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**Many faces of cutaneous leishmaniasis**

Arfan Ul Bari, Simeen Ber Rahman .....

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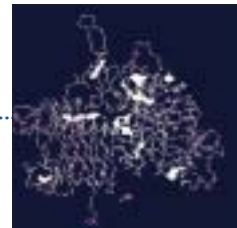


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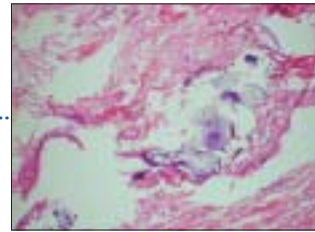
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## Preventing steroid induced osteoporosis

**Jyotsna Oak**

Division of Rheumatology, Department of Medicine, LTM Medical College & LTM General Hospital, Sion, Mumbai, India

**Address for correspondence: Dr. Jyotsna Oak, Department of Medicine, LTM Medical College and LTM General Hospital, Sion, Mumbai - 400 022, Maharashtra, India. E-mail: jyotsnaoak@gmail.com**

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The most frequently encountered secondary cause of osteoporosis is steroid induced osteoporosis (SIOP). The association between Cushing's syndrome and osteoporosis has been recognized since 1932 when Harvey Cushing first described the clinical features of endogenous hypercortisolism.

Since corticosteroids were first used therapeutically in 1948, it has been apparent that exogenous steroids are associated with development of osteoporosis. The extent of bone loss with steroid therapy depends on underlying disease and duration of therapy. Steroids have greater effect on trabecular than on cortical bone, thus the reduction in bone mass is more marked in the spine and proximal femur than in forearm or femoral shaft. The reduction in bone mass associated with CS therapy appears to be greater in rheumatoid arthritis (RA) than in asthma or polymyalgia rheumatica, because of the effect of RA on bone loss. Histological and densitometric studies suggest that the bone loss occurs within a few months of starting CS therapy. The rate of bone loss has been correlated with the daily CS dose, cumulative dose of CS and duration of therapy.

The reduction in bone mass observed with CS therapy is associated with increased risk of fractures, particularly of the vertebrae, ribs and pelvis. The prevalence of fractures in patients treated with oral CS has been reported to be between 11 and 20%.

### THE EFFECTS OF CS ON CALCIUM METABOLISM

High dose of CS therapy (>10 mg of prednisolone/day) decreases calcium absorption by a direct effect on bowel

mucosa which results in secondary hyperparathyroidism and elevation of plasma 1,25 dihydro vitamin D. Exogenously administered CS also suppress the production of androgens which in turn leads to reduction in plasma estrone levels. The release of calcitonin in response to increase in plasma calcium may also be decreased by CS therapy. These changes lead to increased bone resorption, but CS may also increase bone resorption by a direct effect on osteolysis. CS therapy also causes hypercalciuria but it is not clear, if it is due to increased bone resorption or decreased renal tubular resorption.<sup>[1,2]</sup>

### PREVENTION OF CS INDUCED BONE LOSS

Studies show that many patients treated with CS do not receive treatment to prevent bone loss. Approximately 0.5% of the general population and 2% of medical outpatients receive prolonged steroid therapy at some time. Of these only a minority are considered for medical measures to prevent SIOP. Several guidelines are now available to manage SIOP.<sup>[3]</sup>

Recommendations for all the patients receiving CS would include identification and correction of lifestyle risk factors such as smoking and lack of habitual exercise and supplementation with vitamin D and calcium. In 1996, American College of Rheumatology (ACR) summarized available information about the pathophysiology, diagnosis, prevention and treatment of SIOP and developed recommendations of clinical practice.<sup>[4]</sup>

As per the guideline, it is essential to have a baseline measurement of bone mineral density (BMD) at the lumbar spine or hip when initiating the long term (i.e. >6 months)

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CS therapy. The BMD should be repeated after every six months of monitoring therapy. For the patients who are receiving preventive therapy already; annual measurements are probably sufficient. DEXA (dual energy X-ray absorptiometry), QCT (quantitative computed tomography), single X-ray absorptiometry or quantitative ultrasound of bone could be done for the assessment of therapy for which DEXA is the gold standard.<sup>[5]</sup>

## **CALCIUM AND VITAMIN D**

Supplementation with calcium and vitamin D either plain or in activated form (alfacalcidol) at 0.5-1 microgram/day can preserve bone mass in patients receiving long term treatment of glucocorticoids at an average dosage of <15 mg/day. However, calcium alone does not prevent bone loss in patients receiving glucocorticoid therapy.<sup>[6-8]</sup>

Therefore both calcium and vitamin D should be prescribed for SIOP. The patients should be closely monitored for the development of hypercalcemia and hypercalciuria if activated vitamin D is used. If adverse effects develop the dose of activated vitamin D supplement should be reduced.

## **ANTIRESORPTIVE AGENTS**

A number of antiresorptive agents are available to both prevent and treat SIOP

### **Gonadal sex hormones**

Patients receiving prolonged glucocorticoid therapy may develop hypogonadism due to inhibition of secretion of LH and FSH from the pituitary gland as well as the direct effect on hormone production by ovary and testis. All patients receiving steroids should be assessed for hypogonadism and if present; should be corrected if possible. In a trial of postmenopausal women with RA, who were taking prednisolone and were randomised to receive either HRT or placebo, those who had received HRT had a significant (3-4%) increase in the lumbar spine BMD compared with controls while there was no change in BMD at femoral neck.<sup>[9]</sup>

There is less information available regarding men with hypogonadism. A randomized cross over trial demonstrated the effectiveness of testosterone therapy in 15 men with glucocorticoid treated asthma.<sup>[10]</sup> Lumbar spine BMD was significantly increased (4%) after 12 months of monthly intramuscular testosterone injection. Thus men with low serum levels of testosterone (<300 ng/ml) who are receiving CS should receive replacement therapy.<sup>[11]</sup>

Premenopausal women who experience menstrual irregularities (oligo or amenorrhea) while taking CS should be offered OC'S or cyclic estrogen and progesterone if contraindications are not present. No data is available on the efficacy of selective estrogen receptor modulator (SERM) in the prevention or treatment of SIOP. The SERM raloxifene (60 mg/day) is approved by the FDA and is available for the prevention and treatment of postmenopausal osteoporosis.<sup>[12]</sup> A SERM could be used to prevent SIOP in women who either have contraindications to or do not wish to take HRT or other antiresorptive medication. The phase III clinical trials of new SERM, bazedoxifene (20 mg or 40 mg/day) are underway for the treatment of postmenopausal osteoporosis.

### **Bisphosphonates**

Results from five large randomized controlled clinical trials provide evidence that bisphosphonates; etidronate, alendronate and risedronate are effective in both prevention and treatment of SIOP.<sup>[8,13,14]</sup> Significant increase in BMD with bisphosphonates treatment, most consistently observed in lumbar spine were seen in patients of polymyalgia rheumatica and RA treated with glucocorticoids. The improvement in BMD was irrespective of patient's age, sex and menopausal status in women. Also it was observed that there was statistically significant reduction in the absolute risk and relative risk of incident radiographic vertebral fracture (0.7%) with alendronate in SIOP over a period of two years. Drug toxicities were uncommon except a slight increase in non-serious upper GI adverse events.

Bisphosphonates should be used in conjunction with calcium and vitamin D supplementation in the following group of patients:

1. Patients in whom glucocorticoid therapy is being newly initiated to prevent bone loss.
2. Patients receiving long term glucocorticoid therapy with documented osteoporosis on BMD or presence of osteoporotic fractures.
3. Patients on long term CS therapy who have had fractures while on HRT or in whom HRT is not tolerated.

Bisphosphonates not only prevent the bone loss but also prevent apoptosis of osteocytes and osteoblasts.<sup>[15]</sup>

### **Calcitonin**

Calcitonin given either subcutaneous injection or intranasal inhalation has not been shown consistently to prevent bone loss compared to vitamin D and calcium supplementation in



patients starting CS therapy. However calcitonin is shown to increase the BMD at lumbar spine but not at femoral neck in patients of long term CS therapy.<sup>[16]</sup> Calcitonin does not reduce the risk of radiographic vertebral fracture. Thus calcitonin can be considered a second line agent for treatment of patients with low BMD who are receiving long term CS therapy and could be used in patients who have contraindication to or cannot tolerate bisphosphonates. Calcitonin is not recommended for prevention of bone loss in patients being initiated on CS treatment.

### Anabolic agents

Anabolic agents stimulate new bone formation beyond the filling in of remodeling space. Fluoride, (20 mg/day) an anabolic agent has been evaluated in SIOp. In a randomized controlled trial of CS treated patients (average prednisone dosage 1522 mg/day), sodium fluoride (50 mg/day) or placebo were given with calcium and VIT D supplementation. On comparison after two years in the fluoride group and placebo treated group the BMD of femoral neck had increased by 2.2% and decreased by 3.8% respectively.<sup>[17]</sup> Fluoride increases BMD at the lumbar spine but has no effect at the hip. Another anabolic agent is para thyroid hormone (PTH), which has been used in SIOp. Anabolic steroids are potentially useful agents in SIOp. For example, nandrolone decanoate showed increases in forearm bone mass, without significant masculinizing side effects.

Glucocorticoids are used to treat a wide variety of allergic and inflammatory diseases and are prescribed by both specialists and generalists. The best preventive measure for SIOp is to avoid systemic steroids if possible. Whenever possible, local steroid preparations e.g. inhalers in asthma should be used. In some cases steroid sparing agents such as azathioprine or methotrexate may be used. 'Long term steroid treatment' i.e. use of prednisolone  $\geq 5$  mg/day for six months or longer or its equivalent should be accompanied by preventive therapy for SIOp.

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