EJR European Journal of Rheumatology

DOI: 10.5152/eurjrheum.2025.24047

Original Article

Glucocorticoid-Induced Adiposity Is a Predictor of Fragility Fractures in a Large Cohort: A Cross-Sectional Study

Hamzah Amin[1], Muhammed Aqib Khan[2], Zain Sultan[2], Marwan Bukhari[1,2]

Abstract

Background: Glucocorticoids (GCs) predispose individuals to fractures by reducing bone mineral density (BMD) and inducing adiposity. Emerging evidence highlights the harmful effects of adiposity on bone and increased fracture risk. This study aims to investigate GC-induced adiposity as a predictor of fragility fractures in a large population-based cohort.

Methods: Data were collected from patients referred to a regional Dual-energy X-ray absorptiometry (DEXA) scanner in the northwest of England from 2004 to 2019 for their first DEXA scan. Patients underwent bilateral femoral and lumbar spine DEXA scans, providing fat percentage data for the abdomen (FPA), left hip (FPLH), right hip (FPRH), and partial body fat (PBF), as well as BMD data over the scanned areas. A questionnaire was administered to gather demographic variables, Fracture Risk Assessment (FRAX) risk factors, and self-reported history of fragility fractures (defined as a fall from standing height after the of age 50). Comparative statistics were conducted between GC and non-GC populations, with further analysis of fractured and non-fractured GC subgroups. A multivariate logistic regression modeled PBF, FPA, FPLH, and FPRH as predictors of non-spine fractures, adjusting for FRAX risk factors: age, sex, body mass index (BMI), smoking, excess alcohol use (defined as >3 units per day), rheumatoid arthritis, history of fracture, family history of fracture, and the left femoral T-score.

Results: A total of 8023 GC and 23 523 non-GC patients were referred for scans. The mean age of GC patients was 67.3 years (SD 28.3) compared to 63.8 years (SD 13.7) for non-GC patients. Fractured GC patients had higher BMIs, weighed more, and had higher levels of adiposity than non-GC patients. In the GC population, only peripheral fat was significantly different between fractured and non-fractured groups. Partial body fat% (odds ratio [OR] 3.8, 95% CI 1.2-12.2), FPA% (OR 2.4, 95% CI 1.1-4.8), and FPLH% (OR 3.5, 95% CI 1.1-10.6) all predicted non-spine fractures.

Conclusion: A key limitation of this study is the lack of data on the duration and dosage of glucocorticoid (GC) administration, which may have influenced the results. Despite this, GC-induced adiposity was a significant predictor of non-spine fractures, except for fractures in the Fat percentage at the right high (FPRH). High peripheral fat, particularly at the left hip, may indicate regional muscle loss, leading to instability and increased fracture risk. Further research is needed to investigate the relationship between GC dosage, timing, adiposity, and fracture risk, as well as to explore interventions aimed at reversing compositional changes in patients with a history of GC use to prevent fractures.

Keywords: Adiposity, body composition, fracture, glucocorticoids, sarcopenia

ORCID iDs of the authors:
H.A. 0000-0002-2287-381X;
M.A.K. 0000-0002-5323-2484;
Z.S. 0000-0001-7274-2837;
M.B. 0000-0003-4311-5222.

Cite this article as: Amin H, Khan MA, Sultan Z, Bukhari M. Glucocorticoid-induced adiposity is a predictor of fragility fractures in a large cohort: A cross-sectional study. *Eur J Rheumatol.* 2025, 12(3), 0047, doi: 10.5152/eurjrheum.2025.24047.

[1] Lancaster Medical School, Lancaster University, Lancaster, UK
[2] Department of Rheumatology, University Hospitals of Morecambe Bay NHS Foundation Trust, Lancaster, UK

Corresponding author:
Hamzah Amin
E-mail: h.amin1@lancaster.ac.uk

Received: May 30, 2024
Revision Requested: August 5, 2024
Last Revision Received: November 4, 2024
Accepted: September 25, 2025

Introduction

Glucocorticoids (GC) are a class of medications that are widely used in several medical disciplines. Pharmacologically, they mimic the action of the body's endogenous glucocorticoid cortisol. They are clinically useful as they reduce the body's inflammatory response.[1] Hence, they are used in a multitude of diseases where inflammation is a key part of the pathogenesis, including autoimmune diseases such as rheumatoid arthritis and systemic lupus erythematosus. Patients can often be prescribed prolonged doses of GCs, predisposing them to harmful side effects. These side effects include central obesity, hyperglycemia, muscle wasting, and crucially loss of bone mineral density (BMD).[2] The principal mechanism of bone loss is mediated through a decrease in osteoblast activity while increasing the function of osteoclasts.[3] Initial use of GCs primarily affects the trabecular bone; however, prolonged use can also affect cortical bone.[4] The prevalence of fragility fractures as a result of chronic GC use is reported to be as high as 50%, making it a massive public health concern.[5]

Traditionally, adiposity has been thought to be protective against fragility fractures by cushioning falls[6] as well as increasing the mechanical load going through the bones.[7] Furthermore, adiposity's role in the production of estrogen in postmenopausal women is also an important factor in bone health.[8] Current approaches to fracture risk estimation, including FRAX® and QFracture®, indirectly consider adiposity through the use of body mass index (BMI), with increases in BMI being protective against fractures. However, research has highlighted that approaches using BMI may underestimate fracture risk in clinically overweight populations by not considering the true composition of patients.[9] Furthermore, there is growing evidence highlighting both the deleterious effects of adiposity on bones as well as an increased risk of fracture in clinically overweight and obese patients.[9-11]

While the interactions between adipose tissue and bone are complex and not fully known, emerging research is highlighting that adiposity may predispose individuals to fracture by causing a reduction in BMD.[11] Adiposity is thought to reduce BMD by inducing a low-grade chronic inflammatory state[12] as well as through the disruption of hormones important in bone metabolism, such as oestrogen.[8] The location of adipose tissue (subcutaneous versus visceral) is yet another important factor in bone health, with high levels of visceral fat causing bone loss[13] and increasing the risk of fracture.[14] Furthermore, patients with high levels of adiposity may be more likely to fracture due to biomechanical instability.[15] Hence, multiple pathophysiological mechanisms make overweight and obese populations at increased risk of fragility fractures, which may not be accounted for in current approaches using BMI.

Adiposity has yet to be studied as a predictor of fracture in patients taking GCs. This is particularly important given that GC-induced adiposity causes central obesity with an increase in visceral fat.[16] Hence, while GCs directly cause bone loss through disruption of osteoblast and osteoclast activity,[2] GC-induced adiposity may also play a role in precipitating bone loss. Furthermore, additional mechanisms may make GC patients particularly susceptible to fracture. Principally, GC-induced compositional changes at the gluteal sites, including an increase in fat mass, muscle atrophy, and fatty infiltration of muscle, may predispose GC patients to fracture by making them more unstable. However, this has yet to be proven in GC patients and remains a novel hypothesis. Therefore, an understanding of regional fat deposition in patients taking GCs and its impact on fracture risk is important in allowing improvement of fracture risk calculators for this population of patients.

Main Points

- Glucocorticoids cause changes in body composition, including increased regional adiposity.
- We found that increased regional adiposity, except at the right hip, was associated with an increased odds of non-spinal fractures.
- Further focus should be placed on improving body composition in patients on glucocorticoids.

Aims

The aim of this study is to investigate GC-induced central and peripheral (gluteal) adiposity as predictors of fragility fractures in patients with a history of GC use.

Material and Methods

Data Collection

Patients were referred from primary and secondary care in the Northwest of England to the GE Lunar DPX DEXA scanner between 2004 and 2019 under the pretext of suspected osteoporosis. The GE Lunar DPX scanner precisely measures BMD and body composition, including body fat percentage. Fat percentage calculations are based on the differential attenuation of X-rays through various tissues, with the scanner outputting fat mass and lean mass over the scanned regions. Most patients received bilateral femoral and lumbar spine (L1-L4) scans. This provided fat mass, lean mass, and BMD data for these sites, allowing for the calculation of local and total fat percentages. However, data on muscle mass and visceral adipose tissue were not available.

Data on osteoporosis risk factors were collected via a questionnaire at the time of the scan. Risk factors included a history of self-reported fragility fractures (fractures from standing height or less) and other risk factors considered in the Fracture Risk Assessment Tool (FRAX), such as family history of fractures, smoking, alcohol use, and secondary osteoporosis (e.g., type 1 diabetes, osteogenesis imperfecta in adults, untreated long-standing hyperthyroidism, hypogonadism or premature menopause <45 years, chronic malnutrition or malabsorption, and chronic liver disease). Height and weight data were used to calculate BMI (kg/m^2).

Patients were also asked whether they were currently on or had been on glucocorticoids. Information on the dosage and treatment was not collected. Patients who were currently on or had previously been on glucocorticoids were eligible for inclusion in the primary analysis.

Data were stored in a Microsoft Access relational database until extraction for analysis. Full ethical approval for pseudonymized data extraction in the absence of informed consent was obtained from the local ethics committee, National Research Ethics Service Committee Northwest Preston (project number 14/NW/1136), 2014.

Data Analysis

Baseline characteristics were compared between patients with and without a history of glucocorticoid (GC) use as well as GC patients reporting versus not reporting a fracture. Comparative statistics included the Student's *t*-test for normally distributed continuous data and Pearson's chi-squared test for categorical data.

The primary analysis included patients with a history of GC use. A stepwise logistic regression model, reporting odds ratios (OR), was fit to predict non-spine fractures with the primary predictive variables being the partial body fat percentage (PBF), fat percentage at the abdomen (FPA), left hip (FPLH), and right hip (FPRH). All results were adjusted for age, sex, BMI, smoking, excess alcohol use (defined as >3 units per day), rheumatoid arthritis, history of fracture, family history of fracture, and the left femoral T-score.

Results

A total of 31 546 total patients were referred to the scanner. This included 8023 with a history of GC use. Baseline characteristics were compared between the GC and non-GC referred populations to the scanner. The results of this analysis can be seen in Table 1. The GC population displayed many differing characteristics. Such patients were heavier, with higher fat percentages at the abdomen, left, and right femurs. Patients on GCs also had a higher BMD but were younger at the time of the scan. Furthermore, patients on GC also reported a decrease in hip and non-hip fractures compared to patients not on GC. All results, with the exception of smoking status, were statistically significant.

Table 1. Baseline Characteristics Between Patients on Glucocorticoid Versus Patients not on Glucocorticoid

	Glucocorticoid Referred Population	Non-Glucocorticoid Referred Population	*P*
Number of patients referred	8023 (25.4%)	23 523 (74.6%)	
Hip fracture	261 (3.3%)	1104 (4.7%)	.000
Non-hip fracture	2227 (27.8%)	8826 (37.5%)	.000
Age	64.6 (SD 13.4)	65.1 (SD 12.7)	.007
Height	162.8 cm (SD 9.10)	161.8 cm (SD 8.37)	.000
Weight	73.3 kg (SD 16.9)	70.4 kg (SD 15.6)	.000
Body mass index	27.6 (SD 7.3)	26.9 (SD 7.6)	.000
Fat percentage at the abdomen, %	31.7% (SD 11.0)	29.8% (SD 10.9)	.000
Fat percentage at the left hip, %	30.3% (SD 7.6)	29.5% (SD 7.0)	.000
Fat percentage at the right hip, %	29.7% (SD 7.5)	29.0% (SD 6.9)	.000
Left femoral t-score	- 0.9 (SD 1.3)	- 1.0 (SD 1.2)	.000
Current smoker	1071 (13.3%)	3078 (13.1%)	.546
Excess alcohol use (>3 units per day)	401 (5.0%)	1353 (5.6%)	.011

Within the GC population, comparative statistics was performed between the patients who reported a fracture versus non-fractured patients with a history of GC use. The results of this analysis can be found in Table 2. Body mass index, FPA, and weight were not significantly different between the groups, while the FPLH and FPRH were significantly different. Furthermore, GC patients who reported a fracture were older and had a lower left femoral T-score of −1.2 (SD 1.2) versus a T-score of −0.8 (SD 1.3) in the non-fractured GC population.

Our primary analysis demonstrated that the PBF% (OR 3.8, 95% CI 1.2, 12.2), FPA% (OR 2.4, 95% CI 1.1, 4.8) and FPLH% (OR 3.5, 95% CI 1.1, 10.6) all predicted non-spine fractures after adjustment of confounders. This can be seen in table 3.

Discussion

It is important that the results are interpreted within the study design. A referred GC population was analyzed where there was no access to the dosage or length of administration of GCs. Furthermore, given that the GC-referred population also had higher BMDs on average as can be seen in table 1, it could be argued that the study does suffer from confounding by indication. However, on the contrary, it is believed that the results could underestimate the effect size, as patients were referred likely on a proactive basis and hence were less likely to have fractured, as seen in the dataset. Hence, in the future, prospective follow-up of GC patients with adjustment of GC dosages and length of administration would be needed to confirm the findings. Nonetheless, it is believed that the message in this large dataset offsets the limitations and is worthy of reporting.

Table 2. Comparison of Baseline Characteristics Between Fractured and Unfractured Glucocorticoid Patients

	Glucocorticoid and Fracture	Glucocorticoid and Non-Fracture	*P*
Number of patients referred	2373 (29.6%)	5650 (70.4%)	
Gender	1509 females 360 males	4530 females 1624 males	.000
Age	67.3 (SD 28.3)	63.8 (SD 13.7)	.000
Height	161.4 cm (SD 8.7)	163.3 cm (SD 9.1)	.000
Weight	72.8 kg (SD 17.5)	73.4 Kg (SD 16.7)	.190
Body mass index	27.9 (SD 6.2)	27.6 (SD 7.6)	.077
Fat percentage at the abdomen, %	32.3% (SD 10.9)	31.6% (SD 11.0)	.185
Fat percentage at the left hip, %	31.8% (SD 7.5)	29.9 % (SD 7.5)	.000
Fat percentage at the right hip, %	31.1% (SD 7.4)	29.3% (SD 7.5)	.000
Left femoral t-score	- 1.2 (SD 1.2)	- 0.8(SD 1.3)	.000

In the analysis seen in Table 1, comparative statistics demonstrated that patients with a history of GC use were, on average, heavier than the rest of the population, with higher BMIs as well as increased FPA, FPLH, and FPRH. Based on previous literature, this could be expected to be seen as GC induces adiposity.[2] Specifically, GC-induced adiposity is thought to mainly deposit centrally.[16] Interestingly, it was also found that GC patients had greater levels of peripheral adiposity than the general population. A contradictory finding in the dataset was that GC patients had higher levels of BMD and lower rates of fractures than patients not on GCs. This could be due to proactive screening by clinicians (confounding by indication) or due to other confounders not measured in the study, including dose and length of treatment.

In Table 2 of the analysis, where fractured versus non-fractured patients on GCs were compared, it was found that weight, BMI, and FPA were not significantly different between either group. This could indicate that BMI and weight are relatively insensitive measures of fracture risk in GC patients. FPA was also found not to be different between the groups, which could be explained by the nature of GC-induced adiposity preferentially depositing in abdominal sites.[16] However, fractured patients had higher levels of peripheral adiposity, which could lend support to the hypothesis that peripheral adiposity may cause instability, predisposing individuals to falls and hence may be a strong predictor of fracture in this population. Ultimately, though, this is a novel finding and would need to be supported with further data from larger prospective cohorts.

The primary analysis as seen in Table 3 revealed that PBF% (OR 3.8, 95% CI 1.2-12.2), FPA% (OR 2.4, 95% CI 1.1-4.8), and FPLH% (OR 3.5, 95% CI 1.1-10.6) predicted non-spine fractures. However, the FPRH did not predict non-spine fractures. The FPA% results demonstrated the narrowest CI, indicating a higher level of statistical certainty. The FPA% may be an appropriate site to help predict non-spine fractures as high levels of abdominal fat could be indicative of

Table 3. The Primary Analysis Using a Stepwise Logistic Regression, with Regional Adiposity as the Main Predictor of Fracture

Stepwise Regression Analysis Looking At the Relationship Between Non-Spine Fractures and Local Variations in Fat Percentage				
	PBF%	FPA%	FPLH%	FPRH%
Unadjusted	OR 14.0 95% CI 7.2, 27.0	OR 1.7 95% CI 1.1, 2.7	OR 40.4 95% CI 20.0, 81.4	OR 41.2 95% CI 20.2, 84.0
Adjusted for:				
Age; BMI; biological sex	OR 2.7 95% CI 1.1, 6.4	OR 1.0 95% CI 0.6,1.8	OR 7.7 95% CI 2.8, 20.9	OR 5.7 95% CI 2.2, 14.6
Age; BMI; biological sex; current smoker; current alcohol excess; current steroid therapy	OR 2.6 95% CI 1.1, 6.3	OR 1.1 95% CI 0.6, 1.9	OR 6.8 95% CI 2.6, 18.2	OR 5.2 95% CI 2.0, 13.4
Age; biological sex; current smoker; current alcohol excess; current steroid therapy; rheumatoid arthritis; family history of fracture; previously reported fracture	OR 2.8 95% CI 1.2, 6.6	OR 1.1 95% CI 0.6, 2.0	OR 7.4 95% CI 2.7,19.9	OR 5.5 95% CI 2.1, 14.1
Age; biological sex; current smoker; current alcohol excess; rheumatoid arthritis; family history of fracture; previously reported fracture; left femoral T-score	OR 3.8 95% CI 1.2, 12.2	OR 2.4 95% CI 1.1, 4.8	OR 3.5 95% CI 1.1, 10.6	OR 2.7 95% CI 0.9, 8.7

BMI, body mass index; FPA, fat percentage at the abdomen; FPLH, fat percentage at the left hip; FPRH, fat percentage at the left hip; OR, odds ratio; PBF, partial body fat.

patients experiencing the cushingoid effects of glucocorticoids. Consequently, these patients may be at increased risk of fractures due to muscle wasting and lower BMD. These findings align with previous research indicating that abdominal obesity is associated with an increased risk of fractures,[17] and the results of this paper extend this association to patients on glucocorticoids.

While FPLH% predicted fractures, the FPRH% did not after adjusting for the left femoral T-score. This could suggest that the left femoral T-score and right hip fat might not be related, potentially affecting the model's ability to predict fractures. Therefore, a regional approach that considers the right femoral T-score with right hip fat might be more effective; however, no analysis has been performed. The results do imply that FPLH may be a good predictor of non-spine fractures in patients on glucocorticoids. Reasons for this could include that patients with high local fat percentages may have less muscle mass, which could correlate to bone loss locally[18] and precipitate the risk of fracture due to instability. However, this remains a hypothesis and further research is needed to correlate how doses and timing of GCs correlate with local hip fat and how this influences fracture risk in patients on GC. Furthermore, research should also focus on reversing these compositional changes induced in patients with a history of GC and whether these interventions can lower the risk of fractures in patients.

Strengths and Limitations

The study design is limited as there was no information on doses and length of administration of GCs, which may impact adiposity and fracture risk. There was also no follow-up with any of the patients, which limits the ability to make conclusions. The cross-sectional design could mean adiposity could have occurred post-fracture as an unmeasured confounder. The referred population was also very homogeneous, containing nearly 99% Caucasian patients. Hence, more diverse representative samples are needed in further studies.

The study has included many patients, all of whom who underwent the gold standard DEXA scanning. Hence, the large sample means that the study has sufficient power to detect an association. The methods also have the strength that more clinically meaningful outcome of fragility fracture rather than bone loss was used.

In conclusion, the analysis shows that patients on glucocorticoids (GCs) have higher BMIs, weigh more and have higher levels of adiposity than those patients who never have GC use. It was found that overall body fat (PBF%), abdominal fat (FPA%), and left hip fat (FPLH%) were predictive of non-spine fractures. It was hypothesized that high abdominal fat could suggest a cushingoid side effect of GC use, hence predisposing to fractures, while peripheral fat could be a surrogate marker for muscle mass loss at the hip, which would cause lower BMD locally and precipitate fractures in patients on GCs. Further research is needed on GC dosage, timing, and regional fat distribution to improve fracture risk prediction models. Interventions to reverse the compositional changes associated with GC use should be trialed to lower fracture risk in patients with a history of GC use.

Data Availability Statement: The data that support the findings of this study are available upon request from the corresponding author.

Ethics Committee Approval: This study was approved by the National Ethics Research Service Committee of NorthWest - Preston (Approval no.: 14/NW/1136; Date: 2014).

Informed Consent: Informed consent was not obtained from the patients/patient as per our ethical approval which allowed for extraction and pseudo-anonymisation of data.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – H.A., M.B.; Design – H.A.; Supervision – M.B.; Resources – M.B.; Materials – M.B.; Data Collection and/or Processing – Z.S., M.A.K.; Analysis and/or Interpretation – H.A.; Literature Search – H.A., M.A.K.; Writing Manuscript – H.A., M.A.K.; Critical Review – H.A., M.A.K.

Declaration of Interests: HA, MGS and MAK have no disclosures. MB has the following disclosures: M.B. has been sponsored to attend regional, national and international meetings by UCB Celltech, Roche/Chugai, Pfizer, Abbvie, Merck, Mennarini, Janssen, Bristol-Myers Squib, Novartis and Eli Lilly. He has received honoraria for speaking and attended advisory boards with Bristol-Myers Squib, UCB Celltech, Roche/Chugai, Pfizer, Abbvie, Merck, Mennarini, Sanofi-Aventis, Eli-Lilly, Janssen, Amgen, Novartis and Gilead. He has received honoraria from educational groups Revalidaid and TREG consultants.

Funding: The authors declared that this studyhas received no financial support.

References

1. Coutinho AE, Chapman KE. The anti-inflammatory and immunosuppressive effects of glucocorticoids, recent developments and mechanistic insights. *Mol Cell Endocrinol*. 2011;335(1):2-13. [CrossRef]
2. Oray M, Abu Samra K, Ebrahimiadib N, Meese H, Foster CS. Long-term side effects of glucocorticoids. *Expert Opin Drug Saf*. 2016;15(4):457-465. [CrossRef]
3. Compston J. Glucocorticoid-induced osteoporosis: an update. *Endocrine*. 2018;61(1):7-16. [CrossRef]
4. Kobza AO, Herman D, Papaioannou A, Lau AN, Adachi JD. Understanding and managing corticosteroid-induced osteoporosis. *Open Access Rheumatol*. 2021;13:177-190. [CrossRef]
5. Briot K, Roux C. Glucocorticoid-induced osteoporosis. *RMD Open*. 2015;1(1):e000014. [CrossRef]
6. Pana TA, Kioh SH, Neal SR, et al. Body fat percentage and the long-term risk of fractures. The EPIC-Norfolk prospective population cohort study. *Maturitas*. 2023;168:71-77. [CrossRef]
7. Zaslavsky O, Li W, Going S, Datta M, Snetselaar L, Zelber-Sagi S. Association between body composition and hip fractures in older women with physical frailty. *Geriatr Gerontol Int*. 2017;17(6):898-904. [CrossRef]
8. Gkastaris K, Goulis DG, Potoupnis M, Anastasilakis AD, Kapetanos G. Obesity, osteoporosis and bone metabolism. *J Musculoskelet Neuronal Interact*. 2020;20(3):372-381.
9. Amin H, Syed FA, Khan MA, Sultan Z, Bukhari M. Partial body fat percentage as a predictor of fragility fractures in a large cohort: a cross sectional study. *Rheumatol Adv Pract*. 2024;8(1):rkae010. [CrossRef]
10. Hsu YH, Venners SA, Terwedow HA, et al. Relation of body composition, fat mass, and serum lipids to osteoporotic fractures and bone mineral density in Chinese men and women. *Am J Clin Nutr*. 2006;83(1):146-154. [CrossRef]
11. Chen R, Armamento-Villareal R. Obesity and skeletal fragility. *J Clin Endocrinol Metab*. 2024;109(2):e466-e477. [CrossRef]
12. Lumeng CN, Saltiel AR. Inflammatory links between obesity and metabolic disease. *J Clin Invest*. 2011;121(6):2111-2117. [CrossRef]
13. Kim JH, Choi HJ, Ku EJ, et al. Regional body fat depots differently affect bone microarchitecture in postmenopausal Korean women. *Osteoporos Int*. 2016;27(3):1161-1168. [CrossRef]
14. Machado LG, Domiciano DS, Figueiredo CP, et al. Visceral fat measured by DXA is associated with increased risk of non-spine fractures in nonobese elderly women: a population-based prospective cohort analysis from the São Paulo Ageing & Health (SPAH) Study. *Osteoporos Int*. 2016;27(12):3525-3533. [CrossRef]
15. Ghezelbash F, Shirazi-Adl A, Plamondon A, Arjmand N, Parnianpour M. Obesity and obesity shape markedly influence spine biomechanics: A subject-specific risk assessment model. *Ann Biomed Eng*. 2017;45(10):2373-2382. [CrossRef]
16. Lee MJ, Pramyothin P, Karastergiou K, Fried SK. Deconstructing the roles of glucocorticoids in adipose tissue biology and the development of central obesity. *Biochim Biophys Acta*. 2014;1842(3):473-481. [CrossRef]
17. Zhu XW, Liu KQ, Yuan CD, et al. General and abdominal obesity operate differently as influencing factors of fracture risk in old adults. *iScience*. 2022;25(6):104466. [CrossRef]
18. Kawao N, Kaji H. Interactions between muscle tissues and bone metabolism. *J Cell Biochem*. 2015;116(5):687-695. [CrossRef]