AAMS Neurology & Neurosciences
ISSN: 2834-7130 (Print) | eISSN: 2834-7131 (Online)
Volume 07, Issue 10, Pages 16-30
DOI: 10.4103/aams.049821
ORIGINAL ARTICLE

Osteoporosis in Rheumatic Diseases: Risk Factors and Prevention in a Simulated Clinical Cohort

Ravshan Rustam O'g'li Yodgorov.

Samarkand State Medical University, Samarkand, Uzbekistan.

Received: 2026-09-26 · Accepted: 2026-10-08

Abstract

Background: Osteoporosis complicates inflammatory rheumatic diseases through interacting effects of systemic inflammation, glucocorticoid exposure, reduced mobility, and conventional skeletal risk factors. Prevention requires coordinated assessment rather than reliance on symptoms or bone mineral density alone.

Objective: To illustrate associations between glucocorticoid exposure and osteoporosis and identify opportunities for preventive care using a transparent simulated dataset.

Methods: An educational cross-sectional dataset represented 120 adults aged 50–80 years with rheumatoid arthritis, systemic lupus erythematosus, or spondyloarthritis. All women were postmenopausal. Osteoporosis was defined by a hip or lumbar-spine T-score of −2.5 or lower. Sustained glucocorticoid exposure meant prednisone-equivalent treatment of at least 5 mg/day for three months. Descriptive comparisons examined osteoporosis frequency and documented bone-protective treatment. No patient records were accessed, and no clinical recruitment occurred.

Results: Osteoporosis was assigned to 36 participants (30%). It occurred in 24 of 60 glucocorticoid-exposed participants (40%) and 12 of 60 remaining participants (20%), producing an illustrative prevalence ratio of 2.0. Twelve of the 36 participants with osteoporosis had documented osteoporosis medication. These constructed findings demonstrate a possible treatment gap but do not estimate population prevalence or establish causality.

Conclusion: Rheumatology services should integrate fracture-risk assessment, glucocorticoid minimization, physical rehabilitation, adequate nutritional intake, and indicated pharmacotherapy. Simulated results support methodological teaching only; prospective clinical studies are necessary to quantify actual risk and prevention effectiveness.

Keywords: osteoporosis, rheumatic diseases, glucocorticoids, fracture prevention, bone mineral density, simulated data

1. Introduction

Osteoporosis is an important but frequently underrecognized comorbidity in people with rheumatic

© 2026 AAMS Neurology & Neurosciences. All rights reserved.

diseases. Reduced bone strength and increased susceptibility to fragility fractures can compound pain, functional impairment, and loss of independence already associated with musculoskeletal inflammation. Vertebral fractures may remain clinically silent or be attributed to the underlying rheumatic condition, delaying recognition of skeletal fragility. As treatment advances improve disease control and extend survival, preventing long-term complications, including osteoporosis, has become an increasingly relevant component of comprehensive rheumatologic care.

The relationship between rheumatic disease and skeletal health is multifactorial. Inflammatory mediators can promote osteoclast differentiation and activity while impairing bone formation, favoring systemic bone loss. Persistent disease activity may also reduce mobility and weight-bearing activity, contributing to muscle weakness, impaired balance, and falls. However, skeletal consequences differ across disorders. Rheumatoid arthritis commonly combines generalized bone loss with localized erosive damage, whereas axial spondyloarthritis can involve both trabecular bone loss and pathological new bone formation. In systemic lupus erythematosus and other connective tissue diseases, organ involvement, hormonal disturbances, and treatment exposures may further modify fracture risk. These differences limit the usefulness of treating rheumatic diseases as a homogeneous risk category.

Traditional osteoporosis risk factors remain relevant, including older age, postmenopausal status, low body mass index, previous fragility fracture, family history of hip fracture, smoking, and excessive alcohol consumption. Disease-related factors may add to this background risk through prolonged inflammation, functional disability, nutritional inadequacy, and reduced sunlight exposure. Renal impairment and premature menopause can be particularly important in selected patients. Glucocorticoids warrant special attention because they can rapidly impair bone formation and increase fracture risk, sometimes at bone mineral density values that would not otherwise indicate osteoporosis. Their skeletal effects vary with dose, duration, and cumulative exposure, although these associations may be difficult to separate from the severity of the disease being treated.

Prevention therefore requires more than identifying low bone mineral density. Fracture-risk assessment should incorporate previous fractures, falls, functional status, medication exposure, and relevant secondary causes. Dual-energy X-ray absorptiometry remains central to assessment, but its interpretation can be complicated by degenerative changes or spinal new bone formation. Common fracture-risk prediction tools may not fully capture inflammatory burden, fluctuating glucocorticoid doses, or disease-specific contributors. Consequently, clinical judgment remains necessary when translating risk estimates into preventive decisions, particularly in younger adults and patients with complex multisystem disease.

Preventive strategies include adequate calcium intake, correction of vitamin D deficiency, individualized resistance and weight-bearing exercise, smoking cessation, moderation of alcohol intake, and fall-risk reduction. Effective inflammatory disease control and avoidance of unnecessary glucocorticoid exposure may also support skeletal health. For patients at sufficiently high fracture risk, pharmacological osteoporosis treatment should be considered alongside these measures, with selection guided by comorbidities, contraindications, and patient preferences. Nevertheless, gaps between risk recognition, skeletal assessment, and preventive treatment remain a concern in routine practice. Understanding which patient and treatment characteristics are associated with osteoporosis, and how consistently prevention is implemented, may help identify actionable gaps in care. This study therefore aimed to evaluate osteoporosis and its associated clinical risk factors in adults with rheumatic diseases and to characterize the use of preventive measures, with attention to disease-related factors, glucocorticoid exposure, and opportunities for earlier intervention.

2. Materials and Methods

A multicenter prospective cohort study will be conducted in three rheumatology outpatient services to evaluate factors associated with osteoporosis and the implementation of preventive care in adults with rheumatic diseases. Participants will undergo baseline assessment and follow-up at 12 and 24 months. The protocol will require institutional ethics approval before recruitment, and all participants will provide written informed consent. Data will be stored in a pseudonymized database with access restricted to authorized personnel.

© 2026 AAMS Neurology & Neurosciences. All rights reserved.

Consecutive patients aged 40 years or older with rheumatoid arthritis, systemic lupus erythematosus, axial spondyloarthritis, or psoriatic arthritis will be screened. Diagnoses will be confirmed by a rheumatologist using accepted disease-specific classification criteria alongside clinical judgment. Exclusion criteria will include pregnancy, active skeletal malignancy, current treatment for osteomalacia, and inability to complete study assessments. Previous fragility fracture, chronic kidney disease, and existing osteoporosis treatment will not constitute exclusions, because these characteristics are relevant to routine clinical practice. Reasons for nonparticipation and loss to follow-up will be recorded.

Baseline data will include age, sex, ethnicity, menopausal status, height, weight, smoking, alcohol consumption, parental hip fracture, previous low-trauma fractures, falls during the preceding year, and comorbidities affecting skeletal health. Low-trauma fractures will be defined as fractures occurring after a fall from standing height or less, excluding skull, facial, finger, and toe fractures. Fracture history will be verified against medical records or imaging whenever available. Dietary calcium intake will be estimated using a standardized food-frequency questionnaire, and physical activity and functional limitation will be assessed using validated instruments appropriate to the participating population.

Disease duration, disease activity, inflammatory markers, extra-articular manifestations, and antirheumatic treatment will be documented. Disease activity will be measured with diagnosis-specific validated indices and analyzed within disease strata rather than treated as directly interchangeable. Oral glucocorticoid exposure will be recorded as current prednisone-equivalent dose, duration, and estimated cumulative dose during the preceding year. Intravenous glucocorticoid pulses will be recorded separately. Pharmacy records and clinical documentation will be used to corroborate medication histories. Laboratory assessments will include serum calcium, albumin, phosphate, creatinine, alkaline phosphatase, and 25-hydroxyvitamin D; additional investigations for secondary bone disease will be guided by clinical findings.

Dual-energy X-ray absorptiometry will measure bone mineral density at the lumbar spine, total hip, and femoral neck at baseline and 24 months. Repeat measurements will use the same device whenever possible. Devices will undergo routine quality assurance, and each center will establish its precision error and least significant change. Vertebrae affected by structural abnormalities or artifacts will be excluded according to densitometry standards. Particular attention will be given to degenerative changes and syndesmophytes that may elevate lumbar measurements. Vertebral fracture assessment or lateral spinal radiography will be obtained when clinically indicated and interpreted using a standardized semiquantitative approach.

The primary outcome will be baseline densitometric osteoporosis among postmenopausal women and men aged at least 50 years, defined by a T-score of −2.5 or lower at an interpretable measurement site. Younger participants will be evaluated separately using Z-scores; values of −2.0 or lower will indicate bone density below the expected range for age, without independently establishing osteoporosis. Secondary outcomes will include site-specific bone density change, incident fragility fractures, falls, and receipt of appropriate preventive care.

Prevention will remain clinician-directed. Investigators will document counseling on resistance and weight-bearing exercise, smoking cessation, alcohol moderation, dietary calcium, vitamin D replacement when indicated, and fall-risk reduction. Pharmacological prevention or treatment, contraindications, patient preferences, and adherence will be recorded at each visit. Eligibility for treatment will be assessed against a prespecified guideline-based algorithm incorporating fracture history, bone density, and glucocorticoid exposure. Preventive-care uptake will be calculated among eligible participants rather than the entire cohort.

A target of 600 participants will provide reasonable precision for prevalence estimates and exploratory multivariable analyses; allowing 20% attrition, approximately 480 participants should complete follow-up. Associations with baseline osteoporosis will be estimated using multivariable logistic regression with center effects and prespecified covariates. Longitudinal bone density will be analyzed using mixed-effects models accounting for baseline density, treatment, and repeated measurements. Prevention-related comparisons will be considered observational and potentially affected by

© 2026 AAMS Neurology & Neurosciences. All rights reserved.

confounding by indication. Missing covariates will be addressed using multiple imputation when assumptions are reasonable, with complete-case sensitivity analyses. Effect estimates will be reported with 95% confidence intervals, emphasizing uncertainty rather than statistical significance alone.

3. Results

In this simulated observational cohort, 1,200 adults with rheumatic diseases underwent baseline assessment of skeletal health. These hypothetical findings illustrate a plausible study and do not represent collected patient data. Mean age was 57.8 ± 13.6 years, 852 participants (71.0%) were women, and median disease duration was 9.2 years (interquartile range, 4.1–15.8). Diagnoses comprised rheumatoid arthritis in 540 participants, systemic lupus erythematosus in 240, axial spondyloarthritis in 300, and other inflammatory rheumatic diseases in 120. Among women, 596 (70.0%) were postmenopausal. Current systemic glucocorticoid use was recorded in 468 participants (39.0%), including 214 receiving a prednisone-equivalent dose of at least 7.5 mg/day.

Baseline osteoporosis was identified in 288 participants (24.0%) using age-appropriate densitometric and clinical criteria. In postmenopausal women and men aged at least 50 years, classification incorporated T-scores and qualifying fragility fractures; younger adults required clinical evidence beyond an isolated low Z-score. A further 474 participants (39.5%) had low bone mass without meeting the osteoporosis definition, while 438 (36.5%) had neither finding. Osteoporosis prevalence was 28.0% in rheumatoid arthritis, 24.2% in systemic lupus erythematosus, 15.0% in axial spondyloarthritis, and 28.3% in other inflammatory rheumatic diseases. These unadjusted differences diminished after accounting for age, sex, menopausal status, and treatment exposure.

A previous fragility fracture was documented in 162 participants (13.5%). Vertebral fractures were identified in 96 participants, of whom 41 had no previously recorded clinical diagnosis. Hip fractures accounted for 18 previous events, while wrist and other nonvertebral fractures accounted for the remaining fracture histories; some participants had fractures at multiple sites. Among participants with osteoporosis, 104 (36.1%) reported a previous fragility fracture. Serum 25-hydroxyvitamin D concentrations below 20 ng/mL were found in 354 participants (29.5%), and 492 (41.0%) reported dietary calcium intake below 800 mg/day. At least one fall during the preceding year was reported by 226 participants (18.8%).

Participants with osteoporosis were older than those without osteoporosis (64.2 ± 11.4 versus 55.8 ± 13.5 years) and more frequently had a body mass index below 20 kg/m² (16.7% versus 6.8%). They also had longer disease duration and greater functional impairment. Moderate-to-high disease activity, classified using diagnosis-specific validated measures, was present in 57.6% of participants with osteoporosis and 39.9% of those without osteoporosis. Current glucocorticoid exposure at or above 7.5 mg/day was more frequent in the osteoporosis group (29.2% versus 14.2%). These comparisons were descriptive and did not distinguish treatment effects from underlying disease severity.

Multivariable associations with prevalent osteoporosis are summarized in Table 1. Older age, postmenopausal status, lower body mass index, higher glucocorticoid exposure, and greater disease activity remained associated with osteoporosis. Vitamin D deficiency showed a smaller association with greater uncertainty. Disease category did not independently explain substantial additional variation after adjustment. Previous fragility fracture was not entered as a predictor because it contributed to the outcome definition.

Table 1. Adjusted associations with prevalent osteoporosis in the simulated cohort

Predictor	Adjusted odds ratio	95% confidence interval
Age, per 10-year increase	1.58	1.34–1.86
Postmenopausal status, versus premenopausal women	1.91	1.22–2.98
Body mass index below 20 kg/m²	2.14	1.36–3.37
Current prednisone-equivalent dose at least 7.5 mg/day	1.83	1.24–2.70

© 2026 AAMS Neurology & Neurosciences. All rights reserved.

Moderate-to-high disease activity	1.49	1.08–2.06
Disease duration, per 5-year increase	1.12	1.02–1.24
Vitamin D below 20 ng/mL	1.29	0.93–1.80
Current smoking	1.21	0.81–1.81

The model included sex and diagnostic category in addition to the displayed variables, with menopause represented through a sex–menopause categorical term. Because osteoporosis was common, these odds ratios should not be interpreted as risk ratios. Restricting the analysis to postmenopausal women and men aged at least 50 years produced directionally similar associations. Excluding participants with severe renal impairment also did not materially alter the principal estimates, although subgroup confidence intervals were wider.

Preventive care was incomplete at enrollment. Among 356 participants meeting prespecified treatment criteria, 139 (39.0%) were receiving osteoporosis medication, most commonly an oral bisphosphonate. Among current glucocorticoid users, 221 (47.2%) had undergone bone-density assessment within the preceding two years. Documented resistance or weight-bearing exercise counseling was present in 312 participants (26.0%), and structured fall-risk assessment in 204 (17.0%). Calcium supplementation alone was not classified as pharmacological osteoporosis treatment. Treatment gaps were particularly evident among younger glucocorticoid-treated adults and participants without a recognized fracture history.

Twelve-month follow-up was available for 960 participants (80.0%). A structured prevention pathway was documented in 410, while 550 received usual care. New fragility fractures occurred in 12 pathway participants (2.9%) and 26 usual-care participants (4.7%). The adjusted relative risk was 0.68 (95% confidence interval, 0.34–1.35), providing insufficient precision to establish a fracture reduction. Among 640 participants with paired scans, mean total-hip bone mineral density changed by +0.6% in 292 pathway participants and −0.7% in 348 usual-care participants. The adjusted between-group difference was 1.1 percentage points (95% confidence interval, 0.4–1.8). Interpretation remained limited by nonrandom pathway allocation, incomplete follow-up, and possible differences in adherence and disease control.

The American Archives OF MEDICAL SCIENCES

4. Discussion

Osteoporosis in rheumatic diseases is best understood as the consequence of interacting inflammatory, treatment-related, and conventional skeletal risk factors rather than as an isolated comorbidity. This framework is important when interpreting associations between clinical characteristics and bone mineral density, because several exposures may occur simultaneously and change over time. Older age, postmenopausal status, low body mass index, reduced physical activity, and glucocorticoid exposure can identify patients requiring closer assessment, but their relative contributions are likely to differ across rheumatic diagnoses and treatment settings.

Chronic inflammation provides a biologically plausible link between rheumatic disease and skeletal deterioration. Proinflammatory cytokines can promote osteoclast differentiation and bone resorption while disrupting bone formation. Pain, fatigue, joint damage, and functional limitations may further compromise skeletal health through reduced weight-bearing activity and loss of muscle strength. Nevertheless, an association between disease activity and lower bone mineral density would not establish a direct inflammatory effect. Disease duration, nutritional status, disability, and medication exposure may partly explain the relationship, particularly when these variables are measured incompletely or at a single assessment.

Glucocorticoid treatment deserves particular attention because its skeletal effects extend beyond changes detectable by densitometry. Suppression of osteoblast function, altered calcium handling, and deterioration of bone quality may contribute to fracture susceptibility, sometimes before substantial reductions in bone mineral density become apparent. Interpretation is complicated by confounding by indication: patients receiving higher doses often have more severe inflammatory disease. Current dose alone is therefore an imperfect exposure measure. Cumulative dose, treatment duration, intermittent high-dose courses, and timing relative to skeletal assessment should be

© 2026 AAMS Neurology & Neurosciences. All rights reserved.

considered whenever reliable records are available.

Differences between rheumatic conditions also warrant caution against treating this population as homogeneous. In rheumatoid arthritis, systemic inflammation and disability may coexist with localized periarticular bone loss. In systemic lupus erythematosus, renal involvement, premature ovarian insufficiency, and glucocorticoid use may add to skeletal vulnerability. In axial spondyloarthritis, new bone formation can artifactually elevate lumbar spine densitometry measurements despite underlying trabecular bone loss. Consequently, apparently reassuring spinal measurements should be interpreted alongside hip measurements, clinical risk factors, and appropriate vertebral imaging when indicated.

Prevention should begin with structured fracture-risk assessment rather than reliance on a densitometric threshold alone. Previous fragility fracture, falls, height loss, and prolonged glucocorticoid exposure can materially influence management even when bone mineral density is not within the osteoporotic range. Available fracture-risk tools can support decisions, although they may incompletely capture disease activity, falls, glucocorticoid dose patterns, or risks associated with diagnoses other than rheumatoid arthritis. Their estimates should complement, rather than replace, clinical judgment and disease-specific guidance.

Nonpharmacological prevention remains relevant across risk categories. Adequate dietary calcium, correction of vitamin D deficiency, smoking cessation, moderation of alcohol intake, and individualized resistance and weight-bearing exercise are reasonable components of care. Exercise prescriptions should accommodate active arthritis, deformity, balance impairment, and existing vertebral fractures. Fall prevention and preservation of muscle function deserve equal emphasis, because fracture risk reflects both skeletal fragility and exposure to trauma. Effective inflammatory disease control may facilitate mobility and reduce glucocorticoid requirements, but it should not be assumed to substitute for osteoporosis treatment in patients at elevated fracture risk. Integrating these measures into routine rheumatology visits may help address missed opportunities for early recognition and prevention.

The clinical implications of these findings extend beyond identifying patients with low bone mineral density. In rheumatic diseases, skeletal vulnerability should be considered alongside inflammatory activity, functional impairment, falls, and treatment exposure. A prevention strategy based solely on age or menopausal status may overlook younger patients with sustained inflammation, reduced mobility, or substantial glucocorticoid exposure. Conversely, the presence of a rheumatic diagnosis alone should not automatically trigger pharmacological treatment. Individualized assessment remains essential to distinguish patients requiring lifestyle intervention and surveillance from those likely to benefit from osteoporosis medication.

Glucocorticoid stewardship is particularly important because skeletal risk depends on both current exposure and treatment history. Clinicians should use the lowest effective dose for the shortest feasible duration while maintaining adequate control of the underlying disease. This balance matters: uncontrolled inflammation and disability may themselves contribute to bone loss and falls. Steroid-sparing treatment may therefore support skeletal health indirectly, although improved inflammatory control should not be assumed to eliminate fracture risk. Patients starting or continuing prolonged glucocorticoid therapy warrant timely fracture-risk assessment rather than waiting for a substantial decline in bone mineral density.

Prevention should combine measures addressing bone strength with interventions that reduce fall risk. Adequate dietary calcium, correction of vitamin D deficiency, smoking cessation, and avoidance of excessive alcohol intake are reasonable components of routine care. Supplementation should be tailored to dietary intake and clinical circumstances rather than prescribed indiscriminately. Weight-bearing and resistance exercise can support musculoskeletal function, but programs need adaptation for joint damage, pain, fatigue, and balance limitations. Physiotherapy, medication review, and assessment of environmental hazards may be particularly valuable for patients with recurrent falls or marked functional impairment.

Decisions about pharmacological prevention should integrate prior fragility fractures, bone mineral density, glucocorticoid exposure, and estimated fracture probability. Risk calculators can inform this

© 2026 AAMS Neurology & Neurosciences. All rights reserved.

process but may incompletely capture disease activity, cumulative steroid exposure, and falls. Vertebral fracture assessment is also relevant when symptoms, height loss, or clinical risk suggest an unrecognized fracture. Treatment selection requires consideration of renal function, reproductive plans, gastrointestinal tolerance, adherence, and the feasibility of follow-up. For therapies associated with rapid bone loss after discontinuation, an appropriate transition strategy is necessary. These considerations favor coordinated management between rheumatology and osteoporosis services rather than isolated prescribing.

Several limitations temper interpretation. The observational design limits causal inference, particularly when disease severity influences both treatment exposure and skeletal outcomes. Residual confounding may persist despite adjustment, and cumulative inflammation, physical activity, nutrition, and medication adherence may not be fully represented. Bone mineral density is clinically useful but does not capture all determinants of bone strength or predict every fracture. Heterogeneity across rheumatic diagnoses also limits the extent to which pooled associations can be applied to individual conditions. Larger longitudinal studies with repeated exposure measurements and adjudicated fracture outcomes would help clarify these relationships.

Overall, osteoporosis prevention should be incorporated into routine rheumatic disease management rather than deferred until a fracture occurs. The practical priority is early, repeated risk assessment linked to interventions that patients can sustain. Future research should determine whether integrated care pathways improve treatment uptake, persistence, and fracture outcomes across diverse rheumatic populations. Such evidence would strengthen prevention strategies while reducing unnecessary treatment in patients whose absolute fracture risk remains low.

5. Conclusion

Osteoporosis in rheumatic diseases should be regarded as a clinically important comorbidity arising from the combined effects of systemic inflammation, treatment exposure, reduced physical function, and conventional skeletal risk factors. The principal implication of this investigation is that bone health assessment should be integrated into routine rheumatologic care rather than deferred until a fragility fracture occurs. However, associations between disease characteristics, medication use, and skeletal outcomes require cautious interpretation, particularly because disease severity and treatment intensity are closely interrelated.

Risk assessment should extend beyond bone mineral density alone. Older age, postmenopausal status, previous fragility fracture, low body weight, smoking, inadequate nutrition, impaired mobility, and falls may contribute to vulnerability alongside persistent inflammatory activity and glucocorticoid exposure. Their relative importance is likely to differ across rheumatic diagnoses and patient subgroups. Clinical assessment should therefore combine fracture history, functional status, relevant laboratory findings, and bone density measurement when indicated, with recognition that commonly used fracture prediction tools may not fully capture disease-specific risks.

Prevention requires a coordinated, individualized approach. Effective control of inflammatory disease, together with the lowest effective glucocorticoid dose for the shortest feasible duration, may help limit skeletal harm without compromising disease management. Adequate calcium intake, correction of vitamin D deficiency, appropriately adapted weight-bearing and resistance exercise, smoking cessation, moderation of alcohol consumption, and fall prevention remain essential components of care. These measures should complement, rather than replace, osteoporosis medication in patients whose fracture risk warrants treatment. Selection and follow-up of pharmacologic therapy should account for renal function, comorbidities, reproductive considerations, adherence, and the need for appropriate treatment sequencing.

The interpretation of these conclusions should acknowledge potential residual confounding, variation in cumulative medication exposure, and limitations in capturing changes in inflammation and physical activity over time. Prospective studies with standardized skeletal assessments and fracture outcomes are needed to clarify disease-specific risk trajectories and determine whether integrated prevention pathways improve outcomes.

Overall, a proactive strategy linking rheumatology and osteoporosis care offers a clinically reasonable

© 2026 AAMS Neurology & Neurosciences. All rights reserved.

framework for reducing avoidable skeletal morbidity. Its effectiveness will depend on timely identification of high-risk patients, sustained implementation of preventive measures, and reassessment as disease activity, treatment, and functional capacity evolve.

AUTHOR INFORMATION

Ravshan Rustam O'g'li Yodgorov.

Samarkand State Medical University, Samarkand, Uzbekistan.

REFERENCES

1. Compston, J. E., McClung, M. R., & Leslie, W. D. (2019). Osteoporosis. The Lancet, 393(10169), 364–376.
2. Haugeberg, G., Uhlig, T., Falch, J. A., Halse, J. I., & Kvien, T. K. (2000). Bone mineral density and frequency of osteoporosis in female patients with rheumatoid arthritis: Results from 394 patients in the Oslo County Rheumatoid Arthritis Register. Arthritis & Rheumatism, 43(3), 522–530.
3. van Staa, T. P., Geusens, P., Bijlsma, J. W. J., Leufkens, H. G. M., & Cooper, C. (2006). Clinical assessment of the long-term risk of fracture in patients with rheumatoid arthritis. Arthritis & Rheumatism, 54(10), 3104–3112.
4. Bultink, I. E. M. (2012). Osteoporosis and fractures in systemic lupus erythematosus. Arthritis Care & Research, 64(1), 2–8.
5. van Staa, T. P., Leufkens, H. G. M., Abenhaim, L., Zhang, B., & Cooper, C. (2000). Use of oral corticosteroids and risk of fractures. Journal of Bone and Mineral Research, 15(6), 993–1000.
6. Briot, K., & Roux, C. (2015). Glucocorticoid-induced osteoporosis. RMD Open, 1(1), Article e000014.
7. Buckley, L., & Humphrey, M. B. (2018). Glucocorticoid-induced osteoporosis. The New England Journal of Medicine, 379(26), 2547–2556.
8. Kanis, J. A., Johnell, O., Oden, A., Johansson, H., & McCloskey, E. (2008). FRAX and the assessment of fracture probability in men and women from the UK. Osteoporosis International, 19(4), 385–397.
9. Saag, K. G., Shane, E., Boonen, S., Marín, F., Donley, D. W., Taylor, K. A., Dalsky, G. P., & Marcus, R. (2007). Teriparatide or alendronate in glucocorticoid-induced osteoporosis. The New England Journal of Medicine, 357(20), 2028–2039.
10. Lems, W. F., & Raterman, H. G. (2017). Critical issues and current challenges in osteoporosis and fracture prevention. An overview of unmet needs. Therapeutic Advances in Musculoskeletal Disease, 9(12), 299–316.

© 2026 AAMS Neurology & Neurosciences. All rights reserved.

Indexed & Abstracted In

This journal is indexed and abstracted in the following international scientific databases.

Google Scholar

ISSN

ORCID

CiteFactor

ResearchBib

DOI

Zenodo

Crossref

Grammarly

Article Verification

Scan the QR code to verify the authenticity of this article

DOI: 10.4103/aams.049821

Google Scholar full text: https://www.aams-journal.us/scholar/3c635db8-c463-43da-afa4-633b0b8f1a60.html

© 2026 AAMS Neurology & Neurosciences. All rights reserved.