

OSTEONECROSIS OF FEMORAL HEAD IN SLE PATIENT AFTER SHORT PERIOD OF CORTICOSTEROID THERAPY- A CASE REPORT

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Numerous cases of osteonecrosis associated with steroid therapy have been reported and most of these had received large doses for prolonged periods¹⁻⁴. Avascular necrosis of bone occurring after short term steroid treatment is, however, rare⁵⁻⁸.

Many underlying causes have been suggested as being associated with this condition⁴⁻⁶. Gregg et al believe that cases may have avascular necrosis of bone with coincidental steroid treatment for conditions themselves associated with avascular necrosis of bone, for example, Systemic Lupus Erythematosus^{1,9}. We report a case of SLE who was given short term steroid therapy and developed osteonecrosis of femoral head.

CASE REPORT

A sixteen year girl first presented with a history of continuous high grade fever, generalized arthralgia, weight loss, amenorrhoea and alopecia of six months duration. Her past history was unremarkable and she denied having taken any steroids. She was febrile, anaemic, with generalized hyperpigmentation and hepatosplenomegaly. Investigations revealed that Hb was 9 gms% and ESR 100 mm in the first hour. Urine showed proteinuria 500 mg/hrs. SLE was confirmed by a positive ANA & Anti-DNA in a titre of greater than 100. She was given methylprednisolone pulse therapy followed by tab aspirin 300mg QID and prednisolone 10 mg daily to which she responded very well. She was discharged on the same medication and was readmitted one month later for a second pulse therapy. She remained symptom free with no increase in proteinuria and was, therefore, advised to continue the same medication. Two months after this she was given a third pulse therapy. She remained stable throughout and continued on the same treatment. Three months later, i.e., seven months after her admission, she was admitted with one week's complaint of pain in her right hip and thigh aggravated by weight bearing and walking. On examination the right hip joint was tender and movements were restricted and painful. Radiographs of hip revealed avascular necrosis of right femoral head. Considering this to be due to her illness she was given another pulse therapy with no improvement in symptoms at the end of one week. She was then advised bed rest and physiotherapy and after that her symptoms improved. Subsequent radiographs showed no deterioration.

DISCUSSION

The etiology of this entity remains unknown but in 92% cases⁴ it has been found in association with either steroid therapy or any of the conditions such as SLE, sickle cell disease, Gaucher's disease, diabetes mellitus, pancreatitis, liver diseases, idiopathic hyperlipidaemia, gout, alcoholism, drug abuse, Caisson's disease, tumour infiltration and following radiation⁴⁻⁶.

Although, the occlusion of blood supply is known to occur in osteonecrosis, the pathogenesis of steroid induced avascular necrosis remains speculative. Three theories have been postulated¹⁰.

1. A hypercoagulable state associated with vasculitis causing sludging of blood cells and emboliza-

tion^{10,13}

2. Steroid induced osteoporosis resulting in trabecular fractures and compression of subchondral^{2,10}
3. Steroid induced fatty liver with a sequence of hyperlipidaemia, fat micro-emboli and bone infarction^{10-14,16,17,18}.

There is no substantial evidence to support the first two hypotheses^{12,13} but the last one has much experimental support^{10,17}. Abnormal serum lipids have been found in majority of patients with non-traumatic osteonecrosis on long term steroid therapy¹⁴ and similar alterations in lipid metabolism have been observed in work on animal models^{10,17}. A steroid induced increase in diameter of marrow fat cells might be significant in causing embolic occlusion of the microcirculation of the sub-chondral vessels or the compression of sinusoidal vascular bed and the development of avascular necrosis¹⁰. While the evidence supports a relationship between steroid therapy and avascular necrosis of bone, it is important to ascertain why this develops in only a few patients. Most of the cases have been reported after prolonged use particularly in patients with renal transplants^{2,14,18}. Short term high dose steroid therapy has only recently been recognized as 'a cause of osteonecrosis and the reports following its usage are relatively few⁵⁻⁸. The association between daily doses as low as 5 mg prednisolone¹⁹ and the duration of therapy as short as 7 days with the development of avascular necrosis suggests that administration of steroids for short period of time does not prevent the risk of this complication. SLE as a cause of aseptic necrosis has also been described in literature'. Our patient was diagnosed as a case of SLE 7 months before the development of osteonecrosis and had received methylprednisolone pulse therapy in addition to daily low dose prednisolone. Two etiological agents can hence be implicated in the development of this lesion - SLE and Steroid therapy. SLE as a cause of aseptic bone necrosis is less likely as the time period from the onset of symptoms of SLE was only 13 months (mean documented 5.1 years). Secondly our patient responded to rest and physiotherapy and the hip pain was not relieved by a subsequent pulse therapy which should have been so if SLE was the cause'. Steroid therapy, therefore, seems more likely to be the etiological factor. Aseptic necrosis can no longer be viewed as a problem pertaining to patients on long term high dose regimens. Short term usage can be as contributory to the development of this invalidating condition. We, therefore, recommend that indications for the use of steroids should be clearly delineated; the use should be minimal and only when deemed necessary.

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