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Case Report

Corticosteroid Induced Osteoporosis Leading to Fracture Neck Femur in a Case of Type 2 Lepra Reaction

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Corticosteroids are the mainstay of treatment for type 2 lepra reaction. We report a case of 26-year-old woman with fracture neck femur and radiographic evidence of osteoporotic changes, who initially presented with swelling, redness of face and arms with intermittent fever, joint pain and malaise. She was treated for Hansen's disease, one year back, with multibacillary - multi drug therapy. She was suffering from recurrent episodes of type 2 lepra reaction and was on intermittent high doses of corticosteroids for control of type 2 lepra reaction, leading to osteoporosis and fracture neck femur. This report highlights the importance of regularly monitoring the patients on long term corticosteroids for the musculoskeletal adverse effects.

Keywords: Osteoporosis, Hansen's Disease, Erythema Nodosum Leprosum, Steroids

Introduction

Three types of lepra reactions are recognized type 1, type 2 or erythema nodosum leprosum (ENL) and the Lucio phenomenon (Kar & Chauhan 2020). Corticoteroids (CS) are the mainstay of treatment (Narang & Kamat 2020). Chronic use of systemic CS is associated with the development of osteoporosis and an increased risk of fracture (Landis & Adams 2020). Observational data shows that the risk of hip and other non-vertebral fractures are doubled in patients on CS (Cooper et al 1995). Fractures occur in as many as 50% of long term users of CS (Hayat & Magrey 2020). Fracture risk depends upon the cumulative dose and also the highest daily dose of CS. We report a case of spontaneous fracture neck femur in a patient on long term systemic steroids for type 2 lepra reaction (T2LR), which is a rare entity.

Case Report

A 26-year-old married woman, presented with swelling and redness of face (Fig 1) and arms, with intermittent fever, joint pain and malaise, for two months. Patient gave a history of similar complaints for the last two years. She was diagnosed with Hansen's disease with recurrent T2LR and had completed multibacillary - multi drug therapy (MB-MDT) for a year, one year back. In the past two years she had about 11 episodes

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of T2LR for which she had been regularly treated with tablet prednisolone and tapered according to the WHO regimen. Patient was also prescribed calcium and azathioprine, on and off. The cumulative dose of corticosteroids taken by the patient in the last 2 years was around 18 grams of prednisolone. On examination, diffuse edema and erythema of face, upper and lower limbs was present along with bilateral ear lobe infiltration. Ichthyosis of both the legs were seen. Sensory examination revealed near glove and stocking type of anaesthesia. Loss of hypothenar eminence was present. On motor examination, card test was positive with ulnar deviation of little and ring finger of both hands. Peripheral nerve examinations showed enlargement of both ulnar nerves and radial cutaneous nerves with a cystic swelling along the course of ulnar nerve over right elbow. Slit skin smear from the ear lobe showed bacilli arranged in globi (bacteriological index 6+). Diagnosis of Hansen's disease with relapse and recurrent T2LR was made. Patient was admitted and started on injection dexamethasone 8 mg/ day for 5 days along with analgesic / anti-inflammatory drugs. Patient responded well and was discharged with tab prednisolone 40 mg for 15 days and to restart MB-MDT for a further period of one year and report every 15 days.

One week later, the patient presented to outpatient with pain and swelling of the right hip joint with restricted movements for three days. There was no history suggestive of fall or trauma, new skin lesions or fever. Patient was referred to the department of orthopaedics for further evaluation. Radiograph of hip and pelvis was taken, which showed a fractured neck of the right femur (Fig 2). Diagnosis of pathological fracture secondary to osteoporosis due to long term systemic CS was made. She was admitted in the department of orthopaedics, operated with



Fig. 1 : Redness of the face associated with swelling



Fig. 2 : Radiograph of pelvis with both hip - AP view showing fracture neck femur (arrow)

cannulated cancellous screw fixation, and is under regular follow-up.

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Discussion

Lepra reactions result from changes in the immune balance between the host and *M. leprae* (Lastoria & Morgado de Abreu 2014). Type 1 lepra reactions are most typical of borderline cases, while T2LR, are systemic events that occur in borderline lepromatous and lepromatous cases and can cause damage to the nerves, eyes, and skin (Leon et al 2015). Around 50% of patients with leprosy may develop lepra reactions prior to, during or after MDT. These reactions are characterized by sudden reactivation of immune responses with high levels of cytokines.

Although a number of drugs are observed to be effective, CS are still the mainstay of treatment for lepra reactions (Quyum et al 2020). Cases of chronic recurrent T2LR often become steroiddependent, the reason for which are unclear (Schreuder & Naafs 2003). Steroid therapy is associated with many side effects like cushingoid changes, steroid acne, gastritis, fungal infection of the skin, eye changes like cataract and glaucoma, reactivation of tuberculosis, and bone changes like avascular necrosis (AVN) and osteoporosis (Sugumaran 1998).

Osteoporosis compromises bone strength and results in an increased risk of fracture. Corticosteroid induced osteoporosis (CSIO) is the most common cause of secondary osteoporosis. It is a common cause before 50 years of age and the most common iatrogenic cause of osteoporosis (Briot & Roux 2015). Osteoporosis is observed at a higher rate in the femoral and lumbar regions of leprosy patients. In many patients who complete multi-drug therapy for leprosy, the morbidity continues in the form of lepra reactions requiring repeated corticosteroid therapy. It seriously affects bone health and leads to osteoporosis. The inflammatory mediators of lepra reactions negatively affect bone remodeling. Nutritional deficiencies and immobility further increase the risk of osteoporosis. In male patients, testicular involvement results in deficiency of testosterone, which perpetuates osteoporosis. In spite of having so many predilections, osteoporosis in steroid-treated leprosy reaction patients has not been well explored (Quyum et al 2020).

Osteoporotic changes can start as early as two months with a dose of 20 mg of CS (Ponnuswami et al 2020). Though our patient was given tab. azathioprine, as a steroid sparing drug, it did not lead to suppression of T2LR. Because of the lack of availability of thalidomide and clofazimine, patient could not be started on these drugs. Thus, the patient had to be kept on systemic CS for a longer duration to suppress the reaction. Subsequently, our patient developed a fracture neck of the femur secondary to CSIO. Although fracture neck of femur and AVN of head of femur have been reported more in conditions like pemphigus and discoid lupus erythematosus, where a high dose of corticosteroids are used, it is rarely described in T2LR. To avoid these side effects, it becomes necessary to educate the patient against taking a high dose of corticosteroids for a long duration on their own. Our patient was treated with injection zoledronate (bisphosphonates) after the surgery. Awareness of this side effect helps in early recognition and suitable management in these patients.

Conclusion

Based on our experience, it may be concluded that lepra reaction cases on long-term corticosteroids may be at a higher risk of developing a vascular necrosis of head of femur and fracture neck femur. Treating clinicians should keep this complication in mind when managing such cases.

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