Neuropathic Arthropathy of a Knee Joint: A Case Report

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In the 19th century, Jean-Martin Charcot reported on arthropathies due to late syphilis neurologic complications [1]. Growing knowledge about neuropathic arthropathy or "Charcot joint" has led to the diagnosis of this condition in other diseases, such as diabetes mellitus (DM), leprosy, syringomyelia, multiple sclerosis, spinal cord trauma and tumors, acquired immunodeficiency syndrome (AIDS) or AIDS-related treatment with reverse transcriptase inhibitors, and congenital and familial neurologic disorders. In up to 30% of cases, an underlying neurologic or metabolic disorder or toxic agent is unknown [2]. Neurologic deficit and progressive sensory loss with impaired pain perception probably are responsible for the delay in the diagnosis of this condition. In the context of DM, Charcot joint is a well-known problem, especially in patients with poorly controlled hyperglycemia and diabetes-targeting complications. The majority of DM patients develop Charcot joint in the foot and ankle. Neuropathic arthropathy of the knee is a rare entity with only a few cases reported in the literature.

PATIENT DESCRIPTION

A 22-year-old female was admitted to hospital because of the gradual appearance of swelling in the left knee, accompanied by difficulty in walking. The patient had a history of uncontrolled type 1 DM beginning in early childhood, which was complicated by recurrent episodes of ketoacidosis and peripheral neuropathy without retinopathy or nephropathy, hypothyroidism, amenorrhea, and anorexia nervosa (body mass index of 16 kg/m2). Her medications consisted of Insulin injections (Glargine 12U QD, insulin short-acting 12U TID) and levothyroxine 100 mcg QD.

On admission, the patient appeared very thin. Examination of her heart, lung, and abdomen were unremarkable. There was a large skin scar above the right knee due to a burn from a heater in the past (she did not remember any pain from the burn itself). Severe sensory impairment in both lower limbs and prominent quadriceps muscle wasting were noted. Joint assessment revealed left knee swelling with effusion without local tenderness and deformity of the left ankle joint. The patient's gait was severely disturbed and unstable.

Laboratory tests revealed anemia with hemoglobin 10 g/dl; the rest of blood count, blood chemistry including creatinine, uric acid, electrolytes, thyroid stimulated hormone, C-reactive protein and urinalysis were within the normal range. Glucose levels were above 250 mg/dl. Hemoglobin A1C level was higher than the resolution of the measuring device. Anti-nuclear antibodies, rheumatoid factor, and anti-cyclic citrullinated peptide were negative. Blood cultures, tests for syphilis, AIDS, and tuberculosis were negative. Left knee aspiration revealed 10 ml of clear yellow fluid with normal viscosity and 650 leukocytes/µl. Cultures were negative for bacteria and mycobacteria. No crystals were found in the polarized light microscope.

An X-ray of the left knee revealed destruction of the articular surface of the lateral femoral condyle with a large area of subchondral bone resorption and kissing intercondylar lesions on both sides of the femoral-tibial joint [Figure 1A]. Magnetic resonance imaging (MRI) of the left knee [Figure 1B] revealed bone destruction of the lateral femoral condyle with collapse of the articular surface and lateral tibial eminence with mild bone resorption of the tibia accompanied by knee effusion.

The patient was diagnosed with DM-related neuropathic arthropathy. She was managed with non-weight bearing in a hinged knee brace without improvement and developed severe gait disturbance and disability. Several months later, computed tomography of the left knee showed further progression of the knee destruction. The patient was scheduled for surgical intervention, which was postponed according to her wish.

COMMENT

We report on an extreme case of uncontrolled DM, which is complicated by neuropathic arthropathy of the knee joint. Clinically, it appeared as knee monoarthritis without signs of systemic inflammation, infection, or autoimmune disease. The joint synovial fluid indicated non-inflammatory joint effusion. In the context of DM and prominent sensory loss (no joint pain or tenderness, unrecognized

skin burn), the possibility of neuropathic arthropathy Charcot joint was raised and further confirmed by imaging.

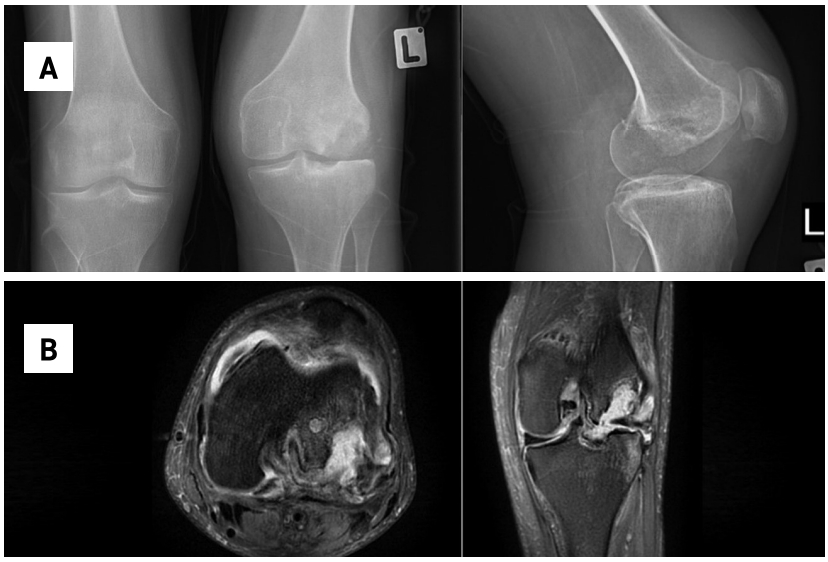
The prevalence of neuropathic arthropathy in patients with DM ranges from 0.08–13.0%. It mainly involves midfoot joints [3]. Diabetic neuropathic arthropathy rarely involves the knee. To the best of our knowledge, only 12 cases of knee joint diabetic neuropathic arthropathy have been reported worldwide. In reported cases, 91.6% were female with a median age of 29.5 years (range 25–69 years), 75% had type 1 DM with long standing disease (7–17 years from the DM diagnosis). All but one patient had poorly controlled DM before the diagnosis of Charcot knee joint. In one report, three young females (all younger than 30 years of age) had poorly controlled DM (hemoglobin A1C was above 9 mg/dl), multiple DM complications, including severe diabetic polyneuropathy. Despite successful conservative treatment of Charcot joint, two patients died before the age of 36 years [4].

The pathogenesis of neuropathic arthropathy remains controversial. Among potentially theories, the neuro-vascular and neuro-traumatic theories are more popular. The first theory suggests that increased osseous blood flow due to the loss of sympathetic innervation in combination with reduced peripheral vascular resistance induces osteoclastic activity; therefore, increasing bone resorption. The second theory suggests that the loss of somatic muscular reflexes leads to repeated micro-trauma and joint destruction. Other theories have suggested the role of local inflammation with increased production of pro-inflammatory cytokines [2,3,5].

The diagnosis of Charcot joint is based on clinical presentation and imaging. X-ray is helpful and may show bone margin deformity, subluxation, fragmentation, bone resorption and formation. X-rays may look normal in the earliest stages of Charcot joint, but later disorganized joint structural changes are quite typical. Early signs of joint damage are better visible on MRI, especially when there is a need to exclude inflammatory synovitis.

Figure 1. Imaging of the left knee

[A] Anterior-posterior and lateral X-ray of the left knee revealed destruction of the articular surface of the lateral femoral condyle with a large area of subchondral bone resorption and kissing intercondylar lesions on both sides of the tibiofemoral joint
[B] Magnetic resonance imaging of the left knee revealed bone destruction of the lateral femoral condyle with collapse of the articular surface and lateral tibia eminence with mild bone resorption of the tibia accompanied by knee effusion



The treatment of DM-related neuropathic arthropathy in general, and of the knee, in particular, is based on non-weight bearing methods, external fixation and casts, with sufficient results in some reported cases and series; in refractory cases, surgical intervention may be required [2,4,5].

CONCLUSIONS

Neuropathic arthropathy may involve the knee joint in patients with severe, long-lasting, and uncontrolled DM. It may mimic inflammatory arthritis. Concomitant diabetic neuropathy and joint instability may be a clue to the diagnosis. Imaging is specific in advanced stages of Charcot joint. A high index of suspicion regarding Charcot joint in patients with sensory deficits of any origin, particularly in complicated DM, is crucial as progressive joint destruction and disability are often a sequela. Control of DM and early recognition of diabetic neuropathic arthropathy, even in an unusual location such as the knee, are essential in the prevention of joint damage and handicap.

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