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Literature Review: Glucocorticoid-Induced Osteoporosis (GIOP) Therapy Based on Current Evidence

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Abstract. *Glucocorticoid-Induced Osteoporosis (GIOP) is the most common form of secondary osteoporosis resulting from long-term glucocorticoid use and is associated with an increased risk of fracture. The management of GIOP continues to evolve as new evidence emerges regarding pharmacological therapy, sequential strategies, and fracture risk-based approaches. This article aims to review glucocorticoid-induced osteoporosis (GIOP) therapy based on the current evidence. This study is a literature review with a search of articles from the PubMed database between 2023 and 2026. The literature used includes original research, systematic reviews, meta-analyses, and clinical guidelines relevant to glucocorticoid-induced osteoporosis (GIOP). Current evidence suggests that glucocorticoid-induced osteoporosis (GIOP) therapy includes bisphosphonates as first-line therapy, denosumab as an alternative in certain at-risk patients, and teriparatide in high-risk patients. Sequential therapy approaches (anabolic-to-antiresorptive) and treat-to-target strategies have been shown to be more effective in increasing and maintaining bone mass. Current evidence-based therapy for glucocorticoid-induced osteoporosis (GIOP) emphasizes an individualized, risk-based approach with a combination of pharmacological therapy, calcium and vitamin D supplementation, lifestyle modification, and regular monitoring. This comprehensive approach is essential to reduce fracture risk and improve quality of life in patients taking long-term glucocorticoids.*

Keywords: *Glucocorticoid-Induced Osteoporosis, Molecular Mechanisms, Bisphosphonates, Denosumab, Anabolic Therapy, Clinical Management*

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INTRODUCTION

Glucocorticoid-induced osteoporosis (GIOP) is the most common form of secondary osteoporosis resulting from long-term glucocorticoid use (Anastasilaki et al., 2023). Glucocorticoids remain an important therapeutic option in clinical practice because of their strong anti-inflammatory and immunosuppressive effects. These drugs are widely prescribed for various inflammatory, autoimmune, allergic, respiratory, and malignant conditions. Despite their clinical benefits, prolonged glucocorticoid exposure is associated with several serious adverse effects, particularly on bone metabolism. One of the most clinically significant complications is the reduction of bone mineral density, which increases skeletal fragility and fracture risk (Jha et al., 2023; Bahat et al., 2026). This risk may occur even at relatively low doses and becomes more concerning when glucocorticoids are used continuously or without adequate monitoring (Noetzlin et al., 2022; Ronchetti et al., 2021).

The clinical burden of GIOP is important because bone loss can occur rapidly after the initiation of glucocorticoid therapy. The risk of fracture, especially vertebral fracture, may increase by approximately 1.7–2.5 times and can appear within the first 3–6 months of treatment (Cho & Sung, 2021; Andreão et al., 2024). This indicates that GIOP is not only a long-term complication but also an early therapeutic concern that requires timely identification and prevention. In Indonesia, the incidence of GIOP is estimated to be relatively high due to the widespread use of glucocorticoids in various clinical and non-clinical settings, including the possibility of prolonged use without strict medical supervision. In addition, GIOP is often asymptomatic in its early stages, so many patients remain undiagnosed until they experience fragility fractures, particularly vertebral or hip fractures (Putri et al., 2026). This condition highlights the need for better awareness, early screening, and preventive strategies among patients receiving glucocorticoid therapy (Laurent et al., 2022; Puksun et al., 2025).

From a pathophysiological perspective, GIOP develops through complex disturbances in bone remodeling. Glucocorticoids act by binding to intracellular glucocorticoid receptors, which then regulate gene expression and suppress inflammatory pathways, including NF-κB and AP-1 (Strickland et al., 2022). However, in bone tissue, this mechanism contributes to harmful skeletal effects. Glucocorticoid exposure increases the expression of receptor activator of nuclear factor kappa-B ligand (RANKL) and decreases osteoprotegerin (OPG), resulting in enhanced osteoclast differentiation and activity. This process accelerates bone resorption. At the same time, glucocorticoids suppress osteoblast differentiation, reduce osteoblast function, and increase apoptosis of osteoblasts and osteocytes (Gado et al., 2022). These combined effects create an imbalance between bone formation and bone resorption, ultimately leading to decreased bone mineral density, impaired bone quality, and increased bone fragility (Chen et al., 2024; Cheng et al., 2022).

The management of GIOP therefore requires an integrated approach that focuses not only on treating established osteoporosis but also on preventing bone loss from the beginning of glucocorticoid therapy (Anastasilaki et al., 2023). A key principle is the use of glucocorticoids at the lowest effective dose and for the shortest possible duration, while still maintaining adequate control of the underlying disease. In addition, non-pharmacological strategies such as lifestyle modification, fall prevention, weight-bearing exercise, smoking cessation, and patient education are important components of care. Supportive therapy with calcium and vitamin D supplementation is commonly recommended to help maintain bone mineral density and reduce fracture risk, particularly in patients who require glucocorticoids for an extended period (Agrawal et al., 2025; Esteves et al., 2022).

Pharmacological therapy also plays a central role in the prevention and treatment of GIOP, especially among patients with moderate, high, or very high fracture risk (Laurent et al., 2022). Current evidence supports the use of bisphosphonates as first-line therapy in many patients because of their ability to inhibit osteoclast-mediated bone resorption and reduce fracture risk. However, newer therapeutic options, including denosumab and anabolic agents such as teriparatide, have become increasingly important in patients who are intolerant to bisphosphonates, have renal impairment, or present with high fracture risk. The selection of therapy should therefore be individualized based on age, glucocorticoid dose and duration, fracture history, bone mineral density, comorbidities, and overall fracture risk.

In this context, updated clinical guidelines from the American College of Rheumatology (ACR) provide evidence-based recommendations for the prevention and treatment of GIOP, including fracture risk stratification and the use of pharmacological therapy according to patient risk level (Humphrey et al., 2023). However, despite the availability of these guidelines, their implementation in routine clinical practice remains suboptimal. Many patients receiving long-term glucocorticoids are not adequately assessed for fracture risk or do not receive appropriate preventive therapy (Laurent et al., 2022). Therefore, a comprehensive review of current evidence is needed to strengthen clinical understanding of GIOP therapy, including first-line treatment, alternative pharmacological options, sequential therapy, supplementation, lifestyle modification,

and monitoring strategies. Based on this background, this literature review aims to examine current evidence regarding glucocorticoid-induced osteoporosis therapy and its implications for clinical management.

METHODS

This study used a literature review method to examine relevant publications on the molecular mechanisms of glucocorticoid-induced osteoporosis (GIOP) and current clinical management recommendations. A literature search was conducted through the NCBI PubMed database using keywords related to "glucocorticoid-induced osteoporosis" OR "glucocorticoid osteoporosis management" OR "glucocorticoid bone loss" OR "bisphosphonate glucocorticoid" OR "denosumab GIOP" OR "teriparatide glucocorticoid." The data used were secondary data that were systematically summarized. Inclusion criteria included original research articles, systematic reviews, and meta-analyses discussing glucocorticoid-induced osteoporosis (GIOP) and its clinical management, published between 2023 and 2026, available in full-text format, and in English. Exclusion criteria included articles without full-text access, pediatric osteoporosis studies, and articles that did not focus on skeletal aspects or were not research articles.

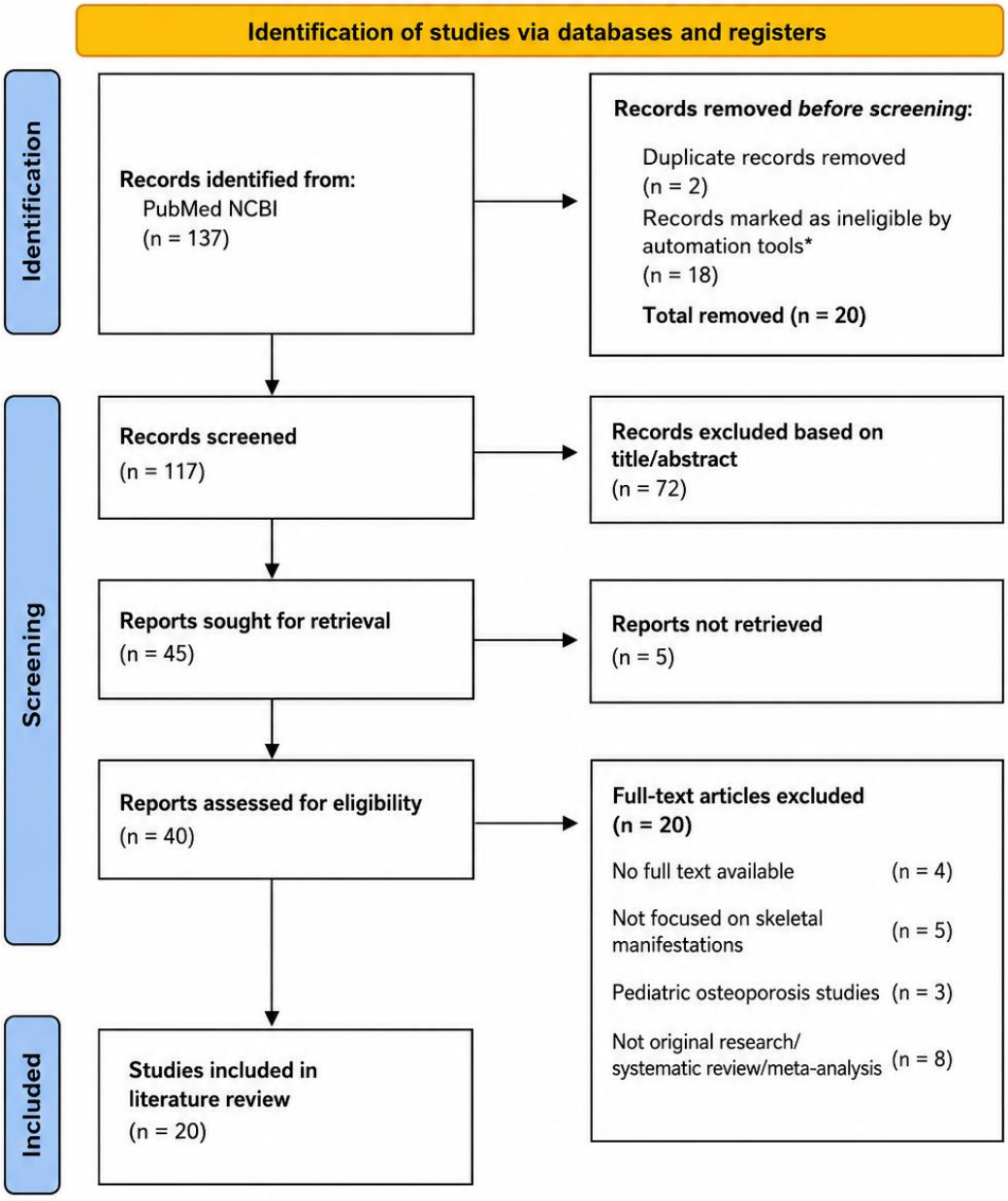

Figure 1. *PRISMA Flowchart*

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The results of this literature review are then synthesized narratively to obtain a comprehensive picture of glucocorticoid-induced osteoporosis (GIOP) therapy based on current evidence, including pharmacological approaches, sequential strategies, and principles of fracture risk-based clinical management.

RESULTS AND DISCUSSION

From the results of a literature search conducted in the PubMed database using the keywords: "glucocorticoid-induced osteoporosis" OR "glucocorticoid osteoporosis management" OR "glucocorticoid bone loss" OR "bisphosphonate glucocorticoid" OR "denosumab GIOP" OR "teriparatide glucocorticoid." Inclusion criteria included publications within the last 4 years (2023-2026), randomized controlled trials (RCTs), meta-analyses, systematic reviews, and relevant clinical practice guidelines:

Table 1. Clinical Recommendations for the Management of Glucocorticoid-Induced Osteoporosis (GIOP) Based on Current Evidence

No	Author and Year of Publication	Source	Topic	Research purposes	Research Design	Research result
1	Florez H, et al. (2025)	*Frontiers in Endocrinology*	*Risk factors for glucocorticoid induced osteoporosis in young adults*	To analyze whether factors associated with the development of glucocorticoid-induced osteoporosis (GIOP) and fragility fractures (FF) differ by age..	*Cross-sectional study*	The management of glucocorticoid-induced osteoporosis (GIOP) requires a holistic approach tailored to the dose and duration of glucocorticoid use, fracture risk, and patient comorbidities. Management includes pharmacological antiosteoporotic therapy, nutritional supplementation, inflammation control, and long-term monitoring to prevent bone loss and fragility fractures.
2	Song X, et al. (2025)	*Medicine (Baltimore)*	*Advances in the Study and Treatment of Glucocorticoid Osteoporosis*	Reviews recent developments in glucocorticoid osteoporosis research and therapy.	*Narrative Review*	Management of glucocorticoid-induced osteoporosis (GIOP) includes calcium supplementation of 1200–1500 mg/day and vitamin D supplementation of 800–1000 IU/day, as well as pharmacological therapy. Bisphosphonates such as alendronate and risedronate are first-line therapies because they are effective in preventing and treating glucocorticoid-induced osteoporosis (GIOP), while zoledronic acid can be used in patients who are intolerant to oral therapy. Teriparatide increases osteogenesis and new bone formation, while denosumab effectively reduces fracture risk and can be used in patients with kidney impairment. Furthermore, the journal also mentions the development of therapies targeting bone remodeling pathways, such as anti-sclerostin, which have the potential to become new therapies for glucocorticoid-induced osteoporosis (GIOP) in the future.
3	Ishihara N, et al. (2024)	*Pharmacy*	*Evaluation of Steroid-Induced Osteoporosis Prevention*	Evaluating the effectiveness of tracing reports as a collaborative tool between hospitals	Retrospective study of collaborative	Pharmacological management of glucocorticoid-induced osteoporosis (GIOP) is based on a fracture risk score, with therapy recommended if the score is ≥3. Bisphosphonates such

			Using Tracing Reports in Collaboration between Hospitals and Community Pharmacists	and community pharmacists to improve adherence to glucocorticoid-induced osteoporosis (GIOP) prevention guidelines	interventio n	as alendronate and risedronate are first-line therapy, while teriparatide, ibandronate, alfacalcidol, and calcitriol are used as alternative therapies. This article also emphasizes that modern management of glucocorticoid-induced osteoporosis (GIOP) does not rely solely on bone mineral density (BMD), as fractures can still occur even if BMD is maintained.
4	Humphrey MB, et al. (2023)	Arthritis & Rheumat ology	*2022 American College of Rheumatology Guideline for the Prevention and Treatment of Glucocorticoid -Induced Osteoporosis*	Develop evidence-based guidelines for the prevention and treatment of glucocorticoid-induced osteoporosis (GIOP) in children and adults using glucocorticoids.	*Clinical Practice Guideline (systematic review & GRADE-based consensus)*	Pharmacological management of glucocorticoid-induced osteoporosis (GIOP) is based on fracture risk stratification. Oral bisphosphonates remain the mainstay of therapy because they have the strongest evidence for preventing vertebral fractures, while PTH/PTHrP and denosumab have demonstrated superior bone mineral density (BMD) gains in high- or very high-risk patients. These guidelines also emphasize the importance of sequential therapy after discontinuation of denosumab, romosozumab, or PTH/PTHrP to prevent rapid bone loss and vertebral fractures.
5	Tanaka Y, et al. (2024)	*Journal of Bone and Mineral Metabolis m*	*The 2023 Guidelines for the management and treatment of glucocorticoid-induced osteoporosis*	To develop and revise guidelines for the management and treatment of glucocorticoid-induced osteoporosis (GIOP) based on current scientific evidence and expert consensus..	*Clinical Practice Guideline berbasis systematic review dan expert consensus (Delphi method)*	Pharmacological therapy for glucocorticoid-induced osteoporosis (GIOP) is recommended for patients with a fracture risk score ≥3. Bisphosphonates are first-line therapy for glucocorticoid-induced osteoporosis (GIOP) with moderate to high fracture risk. In high-risk patients, teriparatide and denosumab are recommended because they are more effective than bisphosphonates in increasing bone mineral density (BMD) and preventing vertebral fractures. Additionally, active vitamin D, such as eldecalcitol, is also beneficial in increasing BMD and preventing nonvertebral fractures. However, there is insufficient evidence to recommend anti-sclerostin antibodies in glucocorticoid-induced osteoporosis (GIOP).
6	Chen JF, et al. (2024)	*Diagnosti cs*	*Development and Comparison of Treatment Decision Tools for Glucocorticoid -Induced Osteoporosis*	Develop a therapeutic decision-making aid (SAFE tool) and compare its performance with existing recommendations from the American College of Rheumatology (ACR), the International	*Observatio nal, case-control, and single-center studies*	The results of this study indicate that the SAFE tool has better accuracy than GC-FRAX and ACR recommendations in identifying patients at high fracture risk. The SAFE tool is considered more sensitive in detecting patients who require early intervention because it considers more specific clinical factors related to glucocorticoid use, such as cumulative dose, duration of

				Osteoporosis Foundation–ECTS, and GC-FRAX in predicting fracture risk in patients using long-term glucocorticoids..		therapy, age, fracture history, and bone mineral density (BMD) values. This study confirms that the use of a more accurate prediction tool can help clinicians determine therapy more quickly, thereby preventing treatment delays and reducing the risk of fracture in patients with glucocorticoid-induced osteoporosis (GIOP)
7	Puksun K, et al. (2025)	*BMC Rheumatology*	*Comparison of Different Intervention Thresholds for the Treatment of Glucocorticoid-Induced Osteoporosis*	Comparing different intervention thresholds for glucocorticoid-induced osteoporosis (GIOP) therapy	*Cross-sectional Study*	The results showed that using a lower threshold increased sensitivity in detecting patients at high fracture risk compared to conventional thresholds. With a lower intervention threshold, more patients would meet the criteria for early antiosteoporosis therapy, thereby reducing the potential for fragility fractures. However, the study also highlighted that using too low a threshold could increase the likelihood of overtreatment in some low-risk patients. Therefore, determining the intervention threshold needs to be tailored to population characteristics, individual risk factors, and the balance between the benefits of therapy and potential side effects of treatment. This study supports the importance of optimizing screening strategies and fracture risk assessment in patients using chronic glucocorticoids.
8	Hofbauer LC, et al. (2025)	*The Lancet Diabetes & Endocrinology*	*Glucocorticoid-Induced Osteoporosis: Novel Concepts and Clinical Implications*	Reviews novel concepts and current clinical implications in glucocorticoid-induced osteoporosis (GIOP)	*Comprehensive Review*	This study emphasizes the development of the concept of precision medicine in the management of glucocorticoid-induced osteoporosis (GIOP), namely a therapeutic approach tailored to the risk profile and biological characteristics of each patient. In addition to bisphosphonates as standard therapy, new anabolic agents such as romosozumab are starting to be considered because they can increase bone formation while reducing bone resorption through sclerostin inhibition. This review also supports the implementation of sequential therapy, namely a strategy of administering anabolic therapy followed by antiresorptive therapy to maintain long-term increases in bone mineral density (BMD). In addition, regular fracture risk monitoring using DXA and FRAX adjusted to the glucocorticoid dose is considered important to improve the success of therapy and prevent fracture complications.

9	Kapszewicz M, et al. (2026)	*Journal of Clinical Medicine*	*Glucocorticoid -Induced Osteoporosis: Pathogenesis, the Impact of Different Administration Routes on Bone Mineral Density, and Fracture Risk and Treatment Options—A Narrative Review*	To evaluate the impact of glucocorticoid (GC) therapy on bone mineral density (BMD) and fracture risk, particularly when glucocorticoids are administered via non-oral routes..	*Narrative Review*	The review results show that decreased BMD and increased fracture risk are not only found with oral glucocorticoid use, but can also occur through non-oral routes such as inhalation and intra-articular administration, although with varying degrees of risk. In the management of glucocorticoid-induced osteoporosis (GIOP), bisphosphonates remain first-line therapy because they have the strongest evidence for increasing BMD and reducing fracture risk. Denosumab has also been reported to be effective, especially in patients with bisphosphonate intolerance or impaired renal function. Furthermore, anabolic agents such as teriparatide and romosozumab have shown promising results in increasing bone formation in patients at very high fracture risk. This review emphasizes the importance of individualized evaluation of the dose, duration, and route of glucocorticoid administration in determining the optimal strategy for the prevention and treatment of glucocorticoid-induced osteoporosis (GIOP)..
10	Gregson CL, et al. (2025)	*Archives Of Osteoporosis*	*The 2024 UK clinical guideline for the prevention and treatment of osteoporosis*	Updating UK clinical guidelines on fracture risk assessment, diagnosis, prevention and management of osteoporosis, including glucocorticoid-induced osteoporosis (GIOP), based on the latest scientific evidence in postmenopausal women and men aged ≥50 years.	*Clinical Practice Guideline*	According to the 2024/2025 NOGG guidelines, first-line therapy for glucocorticoid-induced osteoporosis (GIOP) includes oral bisphosphonates such as alendronate 70 mg/week or risedronate 35 mg/week, and intravenous zoledronate 5 mg/year, as they are considered the most cost-effective. Therapy is recommended to be initiated immediately, along with glucocorticoid use, in high-risk patients, including those with a history of fragility fracture, women aged ≥70 years, postmenopausal women, or men aged ≥50 years receiving high-dose prednisolone ≥7.5 mg/day for >3 months, or patients with FRAX MOF/hip values above the intervention threshold, without the need for DXA examination. Teriparatide is recommended as a first-line alternative in patients with very high fracture risk, such as a FRAX above VHRT, recent or multiple vertebral fractures, and a BMD T-score ≤−3.5. Glucocorticoid-induced osteoporosis (GIOP) has been shown to be superior to alendronate in RCTs. Denosumab 60 mg every 6 months can be used if bisphosphonates are not suitable. The duration of therapy is generally a minimum of 3–5 years for both oral

						and IV bisphosphonates and can be extended in high-risk patients, with sequential therapy planned, including antiresorptive therapy after anabolic therapy and FRAX and BMD monitoring every 12–18 months. This recommendation is supported by bridging studies, meta-analyses, and RCTs showing a significant reduction in the risk of vertebral, non-vertebral, and hip fractures.
11	Dong L, et al (2024)	*Frontiers in Pharmacology*	*Denosumab, teriparatide and bisphosphonates for glucocorticoid-induced osteoporosis: a Bayesian network meta-analysis*	To compare the effectiveness and safety of denosumab, teriparatide, and various bisphosphonates in patients with glucocorticoid-induced osteoporosis (GIOP).	*Systematic Review*	The results of a Bayesian network meta-analysis of 11 randomized controlled trials (RCTs) with a total of 2,877 patients showed that all therapies, namely alendronate, risedronate, etidronate, zoledronate, teriparatide, and denosumab, were effective in increasing bone mineral density (BMD) in patients with glucocorticoid-induced osteoporosis (GIOP). Teriparatide and denosumab showed the highest effectiveness in increasing lumbar spine and femoral neck BMD and were the best in reducing the risk of vertebral fracture compared to other therapies. In addition, alendronate and denosumab provided the best increase in total hip BMD, while alendronate and teriparatide had the lowest rates of adverse events and serious adverse events. This study concluded that teriparatide and denosumab are superior therapies for increasing BMD and reducing the risk of vertebral fracture in patients with glucocorticoid-induced osteoporosis (GIOP).
12	Yuan C, et al (2023)	*Journal of Orthopaedic Surgery and Research*	*Clinical efficacy of denosumab, teriparatide, and oral bisphosphonates in the prevention of glucocorticoid-induced osteoporosis: a systematic review and meta-analysis*	To compare the effectiveness and safety of denosumab, teriparatide, and oral bisphosphonates in patients with glucocorticoid-induced osteoporosis (GIOP), especially in terms of increasing bone mineral density (BMD), reducing the risk of fractures, and the incidence of adverse events..	*Systematic review dan meta-analysis randomized controlled trials (RCTs)*	Teriparatide demonstrated greater effectiveness in increasing lumbar spine BMD and total hip BMD and was more effective in reducing the risk of vertebral fracture compared to oral bisphosphonates. Denosumab also provided a higher increase in lumbar spine BMD compared to bisphosphonates with relatively equivalent safety and tolerability levels between therapies. This study also emphasizes the importance of providing calcium and vitamin D supplementation as basic therapy in all patients with glucocorticoid-induced osteoporosis (GIOP), and recommends sequential therapy after discontinuing anabolic steroids or denosumab to maintain the therapeutic effect and prevent further decline in BMD. Overall, teriparatide and denosumab are considered potential first-line therapy options in

						patients with glucocorticoid-induced osteoporosis (GIOP), especially in patients with a less than optimal response to previous antiosteoporotic therapy.
13	Ahmed AE, et al (2025)	*Cureus*	*Denosumab Versus Bisphosphonates in Glucocorticoid-Induced Osteoporosis: A Systematic Review*	To compare the effectiveness and safety of denosumab with bisphosphonates in adult patients with glucocorticoid-induced osteoporosis (GIOP), particularly with respect to changes in bone mineral density (BMD), bone turnover markers, bone microarchitecture, fracture risk, and side effects of therapy..	*Systematic review*	Denosumab demonstrated superior efficacy compared to bisphosphonates in glucocorticoid-induced osteoporosis (GIOP), particularly in increasing bone mineral density (BMD) in the lumbar spine and total hip. Denosumab also provided stronger suppression of bone turnover markers and better preservation of bone microarchitecture.
14	Yamasaki, S. (2023)	Annals of Hematology	*Bisphosphonate use for glucocorticoid-induced osteoporosis in older patients with immune thrombocytopenia: a clinical perspective*	To review and summarize the current evidence regarding the prevention and management of glucocorticoid-induced osteoporosis (GIOP) in elderly patients with immune thrombocytopenia (ITP), particularly regarding long-term glucocorticoid use and osteoporosis treatment strategies.	*Narrative review*	Bisphosphonates are first-line therapy for the prevention and treatment of glucocorticoid-induced osteoporosis (GIOP) because they have been shown to increase bone mineral density (BMD) and reduce the risk of vertebral fractures. Calcium supplementation of 1000–1200 mg/day and vitamin D supplementation of 600–800 IU/day are also recommended for all patients taking glucocorticoids for ≥3 months to help prevent bone loss. In patients with a high fracture risk or intolerance to bisphosphonates, alternative therapies such as zoledronic acid, teriparatide, and denosumab may be considered. Teriparatide is primarily used for severe osteoporosis (T-score ≤ −3.5 or T-score ≤ −2.5 with fragility fractures), patients who are intolerant to bisphosphonates, or patients who continue to experience fractures after one year of bisphosphonate therapy. Meanwhile, denosumab is an alternative option in patients with high fracture risk, although discontinuation of therapy requires a special strategy due to the risk of rebound vertebral fracture.
15	Chen CL, et al (2024)	*Frontiers*	*Superiority of denosumab over bisphosphonates in preventing and treating glucocorticoid-*	The study aimed to evaluate the effectiveness and safety of denosumab compared to bisphosphonates in the prevention and treatment of	*Systematic review dan meta-analysis*	Denosumab has a higher effectiveness than bisphosphonates in the treatment of glucocorticoid-induced osteoporosis (GIOP). Denosumab provides greater increases in bone mineral density (BMD) in the lumbar spine and total hip and shows a more significant decrease in bone turnover

			induced osteoporosis: a systematic review and meta-analysis with GRADE quality assessment	glucocorticoid-induced osteoporosis (GIOP).		markers such as P1NP, CTx, and TRACP-5b compared to bisphosphonates. Furthermore, the safety profile of denosumab is considered similar to that of bisphosphonates, as it does not significantly increase the risk of adverse events, infections, or fractures. These results suggest that denosumab may be an effective alternative therapy in patients with long-term glucocorticoid use.
16	Kast S, et al (2023)	*Rheumatology Advances in Practice*	*Exercise effects on glucocorticoid-induced bone loss in adults: a systematic review and meta-analysis*	To determine the effect of exercise combined with glucocorticoid therapy on BMD of the lumbar spine and femoral neck in patients with glucocorticoid-induced osteoporosis (GIOP).	*Systematic review dan meta-analysis*	*Exercise, particularly resistance training and lumbar strengthening exercises, can help maintain and increase bone mineral density (BMD) in the lumbar spine in patients with glucocorticoid-induced osteoporosis (GIOP). The combination of exercise and glucocorticoid therapy provides superior results compared to glucocorticoid therapy alone, therefore, exercise is recommended as an adjunct therapy for glucocorticoid-induced osteoporosis (GIOP).*
17	Liang H, et al (2023)	*Thieme*	*Pharmacological Interventions for Glucocorticoid-Induced Osteoporosis: An Umbrella Review*	Evaluate the effectiveness and safety of pharmacological therapy in glucocorticoid-induced osteoporosis (GIOP) based on current evidence to help select the best clinical therapy..	*Umbrella review*	Pharmacological therapies for glucocorticoid-induced osteoporosis (GIOP) include calcium and vitamin D, bisphosphonates, teriparatide, and denosumab. The combination of vitamin D and bisphosphonates has been reported to be more effective than either therapy alone in increasing bone mineral density (BMD). Teriparatide has been shown to be more effective than alendronate in increasing BMD and reducing the risk of vertebral fractures, while denosumab provides better increases in lumbar spine and total hip BMD than bisphosphonates with a similar safety profile. Overall, moderate to high evidence suggests that bisphosphonates, teriparatide, and denosumab are effective therapies in patients with glucocorticoid-induced osteoporosis (GIOP).
18	Zhang T, et al (2023)	*Frontiers*	*Pulsed Electromagnetic Fields in Glucocorticoid-Induced Osteoporosis*	To review the mechanisms and effectiveness of PEMFs in improving bone loss due to glucocorticoid use and to evaluate its potential as a therapy for glucocorticoid-induced osteoporosis (GIOP).	*Narrative review*	*Pulsed electromagnetic fields (PEMFs) are a non-pharmacological, non-invasive therapy with potential adjunctive use in glucocorticoid-induced osteoporosis (GIOP). PEMFs have been reported to reduce bone loss, improve bone quality, and help maintain bone mineral density during long-term glucocorticoid use. However, clinical evidence is still limited.*
19	Squadrito F, et al (2023)	*Elsevier*	*Effects of genistein aglycone in glucocorticoid*	To evaluate the effect of genistein on bone mineral density (BMD), bone	*Randomized clinical trial*	Genistein is a natural phytoestrogen of the isoflavone group that has the potential to be used as an adjunct therapy for glucocorticoid-induced

			induced osteoporosis: A randomized clinical trial in comparison with alendronate	turnover markers, and metabolic profiles in patients with glucocorticoid-induced osteoporosis (GIOP) and compare it with alendronate..		osteoporosis (GIOP). Administration of 54 mg/day of genistein for 24 months has been reported to increase bone mineral density (BMD) in the lumbar spine, femoral neck, and total hip with comparable effectiveness to alendronate. In addition to increasing bone formation markers such as osteocalcin and Bone-ALP, genistein also reduces sclerostin levels and improves patients' metabolic profiles. Therefore, genistein is considered a potential alternative therapy for glucocorticoid-induced osteoporosis (GIOP), although bisphosphonates remain recommended as first-line therapy.
20	Shrestha P, et al (2026)	*Elsevier*	*Management of glucocorticoid-induced osteoporosis in rheumatic diseases*	Reviewing risk factors, prevention, fracture risk evaluation, non-pharmacological therapy, and the latest pharmacological therapy in GIOP based on the latest evidence and guidelines.	*Narrative article*	Management of glucocorticoid-induced osteoporosis (GIOP) includes fracture risk assessment from the start of glucocorticoid use, calcium and vitamin D supplementation, weight-bearing exercise, fall prevention, and pharmacological therapy in moderate- to high-risk patients. Oral bisphosphonates such as alendronate and risedronate are recommended as first-line therapy due to their efficacy and cost-effectiveness, while intravenous zoledronic acid is used if oral therapy is intolerant. Teriparatide provides better bone mineral density (BMD) increases and vertebral fracture reduction than alendronate and is therefore prioritized for patients with very high fracture risk. Denosumab can be used as an alternative if bisphosphonates are not suitable, while romosozumab is starting to be considered as a new anabolic therapy for glucocorticoid-induced osteoporosis (GIOP).

Table 2. Comparison of Pharmacological Therapy in Glucocorticoid-Induced Osteoporosis (GIOP)

Therapy	Mechanism	Indication	Superiority	Weakness
Bisphosphonates (alendronate, risedronate, zoledronic acid)	Inhibits osteoclast activity thereby reducing bone resorption	First-line therapy in patients at moderate–high risk of fracture with glucocorticoid use for ≥3 months	The most extensive evidence, effective in increasing BMD and reducing fracture risk, relatively lower cost	Gastrointestinal side effects, risk of osteonecrosis of the jaw and atypical fractures with long-term use, caution in renal impairment
Denosumab	Inhibits RANKL thereby	Patients at high risk of fracture,	Increased BMD higher than	Risk of rebound bone

	suppressing osteoclast formation and activity.	bisphosphonate intolerance, or renal impairment	bisphosphonates, effective in reducing bone turnover markers, given every 6 months	loss after discontinuation, requiring continued therapy, potential for hypocalcemia
Teriparatide	Anabolic agent that stimulates osteoblast activity and new bone formation	Very high fracture risk, history of multiple fractures, or very low T-score	Very effective in increasing BMD in severe cases, increasing bone formation	Relatively expensive price, daily injections, limited duration of 18–24 months maximum
Romosozumab	Inhibits sclerostin thereby increasing bone formation and decreasing bone resorption.	Potential for very high risk patients or those refractory to other therapies	Anabolic and antiresorptive effects simultaneously, significant increase in BMD	Data on GIOP are still limited, costs are high, concerns about cardiovascular risks

Current Clinical Management Recommendations for Glucocorticoid-Induced Osteoporosis (GIOP)

Management of glucocorticoid-induced osteoporosis (GIOP) focuses on preventing bone loss, early detection, and therapy based on the patient's fracture risk level. Initial evaluation includes bone mineral density (BMD) using dual-energy X-ray absorptiometry (DXA) and fracture risk assessment using FRAX adjusted for glucocorticoid dose (Chen et al., 2024). In addition, history of glucocorticoid use, history of previous fractures, lifestyle, and other risk factors need to be evaluated to determine the optimal therapeutic strategy.

First-Line Therapy for GIOP

Bisphosphonates

Bisphosphonates (e.g., alendronate 70 mg/week or risedronate 35 mg/week, and zoledronic acid 5 mg/year) are first-line therapy in intermediate- to high-risk patients. They are recommended for adult patients taking glucocorticoids for ⩾3 months and with an intermediate fracture risk. The duration of use is generally 1–3 years, depending on clinical response. Bisphosphonates work by inhibiting osteoclasts, thereby reducing bone resorption and have been shown to increase BMD and reduce fracture risk (Tanaka et al., 2024; Laurent et al., 2022).

Calcium and Vitamin D Supplementation

Calcium supplementation of 1000 – 1200 mg/day and vitamin D supplementation of 800 – 1000 IU/day is recommended for all patients taking glucocorticoids for ⩾3 months as either primary therapy or prevention of bone loss. Combining supplementation with antiosteoporotic therapy has been shown to be more effective in maintaining BMD than either therapy alone (Liang et al., 2023).

Therapy for High and Very High Fracture Risk

Denosumab

Denosumab 60 mg subcutaneously every 6 months is used in patients with high fracture risk, bisphosphonate intolerance, or renal impairment. Denosumab works by inhibiting receptor

activator of nuclear factor kappa-B ligand (RANKL), thereby suppressing osteoclast activity. Several systematic reviews and meta-analyses have shown that denosumab provides greater increases in lumbar spine and total hip BMD than bisphosphonates, with a relatively similar safety profile (Ahmed et al., 2025; Yang et al., 2024; Kobayashi et al., 2024; Chai et al., 2025). In addition, denosumab is also more effective in reducing bone turnover markers and maintaining bone microarchitecture.

Teriparatide

Teriparatide 20 µg subcutaneously daily is recommended for very high-risk patients, such as those with a history of multiple fractures or very low T-scores. The duration of use is limited to a maximum of 18–24 months. This drug acts as an anabolic agent that stimulates bone formation through osteoblast activation and has been shown to be more effective in increasing BMD in severe conditions than antiresorptive therapy.

Potential New Therapies and Complementary Therapies

Romosozumab

Romosozumab is beginning to be considered as a new anabolic therapy in patients with glucocorticoid-induced osteoporosis (GIOP) at very high risk of fracture. This drug works by inhibiting sclerostin, thereby increasing bone formation and decreasing bone resorption. Early evidence suggests the potential for significant increases in BMD, although clinical data in GIOP are still limited (Shrestha et al., 2026).

Genistein

Genistein is an isoflavone phytoestrogen with potential as an adjunct therapy for glucocorticoid-induced osteoporosis (GIOP). A randomized clinical trial showed that administering 54 mg of genistein daily for 24 months increased BMD in the lumbar spine, femoral neck, and total hip with an effectiveness comparable to that of alendronate (Squadrito et al., 2023; Squadrito et al., 2023; Athalah et al., 2024). In addition, genistein also increases bone formation markers and improves the metabolic profile of patients.

Non-Pharmacological Therapy

Exercise

Exercise, especially resistance training and weight-bearing exercise, is recommended as an adjunct therapy for patients with glucocorticoid-induced osteoporosis (GIOP). Systematic reviews and meta-analyses show that physical exercise can help maintain and increase lumbar spine BMD compared to glucocorticoid therapy alone (Kast et al., 2023). In addition to improving bone health, exercise also helps improve balance and reduce the risk of falls.

Pulsed Electromagnetic Fields (PEMFs)

Pulsed electromagnetic fields (PEMFs) are a non-invasive therapy that is beginning to be developed as an adjunct to glucocorticoid-induced osteoporosis (GIOP). PEMFs have been reported to reduce bone loss, maintain bone quality, and increase BMD with long-term glucocorticoid use. However, clinical evidence is currently limited, so their use has not yet become a standard therapy (Zhang et al., 2023).

Monitoring and Evaluation

Monitoring is performed every 6–12 months using DXA to assess changes in BMD. Evaluation also includes therapy adherence, vitamin D status, and fall risk. In high-risk patients, monitoring may be performed more frequently depending on the clinical condition (Paccou et al., 2024).

Overall, the management of glucocorticoid-induced osteoporosis (GIOP) requires a comprehensive approach tailored to the dose and duration of glucocorticoid use, the patient's

fracture risk level, and comorbid conditions, through a combination of pharmacological therapy, supplementation, and long-term monitoring (Su et al., 2025; Florez et al., 2025; Ishihara et al., 2024).

CONCLUSION

Based on the results of a literature review, glucocorticoid-induced osteoporosis (GIOP) therapy based on current evidence emphasizes the importance of a comprehensive approach through early detection, fracture risk assessment, and therapy selection tailored to the patient's severity. Long-term glucocorticoid use causes impaired bone remodeling in the form of increased bone resorption and decreased bone formation, which leads to decreased bone mineral density (BMD) and an increased risk of fracture.

SUGGESTION

In the management of glucocorticoid-induced osteoporosis (GIOP), bisphosphonates remain recommended as first-line therapy due to their efficacy, cost-effectiveness, and proven fracture risk reduction. In patients with high to very high fracture risk, denosumab and teriparatide have demonstrated superior efficacy in increasing BMD and reducing vertebral fracture risk compared to conventional antiresorptive therapy. Furthermore, recent evidence suggests the potential of novel therapies such as romosozumab, genistein, and sequential therapy (anabolic-to-antiresorptive) to maintain long-term therapeutic effects. Non-pharmacological interventions also play an important role in the management of glucocorticoid-induced osteoporosis (GIOP), including calcium and vitamin D supplementation, lifestyle modification, exercise, especially resistance and weight-bearing exercise, and fall prevention. Additionally, adjunctive therapies such as pulsed electromagnetic fields (PEMFs) are being developed as a non-invasive therapy with the potential to help maintain bone quality, although clinical evidence remains limited. Routine monitoring through DXA BMD examination, fracture risk evaluation, and adherence assessment are needed to ensure treatment effectiveness. Thus, the application of glucocorticoid-induced osteoporosis (GIOP) therapy based on the latest evidence is expected to reduce the risk of fractures, maintain bone quality, and improve the quality of life of patients optimally.

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