Case Report

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Avascular Necrosis of Femoral Head in Sheehan's Syndrome

Suman Kumar Kotwal*, J.B. Singh*, Annil Mahajan**

Author's Affiliation:
*Consultant, **Professor, Department of Medicine, Government Medical College, Jammu, Jammu and Kashmir

Corresponding Author:
Suman Kumar Kotwal,
H.NO. 55/1, sharika Vihar , Roop Nagar Jammu.
Pin 180013.
E-mail:
sumankk1230@rediffmail.com.

Abstract

Avascular necrosis is clinically characterized by gradual onset of pain in motion and relieved by rest in affected joint. The pathophysiology of avascular necrosis has not been completely elucidated; whereas some cases of disease clearly have direct cause, the pathophysiology is uncertain for most cases. Avascular necrosis in Sheehan's syndrome has not been described in published literature so for and may be due to disease state itself or to chronic corticosteroid therapy. We report a case of Sheehan's syndrome developing avascular necrosis of femoral head. A 42 year women had lactational failure in last pregnancy followed by secondary amenorrhea and other features of hypopituitarism. On clinical and biochemical evaluation she was diagnosed as a case of Sheehan's syndrome and was put on replacement therapy. After 15 years on replacement therapy she started getting pain on left hip with limp on same side and she was diagnosed having avascular necrosis of left femoral head and she was subjected to decompression and now following orthopedician.

Keywords: Sheehan's Syndrome; Avascular Necrosis.

Introduction

Avascular necrosis is clinically characterized by gradual onset of pain in motion and relieved by rest in affected joint with radiation down the affected limb, at times leading to muscle spasm. Radiologically the picture is variable depending upon the stage of the disease, but wedge shaped area of increased radiopacity with the base adjacent to articular cartilage and the apex pointing to head of involved bone. Head of the femur has minimum blood supply with few anastomoses predisposing it to avascular necrosis. Necrosis appears as mottled area and the fibrous zone as a radiolucent band with demineralization of uninvolved bone [1]. The pathophysiology of avascular necrosis (osteonecrosis) has not been completely elucidated; whereas some cases of disease clearly have direct cause (trauma, radiations or caissons disease etc), and the pathophysiology is uncertain for most cases. Multiple investigators have postulated vascular impairment, altered cell physiology etc. [2,3]. Various other mechanisms have been put forth in respect of etiopathogensis of this crippling sideffect, namely increase intraosseus pressure resulting lipocyte hypertrophy and derangements in fatty metabolism [4,5]. How avascular necrosis occurs in Sheehan's syndrome has not been studied extensively, but may be ascribed to reduced bone mineral density, bone mineral content, reduced muscle mass, and altered lipid profile due to growth hormone deficiency. The data on avasular necrosis in Sheehan's syndrome is limited in published literature. Here we report a patient of Sheehan' syndrome with avascular necrosis of left femoral head.

Case Report

A 42 years female gives history of passing blood clots per vagina a day after her last delivery which was conducted at home by local nurse twenty years back. She had history of shock, which required hospitalization and blood transfusions. She also had history of lactational failure in same pregnancy followed by secondary amenorrhea. Her examination

revealed puffiness of face ,cold, shallow and dry skin, pulse of 64 beats per minute, supine blood pressure of 100/70 mmhg. Her pubic and axillary hair was absent and she had atrophic breasts and hung up deep tendon reflexes. Her fundus and fields of vision were normal. In view of above signs and symptoms she was investigated for possible Sheehan's syndrome. Her investigation revealed hemoglobin of 10gm/dl, normal fasting blood glucose and electrolytes. Her hormonal profile showed low T3 and T4 with inappropriately normal TSH levels. Her basal cortisol, prolactin, human growth hormone and FSH and LH levels were low. She was then subjected to insulin tolerance testing, which showed inadequate response for cortisol, prolactin, and human growth hormone and MRI pituitary was showing empty sella (Figure 2). A diagnosis of Sheehan's syndrome was made on the basis of clinical features, hormonal evaluation and imaging. She was put on 100 µgm of levothyroxine, 5mg of prednisolone and estrogen and progesterone containing oral pills. She has been taking all of these medications as advised all along over these fifteen years. Dose variation in levothyroxine was 100-150µgm and 5- 7.5mg in prednisolone over these years. She had increased her prednisolone dose to 10mg only during minor illness for three or four days, three or less occasions per year. She has not received injectable corticosteroid during these years. She started with pain hip region radiating down left limb and had difficulty in walking from last one year or so. On examination she was having limp on left side and there was tenderness over left groin region. Imaging was done which revealed features of avascular necrosis of left femoral head (Figure1). Decompression of left hip was done and now she is following orthopedician.

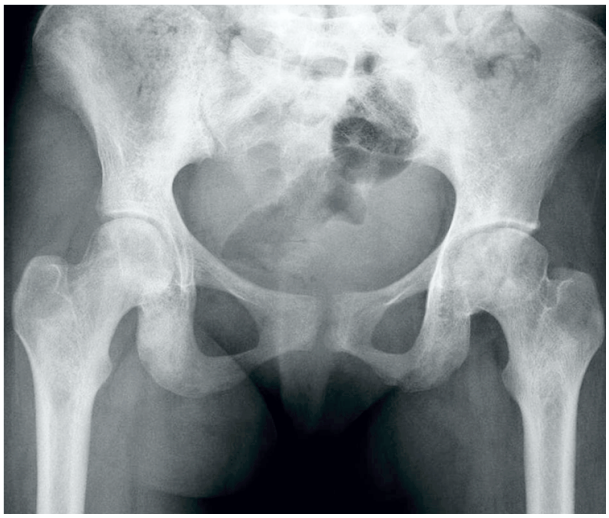

Fig. 1: X-ray of HIP showing avascular necrosis of left femoral head

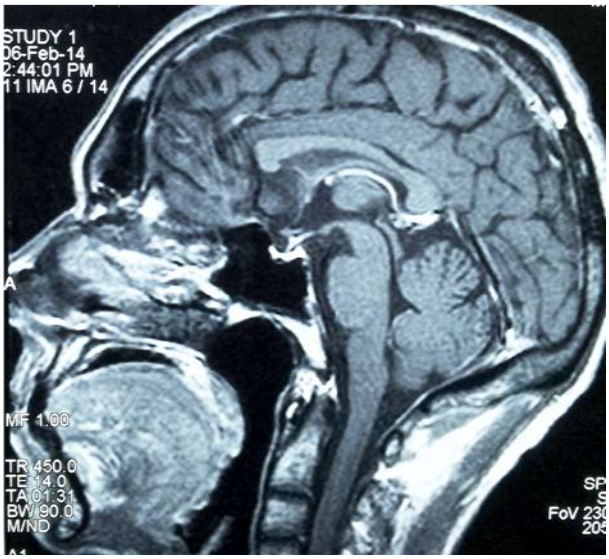

Fig. 2: T1W MRI showing empty sella

Discussion

Avascular necrosis of bone occurs in a wide variety of conditions, its association in some cases with corticosteroids therapy was first described by pietrograude, frieber and colleagues [6].

Nontraumatic avascular necrosis of femoral head occurs in young adults and is frequently progressive. In some cases however disease is static or only progresses very slowly. Clinically symptoms may then remain slight over a long period of time [7].

Assouline Bayan V, Chang C, Greenspan A et al. in their study on pathogenesis and natural history of osteonecrosis described that, the vast majority of cases are secondary to trauma, however for non traumatic cases there remain diagnostic challenge in defining the cause of bone death. Early detection is important because prognosis depends upon stage and location of lesion. The disease should be suspected in patients with history of steroid usage especially with other illnesses that predispose the patient to osteonecrosis [8].

Growth hormone deficiency in adults is associated with abnormal body composition, altered lipid profile, reduced quality of life and osteoporosis. F. Kelestimur, F.tanriverdi, F.Bayram in one of their study on Sheehan's syndrome, which is characterized by severe and long standing growth hormone deficiency demonstrated that growth hormone replacement therapy have beneficial effects in several parameters including lipid profile and waist circumference [9].

Review of literature of avascular necrosis in Sheehan's syndrome gave insufficient data on actual

prevalence of avascular necrosis in such patients and disease specific risk factor for developing the same.

Since patients of Sheehan's syndrome have chronic growth hormone deficiency and other disease specific risk factors for developing avascular necrosis, we therefore like to recommend baseline screening of hip joints at diagnosis and then at regular intervals to screen the patients for any asymptomatic avascular necrosis and early treatment.

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