis on education providers about CN would save limbs and lives.

Keywords: Charcot neuroarthropathy, Secondary infection, Diabetes mellitus

1. Introduction

Diabetes mellitus (DM) is a group of metabolic diseases characterized by hyperglycemia that occurs due to defects in insulin secretion, insulin action or both. Diabetes mellitus (DM) is currently a global health threat. Various epidemiological studies show a tendency to increase the incidence and prevalence of type 2 DM in various parts of the world. The WHO organization predicts a large increase in the number of type 2 DM patients in the coming years. The World Health Organization (WHO) predicts an increase in the number of type 2 DM patients in Indonesia from 8.4 million in 2000 to around 21.3 million in 2030. The RISKESDAS2018 data explains that the national prevalence of DM is 8.5 percent or around 20.4 million Indonesian people diagnosed with DM. DM patients also often experience serious acute and chronic complications and can cause death.¹

Charcot neuroarthropathy was first introduced by Jean-Martin Charcot in 1868 as a complication of syphilis. In 1936, neuroarthropathy began to be associated with DM. Currently, DM is the leading cause of CN. CN of the foot is an inflammatory condition that affects the periarticular soft tissues and bones in people with peripheral neuropathy, resulting in subluxation, dislocation and bone fracture, if the lower extremity is not immobilized. One in four cases of acute Charcot foot is frequently misdiagnosed, most commonly as cellulitis, gout, deep vein thrombosis or sprains that delay diagnosis by up to seven months. Without prompt and appropriate treatment, CN condition often results in foot deformity (ie, rocker-bottom foot) and increases the risk of lower extremity amputation by 15 to 40-fold. Comprehensive management of patients with Charcot foot is very important because it can prevent the progression of the deformity that has occurred and improve quality of life.² ³This case report is intended to provide information

about making a proper early diagnosis of Charcot foot and comprehensive management.

2. Cases

The female patient, Mrs. WA, aged 60 years, came to the ER on December 15, 2021 with the main complaint was a wound on the back of the right leg since 2 weeks ago, the size of the wound with a diameter of about 2 cm, accompanied by pus discharge and getting worse since 5 days ago. Pain in the leg wound felt dull and continuous but did not interfere with the patient's activities. The patient also complained of swelling on the back of the right leg since 2 weeks ago, accompanied by redness and feels hot when held. Initially the patient only felt discomfort on the back of the left leg in the form of a warm feeling, slightly swollen and without being noticed by the patient, over time a wound appeared which was getting bigger and bigger with pus. History of trauma to the foot was denied.

The patient has a history of DM since 8 years ago, early after being diagnosed with type 2 DM, the patient used oral anti-diabetic drugs. After 3 years of using oral anti-diabetic drugs, the patient's treatment was replaced with insulin obtained from the Regional Hospital, the insulin used was Glargine 18 units subcutaneous every 24 hours and insulin Aspart 16 units subcutaneous before meals. Then in the last 1 year the patient used a premix insulin, namely Degludec-Aspart with a dose of 20 units subcutaneously every 24 hours, but since 5 months ago the patient has not routinely controlled and has not used insulin. He denied history of high blood pressure and heart disease. The patient's parents and siblings also suffer from type 2 DM but do not take medication. The patient previously worked as a food seller in the market.

On physical examination, the patient's consciousness was composmentis, general condition was moderately ill, GCS

E4V5M6 with blood pressure 110/70 mmHg, pulse rate 90 times per minute, respiratory rate 20 times per minute, temperature 36.5°C, oxygen saturation 97% room water and VAS 3 lower limb area. The patient's weight was 55 kilograms and the height was 165 cm with a Body Mass Index (BMI) of 20.37 kg/m². General status of the head, neck found no abnormalities, no enlarged lymph nodes, JVP \pm 2 cmH₂O. Examination of the thorax and abdomen was found to be normal. The local status of the examination of the right foot, on inspection it looks swollen, there is no maceration between the toes, the nails appear thickened but there is no discoloration, on the back of the right foot a bulla with a size of 5x3x1cm is found with pus filled. The skin on the feet looks dry and there are no scars on other parts of the feet. There is a rocker bottom on the right leg.

Complete blood count, white blood cells 18.9 x10³/ μ L, hemoglobin 11.7 g/dL and platelets 241 x10³/ μ L, hematocrit 32.3%, absolute neutrophils 15.7 x10³/ μ L, absolute lymphocytes 1.81x10³/ μ L. Blood chemistry Urea N 22 mg/dL, blood creatinine 0.71 mg/dL, albumin 2.35 g/dL, sodium 133 mmol/L and potassium 4.56 mmol/L, HbA1C >12%, Blood sugar 339 g /dl. Record heart with sinus rhythm impression at a rate of 86 beats per minute. Anterior Posterior (AP) and oblique pedis X-ray, there are lytic lesions on the tarsal, proximal and distal metatarsals, digiti phalanges 1, 2, 4, 5 with soft tissue swelling, joint space metatarsophalanges (MTP) 2 narrowed, mineralization down and not visible gas gangrene with the conclusion of Charcot pedis dextra.

This patient was diagnosed with Charcot joint pedis dextra with secondary infection and type 2 DM. During treatment, the patient was given the antibiotic ceftriaxone 3x1 g (iv), Metronidazole 3x500 mg (iv), paracetamol 3x1g (iv) as painkillers, omeprazole 2x40 mg (iv), domperidone 3x10cc (po), aspart 1x12 iu (sc), glargine 3x8 iu (sc). The patient also underwent debridement in the form of a necrotomy and discharge of pus with indications of secondary infection.

3. Discussion

Charcot neuroarthropathy (CN), also known as Charcot foot, is a devastating complication to diabetes. It manifests as an acute aseptic inflammation of bones and joints in the feet. If not diagnosed and treated in time, it may lead to collapse of bones in the foot, which causes deformity, foot ulcers, amputation and death. The risk of amputation with CN is 15% but increases to 35 to 67% in patients with an associated ulcer. This condition has been classified based on clinical and radiologic findings. The typical patient who develops CN is in the 6th decade of life, has been diagnosed with DM for at least 10 years and is morbidly obese. It is estimated that 0.2 to 0.3 per 1, 000 diabetic patients develop CN. The destructive process triggered by CN has a profound and negative impact on health, with severe repercussions on the quality of life of the patient, particularly with regard to physical activity and lower-limb function. Risk factors for CN include DM greater than 10 years, elevated haemoglobin A1c, trauma and obesity. CN is an independent risk factor for lower extremity amputations. The pathophysiology of CN is not entirely known. In 1868 Jean-Martin Charcot was the first to describe Charcot foot as a late sequela of tertiary

syphilis, but it was not described in diabetic patients until almost 70 years later. The two basic theories of its etiology are neurotraumatic and neurovascular. In the neurotraumatic theory, some form of trauma (acute, subacute or cumulative and repetitive) in the neuropathic foot initiates a cascade of inflammation. This then leads to intense osteoclastic activity and joint destruction. In the neurovascular theory, autonomic neuroarthropathy results in vasodilation and increased blood flow. This causes congestion in the venous system and ischemia to the ligaments and tendons, leading to joint instability. This increased blood flow also increases osteoclastic activity. If the patient continues to walk and the process goes unchecked, it results in destruction of the susceptible joint of the ankle or foot. Although diabetes is the major cause, any patients with peripheral neuroarthropathy can develop CN.^{4,5,6,7}

The diagnosis is made based on anamnesis, clinical features, laboratory and radiographic examination. Complaints of thick, tingling, burning sensation may be absent due to loss of sensation. There is usually no history of trauma. The patient may feel pain. Pain is often felt lighter and not proportional to the severity of clinical symptoms. A long history of DM supports the suspicion of diabetic Charcot foot. Patients usually present with redness of the feet, warm to the touch, usually accompanied by foot deformities, such as a falling or descending arch, ankle equinus or walking in an inversion or eversion position of the foot. Suspicion of diabetic Charcot foot, especially if the temperature is increased >2°C compared to the contralateral leg, without open wound or lymphangitis. The examination consisted of patient history, neurological evaluation (achilles reflex), sensory, motor, vascular (dorsal pedis and posterior tibial arteries) and musculoskeletal examinations. In diabetic Charcot foot, circulation is usually adequate. Autonomic neuropathy alters regulation that increases blood flow and forms arteriovenous shunting, resulting in hyperaemia. Autonomic neuropathy also causes glandular dysfunction which causes the skin of the feet to become dry, less flexible and more prone to injury. Motoric evaluation is performed on the intrinsic muscles of the feet which often experience atrophy. Examination includes foot deformity, decreased joint range of motion, difference in leg length, whether there was a previous amputation and evaluation of walking. On evaluation of walking can be found pressure abnormalities in the foot due to deformity that can trigger ulcers and an increased risk of falling. Proprioceptive impairment also causes walking on a wide base and a tendency to look at the floor. Radiographs are the recommended initial imaging study to be done. The characteristic bony changes of CN can take weeks to see on plain X-rays and therefore are not useful for diagnosing CN in the early stage when clinical intervention is critical. It is helpful to take bilateral X-rays to pick up subtle changes in the bone. Patients with peripheral vascular disease are somewhat protected from CN as vasodilation is part of the pathogenesis. Joints are the weak link in the structure of the foot and therefore more susceptible. The midfoot is most often affected as it is subjected to more force during the phases of walking. This is the classic "rocker bottom" deformity. However, any joint of the foot can be affected. Hyperglycemia causes increased risk of ligament and tendon weakening. Patients with diabetes often have lower bone mineral densities, a factor for

development of CN. Munson et al used a big data approach to identify 710 associations of different conditions with CN. In addition, Munson et al discovered that 111 of these medical conditions have direct temporal associations with the development of CN. Not unsurprisingly, the strongest associations to develop CN occurred when those patients had endocrine disorders, namely DM and neurotrophic disorders, which lead to local sensory loss and selective sympathetic denervation. Thus, any patient with endocrine disorders should ultimately be suspect to the possibility of a CN event.^{2,5,7}

In our case, a 50-year-old woman came with complaints of wound on the back of her right leg accompanied by pus, slight pain, swelling on the back of her right leg and feeling warm. The patient also had a history of type 2 DM that was not well controlled. From the physical examination on the local status examination of the right foot, the patient's feet appeared swollen, there was no maceration between the toes, the nails appeared thickened, on the back of the right foot there was an ulcer with a size of 2x2x1cm and was found to be filled with pus and hyperemia was found. The skin on the feet looks dry and there are no scars on the rest of the feet. There is a rocker bottom on the right foot. Anterior Posterior (AP) and oblique pedis X-ray, there are lytic lesions on the tarsal, proximal and distal metatarsals, digiti phalanges 1, 2, 4, 5 with soft tissue swelling, joint space metatarsophalanges (MTP) 2 narrowed, mineralization down and not gas gangrene was seen with the conclusion of Charcot pedis dextra. So based on the anamnesis, the patient's medical history, namely DM, physical examination and supporting examination in the form of X-rays, the patient was diagnosed with Charcot joint pedis dextra.

CN could be present in either the Acute or the Chronic stage. The acute stage is a pathological condition mainly affecting the midfoot. Patients exhibit discomfort, painless, red and warm swollen foot similar to cellulitis. At this stage, the physician examines the foot using an infrared thermometer detecting an increase of 2-6 °C compared to a healthy foot. The chronic stage is distinct from the acute stage. It is defined as an inactive stage since there is no significant difference in an affected foot temperature compared to a healthy foot. However, redness and inflammation subsides, it is frequently replaced by a clear presence of rocker bottom deformity due to collapse of midfoot plantar arch and midfoot prominence at the medial convexity of the foot. These deformities are due to elevated pressure are susceptible to ulceration. The most widely used classification of the Charcot foot is the Eichenholtz system which is also based on radiological features. Eichenholtz, described the three stages as (I) Buildup; (II) Conglutination; and (III) Reestablishment and reconstitution, in which he described the advancement over a period of time lasting from weeks to years. This classification is considered highly descriptive and radiologically useful, but has limited practical application. Clinically, phase 1 is an acute phase, phases 2 and 3 are fixed or reparative grades. All three phases can occur in 2-3 years, although the acute phase may last several months. Shibata and Shella described the presence of grade 0, i. e. there were no radiological changes, but there was swelling and warmth in the feet. Schon and Marks described grade 0 as the patient's risk of neuropathy

and acute ongoing injury, classifying this condition as a pre-Charcot grade.^{2,8}

In our case, on the localized status of the back of the right foot there was an ulcer with a size of 2x2x1cm and was found to be filled with pus, besides that there was also swelling on the back of the foot accompanied by hyperemia. Based on clinical examination, the patient's condition was classified as acute phase of Charcot joint.

Management of CNs based on the acuteness of symptoms, anatomic location and degree of joint destruction. If a clinician is initially unsure about the diagnosis, it is recommended that they treat the condition as CN by offloading until diagnosis confirmed or disproven. Early detection and protection are key to preventing further destruction of the foot. In the acute stage, immobilization and reduction of weight-bearing activities for eight to 12 weeks is the main stay of treatment. The gold standard for immobilization of CN is a total contact cast (TCC), but devices like a removable cast walker (RCW) are also commonly used to offload the foot. Continue immobilization until lower extremity edema and warmth resolve accompanied by evidence of fracture consolidation. In the subacute and chronic stages, recommend devices include the Charcot restrain orthotic walker (CROW) and the patellar tendon-bearing brace (PTB). In the chronic stage, custom-made shoes are indicated. Classically, surgical treatment was reserved for patients in whom the orthopedic treatment had failed. The role of and time for surgical intervention are not clear and intervention has unpredictable results. However, many salvage procedures have been described, including open reduction internal fixation (ORIF), with variable techniques and implants and an external-fixation strategy. Amputation is considered as the last resort intervention. It is performed after the presence of recurrent ulceration or the failure of arthrodesis due to the difficulty in treating the spreading infection.^{5,8,9,10}

In our case, the patient was given insulin to control blood sugar and reduce the progression of DM complications, especially here is the Charcot joint. The patient also underwent debridement in the form of a necrotomy and discharge of pus with indications of secondary infection. In the acute phase of Charcot joint, the gold standard of therapy is mobilization and non-weight bearing (NWB). Proper initiation of NWB will stop the progression of the deformity. Immobilization can be done with Total Contact Cast (TCC), below knee cast or Patellar Tendon Bearing (PTB) with patton bottom. In this case, the patient has not been consulted to the orthopedic department to determine mobilization and non-weight bearing (NWB) therapy, so the possibility of progressive deformity in the patient's foot is still high. The patient was also consulted to the surgical department to be consulted about the wound on the patient's instep and performed debridement, necrotomies and given antibiotics to minimize the risk of infection. Because the patient's Charcot joint was still acute and diagnosed for the first time, surgery in the form of arthrodesis with either internal or external fixation had not yet been performed.

4. Summary

It has been reported the case of a 50-year-old woman with a diagnosis of Charcot joint regio pedis dextra with secondary infection and type 2 DM, with symptoms of swelling of the right leg, dull pain, redness, accompanied by a "rocker bottom" appearance. The patient has a history of DM for 8 years with poor blood sugar control. On imaging examination, a picture of the right leg was obtained with the conclusion Charcot pedis dextra. Patients were given basal and prandial insulin therapy to control blood sugar levels as well as surgical debridement therapy, necrotomy to reduce the risk of infection. The best treatment strategy for CN should be its identification and our focus should be on the prevention of its progression to an unstable foot deformity predisposing patients to ulcerations, infections and amputations.

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