REVIEW ARTICLE

CONTENTS

Hormonal influence on osteoporosis

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ABSTRACT

Osteoporosis is a global health issue that weakens bones and increases the risk of fractures, particularly in postmenopausal women and older adults. This review explores the influence of hormones on osteoporosis, with a focus on estrogen, glucocorticoids, vitamin D, parathyroid hormone, and thyroid hormones. Scientific evidence is analyzed to highlight hormonal mechanisms, therapeutic interventions, and their effects on bone health. The review examines hormonal therapies, including selective estrogen receptor modulators, and discusses the impact of glucocorticoids, essential for managing inflammation but known to significantly increase bone porosity. It underscores the importance of minimizing glucocorticoid use and implementing preventive measures such as calcium and vitamin D supplementation. Additionally, the review addresses the consequences of vitamin D deficiency, which disrupts calcium and phosphate metabolism, contributing to bone fragility. It also explores the regulatory roles of parathyroid and thyroid hormones in bone remodeling and calcium balance, with a focus on their therapeutic applications in osteoporosis management. This review emphasizes the complex interplay between hormonal, genetic, and environmental factors, advocating for holistic and personalized treatment approaches. Future research should aim to bridge existing knowledge gaps regarding hormone-environment interactions and assess the long-term efficacy of therapies to optimize osteoporosis prevention and treatment strategies.

KEY WORDS: osteoporosis, menopause, estrogen, parathormone, thyroid hormones, glucocorticoids

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INTRODUCTION

Osteoporosis is one of the major global health concerns, affecting millions of people worldwide. However, it often does not receive the attention it deserves. This disease gradually weakens bones, increasing their fragility and the risk of fractures. Its impact extends beyond physical health to psychosocial well-being, significantly affecting quality of life. Despite its widespread prevalence, public awareness remains limited regarding the development of osteoporosis, its risk factors, and preventive measures [1].

The human skeleton is a complex organ system that, in addition to its role in hematopoiesis, serves crucial functions related to movement and the protection of internal organs. It undergoes complete reconstruction approximately every ten years through a delicate balance between bone formation and resorption. In osteoporosis, this balance is disrupted, leading to the loss of bone mass and deterioration of its microarchitecture. The condition primarily affects older individuals, particularly postmenopausal women, due to hormonal changes, chronic illnesses, or long-term medication use [2].

Osteoporosis is a metabolic disease characterized by reduced bone mass and structural deterioration. The most common form, primary osteoporosis, is associated with aging and menopause, whereas secondary osteoporosis results from underlying medical conditions or medication side effects. Since osteoporosis primarily affects bone strength, understanding bone structure is crucial. Bone tissue consists of both cortical and trabecular (spongy) layers, with cortical bone providing most of the structural integrity. Hormonal regulation plays a crucial role in maintaining bone health by modulating the activity of osteoblasts (bone-forming cells) and osteoclasts (bone-resorbing cells) [3]. Estrogen, for example, protects against bone loss and is a key factor in the increased prevalence of osteoporosis among

postmenopausal women. Similarly, testosterone is essential for maintaining bone strength in men, and its decline can lead to characteristic fractures of the vertebrae and hip. Other hormones involved in bone metabolism include parathyroid hormone, calcitonin, and thyroid hormones. Imbalances in these hormones, combined with aging and other risk factors, contribute to bone loss and increase the likelihood of fractures [4].

AIM

This review emphasizes the critical role of hormones in osteoporosis, with a particular focus on their influence on bone mineral density (BMD), a key determinant of fracture risk. Understanding these hormonal interactions is essential for developing effective prevention and treatment strategies. By gaining insight into the hormonal mechanisms underlying bone remodeling and implementing preventive measures – such as fall prevention, regular physical activity, and proper nutrition – the risk of osteoporosis-related complications can be significantly reduced, ultimately improving patient outcomes.

MATERIALS AND METHODS

This review aims to evaluate the hormonal influence on osteoporosis by analyzing data from 72 peer-reviewed articles available in the PubMed database. The search strategy involved using specific keywords, such as "Hormonal influence on osteoporosis," "Postmenopausal osteoporosis," "Hormonal balance," "Mineral balance and bones," "Osteoporosis treatment," "Estrogen and osteoporosis," "Glucocorticoids," "GIOP," "Vitamin D," "Parathyroid hormone and osteoporosis," "Calcitonin and osteoporosis," and "Thyroid hormones and osteoporosis."

The inclusion criteria focused on studies published in English, prioritizing original research articles, systematic reviews, and meta-analyses. Articles discussing hormonal mechanisms, therapeutic interventions, and their impact on bone health were included. Exclusion criteria consisted of studies with incomplete data, non-English publications, and studies published more than 20 years ago. All retrieved articles were critically assessed for relevance and quality.

REVIEW

ESTROGEN

Estrogens and androgens have protective effects on bones, playing a significant role in bone formation, with deficiency leading to increased resorption. The primary function of estrogen is regulating bone turnover by decreasing bone resorption. It affects both cortical and trabecular bone, reducing their density [5].

A vast majority of studies have focused on cancellous bone, prompting research on the often-overlooked cortical bone. The effect of estrogen deficiency on cortical bone, and more specifically on its two structures – lamellar and disorganized – was examined by comparing their mechanical and structural properties. Femoral bones from rodents with removed ovaries (test group, with reduced estrogen levels) were compared to those of rodents with intact ovaries (control group). Differences were observed in the stiffness of the bone elements; although the changes were minor, they affected the fundamental components of the skeleton, potentially reducing its strength. Furthermore, there is a risk of intensified osteocyte apoptosis. These findings indicate the general impact of reduced estrogen levels on cortical bone in rats [6, 7]. In women, this estrogen deficiency is most pronounced during menopause, a period marked by profound changes in biology and endocrinology. For women under 60 years old, the most effective treatment is menopausal hormone therapy. Estrogens and selective estrogen receptor modulators (SERMs) inhibit the production of RANKL, a receptor activator of nuclear factor kappa beta ligand, which promotes osteoclast differentiation. This leads to osteoclast apoptosis and encourages the production of osteoprotegerin. Two substances approved by the FDA for osteoporosis treatment, raloxifene and bazedoxifene, help stop bone remodeling [8, 9].

GLUCOCORTICOIDS

Osteoporosis and bone fractures are among the most common side effects of glucocorticoid medications. Patients taking these steroids are exposed to an increased risk of bone loss. It is also important to note that basic diseases, which are treated with these medications, can themselves contribute to bone mass loss. Additionally, factors such as old age, being female, and initially low bone mass further intensify the risk of bone damage. Over a hundred KEGG pathways associated with genes expressed in osteoporotic individuals have been identified [10]. Glucocorticoid-induced osteoporosis (GIOP) is a secondary cause of osteoporosis in adults, with menopause being the primary cause. The pathophysiology of GIOP is more complex than it might initially seem. During the first year of treatment with corticosteroids, rapid bone loss is often observed, typically around the lumbar spine. Several factors contribute to the development of GIOP, including inhibition of the somatotropic and gonadotropic axes, impaired intestinal calcium absorption,

post-glucocorticoid myopathy, cataracts, and fluctuations in bone cell survival. What is the effect of steroids on bone cells? They promote osteoclastogenesis by "taking the osteoclasts' side." The mechanism by which glucocorticoids influence bone cells involves the suppression of Wnt agonists and the upregulation of Wnt signaling inhibitors. The suppression of the Wnt/beta-catenin pathway is crucial in osteoblastogenesis, and blocking this pathway prevents osteoblast differentiation. One of the most important effects of glucocorticoids in treatment is their anti-inflammatory action. These hormones inhibit the production of proinflammatory cytokines, such as IL-1 and IL-6, which induce bone resorption and inhibit bone formation. In this context, glucocorticoids have a protective effect. Moreover, glucocorticoids also act on extraskeletal mechanisms. They suppress insulin-like growth factor 1 (IGF-1), which normally stimulates the synthesis of collagen type 1 and promotes bone formation. As a result, this mechanism leads to collagen degeneration and osteoblast apoptosis [11]. Recent preclinical studies suggest that excessive corticosteroid use may disrupt the intestinal microbiota and the endogenous biological rhythm of glucocorticoid secretion, causing detrimental effects on the skeleton. In GIOP, we observe inhibition of osteoblast differentiation, decreased collagen synthesis by these cells, and induction of osteoblast apoptosis. Exhaustion of the body and limited movement can also impair bone development, particularly in younger individuals. How can we manage GIOP? The first step is to avoid excessive glucocorticoid use. Patients should take the lowest effective dose for the shortest duration and consider local rather than systemic administration of these medications. GIOP prevention is recommended for postmenopausal women, men, and women around 40 years of age who are at high risk of fractures. For patients who have been using glucocorticoids for more than three months or who have experienced fragility fractures, anti-osteoporosis therapy should be initiated. It is also helpful to supplement with 1200-2000 mg of calcium daily and vitamin D. Additionally, platelet lysates containing PDGF, VEGF, transforming growth factor beta, and basic fibroblast growth factor may also aid in managing osteoporosis [12].

VITAMIN D

Currently, one-third of the global population suffers from insufficient levels of 25-hydroxyvitamin D, with concentrations not exceeding 20 ng/mL [13]. An important aspect to consider is that vitamin D increases tissue sensitivity to anti-resorptive therapy [14]. The primary source of vitamin D in humans is cholecalciferol, which is synthesized in the skin upon exposure to ultraviolet B (UVB) radiation. The optimal level of 25(OH)-cholecalciferol is considered to be between 36-75 nmol/L. A low level of 25(OH)2-D causes disturbances in intestinal calcium-phosphate absorption, leading to a decrease in serum concentrations of ionized calcium. As a result, parathyroid hormone (PTH) levels rise, promoting the differentiation of preosteoclasts into osteoclasts. An increase in the number of mature osteoclastic cells leads to bone resorption, loss of bone mineral mass and matrix, and eventually results in osteoporosis. Consequently, bones become fragile and more prone to fractures. Kuchuk and colleagues identified a correlation between vitamin D deficiency and fractures, noting that serum levels of 25(OH)2D below or equal to 30 nmol/L significantly increase the risk of fractures in individuals aged 65-76 years [15].

PARATHORMONE

Parathyroid hormone (PTH) plays a crucial role in skeletal homeostasis by participating in various cellular and molecular mechanisms. It is the most important endocrine regulator of calcium-phosphate balance [16]. PTH is secreted by four small, highly vascularized glands located behind the thyroid gland in the anterior neck [17]. However, excessive secretion of PTH into circulation can have detrimental effects. Functional parathyroid hormone receptors are present on osteoblast lineage cells. PTH stimulates bone resorption and turnover. In this process, bone formation occurs through an increase in osteoblast numbers, enhanced deposition of mineralized matrix, proliferation of osteoblast precursors, suppression of apoptosis, and activation of lining cells [18, 19]. PTH also has significant effects on other organs. In the kidneys, it enhances calcium reabsorption and decreases phosphate reabsorption. Additionally, it stimulates 1-alpha-hydroxylase to synthesize the active form of vitamin D, which is essential for maintaining proper bone structure [18,20]. The action of PTH is mediated through interaction with its receptor (PTH/PTHrP receptor type 1) in target tissues, involving G-protein signaling [20]. Interestingly, PTH has been shown to counteract glucocorticoid-induced suppression of insulin-like growth factor-1 (IGF-1), a key factor in bone formation, as previously discussed in the context of glucocorticoid-induced osteoporosis (GIO). Synthetic parathyroid hormone is widely used as a treatment for osteoporosis, particularly in postmenopausal women and individuals with hypogonadism [17].

THYROID HORMONES

The major thyroid hormones in humans are thyroxine (T4) and triiodothyronine (T3). Their production and

secretion are regulated by thyroid-stimulating hormone (TSH). The human endocrine system is characterized by complex feedback mechanisms, and thyroid hormone receptors are widely distributed across various organs, including the nervous system, pituitary gland, lungs, heart, liver, muscles, bones, testes, placenta, and others [21]. Maintaining hormonal balance is equally critical for children's health. Untreated hypothyroidism in young individuals can result in delayed skeletal development and disrupted endochondral ossification. Both T3 and T4 play crucial roles in cartilage development. TR-alpha receptors, located on osteoblasts and osteoclasts, interact with triiodothyronine. Upon binding with T3, these cells receive stimulatory signals for bone remodeling. Hyperthyroidism is associated with decreased bone mineral density (BMD) and an increased risk of fractures. Due to its significant impact on the bone remodeling cycle, thyroid hormone imbalance can contribute to osteoporosis. Moreover, in women over the age of 65 with hypothyroidism and a history of osteoporosis, levothyroxine supplementation at doses exceeding 150 µg daily has been linked to an increased fracture risk and reduced BMD [22, 23]. Human calcitonin, a peptide hormone produced by parafollicular cells in the thyroid gland, plays a role in calcium homeostasis and bone turnover regulation by activating the calcitonin receptor (CTR). Calcitonin has been utilized in the treatment of postmenopausal osteoporosis, Paget's disease of bone, and hypercalcemia. Extensive research has evaluated the effectiveness of salmon calcitonin as a replacement for human calcitonin, yielding positive results [24-26]. Its mechanism of action involves reducing osteoclast activity, which lowers blood calcium levels while balancing serum creatinine and alkaline phosphatase levels. Salmon calcitonin is typically administered intranasally. However, salmon calcitonin has not demonstrated significant efficacy in improving BMD in the hip or lumbar vertebrae [27]. Currently, calcitonin is approved for patients with at least a five-year history of postmenopausal osteoporosis, although it is not considered a first-line treatment due to an observed increased risk of malignancies among patients using intranasal salmon calcitonin compared to those using a placebo. Bisphosphonates are generally preferred for their superior effects on BMD improvement and bone turnover reduction [26]. Despite this, salmon calcitonin has shown promise in alleviating bone pain in oncology patients with metastases to the skeleton, particularly from breast, lung, kidney, or prostate cancer [28].

DISCUSSION

The findings of this review underscore the pivotal role of hormonal changes, particularly involving estrogen, parathyroid hormones, and other endocrine factors, in the pathogenesis of osteoporosis. These results align with prior studies that emphasize the protective effects of estrogen on bone density, its regulatory role in bone remodeling, and its ability to inhibit osteoclast activity. Research has demonstrated that postmenopausal women experience accelerated bone loss due to a marked decline in estrogen levels [29].

Additionally, this review reinforces the involvement of parathyroid hormone (PTH) in modulating calcium homeostasis and highlights its therapeutic potential in managing osteoporosis [30]. However, it also reveals discrepancies in the existing literature regarding the efficacy of certain hormone-based treatments, thus paving the way for further research into long-term hormone therapies and a more detailed understanding of hormonal pathways. The complexity of these mechanisms highlights the need for a more comprehensive approach to osteoporosis that integrates both systemic endocrine functions and environmental/genetic factors. Studies suggest that environmental and genetic influences may interact with hormonal mechanisms to influence osteoporosis risk [31], though this area remains under-researched. Important questions persist, such as whether specific biomarkers could predict how individuals respond to hormone-based treatments. These questions warrant further exploration, as understanding the hormonal basis of osteoporosis alone is insufficient without considering its interactions with other influencing factors.

While this review emphasizes the critical role of hormones, it also highlights gaps in personalized osteoporosis management. Recent advancements in molecular biology have opened avenues for selective estrogen receptor modulators (SERMs), which mimic estrogen's effects without the associated risks, such as breast cancer [32]. Despite their potential, the clinical application of SERMs remains underexplored, underscoring the need for more research into individualized therapeutic strategies. Furthermore, comparative studies reveal significant geographic and ethnic variability in osteoporosis prevalence and hormonal interactions. For instance, Asian populations, despite having lower average bone mineral density, exhibit reduced fracture rates compared to Caucasians, potentially due to differences in bone microarchitecture and dietary calcium intake [33]. These findings emphasize the importance of considering hormonal influences within broader genetic and environmental frameworks to optimize osteoporosis prevention and treatment strategies.

This review also highlights the significant risks posed by Vitamin D deficiency to bone health across both younger and older populations. Approximately one-third of the

global population has insufficient serum 25-hydroxyvitamin D levels (<20 ng/mL), contributing to disorders such as osteomalacia in adults and rickets in children. Deficiency is also prevalent among patients with joint diseases and the elderly [13]. Vitamin D plays a critical role in calcium-phosphate metabolism and bone homeostasis by promoting intestinal calcium absorption, renal calcium reabsorption, and regulating bone remodeling via osteocalcin expression and parathyroid hormone inhibition. Le Boff et al. examined the effects of vitamin D supplementation on bone mineral density (BMD) and structure, and while vitamin D is important for bone health, supplementation alone may not significantly improve BMD in the general population [34]. This suggests that maintaining adequate vitamin D levels is crucial, but supplementation should be considered alongside other factors influencing bone health. Optimal 25(OH) D levels are essential for maintaining bone strength and enhancing the efficacy of anti-resorptive therapies.

Glucocorticoids are invaluable in reducing inflammation and modulating the immune response, which makes them a first-line treatment for diseases like rheumatoid arthritis, systemic lupus erythematosus, asthma, and inflammatory bowel diseases [35]. However, glucocorticoids significantly increase bone porosity and fracture risk, leading to glucocorticoid-induced osteoporosis (GIOP). These drugs impair calcium metabolism by reducing intestinal absorption, increasing renal loss, and suppressing IGF-1, a key factor in collagen synthesis and bone formation. This cascade results in osteoblast apoptosis, collagen degradation, and compromised bone strength. In younger patients, GIOP commonly results in vertebral fractures [36]. The adverse effects of glucocorticoids on bone health necessitate vigilant monitoring and proactive management to prevent and treat GIOP. Regular DEXA scans are required for monitoring the efficacy of treatments such as bisphosphonates, calcium, and vitamin D supplementation [37].

While the review presents critical insights into the hormonal influences on osteoporosis, it also identifies several limitations. The predominance of observational studies restricts the ability to establish causal relationships between hormonal changes and osteoporosis. Cross-sectional studies offer valuable data snapshots, but they fail to account for the longitudinal effects of hormonal fluctuations over time. Furthermore, study heterogeneity is evident, with a disproportionate focus on postmenopausal women and limited research on osteoporosis in men or premenopausal women. This gender imbalance highlights the need to better understand the hormonal dynamics in male populations, as studies on androgen levels in men, such as those by Zitzmann [38], remain underrepresented.

Another limitation is the reliance on bone mineral density (BMD) as the primary diagnostic tool for osteoporosis. Emerging research incorporates advanced markers of bone quality, such as the trabecular bone score (TBS), which provides a more comprehensive assessment of fracture risk [39]. However, the underutilization of such techniques in reviewed studies may underestimate the complexity of bone health.

The review process itself was constrained by several factors. Excluding studies published more than a decade ago may have omitted foundational research crucial to understanding historical advancements in osteoporosis. Although the focus on recent studies ensures relevance, it risks neglecting earlier evidence that could provide valuable context. Additionally, language restrictions, including only English-language studies, may introduce a Western-centric bias. Excluding research from non-English-speaking regions, such as Japan and South Korea, limits the review's global applicability.

The findings of this review have significant implications for clinical practice, public health, policy development, and future research. Clinically, these results underscore the importance of regular hormonal assessments in individuals at risk for osteoporosis. Routine screening for key hormones, such as estrogen in postmenopausal women and aging men, could facilitate early interventions. Moreover, prioritizing the development and adoption of advanced diagnostic tools, such as TBS and biomarkers of bone turnover, can improve risk stratification and treatment outcomes.

Greater emphasis should also be placed on underrepresented populations, including men, premenopausal women, and diverse ethnic groups, to develop inclusive and equitable strategies. Investigating novel therapeutic approaches, such as gene therapy and personalized medicine, may pave the way for transformative advances in osteoporosis management. Finally, while this review underscores the central role of hormones, it also highlights the complexity of their interactions with genetic, environmental, and lifestyle factors [40].

CONCLUSIONS

Based on the reviewed literature, it can be concluded that hormones significantly impact the development and progression of osteoporosis. In particular, estrogen, testosterone, and glucocorticoids play a major role in both the pathogenesis and progression of the disease. The endocrine system involves a complex network of interactions, where each hormonal imbalance leads to specific consequences. For instance, estrogen has a protective effect on bone mineralization, while glucocorticoids exert a detrimental effect, contributing

to osteoporosis. Therefore, it is crucial to consider the body as a system of interconnected processes. Addressing these multifaceted interactions through focused research, health policy reforms, and clinical innovations offers substantial potential to enhance the prevention and treatment of osteoporosis on a global scale. From a policy standpoint, the findings underscore the importance of incorporating hormonal health into national osteoporosis prevention programs. Public health initiatives should highlight the significance of maintaining hormonal balance, alongside traditional risk factors such as calcium and vitamin D intake.

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CONFLICT OF INTEREST

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