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Journal of Osteoporosis and Physical Activity

Perspective

Steroid-Induced Osteoporosis: Causes, Mechanisms, Risks, Diagnosis, Treatment, and Prevention

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DESCRIPTION

Steroid-induced osteoporosis is a common and serious complication of long-term glucocorticoid therapy, which leads to decreased Bone Mineral Density (BMD) and increased fracture risk. Glucocorticoids, also known as corticosteroids, are frequently prescribed to treat various chronic inflammatory, autoimmune, and allergic conditions, such as rheumatoid arthritis, asthma, systemic lupus erythematosus, and inflammatory bowel disease. While effective in reducing inflammation and controlling disease activity, these medications adversely affect bone health, often resulting in rapid bone loss and structural deterioration. Steroid-induced osteoporosis is the most common form of secondary osteoporosis and can affect both trabecular and cortical bone, though trabecular bone is typically affected earlier and more severely.

The pathophysiology of steroid-induced osteoporosis involves multiple mechanisms that interfere with the normal bone remodeling process. Bone remodeling is a continuous cycle involving bone resorption by osteoclasts and bone formation by osteoblasts. Glucocorticoids disrupt this balance by both promoting bone resorption and inhibiting bone formation. They increase the lifespan and activity of osteoclasts, leading to enhanced bone breakdown, while simultaneously decreasing the replication, differentiation, and activity of osteoblasts, reducing bone formation. Moreover, glucocorticoids promote osteoblast and osteocyte apoptosis, which further diminishes bone-forming capacity and compromises bone strength.

Another significant effect of glucocorticoids is the reduction in calcium absorption from the gastrointestinal tract and an increase in renal calcium excretion. This causes a decrease in serum calcium levels, which in turn stimulates the secretion of Parathyroid Hormone (PTH). Elevated PTH levels further increase bone resorption in an attempt to normalize serum calcium levels. This secondary hyperparathyroidism adds to the bone loss already caused by glucocorticoid therapy. Additionally, glucocorticoids suppress the production of sex hormones such as estrogen and testosterone, both of which are essential for maintaining bone density. The resulting hypogonadism contributes to further bone loss in both men and women.

Bone loss due to glucocorticoid use is most rapid during the initial months of therapy, with significant reductions in BMD observable within the first three to six months. The degree of bone loss and risk of fracture correlate with the dose and duration of glucocorticoid treatment, but even low doses taken over long periods can increase fracture risk. Patients using a daily dose of 5 mg or more of prednisone (or its equivalent) for more than three months are considered at high risk for steroid-induced osteoporosis. Fractures associated with glucocorticoid-induced bone loss often occur at the spine, hips, and ribs and may happen with minimal trauma or no identifiable cause.

Diagnosis of steroid-induced osteoporosis involves a thorough medical history, assessment of glucocorticoid use, and evaluation of fracture risk. Bone mineral density is measured using Dual-Energy X-ray Absorptiometry (DEXA), with results reported as T-scores. A T-score of -2.5 or lower confirms the diagnosis of osteoporosis. However, in patients on chronic steroids, even T-scores in the osteopenic range (-1.0 to -2.5) warrant clinical concern due to the increased fragility associated with glucocorticoid use. In addition to BMD testing, clinical tools such as the FRAX (Fracture Risk Assessment Tool) can be used to estimate the 10-year probability of fractures, adjusting for steroid exposure.

The management of steroid-induced osteoporosis includes both non-pharmacologic and pharmacologic strategies aimed at preserving bone mass and preventing fractures. Non-pharmacologic measures include ensuring adequate intake of calcium and vitamin D, engaging in regular weight-bearing and resistance exercise, avoiding tobacco use, and limiting alcohol consumption. The recommended daily intake of calcium for adults at risk is 1,200-1,500 mg, and vitamin D supplementation should maintain serum 25-hydroxyvitamin D levels above 30 ng/mL.

Pharmacologic treatment is essential for individuals who are starting or continuing long-term glucocorticoid therapy, especially those with low BMD or a history of fractures.

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Received: 19-Feb-2025, Manuscript No. JOPA-25-38089; **Editor assigned:** 21-Feb-2025, PreQC No. JOPA-25-38089 (PQ); **Reviewed:** 07-Mar-2025, QC No. JOPA-25-38089; **Revised:** 14-Mar-2025, Manuscript No. JOPA-25-38089 (R); **Published:** 21-Mar-2025, DOI: 10.35841/2329-9509.25.13.451

Citation: Gyune S (2025). Steroid-Induced Osteoporosis: Causes, Mechanisms, Risks, Diagnosis, Treatment, and Prevention. J Osteopor Phys Act. 13:451.

Bisphosphonates, such as alendronate, risedronate, and zoledronic acid, are the first-line medications. They work by inhibiting osteoclast-mediated bone resorption and have been shown to increase BMD and reduce vertebral fractures in patients taking steroids. Denosumab, a monoclonal antibody that blocks RANKL, is another effective antiresorptive agent, particularly useful for patients who cannot tolerate bisphosphonates or have impaired kidney function. Teriparatide and abaloparatide, anabolic agents that stimulate new bone formation, may be used in patients with severe osteoporosis or those who fail to respond to antiresorptive therapy. Romosozumab, which has both anabolic and antiresorptive effects, may also be considered in certain high-risk cases.

Monitoring treatment response is important in managing steroid-induced osteoporosis. Repeat DEXA scans are usually performed every one to two years to assess changes in BMD. In addition, adherence to therapy and supplementation should be evaluated regularly. Patients should be reassessed for fracture risk periodically, especially if glucocorticoid dosage or duration changes.

Prevention of steroid-induced osteoporosis is ideally initiated at the start of glucocorticoid therapy. Patients expected to take glucocorticoids for more than three months should be assessed for osteoporosis risk and started on preventive measures early. Proactive management, including education about bone health, lifestyle modifications, supplementation, and timely initiation of pharmacotherapy, is essential to reducing the burden of fractures and improving long-term outcomes.

CONCLUSION

Steroid-induced osteoporosis is a serious and preventable complication of glucocorticoid therapy. It arises from an imbalance in bone remodeling caused by increased resorption and decreased formation, compounded by effects on calcium metabolism and sex hormone suppression. Early recognition, risk assessment, and comprehensive management strategies can preserve bone health and significantly reduce the risk of fractures in patients requiring long-term steroid use.