Clinical features and evaluation of glucocorticoid-induced osteoporosis

AUTHOR: Harold N Rosen, MD
SECTION EDITOR: Clifford J Rosen, MD
DEPUTY EDITOR: Katya Rubinow, MD

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INTRODUCTION

Glucocorticoid therapy is associated with an appreciable risk of bone loss, which is most pronounced in the first few months of use. In addition, glucocorticoids increase fracture risk, and fractures occur at higher bone mineral density (BMD) values in glucocorticoid-induced osteoporosis than in postmenopausal osteoporosis. The increased risk of fracture has been reported with doses of prednisone or its equivalent as low as 2.5 to 7.5 mg daily [1]. The pathogenesis, clinical features, and evaluation of glucocorticoid-induced osteoporosis will be reviewed here. The prevention and treatment of glucocorticoid-induced osteoporosis are presented separately. (See "Prevention and treatment of glucocorticoid-induced osteoporosis".)

PATHOGENESIS

The deleterious effects of glucocorticoid excess on bone result from direct effects on osteoblasts, osteocytes, and osteoclasts. Glucocorticoids increase bone resorption and reduce bone formation [2-4]. The risk of bone loss is most pronounced in the first few months of use, followed by slower but steady loss of bone with continued use [3]. Most agents that increase bone loss, such as thyroxine or sustained elevation of parathyroid hormone (PTH), accelerate not only bone resorption but also formation, albeit to a lesser extent [5]. Because glucocorticoids accelerate resorption while inhibiting formation, their use is associated with

early rapid bone loss [6,7]. With chronic use, osteoclast-mediated bone resorption slows, and suppression of bone formation becomes the predominant skeletal effect [8-10].

- **Increase in bone resorption** – As in other target tissues, glucocorticoids exert their effects on gene expression via cytoplasmic glucocorticoid type 2 receptors [11]. In adult bone, functional glucocorticoid receptors are found in pre-osteoblast/stromal cells, osteoblasts (the cells that produce bone matrix), but not in osteoclasts [12,13]. Glucocorticoids stimulate osteoclast proliferation by suppressing synthesis of osteoprotegerin, an inhibitor of osteoclast differentiation from hematopoietic cells of the macrophage lineage, and by stimulating production of the receptor activator of nuclear factor kappa-B (RANK), which is required for osteoclastogenesis. High glucocorticoid levels also stimulate RANK ligand (RANKL) synthesis by pre-osteoblast/stromal cells, supporting osteoclast differentiation and net bone resorption [13]. In addition, glucocorticoids increase bone resorption by decreasing secretion of androgens and estrogens, mediated primarily by inhibition of gonadotropin secretion [14-17]. (See "Normal skeletal development and regulation of bone formation and resorption", section on 'Osteoclasts'.)

  Glucocorticoids also decrease intestinal calcium absorption, in part by opposing the action of vitamin D and by decreasing the expression of calcium channels in the duodenum [3,18-20]. Glucocorticoids increase renal calcium excretion by decreasing calcium reabsorption [19,21,22]. Both of these actions result in an increase in serum PTH and, subsequently, increased bone resorption.

- **Suppression of bone formation** – With long-term use, the predominant effect of glucocorticoids on the skeleton is reduced bone formation. The decline in bone formation is mediated by direct inhibition of osteoblast proliferation and differentiation and by an increase in the apoptosis rates of mature osteoblasts and osteocytes [2,3,23-25]. This apoptosis may also explain the tendency of glucocorticoids to cause osteonecrosis [26]. In addition, glucocorticoids alter PTH secretory dynamics (reduce tonic secretion and increase the amount released as pulses) [27], antagonize the anabolic action of PTH [23,28], and inhibit production of insulin-like growth factor-1 (IGF-1) [3,25,29] and testosterone [14-16]. The reduction in bone formation is associated with a decrease in the mineral apposition rate [30] and in serum and urine biochemical markers of bone formation [21,31]. (See "Normal skeletal development and regulation of bone formation and resorption", section on 'Osteoblasts'.)

EPIDEMIOLOGY AND RISK FACTORS

Glucocorticoids increase the risk of fracture, particularly vertebral fractures, which occur early in the course of treatment, during the rapid phase of bone loss, and at higher bone mineral density (BMD) levels than in postmenopausal osteoporosis [3,32-35]. Fractures have been reported in as many as 30 to 50 percent of glucocorticoid users [1,3,36]. The incidence of fracture is higher with advanced age, larger dose, and longer duration of glucocorticoid therapy [33,36-39]. However, an increased risk has been reported with doses of prednisone or its equivalent even as low as 2.5 to 7.5 mg daily [32] and with short-term use (<30 days) [40]. The sparse data that have been published on alternate-day prednisone therapy suggest that this regimen is not protective of bone [41-43]. The increased risk of fracture in patients taking glucocorticoids declines rapidly in the first year off therapy [32,44].

- **Inflammatory diseases** – The relationship between glucocorticoid dose and fracture risk is complicated by the observation that the disease for which the glucocorticoids are being given (eg, rheumatoid arthritis, inflammatory bowel disease) may itself lead to loss of bone and fracture. (See "Overview of the systemic and nonarticular manifestations of rheumatoid arthritis", section on 'Osteopenia' and "Metabolic bone disease in inflammatory bowel disease".)

  In a case-control study comparing adults with rheumatoid arthritis who had been treated with prednisone (mean dose 8 mg daily for a mean duration of 6.9 years) with control patients (rheumatoid arthritis who had not received prednisone), there was an increased rate of fractures (particularly in the spine, hips, and ribs) in patients taking prednisone (25 versus 15 percent) [45]. Other studies in patients with rheumatoid arthritis have shown a fracture risk of 34 to 58 percent in patients receiving prolonged prednisone therapy at doses of 5 to 8.6 mg daily [46,47].

- **Adrenal insufficiency** – The effect of glucocorticoid replacement therapy on BMD in patients with adrenal insufficiency is controversial [48-50]. In cross-sectional studies of adults with Addison disease receiving long-term replacement therapy, BMD was lower in men [51] and women [50] with Addison disease than in the reference population. In one study, bone density was inversely related to the hydrocortisone dose per kilogram [51]. The men who lost bone received a mean hydrocortisone dose of 16.4 mg/m^2 per day. This is approximately 1.6 times estimated daily production rate in normal subjects, suggesting they were slightly overtreated (see "Treatment of adrenal insufficiency in adults"). In another cross-sectional study of 32 adults treated with glucocorticoids since childhood for 21-hydroxylase deficiency, bone density was significantly lower at the femoral neck than in the reference population, likely due to slight overtreatment [52].

Although fracture data are not available, these studies highlight the importance of avoiding excessive doses of replacement glucocorticoids.

- **Pulmonary diseases –** Inhaled glucocorticoids have fewer and less severe adverse effects than orally administered glucocorticoids, and they are widely used to treat asthma and chronic obstructive pulmonary disease (COPD). Studies have not found consistent results regarding the impact of inhaled glucocorticoids on risk of osteoporosis and of osteoporotic fracture. This could relate to the fact that patients randomized to inhaled glucocorticoids tend to need fewer courses of systematic glucocorticoids, so that a mild, direct, deleterious effect of high-dose inhaled glucocorticoids could be masked by the benefit of reducing the need for systemic glucocorticoids. Topical therapy (eg, inhaled glucocorticoids) is preferred over enteral or parenteral glucocorticoids whenever possible. This topic is reviewed in detail elsewhere. (See "Major side effects of inhaled glucocorticoids", section on 'Osteoporosis and fracture risk in adults'.)

CLINICAL FEATURES

The clinical manifestations of glucocorticoid-induced osteoporosis are the same as those of other causes of osteoporosis. Most often, there are no clinical manifestations until there is a fracture. Vertebral fractures are most common and are often asymptomatic. These are diagnosed as an incidental finding on chest or abdominal radiograph. In patients who have a symptomatic vertebral fracture, there is often no history of preceding trauma. The typical symptomatic patient presents with acute back pain after sudden bending, coughing, or lifting. (See "Clinical manifestations, diagnosis, and evaluation of osteoporosis in postmenopausal women", section on 'Clinical manifestations' and "Clinical manifestations, diagnosis, and evaluation of osteoporosis in men", section on 'Clinical manifestations' and "Osteoporotic thoracolumbar vertebral compression fractures: Clinical manifestations and treatment", section on 'Clinical manifestations'.)

EVALUATION

Any patient taking any dose of glucocorticoid requires an evaluation. The goal of the evaluation is to identify patients at high risk for fracture who would benefit from intervention. (See "Prevention and treatment of glucocorticoid-induced osteoporosis", section on 'Candidates for pharmacologic therapy'.)

Fracture risk assessment — Assessment of fracture risk should include evaluation of clinical risk factors for fracture and, in selected patients, bone mineral density (BMD) [53]. The absolute risk of fracture can be calculated using a fracture prediction tool, such as FRAX (Fracture Risk Assessment Tool). In individuals taking glucocorticoid doses equivalent to prednisone ≥2.5 mg/day for >3 months, the American College of Rheumatology (ACR) guidelines suggest assessment as soon as possible after initiating glucocorticoids [53]. We and others perform an assessment within three months, as glucocorticoids can increase fracture risk within three months of initiating therapy [1,54]. (See "Fracture risk assessment".)

Clinical risk factor assessment — In addition to glucocorticoid exposure, advancing age, prior history of fragility fracture, low body mass index (BMI), parental history of hip fracture, frequent falls, cigarette smoking, and excess alcohol intake are risk factors that are predictive of fracture. These risk factors are easily discernible from a routine history and physical examination. (See "Fracture risk assessment", section on 'Clinical risk factor assessment'.)

Glucocorticoid exposure — High glucocorticoid exposure can markedly increase fracture risk even in individuals with normal bone mass and without prior history of fracture. The ACR guidelines consider high-dose glucocorticoid therapy (treatment with prednisone ≥30 mg daily for >30 days or cumulative doses ≥5 g per year [or equivalent]) to confer very high fracture risk, irrespective of the presence of other clinical risk factors [53].

BMD — We measure bone mineral density (BMD; dual-energy x-ray absorptiometry [DXA]) of the hip and spine in all individuals taking any dose of glucocorticoid with anticipated duration of ≥3 months (or if duration of therapy is uncertain).

- Patients with a history of a fracture or with osteoporosis on baseline BMD (T-score ≤-2.5) should be evaluated to exclude secondary causes of osteoporosis, including vitamin D deficiency, hyperparathyroidism, or hypogonadism (table 1 and table 2). The evaluation for secondary causes of osteoporosis is reviewed separately. (See "Clinical manifestations, diagnosis, and evaluation of osteoporosis in postmenopausal women", section on 'Evaluation' and "Clinical manifestations, diagnosis, and evaluation of osteoporosis in men", section on 'Evaluation'.)

- For patients who do not have osteoporosis on baseline BMD or a history of fracture, we also measure serum 25-hydroxyvitamin D (25[OH]D). Vitamin D supplementation is recommended for patients initiating or receiving any dose of glucocorticoids for any duration. For patients with normal baseline serum 25(OH)D levels, supplementation with 800 international units/day is adequate. However, patients with low baseline serum 25(OH)D levels will require higher doses. Vitamin D supplementation may be initiated

prior to measurement of serum 25(OH)D. (See "Vitamin D deficiency in adults: Definition, clinical manifestations, and treatment".)

Fracture risk assessment tool — For patients aged ≥40 years **without** established osteoporosis (eg, the absence of prior fragility fracture or BMD T-score ≤-2.5), fracture risk can be quantitated using a fracture prediction tool, such as FRAX (Fracture Risk Assessment Tool). FRAX estimates the 10-year probability of fracture for untreated patients between ages 40 and 90 years, using femoral neck BMD and clinical risk factors, including glucocorticoid exposure. For patients taking prednisone >7.5 mg/day or equivalent, the risk estimate must be corrected for glucocorticoid exposure (increase by 15 percent of the estimated risk for major osteoporotic fracture and by 20 percent for hip fracture) [55]. (See "Fracture risk assessment", section on 'Fracture risk assessment tool'.)

Vertebral imaging — For patients in whom the decision to treat or monitor is not obvious (eg, low bone mass [T-score between -1.0 and -2.5], no known prior fracture, but strong family history of osteoporosis), we obtain imaging of the spine. In a patient who already has an indication for pharmacologic therapy (ie, prior fracture, osteoporosis on BMD), we do not typically obtain radiographic imaging of the spine.

Imaging may be performed with conventional radiography or with vertebral fracture assessment (VFA), a component of the DXA instrument. The benefit of using VFA is that a patient does not require a separate appointment for vertebral imaging. It can be performed at the same time as BMD study. VFA compares favorably with spine radiographs in detecting moderate and severe vertebral fractures, but it does not perform as well for diagnosing mild vertebral fractures. (See "Overview of dual-energy x-ray absorptiometry", section on 'Vertebral fracture assessment'.)

Vertebral fracture is the most common type of fracture in patients taking glucocorticoids, but they are often asymptomatic. The presence of a vertebral fracture is a strong predictor of future fractures of all types [56,57] and is an indication for pharmacologic therapy. Thus, the identification of a previously undetected vertebral fracture may be useful in making a decision to treat with pharmacologic therapy or to monitor. (See "Prevention and treatment of glucocorticoid-induced osteoporosis", section on 'Candidates for pharmacologic therapy'.)

Other imaging studies — Advanced methods of measuring volumetric BMD, including high-resolution micro-computed tomography (microCT) and micro-magnetic resonance imaging (microMRI), allow noninvasive, three-dimensional evaluation of bone architecture. While these methods have provided insight into the skeletal changes that occur with chronic

glucocorticoid exposure, they do not have a role in the clinical evaluation of patients with glucocorticoid-induced osteoporosis [58]. They are used only for research.

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Osteoporosis".)

SUMMARY AND RECOMMENDATIONS

- **Pathogenesis –** Glucocorticoids increase bone resorption, reduce bone formation, decrease intestinal calcium absorption, and increase renal calcium excretion. Glucocorticoid therapy appreciably increases bone loss, which is most pronounced in the first few months of use. (See 'Pathogenesis' above.)

- **Risk factors for fracture –** Glucocorticoids increase the risk of fracture, particularly vertebral fractures, which occur early after exposure, during the rapid phase of bone loss. Fractures due to glucocorticoid use occur at higher bone mineral density (BMD) values than occur in postmenopausal osteoporosis. The incidence of fracture is higher with advanced age, larger dose, and longer duration of glucocorticoid therapy. However, the increased risk of fracture has been reported with doses of prednisone or its equivalent as low as 2.5 to 7.5 mg daily. (See 'Epidemiology and risk factors' above.)

- **Clinical manifestations –** The clinical manifestations of glucocorticoid-induced osteoporosis are the same as those of other causes of osteoporosis. Most often, there are no clinical manifestations until there is a fracture. Vertebral fractures are most common, and they are often asymptomatic. (See 'Clinical features' above.)

- **Evaluation –** Any patient taking any dose of glucocorticoid with an anticipated duration of ≥3 months requires an evaluation. The goal of the evaluation is to identify patients at high risk for fracture who would benefit from intervention. Assessment of fracture risk within approximately three months of initiating glucocorticoids should include evaluation of clinical risk factors for fracture and BMD (dual-energy x-ray absorptiometry [DXA]) of the hip and spine. (See 'Evaluation' above and "Clinical manifestations, diagnosis, and evaluation of osteoporosis in postmenopausal women" and "Clinical manifestations, diagnosis, and evaluation of osteoporosis in men".)

- **Glucocorticoid exposure –** High glucocorticoid exposure can markedly increase fracture risk even in individuals with normal bone mass and without other clinical risk factors for fracture. (See 'Glucocorticoid exposure' above.)

- **BMD measurement –** We measure BMD (DXA) of the hip and spine in all individuals taking any dose of glucocorticoid with anticipated duration of ≥3 months (or if duration of therapy is uncertain). We also measure serum 25-hydroxyvitamin D (25[OH]D) to guide vitamin D supplementation. Additional laboratory evaluation depends upon the results of the BMD study. (See 'BMD' above.)

- **Fracture risk assessment –** For patients aged ≥40 years **without** established osteoporosis (eg, the absence of prior fragility fracture or BMD T-score ≤-2.5), fracture risk can be quantitated using a fracture prediction tool, such as FRAX (Fracture Risk Assessment Tool). (See 'Fracture risk assessment tool' above.)

- **Vertebral imaging –** For patients in whom the decision to administer pharmacologic therapy for fracture prevention is uncertain (eg, low bone mass [T-score between -1.0 and -2.5], no known prior fracture, but strong family history of osteoporosis), we obtain imaging of the spine. Imaging may be performed with conventional radiography or with vertebral fracture assessment (VFA), a component of the DXA instrument. The presence of a vertebral fracture is a strong predictor of future fractures of all types and is an indication for pharmacologic therapy. (See 'Vertebral imaging' above.)

- **Prevention and treatment –** The prevention and treatment of glucocorticoid-induced osteoporosis are presented separately. (See "Prevention and treatment of glucocorticoid-induced osteoporosis".)

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