Avascular Necrosis of Both Femoral Heads Following Short-term High-dose Steroid Treatment for Acute Severe Asthma – A Rare Presentation: Case Report and Review of Literature

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ABSTRACT

A 50-year-old patient who had acute severe asthma presented with avascular necrosis (AVN) of both femoral heads 6 months after short-term high-dose steroid therapy. The use of steroids on a long-term basis can cause AVN of the bone which also occurs in various diseases. A variety of traumatic and nontraumatic factors contribute to the etiology of AVN although long-term exogenous steroid administration and alcoholism are among the most common nontraumatic causes. AVN of both femoral heads presenting after short-term high-dose steroid treatment for acute severe asthma is rare in medical literature; thus, prompting the present case report.

Key words: Acute severe asthma; avascular necrosis; femoral heads; status asthmaticus; steroid

Introduction

Steroid therapy, irrespective of the mode of administration, has its own attendant risks of side effects. AVN of the femoral head is one of the universally recognized side effects of steroid therapy.[1] The etiology of AVN is multifactorial and ranges from traumatic causes namely fracture of the neck of femur,[2,3] traumatic dislocation of hip,[4] slipped femoral capital epiphysis,[5] and osteotomy,[6] to nontraumatic causes such as sickle cell anemia,[7] alcoholism,[8] autoimmune connective tissue disorders which include rheumatoid arthritis, lupus erythematosus, as well as pancreatitis, occlusive vascular disease, pheochromocytoma, infections, radiation, Gaucher's disease, renal transplantation, Cassion's disease, and pregnancy.[6]

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Avascular necrosis (AVN) is clinically characterized by a gradual onset of pain in motion, relieved by rest in the affected joint with radiation down the affected limb; at times leading to muscle spasms.[1] A slight limp, often unilateral with limitation of motion to a variable degree is characteristic. The patient may have an abductor lurch and rotation, with limitation of abduction and adduction.[1] There may be associated atrophy of the proximal muscles.[1] The head of the femur has minimum blood supply with few anastomoses leading to a wedge-shaped area of AVN.[1] Histologically, the involved bone has three zones: Necrotic, granulomatous, and a variable zone.[1] Radiologically, the picture is variable depending on the stage of the disease, but a wedge-shaped area of increased radiopacity with the base adjacent to the articular cartilage and the apex points to head of the involved bone.[1] Necrosis appears as a mottled area and the fibrous zone as a radiolucent band with demineralization of the uninvolved bone.[9]

Various etiopathogenetic mechanisms have been suggested, namely increase in the intraosseous pressure resulting from lipocyte hypertrophy and derangements in fatty metabolism causing deposition of fat in the marrow spaces of the skeleton in patients who were treated with steroids, particularly

in individuals who underwent short-term treatment with high-dose steroid.[10]

Reports of AVN occurring in patients with acute severe asthma following treatment with steroids are very rare. We report a case of AVN of both femoral heads in a high ranking army officer who presented 6 months after treatment with short-term high-dose steroid therapy for acute severe asthma.

Case Report

A 50-year-old, married man who was a high ranking officer in Nigerian army presented with 2-week history of gradual pain in both hips on motion, relieved by rest in the affected joints, with radiation down the affected limbs which at times led to muscle spasms. A slight bilateral limp with limitation of motion was also noted by the patient. There was no history of trauma. The patient neither smoked cigarette nor indulged in alcohol intake. The systemic review was essentially normal.

On physical examination, the patient was not chronically ill looking and was not in respiratory distress. He was afebrile, anicteric, and not pale. Musculoskeletal system examination revealed the patient had abductor lurch and rotation, with limitation of abduction and adduction in both hips. His pulse was 90 beats per min, while the rest of cardiovascular as well as neurological examinations were unremarkable.

However, patient reported he was treated for acute severe asthma 6 months before the onset of the complaints. The details of his treatment retrieved from his attending physician revealed that during the episode he received the following medications; intravenous methylprednisolone 125 mg stat, and albuterol given as handheld nebulizer at a dose of 2.5 mg on presentation to the emergency department. He was subsequently transferred to male medical ward and was placed on maintenance dose of oral prednisone 60 mg q6h to be taken for 9 days. The cumulative dose of prednisone (steroid) received by the patient in 10 days was approximately 2.285 g. He was discharged home on the 7th day of admission for follow-up at medical outpatient department.

The plain radiograph of the hip appeared normal. While coronal T1-weighted and T2-weighted magnetic resonance (MR) images of the hip showed a serpiginous zone of low signal around the avascular areas. The areas of infarcted bone were surrounded by fluid. There was also effusion in the joints [Figures 1 and 2]. However, no collapse of both femoral heads was noted.

Orthopedic evaluation described it as AVN of both femoral heads stage II. Patient was advised bed rest, nonsteroidal anti-inflammatory drugs, and core decompression. However, he opted to travel outside the country for treatment and was lost to follow-up.

Discussion

AVN has long been recognized as a complication of systemic steroid use and was initially believed to occur only in patients who received high doses (equivalent to more 4 g of prednisolone) for extended periods of 3 months or longer.[11,12] Previous sporadic case reports have described patients in whom AVN developed following relatively brief courses (7 days) of high-dose or low-dose, orally administered steroid medication.[13,14] The exact mechanism by which steroids cause AVN remains unclear.[12] Current research has focused on the development of a hypercoagulable state, with subsequent impaired fibrinolysis and venous thrombosis in bone.[15] In addition, steroids cause vasculitis and marked subcortical osteoporosis which further potentiates bone collapse.[16]

In a study of a neurosurgical population on short courses of high-dose steroids, the incidence of development of AVN of the femoral head was 0.3%.[16] The study concluded that this

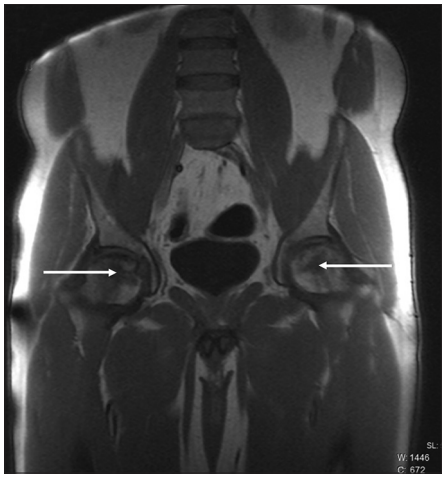

Figure 1: Serpiginous zone of low signal intensity on T1-Weighted image involving both femoral heads (arrows)

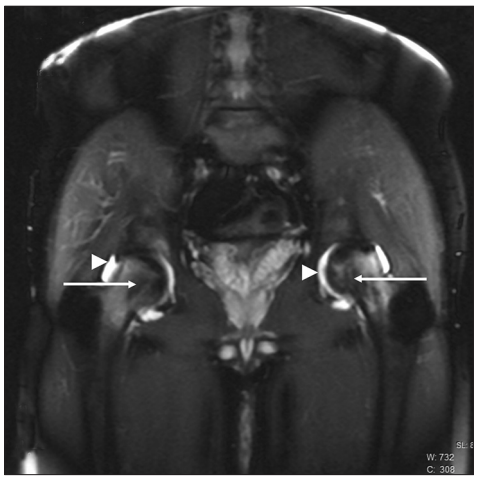

Figure 2: Serpiginous zone of high signal intensity on T2 –Weighted image involving both femoral heads (arrows). There is also bilateral joints effusion (arrow heads)

value is a significant morbidity that should influence clinical decisions on the use of steroid. Reports on AVN, some of which affected unusual or multiple locations indicate that the lesion began to appear in patients after 3-4 months of recovery from severe acute respiratory syndrome.[16] The index case presented with AVN 6 months after recovery from acute severe asthma.

There is no yet established relationship between steroid dosage and the risk of AVN, but experience with long-term steroid management indicates that the duration and dosage of steroids are factors contributing to AVN.[16] The index case had cumulative dose of about 2.285 g in 10 days and the dose was not reduced gradually but was discontinued abruptly, which may have contributed to this complication.

Anatomical changes of bone collapse and deformity may be seen on plain radiographs, computed tomography (CT) scan and magnetic resonance imaging (MRI) scans, but MRI is the most sensitive modality in the diagnosis of early disease in which plain radiographs and CT may appear normal[17] as seen in the index case. Sagittal, coronal, and axial MR images allow optimal assessment of the extent of the disease.[17] The vascular response in healing can also be assessed on MRI.[17] Radioisotope bone scan is also useful in early diagnosis and in assessing healing.[17]

The treatment of AVN depends on the stage of the disease: Stages I, II, and III are offered core decompression or free vascularized fibula grafting.[18] These first three stages are referred to as precollapse stage.[18] The index case falls in this group which was the basis for core decompression. While the treatment for stages IV and V (post-collapse stage) is total hip arthroplasty.[18]

Conclusion

When weighing the risks and benefits of the use of steroids, clinicians should be aware of the potential complications. It would be advisable to minimize both the dosage and the duration of steroid treatment where possible. Patient should be informed of the potential risk of AVN following the use of steroid medication. Complaint of hip pain in patients who have previously been exposed to steroids should lead to a high index of suspicion for underlying AVN of the femoral head. Although early treatment before collapse of the femoral head is beneficial,[18] prevention of this complication is preferable.

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