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GLUCOCORTICOID INDUCED OSTEOPOROSIS: EXPLORING ALLOPATHIC THERAPIES AND TRADITIONAL MEDICINE SOLUTIONS

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ABSTRACT: Osteoporosis is a Skeletal condition that generally develops with aging. The primary osteoporosis is more prevalent in women after menopause which is known as postmenopausal osteoporosis, and it also happens after the age of 75. Secondary osteoporosis is brought on by long-term usage of drugs like glucocorticoids or chronic underlying illnesses. Oral GC use ranges from 0.5 to 0.9% in the community, and it rises to 2.7% among women over 50 years. The key mechanisms by which glucocorticoids directly affect bone formation involve the Wnt/β-catenin signalling pathway and the activation of PPAR-γ. Moreover, glucocorticoids directly contribute to bone resorption by encouraging the production of RANKL, M-CSF and reducing the synthesis of osteoprotegerin ligand by osteocytes and osteoblastic cells. First-line treatments for osteoporosis include weight-bearing physical activity, exercise, and exercises that help with posture and balance, as well as a diet high in calcium and vitamin D, stopping smoking, and consuming alcohol in moderation. Certain medications are required in the second phase such as anabolic, anti-resorptive, and combination drugs. Medicinal plants are essential for preserving health and serve as the main source of ingredients for both conventional and traditional medicine formulations. Traditional therapies, including herbal remedies and natural compounds, may offer benefits such as reducing bone resorption, enhancing bone formation, and minimizing side effects associated with conventional drugs.

INTRODUCTION: Osteoporosis is a Skeletal condition that generally develops with aging. It is typified by a marked decrease in bone strength, bone mass, or bone mineral density, increasing the risk of fractures for the affected person [1]. Around the world, osteoporosis is the most prevalent illness affecting the bones and joints, particularly in the elderly population. Every ethnicity and gender are impacted by osteoporosis, however, to varying degrees [2].

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Osteoporosis is often caused by estrogen shortage in older or postmenopausal women, which is important for maintaining bone homeostasis [4]. An aberrant bone remodelling process between osteoclast and osteoblast is brought on by a female hormone deficiency which results in significant bone loss leading to osteoporosis and osteopenia [4]. Two types of osteoporosis exist: primary and secondary [1].

According to the study, osteoporosis affected roughly one in three female and one in five males. Around 20 crore people worldwide are affected with osteoporosis [5]. The primary osteoporosis is more prevalent in women after menopause which is known as postmenopausal osteoporosis, and it also happens after the age of 75. The ratio of male to female cases is 2:1 [2].

Men and women are equally susceptible to secondary osteoporosis, which can develop at any age. Secondary osteoporosis is brought on by long-term usage of drugs like glucocorticoids or chronic underlying illnesses [2]. Numerous illnesses, such as asthma, autoimmune diseases, ulcerative colitis and rheumatoid arthritis are treated using glucocorticoids [8].

The several studies showed that children who got more than four sessions of glucocorticoids had an increased risk of fracture [3]. Glucocorticoids treatment increases bone resorption, lowers gastrointestinal absorption of calcium, suppresses gonadal hormones like estrogen, and inhibits osteoblast function, all of which contribute to increased bone loss [6]. The synthetic glucocorticoid hormone dexamethasone has been shown to promote the synthesis of collagenase while simultaneously suppressing the synthesis of collagen and fibronectin [7]. Glucocorticoids shorten osteoblasts' and osteocytes' lifespans and suppress osteoblastogenesis [9]. Apoptosis brought on by dexamethasone is linked to the activation of many caspase gene types [7]. Because of the long-term negative effects or ineffectiveness of synthetic drugs, scientists and physicians are interested in finding traditional medicines to treat osteoporosis [9].

Herbs have been utilized for thousands of years by various civilizations to treat a variety of illnesses [10]. Researches has indicated that a number of naturally occurring substances function as antioxidants to prevent osteoporotic effects [11]. Medicinal plants are essential for preserving health and serve as the main source of ingredients for both conventional and traditional medicine formulations [12]. The greatest source of a wide range of medications used to treat serious illnesses in India is medicinal plants [12]. This review aims to highlight how glucocorticoids induce osteoporosis and the effectiveness of herbal plants in treating osteoporosis and to outline conventional medicine for osteoporosis from various scholarly publications.

Composition of Bone: Bone comprises a mixture of extracellular matrix and cells. The extracellular matrix's composition determines bone's mechanical characteristics and the architectural and geometric features brought about by this tissue's spatial distribution. Bone cells are osteocytes, osteoclast, osteoblast and osteoprogenitor cells. The cells create bone and regulate its growth and decomposition. Mineralised and nonmineralized components are present in the extracellular matrix. Calcium hydroxyapatite is a crystalline substance that makes up the mineralised component. Phosphate and calcium ions are the key components of calcium hydroxyapatite. Osteoid refers to the nonmineralized component. Osteoid generates and secretes a non-mineralized substance. Proteins that are collagenous and non-collagenous make up osteoid [13].

Osteoporosis Brought on by Glucocorticoids: For many years, clinical practice has employed synthetic glucocorticoids (GCs) to treat gastrointestinal disorders, rheumatism, organ transplants, autoimmune disorders, and cancers [3]. Glucocorticoid (GC) exposure in both past and present, raises the risk of bone loss and fracture [14]. Oral GC use ranges from 0.5 to 0.9% common in the community, and it rises to 2.7% among women over 50 years [14]. During the first phase of treatment, chronic oral administration of glucocorticoid medication is linked to dose-related fast reduction in bone mass and an elevated risk of fracture. The risk of fractures stays high while on glucocorticoid medication but decreases when it is stopped, however it is uncertain if it returns to its initial levels [15]. Fractures were 29% less common among individuals who stopped taking glucocorticoids at 60 to182 days [15]. At doses of less than 15 mg per day, the incidence of fracture ranged from 4 to 10 per 1000 person-years; at doses of 15 mg per day, it was 16 and at cumulative doses of more than 5400 mg, it was 13.4 [15]. Despite the availability of several glucocorticoid induced osteoporosis guidelines, a small percentage of patients treated with glucocorticoids are still undergoing prophylactic measures for bone problems [14].

Pathophysiology: Two types of osteoporosis exist: primary and secondary [1]. The primary osteoporosis is more prevalent in women after menopause which is known as postmenopausal osteoporosis, and it also happens after the age of 75. Men and women are equally susceptible to secondary osteoporosis, which can develop at any age.

Secondary osteoporosis is brought on by long-term usage of drugs like glucocorticoids or chronic underlying illnesses. The annual loss of bone density in men is 0.30%, whereas in women it is 0.50%. Following menopause, bone loss can reach 4–6% annually, especially in the first five years following the commencement [13].

When glucocorticoids cause osteoporosis, there is a decline in bone production (osteoblast) together with an early but temporary rise in bone resorption (osteoclast) [15]. The distinction between bone mineral density (BMD) and fracture risk and GCs are characteristics of bone fragility in glucocorticoid induced osteoporosis. The aetiology of glucocorticoid induced osteoporosis explains these two points [14]. External mechanical pressures brought on by physical activity and endogenous hormonal changes both affect bone homeostasis. The capacity of bone to support specific loads is significantly reduced by processes that lower collagen or bone mineral levels. When the maximum stress levels are reached, bones break and fractures happen [13].

The key mechanisms by which glucocorticoids directly affect bone formation involve the Wnt/β-catenin signalling pathway and the activation of PPAR-γ (Peroxisome Proliferator-Activated Receptor Gamma). Moreover, glucocorticoids directly contribute to bone resorption by encouraging the production of RANKL (Receptor Activator of Nuclear Factor Kappa-B Ligand) and M-CSF (Macrophage Colony-Stimulating Factor) and reducing the synthesis of osteoprotegerin ligand (OPG) by osteocytes and osteoblastic cells. This leads to an increase in osteoclast activity and quantity. The impact gradually fades, likely because of a reduction in the number of osteoblasts and osteocytes. And few studies suggesting glucocorticoids influence the shape and mineralisation of osteocytes comes from animal models [15]. Because glucocorticoids cause ROS to accumulate, it causes oxidative stress. ROS triggers RANK/TRAF6/RANKL signalling, which enhances the IκB kinase/ nuclear factor kappa-B (IKK/NF-κB) pathway, nuclear factor of activated T cells of type 1 (NFATc1) expression, and genes unique to osteoclasts, such as acid phosphatase (ACP) activity. Meanwhile, glucocorticoids inhibited the activation of nuclear factor erythroid 2-related factor 2/heme oxygenase-1 (Nrf2/HO-1). Kelch-like ECH-associated protein 1 (Keap1) sequesters Nrf2 and also aids in its proteasomal degradation and ubiquitination within the cytoplasm. But under oxidative stress, the cytoprotective potential begins with Keap1's separation from Nrf2, which then promotes Nrf2 nuclear translocation, modifies ARE-dependent gene expression, and triggers HO-1 production, which is in charge of scavenging oxidative stress and ROS. One major factor contributing to the development and onset of osteoporosis is bone resorption, which is triggered by increased RANKL/RANK signalling and inhibited Nrf2/HO-1 signal [16].

Sien Lin *et al.* demonstrated that rats given the maximum dosage of prednisone had considerably lower levels of serum insulin-like growth factor 1 (IGF-1) and longitudinal bone growth (LGR). Drug effects on young patients can be assessed using growing rats as an animal model of glucocorticoid-induced osteoporosis [3]. Forough Pirhadi *et al.* showed that when rats were given dexamethasone, their femoral bone's metaphyseal trabeculae thickness was significantly lower than that of the control group. This resulted in a notable decline in histomorphometric indices, indicating the onset of osteoporosis [1]. Nabil A. Hasona *et al* showed that reduced catalase activity and a markedly lower hepatic GSH level are signs of enhanced oxidative stress brought on by dexamethasone [17]. Glucocorticoids indirectly produce osteoporosis by decreasing the sex steroid hormone synthesis, and hypogonadism alone can cause increased bone resorption. Increased calcium losses in the kidney and decreased intestinal absorption of calcium[14].

Diagnosis of Osteoporosis: In order to estimate bone mineral density, non-invasive dual-energy X-ray absorptiometry (DEXA) can be used. By using the bone mineral density test's T-score, osteoporosis can be differentiated from osteopenia. A bone mineral density test T-score of -2.500 or less indicates osteoporosis, while a T-score between -1.01 and -2.49 indicates osteopenia. Although it is crucial to remember that the findings of bone mineral density tests do not necessarily correspond with the likelihood of fractures, early detection of low bone mineral density can guide clinical intervention [18].

Osteoporosis Management: First-line treatments for osteoporosis include weight-bearing physical activity, exercise, and exercises that help with posture and balance, as well as a diet high in calcium and vitamin D, stopping smoking, and consuming alcohol in moderation. Certain medications are required in the second phase. There are three categories of medications: anabolic, anti-resorptive, and combination treatments. Sclerostin inhibitors and parathyroid hormone (PTH) analogues are examples of anabolic medication. Denosumab, SERMs (Selective estrogen receptor modulators), Bisphosphonates and Calcitonin are examples of anti-resorptive medications [18, 19, 20].

Anti-resorptive Drugs: Fractures can be prevented by up to 50% with antiresorptive medication [20]. Bisphosphonates are frequently used to raise BMD in primary and secondary osteoporosis, they do have an impact on bone flexibility [19]. Bisphosphonates stop the resorption of bone by blocking the activation of osteoclasts [20]. Clodronate, Etidronate, and tiludronate are a few types of first-generation bisphosphonates. Third- and second-generation bisphosphonates include zoledronic acid, pamidronate, ibandronate, risedronate, and alendronate. The way these medications work is by strongly binding to bone mineral and attaching to hydroxyapatite crystals, effectively preventing osteoclast activation and decreasing bone resorption, thereby reducing bone loss. Bisphosphonates can be given intravenously or orally. In postmenopausal women, alendronate reduces the risk of hip, nonvertebral, and vertebral fractures in comparison to vitamin D and calcium supplementation [18].

Denosumab, a human monoclonal antibody, prevents bone resorption by binding to and blocking RANKL (Receptor Activator of Nuclear Factor Kappa B Ligand), inhibiting the maturation of osteoclast precursors into adult osteoclasts [21]. Every six months, denosumab is injected subcutaneously [20]. Some side effects of denosumab include weakness and fatigue. Calcitonin, a thyroid hormone, is released by the thyroid gland, is recommended for treating osteoporosis in women who have been postmenopausal for at least five years [18]. Calcitonin is administered intranasally. The calcitonin binds to osteoclast precursors' calcitonin receptors which inhibits their maturation and functions [21]. One SERMs (selective oestrogen receptor modulator) that lessens postmenopausal bone loss is raloxifene. Women who have postmenopausal osteoporosis can use raloxifene instead of denosumab or bisphosphonates. Since raloxifene lowers the risk of breast cancer, it may be taken into consideration for women who are at high risk for the disease. Nevertheless, raloxifene raises the incidence of DVT (deep vein thrombosis) and there is a modest increase in mortality following stroke [20].

Anabolic Drugs: Teriparatide and abaloparatide are two examples of parathyroid hormone or parathormone analogues, which are anabolic treatments given subcutaneously once a day [21]. Teriparatide is approved to treat osteoporosis resulting from glucocorticoid use in those with a high fracture risk [6]. Teriparatide and abaloparatide act as an agonist on parathyroid hormone type 1 receptor. When the parathyroid hormone receptor is frequently stimulated, the Wnt pathway is activated, which has a net anabolic effect on bone. Additionally, osteoprotegerin (OPG) transcription is upregulated, which binds to RANKL and has an anti-resorptive impact [21]. Headaches, nausea, and short-term hypercalcemia are the most frequent side effects of parathormone analogues, occurring more often with teriparatide compared to abaloparatide. A humanized monoclonal antibody, romosozumab is an anabolic agent that prevents the activity of sclerostin. The role of sclerostin is to block Wnt pathway. Thus, romosozumab-induced sclerostin inhibition activates Wnt signalling pathway, resulting in an anabolic action that promotes bone growth and prevents bone resorption. Romosozumab may cause side effects such as injection site discomfort, necrosis of the jaw bone, dermatitis, and cardiovascular complications [17, 20, 21].

Role of Traditional Medicines in Glucocorticoid Induced Osteoporosis: Dieckol markedly changed OPG and RANKL levels and implied a protective effect on bones. Therefore, dieckol showed that it had an anti-osteoporosis effect on rats that had glucocorticoid induced osteoporosis [5]. Prednisolone-induced osteoporosis in rats was prevented by grape fruit juice, potentially through the RANKL/OPG pathway [8]. Mollugin could potentially be able to treat glucocorticoid-induced

osteoporosis through a mechanism that involves the PI3K/Akt pathway [11]. Ethanolic purslane extract and aqueous chicory extract counteracted dexamethasone induced bone deterioration by stimulating the Nrf2/HO-1 signalling pathway and restraining osteoclast differentiation prompted by RANKL [16]. Pinoresinol diglucoside increases bone mass and controls bone metabolism to prevent dexamethasone-induced osteoporosis [22]. Korean red ginseng (KRG) inhibited caspase-3 and -9 activation, preventing MC3T3-E1 cells from undergoing apoptosis caused by dexamethasone. Additionally, KRG promoted bone growth in animals with glucocorticoid -induced osteoporosis [23]. The processes that underlie curcumin's ability to prevent osteoporosis caused by dexamethasone include the activation of the Wnt/β-catenin pathway [24]. According to a study, annatto tocotrienol may benefit individuals receiving long-term glucocorticoid medication by preventing osteoporosis brought on by glucocorticoids [25]. Basil, chicory and parsley aqueous extracts contributed to bone preservation in rats affected by glucocorticoid exposure [26].

CONCLUSION: Glucocorticoid induced osteoporosis (GIOP) presents a major challenge in managing patients on long-term glucocorticoid therapy, requiring effective strategies to prevent bone loss and fractures. While conventional treatments such as bisphosphonates, denosumab, and teriparatide have proven efficacy in managing GIOP, there is growing interest in the role of traditional medicines as complementary or alternative options. Traditional therapies, including herbal remedies and natural compounds, may offer benefits such as reducing bone resorption, enhancing bone formation, and minimizing side effects associated with conventional drugs. However, while some traditional medicines show promise, further clinical studies and rigorous trials are necessary to establish their safety, efficacy, and optimal use in GIOP management. Ultimately, an integrative approach that combines the strengths of both conventional and traditional treatments, under careful medical supervision, could offer a more holistic solution to managing this debilitating condition.

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CONFLICT OF INTEREST: NIL

REFERENCES:

1. Forough Pirhadi, Ghasem Farjanikish, Mohammad Kamalpour and Alireza Rocky: The therapeutic effect of honey on dexamethasone induced osteoporosis in rats. International Journal of Nutrition Science 2023; 8(4): 252-259.
2. Fina Luthfiana, Riza Ambar Sari, Irawati Sholikhah, Katsuyoshi Matsunami, Sukardiman, Retno Widyowati: Effects of *Eleutherine bulbosa* (mill.) urb. bulb extract on mice glucocorticoid-induced osteoporosis models. Journal of Public Health in Africa 2023; 14(1): 2507.
3. Xiaofeng Jiang, Xiaojun Qi and Chao Xie: Lactobacillus plantarum LP45 inhibits the RANKL/OPG signaling pathway and prevents glucocorticoid-induced osteoporosis. Food & Nutrition Research 2023; 67: 9064.
4. Eunkuk Park and Chang Gun Lee: Osteoprotective effects of loganic acid on osteoblastic and osteoclastic cells and osteoporosis-induced mice. International Journal of Molecular Sciences 2020; 22: 233.
5. Hao Wang, Leigang Yang and Junwei Chao: Antiosteoporosis and bone protective effect of dieckol against glucocorticoid induced osteoporosis in rats. Frontiers in Endocrinology 2022; 13: 932488.
6. LeBoff MS, Greenspan SL, Insogna KL, Lewiecki EM, Saag KG, Singer AJ and Siris ES: The clinician's guide to prevention and treatment of osteoporosis. Osteoporosis International 2022; 33: 2049–2102.
7. Jimmy Zhu and Lyn March: Treating osteoporosis: risks and management. Australian Prescriber 2022; 45: 150–7.
8. Eslam Muhammad Bastawy and Rasha Rashad Ahmed: Grapefruit juice exerts anti-osteoporotic activities in a prednisolone-induced osteoporosis rat femoral fracture model, possibly *via* the RANKL/OPG axis. Cytotechnology 2019; 71: 769–783.
9. Renuka Munshi, Tanvi Patil, Chetan Garuda and Dushyant Kothari: An experimental study to evaluate the antiosteoporotic effect of Panchatikta Ghrita in a steroid induced osteoporosis rat model. Indian Journal of Pharmacology 2016; 48: 298-303.
10. Liping An and Liqiang Shi: Protective effect of Sika Deer bone polypeptide extract on dexamethasone-induced osteoporosis in rats. Electronic Journal of Biotechnology 2021; 52-58.
11. Zhen Jia, Ge Yan, Yusi Wang and Pan He: Mollugin attenuates glucocorticoid-induced osteoporosis in rats via Akt/P13K pathway. Tropical Journal of Pharmaceutical Research 2018; 17 (9): 1765-1770.
12. Bhumika, Sakshi Sharma and Rajesh Sharma: A review on pharmacological action of arjuna (*Terminalia arjuna* (Roxb.) Wt. and Arn.). World Journal of Pharmaceutical Research 2022; 11(9): 1760-1778.
13. Samar R. Saleh and Omnia M. Saleh: The therapeutic potential of two egyptian plant extracts for mitigating dexamethasone-induced osteoporosis in rats: nrf2/ho-1 and rank/rankl/opg signals. Antioxidants 2024; 13: 66.

14. Devon Patel, Courtney Gorrell, Jordan Norris and Jiayong Liu: A narrative review of the pharmaceutical management of osteoporosis. Annals of Joint 2023; 8: 25.
15. Juliet Compston: Glucocorticoid-induced osteoporosis: an update. Endocrine 2018; 61: 7–16.
16. Nabil A. Hasona, Ahmed A. Alrashidi, Thamer Z. Aldugieman, Ali M. Alshdokhi and Mohammed Q. Ahmed: *Vitis vinifera* extract ameliorate hepatic and renal dysfunction induced by dexamethasone in albino rats. Toxics 2017; 5: 11.
17. Shan-Shan Li, Shi-Hao He, Peng-Yu Xie, Wei Li, Xin-Xin Zhang, Tian-Fang Li and Dai-Feng L: Recent progresses in the treatment of osteoporosis. Frontiers in Pharmacology 2021; 12: 717065.
18. Yulita Bong, Sri Angky Soekanto and Erik Idrus: Effects of *Centella asiatica* (L.) leaf extract on bone calcium and phosphate levels of ovariectomized rats. International Journal of Applied Pharmaceutics 2019; 11(1): 2019.
19. Michael Weiser, William Johns, Bryan Hozack: Osteoporosis treatment strategies: current concepts. Surgi Coll 2024; 2(2).
20. Zhan-Feng Zhang, Ji-Kang Min, Dan Wang and Jian-Ming Zhong: Pinoresinol diglucoside exhibits protective effect on dexamethasone-induced osteoporosis in rats. Tropical Journal of Pharmaceutical Research 2016; 15(11): 2451-2457.
21. Clifford J and Rosen MD: The epidemiology and pathogenesis of osteoporosis. Endotext 2020;https://www.ncbi.nlm.nih.gov/books/NBK279134/
22. Zhiguang Chen and Jinqi Xue: Curcumin alleviates glucocorticoid-induced osteoporosis through the regulation of the Wnt signaling pathway. International Journal of Molecular Medicine 2015; 37: 329-338.
23. Ima Nirwana Soelaiman, Elvy Suhana Mohd Ramli, Farihah Suhaimi and Fairus Ahmad: Potential benefits of annatto tocotrienol in glucocorticoid induced osteoporosis: an animal study. Journal of Osteoporosis & Physical Activity 2017; 5: 2.
24. Walaa G. Hozayen and Mohamed A. El-Desouky: Antiosteoporotic effect of *Petroselinum crispum, Ocimum basilicum* and *Cichorium intybus* L. in glucocorticoid-induced osteoporosis in rats. BMC Complementary and Alternative Medicine 2016; 16: 165.
25. Zinan Qin and Siyu Li: Combination therapy of Wuweizi (*Schisandrae chinensis* fructus) and dexamethasone alleviated dexamethasone-induced glucocorticoid osteoporosis in rats with idiopathic pulmonary fibrosis. Hindawi BioMed Research International 2020; 2020, Article ID 6301697, 9 pages.
26. Syed Aamer Nawaz and Ayesha Saffura: Osteoporosis and its treatment using phytoestrogens of cowpeas. Journal of Pharmacognosy and Phytochemistry 2019; 8(5): 2165-2167.
27. Tsung-Rong Kuo and Chih-Hwa Chen: Bone biomarker for the clinical assessment of osteoporosis: recent developments and future perspectives. Biomarker Research 2017; 5: 18.
28. Amr Samir Elbahnasawy and Emiliya Ramzievna Valeeva: Protective effect of dietary oils containing omega-3 fatty acids against glucocorticoid-induced osteoporosis. Journal of Nutrition and Health 2019; 52(4): 323-331.
29. Martiniakova M, Babikova M and Omelka R: Pharmacological agents and natural compounds: available treatments for osteoporosis. Journal of physiology and Pharmacology 2020; 71(3): 307-320.

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