Table 1. Characteristics of the included studies on the efficacy of treatments for glucocorticoid-induced osteoporosis

Author, year, and study design	Population and age	Intervention (name), n, and dose	Intervention outcome	Comparator (name), n, and dose	Main outcome
Reid *et al.* (2001). Randomized clinical trial (6)	Men, outpatient setting, aged 18 to 85 years.	RIS* n = 124 Tab 2.5 mg (n = 61), Tab 5 mg (n = 63).	Increase in BMD* by 4.8% in the lumbar spine with 5 mg of RIS*.	Placebo n = 60	RR*: Vertebral fractures: 82.4% lower risk with RIS* (95% CI: 36.6% – 95.1%, p = 0.008). MD* Increase in BMD* with RIS* 5 mg: Lumbar spine: +4.8% Femoral neck: +2.1% Trochanteric region: +2.6%. BMD* loss in the placebo group: Lumbar spine: −3.4% Femoral neck: −3.3% Trochanteric region: −3.4%
Hoes *et al.* (2010). Cohort study (7)	Patients with an inflammatory rheumatic disease. Outpatient setting.	ALN* n = 116 Tab 10 mg orally daily or ALF* cap 1 µg orally daily. Patients were not categorized by intervention type in the summary.	24% of patients developed at least one new vertebral fracture.	N/A	OR*: Vertebral fractures: 1.00016 (95% CI: 1.00003 – 1.00029; p < 0.05) per mg of cumulative GC* dose. Age: 1.14 (95% CI*: 1.05 – 1.23; p < 0.05) per year of age increase. MD*: Lumbar spine: +0.4% ± 5.9% Femoral neck: −0.18% ± 5.4% Total hip: +0.07% ± 4.8% RR*: 24% of patients developed at least one new vertebral fracture.

Eastell *et al.* (2010). Randomized clinical trial (8)	Men and women ≥ 21 years. Outpatient setting.	TPTD*, n = 97 Amp 20 µg/day subcutaneous administration.	Increase of 92% in OC* and 108% in PINP*	ALN* n = 100 Tab 10 mg/day orally.	OR*: Vertebral fractures with TPTD* vs. ALN*: 0.22. MD*: Increase in BMD* at 12 months: Lumbar spine: TPTD*: Up to 11%. ALN*: Up to 3% RR: TPTD*: 0.22 for vertebral fractures.
Burshell *et al.* (2010). Randomized clinical trial (8)	Men and women aged 22 to 89 years. Outpatient setting.	TPTD* n = 80 Amp 20 µg/day subcutaneous administration.	Significant increase in lumbar spine BMD* (mean ± SE) of +7.2% and femoral neck BMD* of +3.8% at 18 months.	ALN* n = 77 Tab 10 mg/day orally.	MD* TPTD*: Lumbar spine: Increase in BMD*: $p < 0.001$. Femoral neck: Increase in BMD*: $p < 0.01$
Payer *et al.* (2018). Non-randomized clinical trial (9)	Men, mean age: 57.3 ± 12.1 years. Women, age: 59.7 ± 14.8 years. Outpatient setting.	TPTD*, n = 129 Amp 20 µg/day subcutaneous administration Ca* supplements: 500-1000 mg/day orally V.D3*: 400-800 IU/day orally.	Increase in BMD* in lumbar spine (n = 122): +8.3% at 12 months ($p < 0.001$) and +9.28% at 18 months ($p < 0.001$). Increase in BMD* in total hip (n = 116): +3.85% at 18 months ($p < 0.001$). Increase in BMD* in femoral neck: +3.26% ($p < 0.001$).	N/A	MD*: Increases in BMD* after 18 months of treatment with TPTD: Lumbar spine: +9.28% ($p < 0.001$). Total hip: +3.85% ($p < 0.001$). Femoral neck: +3.26% ($p < 0.001$)
Kasayama *et al.* (2005). Randomized clinical trial (13)	Japanese postmeno-pausal women diagnosed with bronchial asthma, with no recent use of systemic corticosteroids. Outpatient setting.	ALN* n = 18 Tab 5 mg/day orally.	Increase in BMD*: Lumbar spine: +4.9% ± 4.5%. Total hip: +2.4% ± 2.2%. Ward's triangle: +3.6% ± 5.2% at 12 months.	ALF* n = 10 Tab 1 µg/day orally.	MD*: Increases in BMD* after 12 months of treatment: Lumbar spine: ALN*: +4.9% ($p = 0.0005$). ALF*: +1.7% (not significant, $p = 0.255$). Total hip: ALEN*: +2.4% ($p = 0.0005$). ALF*: +0.1% (not significant, $p = 0.949$). Ward's triangle: ALN*: +3.6% ($p = 0.02$). ALF*: +2.0% (not significant, $p = 0.484$).

Struys *et al.* (1995). Randomized clinical trial (10)	Men and women. Outpatient setting.	ETD*n = 19 Tab 400 mg/ day for 14 days, followed by Ca* 500 mg/day for 76 days (4 cycles).	Increase of 5.7% in lumbar spine (L1-L4) BMD*. Increase of 6.8% in proximal femur (total hip) BMD*.	Ca* supplemen-tation n = 20	Significant BMD* loss: 3.4% in the lumbar spine and 4.1% in the proximal femur.
Ringe *et al.* (2003). Randomized clinical trial (11)	Men and women, mean age: 64 years. Outpatient setting.	IBN* n = 58 Ampoule 2 mg, intravenous injections every 3 months.	Increase of 13.3% in lumbar spine BMD*. 62.3% reduction in the risk of new vertebral fractures (8.6% incidence). Increase of 5.2% in femoral neck BMD*. Reduction in lumbar pain in 86.2% of patients.	ALF* n = 57. Tab 1 mcg orally daily.	MD*: Increase in BMD* after 12 months. Lumbar spine: ETD*: +5.7% ($p < 0.02$ vs. baseline, $p < 0.001$ vs. Ca* group). Ca*: -3.4% ($p < 0.02$ vs. baseline). Proximal femur (total hip): ETD*: +6.8% ($p < 0.02$ vs. baseline, $p < 0.001$ vs. Ca* group). Ca*: -4.1% ($p < 0.02$ vs. baseline).
Devogelaer *et al.* (2013). Randomized clinical trial (12)	Men and women aged 18 to 85 years.	ZOL* 5 mg IV annually. Treatment subpopulation: n = 272.	Reduction in b-CTx*: 62%, 59%, 65%. NTx* reduction: 20-33% P1NP* reduction: 9-33% BSAP* reduction: 4-14%.	Risedronate 5 mg orally daily 417 patients received RIS*	MD*: Reduction in bone turnover markers after 12 months: b-CTx*: ZOL*: 40-62% ($p < 0.05$). RIS*: 30-46%. NTx*: ZOL*: Reduction of 20-30%. Bone formation markers: P1NP*: ZOL*: 22% ($p < 0.05$). BSAP*: ZOL*: 14-20% ($p < 0.05$).
Kasayama *et al.* (2005). Randomized clinical trial (13)	Japanese postmenopausal women diagnosed with bronchial asthma, with no recent use of systemic corticosteroids, among others.	Alendronate: n = 16, 5 mg/ day.	Oral ALN* 5 mg/d for 12 months significantly increased BMD*: lumbar spine +4.9% ($p = .0005$), total hip +2.4% ($p = .0005$), Ward's triangle +3.6% ($p = .02$).	ALF* n = 9, 1 µg/day.	MD*: Increase in BMD* with ALN* 5 mg: Lumbar spine: +4.9%, Total hip: +2.4%, Ward's triangle: +3.6%. Change with ALF* 1 µg: Lumbar spine: +1.7%, Total hip: +0.1%, Ward's triangle: +2.0%. Markers: Greater reduction in bone turnover markers (NTx, OC*, AF*) with Alendronate.

Lems *et al.* (1997). Randomized clinical trial (14)	Patients on corticosteroids ≥7.5 mg/day.	NaF* + cyclical ETD*, n = 23, NaF 25 mg BID + ETD* 200 mg BID* for 2 weeks every 3 months.	Lumbar spine BMD* ↑ +9.3% (95% CI*: +2.3% to +16.2%, p < 0.01); Hip BMD*: no significant change (−2.5%, NS).	Placebo + cyclical ETD*, n = 24, placebo BID* + ETD* 200 mg BID* for 2 weeks every 3 months.	MD*: Lumbar spine BMD* ↑ +9.3% (95% CI: +2.3% to +16.2%, p < 0.01) vs. +0.3% (95% CI*: −2.2% to +2.8%) with placebo. Δ between groups: +8.9% (95% CI: +1.9% to +16.0%, p < 0.01). Hip BMD* ↓ −2.5% (NS*) vs. −4.0% (p < 0.01), Δ = +1.5% (NS*).
Fuji *et al.* (2007). Randomized clinical trial (15)	CKD* patients with GIOP*, age: 42.5 ± 16.6	RIS*, n = 50 (with aVD*), 2.5 mg/d PO* RIS*, n = 28 (without aVD*), 2.5 mg/d PO*	Lumbar spine BMD* ↑ 2.8% (with aVD*) and ↑ 2.1% (without aVD*) at 12 mo S-NTX* ↓ ~20%, ALP* ↓ ~11% at 6 mo*	aVD* only, n = 37, mean dose 0.36 mg/d	Lumbar spine BMD* ↓ 1.2% (NS*) S-NTX* unchanged, ALP* ↑ 26.9% RIS improved BMD* vs aVD* (p < 0.05)
Kikuchi *et al.* (2007). Randomized clinical trial (16)	Patients with glomerular diseases; mean age 42 ± 16 years.	Group R: Risedronate, n=12, 2.5 mg/ day Group R+A: Risedronate + Alfacalcidol, n=11, 2.5 + 0.5 mg/day.	R: No significant change in BMD* R+A: BMD* ↑ +2.0% overall, +2.1% in patients receiving steroid pulses.	Group A: Alfacalcidol only, n=15, 0.5 mg/day.	Lumbar BMD changes: R: stable R+A: Significant increase A: Significant decrease (−5.6%) Urinary NTx*: decreased in R and R+A, unchanged in A Bone formation markers (OC*, ALP*): decreased in all groups Baseline urinary NTx* was higher in patients with BMD* loss.
Lane *et al.* (2000). Randomized, controlled, parallel-group trial (17)	Postmenopausal women; age 50–82 yrs, with GIOP* on ≥1 year of HRT and prednisone 5–20 mg/day.	PTH (1–34)*: n=28, 400 IU/ day SC*	↑ Spine BMD*: +35% (QCT*), +11% (DXA*) at 12 mo (p < 0.001); sustained ↑ at 24 mo. ↑ bone markers (BSAP*, OC*); delayed ↑ in DPD.	Control group: n=21; no PTH*; continued HRT*, glucocorticoids, calcium (1500 mg/day) + Vitamin D (800 IU/day)	Early ↑ in bone formation markers (BAP*, OC*) predicted spine BMD* gains. Marker response (1–6 mo) correlated with 12–24 mo BMD* ↑. ROC* AUC* >0.79 for QCT* prediction.

Farahmand *et al.* (2013). Controlled clinical trial (18)	Ambulatory men with GIO*; mean age 56.3 yrs (range 25–82).	Teriparatide, n = 45, 20 µg/ day SC*	↑ P1INP* and CTx*; ↑ vertebral strength in all loading modes; strong positive correlation between ↑ P1INP* and vertebral strength at 18 months.	Risedronate, n=47, 35 mg/ week orally.	TPTD* ↑ vertebral strength vs. RIS* at 18 mo in all FEA* modes (e.g., axial compression: 7.08 ± 3.48 vs. 5.95 ± 2.2 kN, p = 0.015); PINP* ↑ significantly with TPTD* (p < 0.001); early PINP* changes correlated with strength gains (e.g., r = 0.560 at 6 mo), supporting PINP* as a surrogate marker of bone strength in GIOP*.
Thomas *et al.* (2013). Observational cohort study using administrative claims data (19)	Women ≥65 yrs on glucocorticoids (GC).	Alendronate, n = 6.359, 70 mg/week Risedronate, n=4,648, 35 mg/week.	ALN: ↓ non-vertebral fracture rate by 33% (from 5.22 to 3.51/100 PY), ↓ vertebral fracture rate by 59% (p < 0.05) RIS: ↓ non-vertebral fracture by 28% (from 5.51 to 3.95/100 PY), ↓ vertebral fracture by 54% (p < 0.05).	N/A*	Both ALN and RIS significantly reduced clinical vertebral and non-vertebral fracture incidence over 12 mo vs. baseline 3-mo rate in women with GIO. Study not designed for head-to-head comparison.
Li *et al.* (2010). RCT, double-blind, placebo-controlled (20)	Chinese women with SLE* on long-term glucocorticoids; mean age 44 ± 8.7 yrs.	Ibandronate, n = 20, 150 mg/ month + daily Alfacalcidol 1 µg + calcium 500 mg/day.	↑ Cortical bone density (Dcomp) (p = 0.023); ↑ aBMD* lumbar spine (+4.9%, p < 0.0001) and hip (+1.0%, p < 0.005); stable microarchitecture.	Placebo, n=20, monthly placebo + daily alfacalcidol 1 µg + calcium 500 mg/day.	IBAN* ↑ cortical Dcomp and preserved microarchitecture (HR-pQCT*); ↑ aBMD* LS vs placebo (4.9% vs 2.6%, p = 0.024); ↓ Tb.Sp* only in placebo (p = 0.04).
Stoch *et al.* (2009). RCT*, double-blind, placebo-controlled, multicenter. (21)	Patients (ALN*: n = 114, placebo: n = 59); mean age ~52–55 yrs; men and women on ≥7.5 mg/d prednisone for ≥12 months.	Alendronate (ALN), n = 114, 70 mg/ week + calcium 1000 mg/day + vitamin D 400 IU/day.	↑ BMD* lumbar spine: +2.45% (p ≤ 0.001); trochanter:+1.27% (p = 0.007); total hip: +0.75% (p = 0.008); ↓ NTX/ Cr*: −32.2% (p ≤ 0.001).	Placebo, n = 59, + same calcium and vitamin D regimen.	ALN ↑ BMD vs. placebo at 12 mo in LS (+2.92%, p ≤ 0.001), trochanter (+1.66%, p = 0.007), and total hip (+1.19%, p = 0.008); ↓ BSAP (−14.5%, p ≤ 0.001); ALN well tolerated, no sig. difference in AE vs placebo.

Buxton *et al.* (2004). RCT, parallel-group, open-label (22)	Postmenopausal women with GIOP*; age 50–82 yrs.	PTH*(1–34), n = 28; 400 U/day (~40 µg), SC for 12 months + HRT* + calcium (1500 mg) + vitamin D (800 IU).	↑ sRANKL* (+250% at 3 mo, p < 0.05), ↑ IL-6 (+130% at 3 mo, p < 0.05), ↑ IL-6sR (peak at 1 mo*, p < 0.05), ↓ OPG (–45% at 12 mo); ↑ OC & BSAP* > 100%	HRT alone, n = 23; calcium + vitamin D.	PTH (1–34)* rapidly ↑ bone formation markers (OC*, BSAP*) and osteoblast-derived cytokines (sRANKL*, IL-6*, IL-6sR*), preceding DPD* ↑; supports uncoupling of remodeling in favor of formation during early GIOP* treatment.
Nordborg *et al.* (1997). RCT*, double-blind, placebo-controlled (23)	Patients with GCA on GC*; 21 women / 6 men; mean age 70.2 yrs.	Clodronate, n = 14; 800 mg PO* daily, cyclically every second month (months 1,3,5,7,9,11) + calcium 500–750 mg/day.	No significant effect on BMD or BMC at any time point; ↓ OC at 12 mo vs. control (5.81 ± 2.21 vs. 6.69 ± 2.60 ng/mL, p < 0.05).	Placebo, n = 13; same calcium regimen.	Cyclic oral clodronate did not prevent GC-induced bone loss better than calcium alone; BMD and BMC recovered similarly in both groups.
Losada *et al.* (2009). RCT, double-blind, double-dummy, active comparator-controlled (24)	Patients: Mean age ~58 yrs; men and women ≥21 yrs with GIO on ≥5 mg/d prednisone for ≥3 months.	TPTD* n = 214; 20 µg/day SC* + calcium 1000 mg/d + vitamin D 800 IU/d.	↑ LS* BMD* +9.8% ±1.7 vs ALN* +4.2% ±1.4 (p < 0.001); ↑ TH* BMD* +5.9% vs +1.3% (p < 0.001); ↑ FN* BMD* +4.3% vs +2.0% (p = 0.228).	ALN*, n = 214; 10 mg/day PO + calcium 1000 mg/d + vitamin D 800 IU/d.	TPTD* showed significantly greater ↑ in LS* and TH* BMD* vs ALN* at 18 mo in both Hispanic and non-Hispanic cohorts; fracture incidence and AEs were similar.
Braith *et al.* (2003). RCT*, prospective, 3-arm, controlled (26)	Heart transplant recipients; all on GC*; mean age: 54–56 yrs.	ALN*+TRN*: n = 8; Alendronate 10 mg/day + supervised resistance training for 6 mo*, starting 2 mo* post-transplant.	BMD* restored to near-baseline at 6 mo*: total body –0.9%, femoral neck –2.1%, lumbar spine –3.4%; significant ↑ vs ALN* or control.	ALN*: n = 8; Alendronate 10 mg/day only. Control: n = 9; no intervention.	ALN*+TRN* more effective than ALN alone in reversing GC-induced BMD* loss in HTR*; combined therapy restored BMD* nearly to baseline at all skeletal sites.
Sambrook *et al.* (2012). RCT*, double-blind, double-dummy, active-controlled (27)	Men (mean age 56.4 yrs) receiving glucocorticoids.	ZOL*, n = 131; 5 mg IV once + daily calcium (1000 mg) + vitamin D (400–1200 IU).	At 12 mo*, ↑ LS* BMD*: +2.46% (prevention), +4.69% (treatment); ↑ TH* BMD*: +1.10% and +1.82%; ↓ β-CTx* & P1NP* > RIS* at all timepoints (p < 0.05–0.001).	RIS*, n = 134; 5 mg PO* daily + same supplements.	ZOL* produced greater ↑ in LS* and TH* BMD* vs. RIS* in both prevention and treatment arms (e.g., LS: +4.69% vs. +3.27%, p = 0.0232); greater ↓ in bone turnover markers.

Saag *et al.* (2007). RCT, double-blind, double-dummy, active-controlled (28)	Adults (345 women, 83 men), aged 22–89 yrs, with GIOP (on ≥5 mg/d prednisone ≥3 mo).	TPTD*, n = 214; 20 µg/day SC* + calcium 1000 mg/day + vitamin D 800 IU/day.	↑ LS* BMD* +7.2% ±0.7 vs ALN* +3.4% ±0.7 (p < 0.001); ↑ TH* BMD* +3.8% ±0.6 vs ALN* +2.4% ±0.6 (p = 0.005); ↑ bone formation markers, ↓ new vertebral fx (0.6%).	ALN*, n=214; 10 mg/day PO* + same calcium and vitamin D regimen.	TPTD* significantly ↑ LS* and TH* BMD* vs ALN* and ↓ new vertebral fractures (0.6% vs 6.1%, p = 0.004); similar nonvertebral fx rate (5.6% vs 3.7%, p = 0.36).
Saag *et al.* (2019). RCT, double-blind, double-dummy, active-controlled (29)	Patients with GIO; age ~61–67 yrs.	DMAb*, n=398; 60 mg SC* every 6 mo + daily oral placebo + calcium ≥1000 mg + vitamin D ≥800 IU.	At 12 mo*, ↑ LS BMD*: +4.4% (GC*-continuing), +3.8% (GC*-initiating); TH*: +1.5%; FN*: +1.0% and +1.1%; ↓ CTX* & P1NP* > RIS* (p < 0.0001–0.046).	RIS*, n = 397; 5 mg/day PO + SC placebo every 6 mo + same supplements.	DMAb* was non-inferior and superior to RIS* in ↑ LS*, TH*, and FN BMD* at 12 mo (e.g., LS* diff. +2.2% [95% CI* 1.4–3.0]); similar fracture and AE* rates in both arms.
Saag *et al.* (2009). RCT, double-blind, double-dummy, active-controlled (30)	Adults aged 22–89 yrs, with GIOP*; all on ≥5 mg/d prednisone for ≥3 mo*	TPTD*, n = 214; 20 µg/day SC + calcium 1000 mg/day + vitamin D 800 IU/day.	At 36 mo*: ↑ LS BMD* +11.0%, ↑ TH* BMD* +5.2%, ↑ FN* BMD* +6.3%; ↑ bone formation markers (P1INP*, OC*, ALP*) throughout; ↓ new vertebral fx (1.7% vs 7.7%, p = 0.007).	ALN*, n=214; 10 mg/day PO + same supplements.	TPTD* led to significantly greater ↑ in BMD* (LS*, TH*, FN*) and fewer vertebral fx vs ALN* over 36 mo*; nonvertebral fx rates similar (7.5% vs 7.0%, p = 0.843); TPTD* ↑ serum calcium more frequently.
Saag *et al.* (2016). Subanalysis of RCT*, double-blind, double-dummy, active-controlled (31)	Patients with GIOP*; mean age 58 yrs; median prednisone dose 7.5 mg/day; ~81% female.	TPTD*, n = 56; 20 µg/day SC* + calcium 1000 mg/day + vitamin D 800 IU/day.	↑ LS* BMD* +10.3% and ↑ TBS* +3.7% at 36 mo* (p < 0.05); ↑ TBS* from 18 mo* onward (p < 0.05 vs baseline and ALN*); weak correlation between BMD* and TBS* changes.	ALN*, n = 53; 10 mg/day PO + same supplements.	TPTD* ↑ both BMD* and TBS*, while ALN* ↑ only BMD*; TBS* was more sensitive to anabolic treatment effects and may better reflect microarchitectural improvements in GIOP*.
Roux *et al.* (2012). Post hoc analysis of RCT*, double-blind, double-dummy, active-controlled (32)	Patients with GIO*; aged 18–85 yrs; on ≥7.5 mg/day prednisone; stratified in treatment.	ZOL*, n = 416; 5 mg IV single infusion + daily PO* placebo + calcium (1000 mg) + vitamin D (400–1200 IU).	At 12 mo*, ZOL* ↑ LS* BMD* significantly vs RIS* in most subgroups (age 35–74, both sexes, postmenopausal women, Vit D >20 ng/mL, CrCl ≥60 mL/min, PPI* use ; p < 0.05).	RIS*, n = 417; 5 mg/day PO* + IV placebo + same supplements.	ZOL* was more effective than RIS* in ↑ LS* BMD* across multiple GIO* subgroups (gender, age, Vit D, steroid dose/duration, renal fxn); effect maintained regardless of baseline BMD*.

Devogelaer *et al.* (2010). Post-hoc analysis of RCT*, double-blind, double-dummy, active-controlled (34)	Adults with GIO*; mean age ~56 yrs; baseline GC* categorized as low (≤5 mg/d), medium (>5–<15 mg/d), or high (≥15 mg/d).	TPTD*, n = 195; 20 µg/day SC + calcium 1000 mg/day + vitamin D 800 IU/day.	LS* BMD* ↑: +8.1% (low), +6.6% (medium), +4.6% (high GC* dose); greater ↑ than ALN* at all dose categories; greatest difference in low GC* group (4.5%, p < 0.001).	ALN*, n = 192; 10 mg/day PO* + same supplements.	TPTD* led to significantly greater LS* BMD* gains than ALN overall (+3.6%, p < 0.001), especially in low and medium GC* dose groups; effect diminished at high GC* doses.
Liu *et al.* (2020). NMA* of 51 RCTs* (35)	Patients with GIOP* from 51 RCTs*; age range varies across trials.	Multiple drugs studied: teriparatide, denosumab, raloxifene, ibandronate, pamidronate, etc.; doses per standard RCT protocols (varies by trial).	TPTD* (SUCRA* 95.9%) best at ↓ vertebral fractures (RR* 0.06, 95% CI*: 0.01–0.27); RAL* ↑ LS* BMD* (SMD* 12.56); DEN* ↑ TH BMD (SMD* 12.63).	Placebo and active comparators depending on trial (ALN*, RIS*, CT*, Ca*, VD3*, etc.); variable n/doses across included studies.	TPTD* and IBA* most effective in reducing vertebral and non-vertebral fractures, respectively; RAL* best for LS* BMD* gain, DEN* for TH* BMD*.
Rehman *et al.* (2003). RCT*, prospective, randomized, controlled, open-label (33)	Postmenopausal women with GIO* on chronic HRT* and GC*; mean age: 62 yrs.	hPTH (1–34)*, n = 28; 40 µg/day SC for 12 months + HRT* + calcium 1500 mg + vitamin D 800 IU.	↑ VCSA* +4.8% at 12 mo (p < 0.001), maintained at +2.6% at 24 mo (p < 0.05); ↑ BMD* by QCT* +34.9% at 12 mo, +44.1% at 24 mo*; ↑ vertebral strength +200%	HRT* alone, n = 23; continued estrogen + same calcium/vitamin D regimen.	hPTH (1–34)* significantly ↑ vertebral size and compressive strength vs. HRT* alone; effect sustained 1 year post-treatment, likely due to periosteal apposition.
Inoue *et al.* (2013). RCT, open-label, parallel-group, prospective (38)	Japanese patients with IgA nephropathy on high-dose GC; 10 men; mean age 28.6 yrs; normal renal function.	RIS*, n = 4; 2.5 mg/day PO* (some changed to ALN* 35 mg/week).	MDCT* detected ↑ BV/TV* (+1.5%, p = 0.004), ↓ Tb.Sp* (–13.1%, p = 0.015), ↓ SMI* (–0.13, p = 0.009), ↑ fracture load (+6.5%, p = 0.034); no sig. changes in BMD* or BTMs*.	Calcitriol, n = 5, 0.5 µg/day; Menatetrenone, n = 4, 45 mg/day.	Only MDCT* detected microarchitectural preservation with RIS*; MDCT* superior in assessing short-term bisphosphonate effect in early GIO*.
Soen *et al.* (2020). RCT, multicenter, open-label, parallel-group, comparative study (39)	Patients with GIO*; mean age ~64 yrs (range 34–85); all on ≥5 mg/d prednisone for ≥3 months.	MIN-M* + ALF*, n = 75; MIN-M* 50 mg PO every 4 weeks + ALF* 1 µg/day.	At 24 mo: ↑ LS BMD* +4.0% (p = 0.0003), ↑ TH* BMD* +3.2% (p < 0.0001); ↓ TRACP-5b* (–57%) and P1NP* (–42%) at 3 mo*; effects sustained at 24 mo*	ALF*, n = 77; 1 µg/day.	MIN-M*+ALF* superior to ALF* alone in ↑ LS* and TH* BMD* and ↓ bone turnover markers; vertebral fx incidence not significantly different (HR* 3.3; 95% CI 0.7–16.1, p = 0.089).

Hirooka *et al.* (2020). Prospective, open-label, non-randomized clinical trial (36)	Patients with GIO* and prior bisphosphonate use; mean age: 66.7 (DEN*) vs. 61.1 yrs (TPTD*); all on ≥5 mg/d prednisone; various connective tissue diseases.	TPTD*, n = 21; 20 µg/day SC*	↑ LS* BMD* +7.9% ($p < 0.001$), ↑ FN* BMD* +6.6% ($p < 0.05$), ↑ TH* BMD* +3.3% (NS*); ↑ TRACP-5b* and P1NP* throughout.	DEN*, n = 20; 60 mg SC every 6 mo*	TPTD* showed greater early and sustained ↑ in LS* and FN* BMD* than DEN*; both ↑ LS* BMD* at 24 mo, but only TPTD* ↑ FN* BMD*; DEN* ↓ BTMs*, TPTD* ↑ BTMs*; 2 fx in DEN* group.
Hirooka *et al.* (2021). Prospective, open-label, non-randomized clinical trial (37)	Patients with GIO* and prior bisphosphonate use; mean age ˜63 yrs; connective tissue diseases; all on glucocorticoids (mean PSL ˜3 mg/day).	Teriparatide → Denosumab (Switch group), n = 18; TPTD* 20 µg/day SC* for 24 mo*, then switched to DEN* 60 mg SC every 6 mo*	↑ LS* BMD* +14.2% ($p < 0.001$), ↑ FN* BMD* +11.2% ($p < 0.05$), ↑ TH* BMD* +7.2% ($p < 0.01$); ↑ BTMs* (TRACP-5b*, P1NP*) with TPTD*, then ↓ with DEN*	Continuous denosumab, n = 16; DEN* 60 mg SC* every 6 mo* for 48 mo*	TPTD*→DEN* produced greater ↑ in FN* BMD* vs continuous DEN* at 48 mo ($p < 0.05$); both ↑ LS* and TH* BMD*; suggests benefit of anabolic-to-antiresorptive sequence in GIO*
Iseri *et al.* (2018). RCT*, single-center, open-label, randomized controlled trial (40)	Patients with GIO* and glomerular disease; median age 66 yrs; all on PSL*; bisphosphonate-naïve.	DMAb*, n = 14; 60 mg SC every 6 months + daily calcitriol 0.25 µg.	↑ LS* BMD* +5.3% at 12 mo ($p < 0.01$); ↓ TRACP-5b* −57.7%, BAP* −30.9%, t-PINP* −57.4% (all $p < 0.01$); no sig. ↑ in FN* or UD* BMD*	ALN*, n = 14; 35 mg PO weekly + daily calcitriol 0.25 µg.	DMAb* produced significantly greater ↑ in LS* BMD* vs ALN* at 12 mo* ($p < 0.05$); stronger suppression of BTMs*; no fx* difference; 1 FN* fx in DMAb* group.
Jinnouchi *et al.* (2000). RCT, single-center, open-label, comparative trial (41)	Patients (Group A: n = 9; Group B: n = 16) with GIO* and diffuse connective tissue disease; age ≥20 yrs; all on ≥5 mg/d prednisone.	Group B: Etidronate 200 mg/day PO* ×14 days every 3 months + V.D3 1 µg/day.	↓ DPD* (−18.1 to −20.2%, $p < 0.05$), ↑ YAM* +5.17% and +5.54% at 24 and 48 weeks ($p < 0.01$ vs baseline; $p < 0.05$ vs control), ↓ new fractures (6.3% at 1 yr vs 31.3% at baseline).	Group A: V.D3 1 µg/day alone.	Combination therapy with etidronate + V.D3* significantly inhibited bone resorption, improved bone mineral level, and tended to reduce new vertebral fractures.
Kanis *et al.* (2007). Systematic review and cost-utility analysis (42)	Postmenopausal women aged ≥50 years in various European countries, primarily at risk of osteoporotic fractures.	Risedronate; modeled cohort, not a clinical trial — typical dose: 5 mg daily.	Reduction in fracture risk (vertebral, non-vertebral, hip); improved cost-effectiveness profile under specific risk thresholds.	Placebo or no treatment; modeled scenario.	Risedronate (5 mg/day) significantly reduced vertebral fracture risk (RR = 0.33; 95% CI: 0.14–0.80) and was cost-effective in women ≥75 years with prior fractures, with an ICER of £35,000/QALY (95% CI: £25k–£106k).

Langdahl *et al.* (2009). RCT, multicenter, double-blind, double-dummy, active-controlled (43)	Patients with GIO*; postmenopausal women, 67 premenopausal women, 83 men; ≥21 years; on ≥5 mg/day, glucocorticoids for ≥3 months.	TPTD* n = 214; 20 µg/day SC + calcium 1000 mg/day + vitamin D 800 IU/day.	LS* BMD* ↑: +7.8% in postMP* women (vs ALN* +3.7%, p < 0.001); +7.0% in preMP* (vs +0.7%, p < 0.001); +7.3% in men (vs +3.7%, p = 0.03); ↓ new vertebral fractures.	ALN*, n = 213; 10 mg/day PO* + same supplements.	TPTD* significantly increased LS* BMD* and reduced vertebral fracture incidence compared to ALN* in all subgroups; effects were independent of sex or menopausal status.