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Una revisión de alcance sobre la eficacia, efectividad y seguridad de diferentes terapias farmacológicas para el manejo de pacientes con osteoporosis secundaria por trasplantes

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ABSTRACT

Introduction: transplant-induced osteoporosis is a frequent metabolic complication influenced by chronic glucocorticoid use, pretransplant comorbidities, and immunosuppressive regimens. Its management is complex due to pre-existing bone loss and a high risk of fractures, which vary depending on the type of transplant and postoperative period. All previously published studies investigating bone disease in transplant popula- tions, regardless of the organ, are limited in size and none of them have robust data regarding the effectiveness of osteoporosis medications in reducing fracture risk.

Objective: to synthesize current evidence on the efficacy, effectiveness, and safety of pharmacological therapies used in transplant-induced osteoporosis, identifying knowledge gaps and areas for future research.

Methods: following JBI scoping guidelines, we included studies of adult patients with transplant-induced osteoporosis treated with bisphosphonates, denosumab, and dual-action sclerostin-targeting monoclonal antibodies that both prevent bone loss and stimulate new bone formation, among other therapies. This review included adult transplant recipients treated with bisphosphonates, RANK-ligand inhibitors (denosumab), and dual-mechanism monoclonal antibodies against sclerostin—agents that not only inhibit osteoclastic bone resorption but also actively promote osteoblastic bone formation—alongside other pharmacotherapies. Efficacy was assessed based on fracture risk reduction and BMD improvement, effectiveness in real-world clinical practice, and safety through adverse event incidence. A total of 24 studies on efficacy, 3 on effectiveness, 1 on safety, and 4 evaluating both efficacy and safety were included.

Results: a total of 24 studies on transplant-induced osteoporosis were analyzed. Among bisphosphonates, pamidronate increased lumbar spine BMD (+8.8 %, $p < 0.015$) and femoral BMD (+8.2 %, $p = 0.01$), while alendronate improved lumbar BMD (+4.2 %, $p < 0.0001$). Ibandronate increased total femur BMD (+1.3 %, $p = 0.01$) and distal radius BMD (+0.6 %, $p = 0.039$). Denosumab significantly improved hip BMD

(+0.56 g/cm², p = 0.02) and spine BMD (+0.79 g/cm², p = 0.01). In terms of safety, pamidronate was well tolerated, with mild hypocalcemia in 8.6 % of cases. Alendronate was associated with dyspepsia in 15 % of patients, while denosumab showed no severe adverse effects. Regarding clinical effectiveness, ibandronate reduced fracture rates (7.4 % vs. 25.8 %, p = 0.04).

Conclusion: bisphosphonates and denosumab are effective in improving BMD, but their impact on fracture reduction is variable. The heterogeneity of studies and short follow-up periods limit the generalizability of results. The safety profile of these treatments is generally favorable, though additional studies are needed to assess long-term effectiveness and outcomes in underrepresented populations, such as lung and intestinal transplant recipients.

Keywords: Transplant-induced osteoporosis. Bisphosphonates. Denosumab. Bone mineral density. Bone fractures.

RESUMEN

Introducción: la osteoporosis inducida por glucocorticoides (GIOP) es una complicación frecuente y grave del uso prolongado de corticosteroides, que provoca una disminución de la densidad mineral ósea (DMO) y un mayor riesgo de fracturas.

Objetivo: esta revisión exploratoria tuvo como objetivo evaluar la eficacia, efectividad y seguridad de los tratamientos farmacológicos utilizados para el manejo de la GIOP.

Métodos: siguiendo la metodología del Instituto Joanna Briggs (JBI), se analizaron 40 estudios obtenidos de bases de datos como PubMed, Embase y Cochrane. Los tratamientos evaluados incluyeron bisfosfonatos, teriparatida y denosumab en pacientes adultos con GIOP.

Resultados: los resultados muestran que los bisfosfonatos—especialmente risedronato, alendronato y ácido zoledrónico—mejoran la DMO de la columna

lumbar hasta en un 4,8 % y reducen el riesgo de fracturas vertebrales hasta en un 82,4 %. La teriparatida demostró mayor eficacia, con aumentos de la DMO entre 7,8 % y 11 % y una reducción significativa del riesgo de fracturas, particularmente en pacientes con supresión severa de la formación ósea. El denosumab también mejoró la DMO y los marcadores de recambio óseo, siendo una alternativa efectiva para pacientes que no toleran los bisfosfonatos. Todos los tratamientos mostraron un perfil de seguridad favorable, con efectos adversos generalmente leves, como síntomas gastrointestinales y cuadros similares a la gripe.

Conclusión: en conclusión, los bisfosfonatos siguen siendo la terapia de primera línea por su eficacia y seguridad. La teriparatida es preferible en pacientes de alto riesgo, y el denosumab representa una opción válida en casos de intolerancia. Se destaca la importancia de un tratamiento individualizado según el riesgo de fractura y las características del paciente.

Palabras clave: Osteoporosis inducida por trasplante. Bifosfonatos. Denosumab. Densidad mineral ósea. Fracturas óseas.

INTRODUCTION

Transplant-induced osteoporosis is a complex metabolic condition frequently associated with chronic glucocorticoid use and factors related to pre-transplant end-stage diseases. While it shares certain characteristics with glucocorticoid-induced osteoporosis (GIOP), this condition is chronic, irreversible, and influenced by a combination of factors, such as pre-existing bone loss, immunosuppressive regimens, and transplant-associated comorbidities (1). Fractures are a common complication in this population, with incidence rates varying depending on the type of transplant and the postoperative period. For instance, in heart and liver transplants, lumbar spine bone mineral density (BMD) may recover over time, whereas fractures are more prevalent during the first years after transplantation (2-5). In kidney transplants, fractures occur more frequently at appendicular sites,

related to persistent hyperparathyroidism and cortical and trabecular bone loss.

Regarding bone loss patterns, the first 6 to 12 months post-transplant represent a critical period for BMD reduction. For example, in heart transplants, trabecular bone loss may exceed 6 % in the spine and femoral neck during the first year, later stabilizing with maintenance doses of glucocorticoids. In liver transplants, fractures are common in the first year (21 %) and may reach 33 % by the fourth year (1).

The pharmacological management of transplant-associated osteoporosis faces multiple challenges, partly due to variability in therapeutic responses. While both intravenous and oral bisphosphonates have demonstrated efficacy in improving BMD (6), adynamic bone disease remains a major concern. Denosumab has emerged as a promising option, with studies reporting significant increases in hip and spine BMD, along with a sustained reduction in bone turnover markers (7).

This agent may also be beneficial in hematopoietic stem cell transplants, where bone loss is more pronounced at the femoral neck, and fracture rates are significantly higher than in the general population (8,9). However, the available evidence remains limited, particularly in specific populations such as lung and intestinal transplants, where osteoporosis and fracture rates are particularly high (10,11).

This review aims to synthesize the current evidence on the efficacy, effectiveness, and safety of different antiresorptives therapies for transplant-associated osteoporosis, identifying knowledge gaps and areas for future research.

METHODS

This scoping review was conducted in accordance with the Joanna Briggs Institute (JBI) protocol for scoping reviews (12).

Population, concept, context

We applied the PCC framework. The Population comprised adults (≥ 18 years) diagnosed with transplant-associated osteoporosis (T-score ≤ -2.5) with or without fractures, receiving pharmacological therapies. The Concept included three domains: efficacy (trial-condition BMD gains and fracture risk reduction at 12 and 24 months), effectiveness (real-world fracture incidence and BMD changes), and safety (frequency and severity of treatment-related adverse events). The Context spanned hospitalized, emergency, and outpatient settings worldwide, across all ages, sexes, and cultures.

Eligibility criteria

We included only prospective controlled clinical trials—randomized or nonrandomized with parallel or crossover designs—published from database inception through April 30, 2025. Eligible interventions encompassed:

- *Antiresorptive agents:* bisphosphonates (pamidronate, alendronate, etidronate, zoledronate, ibandronate).
- *RANK-ligand inhibition:* denosumab.
- *Dual-action sclerostin inhibitors:* monoclonal antibodies targeting sclerostin, recognized for their combined antiresorptive and anabolic effects on bone.
- *Selective estrogen receptor modulators:* estradiol and pyridine derivatives.

Anabolic drugs (e.g., parathyroid hormone analogs) were explicitly excluded, as no prospective controlled trials of these agents in transplant-associated osteoporosis were identified. Studies were required to confirm osteoporosis by densitometry (T-score ≤ -2.5) and include a comparator arm (placebo, calcium ±vitamin D, or active comparator).

Information sources and search strategy

We searched Medline (via PubMed), Embase, Cochrane CENTRAL, ClinicalTrials.gov, Scopus, Web of Science Core Collection, Google Scholar, and OpenGrey from inception through April 30, 2025. No language or

publication-date limits were applied. Key terms were: Osteoporosis OR “bone loss” AND Transplantation OR graft AND Drug Therapy OR pharmacotherapy OR medication OR drugs.

All references were imported into Rayyan (2016) for duplicate removal and screening.

("Osteoporosis"[MeSH] OR osteoporosis[tiab] OR "bone loss"[tiab]) AND ("Transplantation"[MeSH] OR transplant[tiab] OR graft[tiab]) AND ("Drug Therapy"[MeSH] OR pharmacotherapy[tiab] OR medication[tiab] OR drugs[tiab]).

Embase and Lilacs strategies were analogous, using their respective subject headings and title/abstract fields.

Study selection

Two reviewers (JP, GT) independently screened titles and abstracts in Rayyan, then assessed full texts against inclusion criteria. Discrepancies were resolved by discussion or by a third reviewer (LT).

Data extraction

Data from included studies were captured in a standardized Excel sheet: publication details (author, year, country, funding), design, sample size, intervention (agent, dose, duration), comparator, outcome measures (BMD change, fracture incidence at 12 and 24 months, adverse events), and follow-up. JP and GT performed independent extraction; LT adjudicated any discrepancies.

In total, 24 prospective trials evaluated efficacy, three assessed real-world effectiveness, one addressed safety alone, and four reported both efficacy and safety. This rigorous, reproducible approach ensures that our synthesis reflects the highest-quality prospective controlled evidence for transplant-induced osteoporosis.

RESULTS

A total of 24 studies on transplant-associated osteoporosis were analyzed, evaluating various pharmacological interventions in patients with low bone mineral density (BMD). Of these, 19 studies assessed efficacy, 3 analyzed clinical effectiveness, 3 combined efficacy and safety analysis, and 1 focused exclusively on the safety of interventions. The results showed that different interventions, such as Pamidronate, Alendronate, Etidronate, Neridronate, Ibandronate, and Denosumab, had varying effects on improving BMD and reducing fracture risk.

Efficacy

Several randomized and nonrandomized studies demonstrated that bisphosphonates and related agents significantly improved bone mineral density (BMD) in transplant recipients. In patients receiving pamidronate it is (13) observed a mean increase of +8.8 % in lumbar spine BMD and +8.2 % in femoral BMD compared with calcium-vitamin D controls ($p < 0.015$), and a long-term trial (30) reported that, at four years post-transplant, those without pamidronate prophylaxis lost 12.3 % at the femoral neck ($p < 0.01$), whereas the pamidronate group maintained stable BMD. Etidronate improved lumbar BMD by +4.3 % ($p < 0.03$) and trochanteric BMD by +10.3 % ($p < 0.02$) without affecting femoral-neck density (14). In a head-to-head trial, Jeffery et al. (2003) (15) showed that alendronate increased lumbar BMD by +4.2 % ($p < 0.0001$) and femoral BMD by +3.3 % ($p < 0.001$), whereas the calcitriol group experienced smaller gains. Another study (21) found that combining alendronate with alfacalcidol produced even greater benefits, with +7.9 % in lumbar and +8.0 % in femoral BMD ($p \leq 0.01$ for both). Neridronate delivered monthly intramuscularly achieved +8.6 % in lumbar spine BMD at 12 months ($p = 0.005$) compared to placebo's +4.2 % (17). Zoledronate was associated with +8.6 % ± 7 % in lumbar ($p < 0.01$) and +5.4 % ± 2.2 % in femoral-neck BMD ($p = 0.039$) (18). A systematic review and meta-analysis of multiple bisphosphonates suggested a possible clinical effect on lumbar BMD beyond the first year,

although pooled analyses did not reach significance (SMD -0.29; $p = 0.22$) (19). Ibandronate produced modest but significant gains of +1.3 % in total femur ($p = 0.013$) and +0.6 % in ultradistal radius ($p = 0.039$) (20). In heart-transplant recipients, both alendronate and calcitriol maintained stable BMD for over one year (16). All these efficacy findings are detailed in table I.

Safety

Through multiple studies, pharmacological therapies were generally well tolerated. Pamidronate was associated with mild hypocalcemia in 8.6 % of patients, which was effectively managed (31). Clodronate did not produce severe adverse events in heart-transplant recipients (32). Denosumab did not trigger rejection or major events, though it elicited a slight PTH increase ($p = 0.009$) (26). Alendronate caused no serious adverse effects or renal-function deterioration (23). In kidney-transplant cohorts, 15 % of alendronate recipients experienced transient dyspepsia, whereas none did with pamidronate, and there were no significant differences in creatinine or GFR between treatments ($p = 0.49$ and $p = 0.41$, respectively) (27). A full summary of safety outcomes is provided in table II.

Efficiency

When focusing on bone-loss prevention, pamidronate reduced hip BMD loss to -1.9 % versus -7.3 % in controls ($p = 0.09$) (22) and provided durable protection at four years (30). In kidney-transplant patients, alendronate increased lumbar BMD by +0.035 g/cm² compared with +0.003 g/cm² in untreated subjects (23). A comparison of intravenous pamidronate versus oral alendronate showed that pamidronate preserved femoral-neck density (-1.42 % vs. -2.03 %; $p = 0.003$) and total femur (-1.40 % vs. -1.83 %; $p = 0.03$) more effectively (27). Clodronate achieved an +11.7 % increase in lumbar BMD ($p = 0.02$) while placebo produced no change (24). Although ibandronate's lumbar gain of +4.42 % did not reach statistical significance ($p = 0.13$), treated patients experienced significantly fewer vertebral

deformities, less height loss, and fewer acute-rejection episodes than controls (28); (25). Risedronate increased lumbar BMD by +5.9 % in 12 months and stabilized femoral-neck density, in contrast to declines in controls ($p < 0.05$) (29). Regarding clinical effectiveness, calcidiol reduced vertebral-fracture incidence by 30 % ($p < 0.05$) (33); ibandronate and risedronate lowered NTX levels by 34 % and 28 %, respectively ($p < 0.05$) (34); and pamidronate did not significantly change fracture rates (8 % vs. 8 %; $p = 0.40$) but did mitigate BMD loss (35). These efficiency and fracture-outcome data appear in table III.

Table I. Characteristics of the included studies on the efficacy of therapies for transplant-associated osteoporosis

ID	Author, year, design	Population	Intervention (name), dose, *n*	Intervention outcome	Comparator (name), *n*, dose	Comparator outcome
1	Aris RM et al. (2000). Clinical trial (13)	Outpatient adults (men and women, 18-38 years) with CF*, recruited after lung transplantation, with low bone mineral density (T-score ≤ -2.5)	Pamidronate 60 mg single dose. *n*: 16	Change in lumbar spine BMD: +8.8 ± 2.5 %, *p* = 0.015. Change in femoral BMD: +8.2 ± 3.8 %, *p* = 0.01. Type I collagen N-telopeptide levels:	Calcium (1 g/day) +vitamin D (800 IU/day). *n* = 18 (men, women, aged 18-38 years)	Change in lumbar spine BMD: +2.6 ± 3.2 %, *p* = 0.015. Change in femoral BMD: +0.3 ± 2.2 %, *p* = 0.01. Osteocalcin levels: *p* < 0.001

				Significant decrease of 53.7 ± 39 %, $p < 0.001$. Osteocalcin levels: Increase, $p < 0.001$		
2	Arlen DJ et al. (2001). Retrospective cohort study (14)	Outpatient adult patients (men and women aged 18 to 85 years) who received a kidney transplant. With femoral osteoporosis	Etidronate 400 mg/day for 2 weeks every 12 weeks. *n*: 49	Change in lumbar spine BMD: +4.3 ± 6.1 %, $p < 0.03$. Change in trochanteric BMD: +10.3 ± 11.9 %, $p < 0.02$. Change in	Calcium +vitamin D (dose not specified). *n* = 24 (15 men, 9 women, mean age 42 years)	Change in lumbar spine BMD: +0.55 ± 5.3 %. Change in trochanteric BMD: +2.2 ± 5.7 %. Change in femoral neck BMD: +3.2 ± 6.4 %

		(T-score < -2.5)		femoral neck BMD: +3.4 ± 6.5 % (not significant)		
3	Jeffery JR et al. (2003). Randomized Clinical Trial (15)	Outpatient adult patients (men and women, mean age 45 years) who received a kidney transplant. With low bone mineral density (T-score ≤ -2.5 in the	**Alendronate:** 10 mg/day + 500 mg calcium. *n* = 46.	Increase in lumbar spine BMD: +4.2 %, $p < 0.001$. Increase in femoral BMD: +3.3 %, $p < 0.001$	**Calcitriol:** 0.25 µg/day + 500 mg calcium. *n* = 51	Increase in lumbar spine BMD: +2.0 %, $p = 0.002$. Increase in femoral BMD: +3.3 %, $p = 0.023$

		lumbar spine or femur)				
4	Cohen A et al. (2006). Extension Study of a Randomized Clinical Trial (16)	Outpatient adult patients (men and women, mean age 55 years) who received a heart transplant. With baseline bone mineral density (mean lumbar T-score: -	*Alendronate:* 10 mg/day + calcium (315 mg TID) andvitamin D (1,000 IU/day). *n* = 34	Stable BMD in the lumbar spine, hip, and distal radius ($p > 0.05$). Increase of 32 % in Bone-Specific Alkaline Phosphatase (BSAP, $p = 0.001$). No significant changes in NTX (bone	*Calcitriol:* 0.25 µg twice daily + calcium (315 mg TID) andvitamin D (1,000 IU/day). *n* = 25	Stable BMD in the lumbar spine and hip ($p > 0.05$). Increase of 27 % in NTX ($p < 0.001$). Increase of 58 % in BSAP ($p < 0.001$)

		0.31 ± 0.2)		resorption marker, p = 0.25)		
5	Giannini S et al. (2021). Randomized Clinical Trial (17)	Outpatient adult patients (men and women, mean age 49.3 ± 9.1 years) with heart, liver, or lung transplant and osteopenia (T-score < -2.0)	*Neridronate:* 25 mg intramuscular monthly + calcium (500 mg/day) +vitamin D 3 (400 IU/day). n = 22	Significant increase in lumbar spine BMD: +7.3 % at 12 months (p = 0.005). Decrease in total alkaline phosphatase (-31.6 %, p = 0.002), bone-specific alkaline phosphatase (-49.3 %, p < 0.001),	*Placebo:* Monthly intramuscular isotonic solution + calcium (500 mg/day) +vitamin D 3 (400 IU/day). n = 17	Lumbar spine BMD: +1.7 % at 12 months (not significant). No relevant changes in bone turnover markers (total alkaline phosphatase -1.1 %, CTX -4.6 %)

				and CTX (-62 %, $p < 0.001$)		
6	Tauchmanova L et al. (2006). Randomized Clinical Trial (18)	Outpatient young female patients (mean age 26 years, mean lumbar BMD: 0.91 g/cm², T-score -1.3), recipients of allogeneic stem cell transplants with ovarian failure.	*Risedronate:* 35 mg weekly orally + calcium (1,000 mg/day) +vitamin D (800 IU/day). $n = 15$	Significant increase in lumbar spine BMD: +5.8 % ± 2.1 %, $p < 0.035$. Prevention of femoral neck bone loss: +1.3 % ± 1.2 %, $p = 0.6$	*Calcium:* 1,000 mg/day, orally administered. *Vitamin D:* 800 IU/day, orally administered. $n = 15$	Significant decrease in lumbar spine BMD: -4.3 % ± 2.3 %, $p = 0.046$. Decrease in femoral neck BMD: -4.2 % ± 1.6 %, $p = 0.046$
	Lip A et al.	Outpatient	*Alendronat*	No	*Calcium:*	No significant

7	(2019). Systematic Review and Meta-Analysis (19)	adult patients (> 18 years, men and women, *n* = 1,762) who received a kidney transplant, followed for > 12 months, with T-score < -1 (osteopenia) or < -2.5 (osteoporosis)	*e:* 10 mg/day orally. *Pamidronate:* 60 mg intravenously every 3 months. *Zoledronate:* 4 mg intravenously every 12 months. *Ibandronate:* 150 mg orally once a month. *Etidronate:* 400 mg/day orally for 14 days every	significant increase in lumbar spine BMD at 12-98 months post-transplant: SMD -0.29 (-0.75 to 0.17), *p* = 0.22. **Fractures:** 2.8 % (*n* = 12/683)	1,000 mg/day.vitamin D*:* 400-800 IU/day. *n = 1,079*	improvement in lumbar spine BMD. *Fractures:* 2.7 % (*n* = 31/1,079)

			3 months. *n = 683*			
8	Smerud KT et al. (2012). Randomized Clinical Trial (20)	Outpatient adult patients (men and women, mean age 51.4 ± 13.8 years), kidney transplant recipients with stable renal function (eGFR ≥ 30 mL/min). Baseline lumbar spine BMD:	*Ibandronate:* 3 mg i.v. every 3 months + calcium (500 mg b.i.d.) + calcitriol (0.25 µg/day). *n = 66*	Increase in lumbar spine BMD: +1.5 % ± 0.06 %, p = 0.28. Significant increase in total femur BMD: +1.3 % ± 0.04 %, p = 0.013, and distal radius BMD: +0.6 % ± 0.03 %, p = 0.039. Reduction	*Placebo:* Isotonic i.v. solution every 3 months + calcium (500 mg b.i.d.) + calcitriol (0.25 µg/day). *n = 63*	Increase in lumbar spine BMD: +0.5 % ± 0.08 %, p = 0.33

		1.184 ± 0.171 g/cm² (T-score: -0.50 ± 1.36)		in bone turnover markers: *PINP:* -13.1 ± 56.4, *p* = 0.0003. *Osteocalcin:* -5.5 ± 21.5, *p* = 0.0004		
9	Trabulus S et al. (2008). Randomized Clinical Trial (21)	Outpatient adult patients (men and women, mean age 34 ± 10 years), kidney transplant	*Alendronate + alfacalcidol:* 10 mg/day alendronate + 0.5 µg/day alfacalcidol + 1,000 mg/d	Non-significant increase in lumbar spine BMD: +0.7 %, *p* = 0.8. Non-significant decrease in femoral	**Alendronate alone:** 10 mg/day alendronate + 1,000 mg/day calcium. ***n* = 12.** Increase in lumbar	Non-significant increase in lumbar spine BMD: +0.1 %, *p* = 0.7. Non-significant decrease in femoral BMD: -2.1 %, *p* = 0.8

		recipients with low BMD (T-score ≤ -2.5)	ay calcium. *n = 17* Increase in lumbar spine BMD: +7.9 %, *p* = 0.006. Increase in femoral BMD: +8.0 %, *p* = 0.01. Significant improvement in lumbar T-score (*p* = 0.003) and femoral T-score (*p* = 0.02). *Alfacalcidol alone:*	BMD: -1.8 %, *p* = 0.4.	spine BMD: +4.4 %, *p* = 0.2. Increase in femoral BMD: +6.5 %, *p* = 0.09. Significant improvement in lumbar T-score (*p* = 0.009) and femoral T-score (*p* = 0.005). *Control:* 1,000 mg/day calcium. *n = 9*	

			0.5 µg/day alfacalcidol + 1,000 mg/day calcium. *n = 21*			
10	Kananen K et al. (2006). Randomized Clinical Trial (22)	Outpatient adult patients (men and women, mean age 41-46 years), recipients of allogeneic stem cell transplants with baseline lumbar BMD: 0.91 ±	*Pamidronate:* 60 mg intravenous every 1-3 months + calcium (1,000 mg/day) +vitamin D (800 IU/day). *n = 14*	Lumbar bone loss: -0.7 % at 12 months ($p = 0.28$). Total hip bone loss: -4.6 %, $p = 0.008$. Significant reduction in PINP (-86.2 %, $p = 0.0003$)	*Calcium +vitamin D:* 1,000 mg/day calcium + 800 IU/dayvitamin D. *n = 16*	Lumbar bone loss: -3.5 % at 12 months ($p = 0.07$). Total hip bone loss: -7.3 %, $p = 0.03$. Moderate reduction in PINP (-38.5 %, $p = 0.06$)

		0.14 g/cm² (T-score: -1.3 ± 1.3)				
11	Huang W-H et al. (2012). Case-Control Study (23)	Outpatient adult patients (men and women, mean age 47 ± 13 years), kidney transplant recipients with baseline lumbar BMD: 0.90 g/cm² (mean T-score: -1.53)	*Alendronate (Fosamax):* 70 mg weekly orally + calcium (1,000 mg/day) +vitamin D (800 IU/day). *n = 34*	Significant increase in lumbar spine BMD: +2.2 % (from 0.90 g/cm² to 0.92 g/cm², $p < 0.001$). Significant increase in total hip BMD in men ($p = 0.03$)	Calcium (1,000 mg/day) +vitamin D (800 IU/day). *n = 42*	• No significant changes in lumbar spine BMD (+0.5 %, $p = 0.33$) or hip BMD. • 14 % of patients experienced bone deterioration
	Ippoliti G et al.	Outpatient	*Clodronate:*	Significant	*Placebo:*	Lumbar spine BMD

12	(2003). Randomized Clinical Trial (24)	adult patients (56 men, 8 women, mean age 50 years), heart transplant recipients with osteoporosis (T-score < -2.5)	1,600 mg/day in two doses + calcium (2,000 mg/day). *n = 32*	increase in lumbar spine BMD: from 0.77 ± 0.14 g/cm² to 0.86 ± 0.16 g/cm², $p = 0.02$. Reduction in bone isoenzyme of alkaline phosphatase: -35 %, $p = 0.03$	Isotonic solution + calcium (2,000 mg/day). *n = 32*	loss: from 0.75 ± 0.12 g/cm² to 0.73 ± 0.15 g/cm², $p = 0.0001$. *Fracture incidence:* 9.3 % (2 vertebral fractures, 1 hip fracture)
13	Kaemmerer D et al. (2010). Randomized Clinical Trial (25)	Outpatient adult patients (men and women, mean age 51.7 ±	*Ibandronate:* 2 mg intravenous every 3 months + calcium (1,000 mg/d	Increase in lumbar spine BMD: +4.42 % at 24 months, $p = 0.13$. Significant	*Calcium +vitamin D 3:* 1,000 mg/day calcium + 800-1,000 IU/da	Lumbar spine BMD loss: -1.80 % at 24 months, $p = 0.13$ Fracture rate: 25.8 % (8 fractures).

		12.9 years), liver transplant recipients with baseline lumbar T-score: -1.75 ± 1.08	ay) +vitamin D 3 (800-1,000 IU/day). *n = 34*	reduction in fractures: 7.4 % (2 fractures), *p* = 0.04	yvitamin D3. *n = 40*	
14	Alfieri C et al. (2021). Prospective Observational Study (26)	Outpatient adult patients (men and women, median age 62 years), kidney transplant recipients with femoral osteoporosis (T-score < -	*Denosumab:* 60 mg subcutaneous every 6 months + calcium (1,000 mg/day) +vitamin D (800-1,000 IU/day). *n = 32*	Increase in lumbar spine BMD: +9.7 %, *p* = 0.01. Increase in femoral BMD: +5.7 %, *p* = 0.02. Reduction in femoral osteoporosi	No direct comparator	NA

		2.5)		s: from 78 % to 69 %, *p* = 0.001		
15	Bita Omidvar et al. (2011). Clinical Trial (27)	40 kidney transplant patients (27 men, 13 women) with T-score < -2.5 in the lumbar spine, femoral neck, or total hip	*Pamidronate: n = 20*, 90 mg intravenous from the 3rd week post-transplant for 3 months	Reduction of 1.42 % in femoral neck bone density and 1.40 % in the femur (less bone loss than the Alendronate group, *p = 0.003 and 0.03)*	*Alendronate: n = 20*, 70 mg oral weekly for 3 months	Reduction of 2.03 % in femoral neck bone density and 1.42 % in the femur. *Adverse effects:* Gastrointestinal side effects in 3 patients (dyspepsia)
16	Grotz et al. (2001). Randomized	Hospitalized post-kidney transplant	*Ibandronate:* Variable dose. *n: not*	Prevention of BMD loss in the	*Control (without Ibandronate*	Greater BMD loss in the control group (-6.5 % in the lumbar

	Controlled Clinical Trial (28)	patients with reduced BMD, some with osteoporosis (variable T-score)	*specified*	lumbar spine (-0.9 % vs. -6.5 %, *p < 0.0001)* and femur (-10.5 % vs. -27.7 %, *p < 0.0001)*	*): n: not specified*	spine, -27.7 % in the femur, *p < 0.0001)*
17	Tauchmanova et al. (2003). Prospective Randomized Study (29)	Outpatient post-allogeneic stem cell transplant patients with osteoporosis (T-score -2.5)	*Risedronate :* 5 mg/day for 12 months. *n = 17*	Increase in lumbar spine BMD: *+4.4 % ± 1.6 % at 6 months* and *+5.9 % ± 1.7 % at 12 months***.** Stable BMD in the femoral	*Calcium (1 g/day) +vitamin D (800 IU/day). n = 17*	Decrease in lumbar spine BMD: *-4.3 % ± 1.5 %***.** Decrease in femoral neck BMD: *-4.3 % ± 2.1 %*

				neck		
18	Fan et al. (2003). Clinical Trial (30)	Hospitalized post-kidney transplant patients with *T-score < -2.5*, indicative of osteoporosis	*Pamidronate:* 90 mg IV, starting from the 3rd week post-transplant for 3 months. ***n* = 20**	*Reduction in BMD:* Femoral neck: **-**1.42 % (*p* = 0.003) *Femur:* -1.40 % (*p* = 0.03)	*Alendronate:* 70 mg/week orally for 3 months.	*Reduction in BMD:* Femoral neck: *-2.03 % (p = 0.003).* Femur: **-***1.42 % (p = 0.03). Adverse effects:* Dyspepsia in 3 patients

Table II. Characteristics of the studies included for safety in transplant-induced osteoporosis

ID	**Author, year, design**	**Population**	**Intervention (name), *n*, dose**	**Intervention outcome**	**Comparator (name), *n*, dose**	**Comparator outcome**
1	Ippoliti et al. 2003, Clinical Trial (24)	64 patients (56 men, 8 women)	*Clodronate (oral), n* = 32,	Mild gastrointestinal effects:	*Placebo, n* = 32 + *2,000 mg/d*	*New bone fractures: 9.3 %* (2 vertebral fractures, 1 hip

		with bone loss post-heart transplant. *T-score:* -1.43 in the lumbar spine and -4.0 in 1/10 of the forearm	*1,600 mg/day* in two divided doses **+** *2,000 mg/day calcium carbonate*	*nausea and epigastric discomfort in 22 % of patients***.** *New bone fractures: 0 %*	*ay calcium carbonate*	fracture). *Persistent bone pain:* Patients continued requiring analgesics.
2	Walsh SB et al. 2009. Clinical Trial (31)	*Population:* 93 post-kidney transplant patients (46 in the intervention group and 47 in the control group). *Z-*	*Pamidronate, n* = 46, *1 mg/kg IV* perioperatively, then at *1, 4, 8, and 12 months*	5 episodes of transient hypocalcemia (8.6 %)**.**	*No bisphosphonate, n* = 47 (dose not specified)	6 new fractures (12.8 %) in 24 months**.** 0 episodes of transient hypocalcemia

		score < -2.0				
3	Alfieri C et al. 2021. Prospective Observational Study (26)	32 kidney transplant patients (KTxps), *21 women and 11 men*, median age: *62 years*. *T-score:* Femoral -3.0, Vertebral 3.0	*Denosumab, n = 32, 60 mg every six months for one year*	2 cases of new spontaneous vertebral fractures (sVF). 4 urinary tract infections (UTI). No hypocalcemia or graft rejection	No direct comparator group.	*Not applicable*
4	Wen-Hung Huang et al. 2012. Case-Control Study (23)	76 kidney transplant patients. *Osteoporosis:* T-score ≤ -2.5;	Fosamax (Alendronate Sodium), *n = 34,* 70 mg per week	7 patients did not tolerate Fosamax due to side effects (not	Patients without Fosamax, *n = 42*	No significant adverse events reported.

		Osteopenia: between -1.0 and -2.5		specified)		
5	Bita Omidvar et al. 2011. Clinical Trial (27)	40 kidney transplant patients (27 men, 13 women) with *T-score < -2* in the lumbar spine, femoral neck, or total hip	Pamidronate, *n = 20,* 90 mg intravenous from the 3rd week post-transplant for 3 months	No adverse events reported	Alendronate, *n = 20*, 70 mg oral weekly for 3 months	Transient dyspepsia in 3 patients

Table III. Characteristics of the studies included effectiveness in transplant-induced osteoporosis

ID	Author, year (design)	Population	Intervention (agent, dose, *n*)	Comparator (agent, dose, *n*)	Vertebral fracture incidence

					(intervention vs comparator)
1	García-Delgado I et al. (1997) (Randomized Clinical Trial) (33)	Outpatient heart-transplant recipients; mean age 53; T-score ≤ -2.5	Calcidiol 32 000 IU/week + Ca 1 000 mg/day (*n* = 13)	Calcitonin 100 IU/day intranasal + Ca 1 000 mg/day (*n* = 13)	0 % vs 30.8 %
2	Sánchez-Escuredo A et al. (2015) (Prospective Clinical Trial) (34)	Kidney-transplant recipients; mean age 63; lumbar T-score -1.7 ± 0.8; femoral -2.1 ± 0.7	Ibandronate 150 mg monthly + Ca 2 500 mg/day + Vit D 800 IU/day (*n* = 35)	Risedronate 35 mg weekly + Ca 2 500 mg/day + Vit D 800 IU/day (*n* = 34)	Not reported
3	Ninkovic M et al. (2002) (Randomized Clinical Trial) (35)	Outpatient liver-transplant recipients; mean age 53; lumbar T-score -2.0 ± 0.6	Pamidronate 60 mg IV single dose pre-transplant (*n* = 45)	Standard follow-up without pamidronate (*n* = 54)	8 % vs 8 %

DISCUSSION

This scoping review confirms that transplant-induced osteoporosis (TO) arises from a multifactorial interaction among pre-existing bone health, chronic glucocorticoid exposure, immunosuppressive regimens, and transplant-specific factors. Nearly all studies included chronic glucocorticoid use as an underlying contributor to bone loss, yet only a minority explicitly reported corticosteroid dosing or its direct impact on BMD outcomes. Consistently, bisphosphonates (pamidronate, alendronate, etidronate, zoledronate, ibandronate) and denosumab increased BMD across renal, cardiac, and mixed transplant populations (13-21, 26), even though fracture-reduction data remain sparse and variable.

Our efficacy findings align with earlier reports identifying glucocorticoids as central drivers of post-transplant bone demineralization (30,33); which documented up to 12 % femoral BMD loss in renal recipients and high fracture rates in liver transplant patients. However, the wide divergence in fracture outcomes—such as the lower vertebral-fracture incidence reported (25) versus the neutral fracture effect seen (35)—likely reflects methodological heterogeneity (e.g., variable glucocorticoid regimens, follow-up durations, and sample sizes). Notably, although most trials acknowledged patients' glucocorticoid burden, few stratified results by steroid dose or duration, underscoring a gap between recognized pathophysiology and published outcomes.

Overall, pharmacological agents exhibited acceptable safety profiles in the context of concomitant glucocorticoid therapy. Pamidronate was associated with mild, transient hypocalcemia (31), while alendronate caused only minor gastrointestinal discomfort (23). Denosumab did not precipitate rejection or serious adverse events, although modest PTH elevations warrant monitoring (26). Importantly, none of the studies reported glucocorticoid-related exacerbations of adverse effects, suggesting that these antiresorptives can be safely co-administered with glucocorticoids under careful supervision (Table II).

A clear strength of this review is the inclusion of diverse transplant types and pharmacotherapies, offering a panoramic view of current evidence. The rigorous JBI scoping methodology enhanced reproducibility in study selection and data extraction. Conversely, heterogeneity in glucocorticoid dosing regimens, inconsistent reporting of fracture endpoints, and variable follow-up durations limited cross-study comparability. Furthermore, the near-ubiquitous use of glucocorticoids was seldom quantified, impeding nuanced analysis of steroid-specific effects on BMD and fracture risk.

Clinicians should recognize chronic glucocorticoid therapy as a primary risk factor for TO and implement early, individualized bone-preserving strategies. Bisphosphonates remain first-line agents—particularly in kidney and heart transplant recipients—while denosumab offers an alternative for patients that are intolerant of oral bisphosphonates. Routine monitoring of BMD and fracture risk, coupled with judicious tapering of glucocorticoids when feasible, may optimize long-term skeletal health in transplant populations (Table I) (36).

To address current evidence gaps, future studies must standardize reporting of glucocorticoid exposure and incorporate fracture endpoints alongside BMD. Largescale, multicenter randomized trials with uniform definitions of TO, stratified by steroid dose and type, are essential. Extended follow-up beyond two years will capture delayed adverse events and fracture outcomes, while subgroup analyses of underrepresented transplant types (e.g., lung, intestinal) will inform tailored interventions. Cost-effectiveness and patient-reported outcome measures should also be integrated to guide real-world clinical decision-making (37, 38) (Table III).

CONCLUSION

Pharmacological therapies for transplant-induced osteoporosis effectively improve BMD in the setting of chronic glucocorticoid and immunosuppressive use, yet their impact on fracture prevention remains inadequately characterized. Enhanced focus on quantifying glucocorticoid regimens and

standardized fracture reporting will be critical to developing evidence-based, patient-centered strategies that mitigate long-term skeletal complications in transplant recipients.

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