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Corticosteroid Provoked Avascular Necrosis: A complicacy concerning Systemic Lupus Erythematous

Helan Kurian[1], Jobin Kunjumon Vilapurathu[2], G.R Vijaya Sankar[3], Dhanya Paul[1], Chinnu Roy[1]

[1] Pharm D Interns, Department of Pharmacy Practice, Nirmala College of Pharmacy, Muvattupuzha, Kerala, India-686661

[2] Associate Professor, Department of Pharmacy Practice, Nirmala College of Pharmacy, Muvattupuzha, Kerala, India-686661

[3] Associate Professor, Departement of Pharmaceutical Sciences, Vinayaka Missions College of Pharmacy, Salem, Tamilnadu, India-636308

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Abstract

Avascular necrosis (AVN) is the complete death of bone, due to loss of blood flow to the bone. Corticosteroid therapy is the second most significant factor for AVN, and systemic lupus erythematosus (SLE) is the most prevalent underlying disease. Conventional management of AVN entails reduction of weight, complete bed-rest, trimming down the dose of glucocorticoid or abdicating the drug completely. A 19-year old female with a known history of SLE, on methylprednisolone for 2-years, came to the orthopedics outpatient department for pain in her right pelvis. On detailed examination, she was diagnosed with avascular necrosis. The AVN developed as a result of corticosteroid therapy because for the initial 12 months she was on methylprednisolone >20 mg/day (exceeded daily dose). The physician instructed her; complete offload and a trial of bisphosphonate plus aspirin plus statin was given and awaited for revascularization. If the revascularization fails, the physician would suggest total hip replacement.

Keywords: Avascular, corticosteroid, osteonecrosis, femoral head collapse, revascularization.

Corresponding Author
Helan Kurian
Pharm D Intern, Department of Pharmacy Practice
Nirmala College of Pharmacy, Muvattupuzha – 686661
Mobile phone: +919400316412
Email: helankurian11@gmail.com

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Introduction

Avascular bone necrosis (AVN), more kenned as aseptic necrosis of osseins, osteonecrosis, osteochondritis dissecans and ischemic bone necrosis is a malady that leads to significant morbidity, deterioration of quality of life and defacement in the daily function of SLE subjects [1].

According to the diagnostic techniques and the evaluation of symptomatic or asymptomatic subjects in Korea, the prevalence of AVN amongst SLE subjects during 2006-2010 was 31.5-34.2 per 1,000 inhabitants. However, the incidence was reduced to 8.4-9.8 per 1,000 inhabitants between 2008 and 2010 [2, 3].

AVN encompasses progressive destruction of osseins and leads to bone vasculature, loss of fat cells and osteocytes, and revision of bone structure [1]. Avascular necrosis of the femoral head (ANFH) is a disfiguring and progressive condition in young subjects, leading to collapse of femoral head and a total hip arthroplasty [4].

Currently, amongst the idiopathic pathogenic mechanisms, the vascular hypothesis is more reliable, in which local microvascular thrombosis drives to reduction in flow of blood

in the femoral head. The fact that; ANFH is also identified in twins including familial clusters asserts the involvement of ancestral factors and genetic predisposition [4].

90% of all non-traumatic causes of AVN is comprised of Corticosteroid therapy and subsequent alcoholism. Additional non-traumatic circumstances are sickle cell anemia, pregnancy, hyperparathyroidism, thalassemia, Cushing's disease, chronic renal failure, polycythemia, hemophilia, congenital hip dislocation, hypercholesterolemia, pancreatitis, radiation, autoimmune diseases, chronic, caisson disease, and use of potent intravenous Bisphosphonates [1]. The primary aid of therapy in most inflammatory conditions are Glucocorticoids (GCs). They are also present in most chemo protocols. Hence, ANFH is a significant complication for subjects on glucocorticoid therapy [4].

Habitually, there exists a counterbalance between the regenerative and degenerative molecules in the bone environment of the femoral head. The degenerative process is triggered by GC's that leads to suppression of the osteoblast and osteoclast, augment apoptosis of osteocytes and osteoblasts, and restrain the repair. *A2M,* the gene associated with the apoptosis pathway, is a potential biomarker towards the advanced warning of steroid-induced ANFH [4].

Ficat and Arlet classified the four radiographic stages of pelvis AVN as follows: normal radiographic appearance comprises of Stage I, stage II = transition phase, sequestrum with subchondral destruction occurs in Stage III, and stage IV = restrained joint space and collapse of the femoral head [5]. Traditional management involves bed rest, rebate weight-bearing via pharmacological therapy, and canes or crutches. Up-to-date, there exist no ample resources to recommend the conventional use of hyperbaric oxygen or bisphosphonates in glucocorticoid-induced AVN. The surgical approach towards AVN includes bone graft, osteotomy, core decompression and tantalum rod that may benefit to delay AVN progression. The preferred treatment for articular collapse andadvanced AVN is Arthroplasty. As subjects who sustained from glucocorticoid-induced AVN are often young, durability is a concern. [1].

Case Presentation

A 19-year-old female patient came to the orthopedic outpatient department complaining of pain in the right pelvis. Based on physical examination, the Straight-Leg Raising Test (SLRT) was negative with no spinal tenderness; pelvis Range of Motion (ROM) was painful, no solid contortion, flexion at 0-12 degree, abduction at 0-20 degree, adduction at 0-30 degree, internal rotation within 0-30 degree, and external circumrotation within 0-10 degree. She has been diagnosed with Systemic Lupus Erythematous (SLE) for two years and is on Azathioprine 50mg P/O 1/2-0-0, Hydroxychloroquine 300mg P/O 0-0-1, Methylprednisolone P/O 16 mg-0-8mg, Pantoprazole 40 mg P/O 1-0-0, Paracetamol 650mg only if pain persists. Laboratory investigations were performed and the results were as follows: Hb- 11.6 mg/dl, TC- 3720 cells/mm3, ESR- 40 mm/hr, platelet count- 2.5 lakh cells/mm3, SGOT- 36, serum creatinine- 0.8, SGPT- 30 U/L, U/L. X-ray proved no collapse of the femoral head. MRI conclusions confirmed avascular necrosis of the femoral head (ANFH), stage II B of Ficat and Arlet's classification, about 15–30% necrosis of the femoral head. From the investigations, she was diagnosed with avascular necrosis (AVN) and advised of complete offloading. A trial of Aspirin 75mg P/O 0-1-0, Alendronate 10 mg/day P/O 1-0-0, and Atorvastatin 40mg P/O 0-0-1was recommended. In addition, if revascularization failed and collapsed, then she would be advised to proceed to Total Hip Replacement (THR). The physician avoided core decompression, due to progression of stage II B. Review in orthopedics OPD if medical assistants are required.

Discussion

Subjects on long-term glucocorticoid therapy to cure SLE usually get triggered with osteonecrosis or avascular necrosis. In this case report, the patient was a known case of SLE and was on methyl-prednisolone >20mg/day initially for 12-months and then tapered into <20mg/day. Amongst the subjects with SLE reinforced that principally the patients who have surpassed daily doses of prednisone (>20 mg/day) developed AVN, while the patients who prevailed on <20 mg of the daily dose of prednisone did not develop AVN [6].

In this case, complete offloading and a trial of low-dose aspirin, bisphosphonates, and statins were given to the patient and awaited for revascularization; if revascularization fails and collapses proceed to total hip replacement. The assumption from the known anticoagulant properties of aspirin and its positive modulatory and protecting effect on the vascular endothelium suggested that aspirin given for subjects in their early to intermediate stages of non-traumatic ANFH inhibits radiographic progression may reverse the disease process for a short duration [7]. Positive short-term and long-term efficacy of alendronate therapy in adult AVN subjects, were as follows: pain reduction, the revival of articular function, slow bone collapse or progression, and lingers the requirement for arthroplasty. In addition, there were no severe adverse effects associated with alendronate therapy observed during short or long-term follow-up [8]. Reports from 284 subjects who remained on statin therapy throughout the steroid exposure; after 7.5 years with a minimum follow-up of 5 years, barely three patients (1%) develop AVN. Steroid-induced adipogenesis in the marrow may contribute to AVN. Statins may provide some shelter against the development of AVN if steroid therapy is required [9]. Moreover, Lovastatin may further hinder the maturation of steroid-induced AVN [10].

Conclusion

The use of glucocorticoid is one of the most prominent and significant causes of non-traumatic AVN, and it manifests as early as in the initial 12 months. In this case report, the patient was on methylprednisolone >20 mg/day for 12 months, which accelerated AVN. However, statin therapy during glucocorticoid treatment can halter AVN, which is evident from the research of Pritchett and James W. Judicious application of glucocorticoids such as by avoiding prolonged steroid therapy, practicing minimum effective doses, depreciating the use of methylprednisolone intravenous push, and administrating glucocorticoid sparing agents as early as possible may benefit in mitigating the risk of AVN.

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