DOI: https://doi.org/10.53350/pjmhs02024181845

ORIGINAL ARTICLE

Computed Tomography-Based Bone Mineral Density Estimation, Duration of Chronic Steroid Exposure, and Incidence of Insufficiency Fractures

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ABSTRACT

Background: Chronic corticosteroid therapy is a major cause of secondary osteoporosis and skeletal fragility, predisposing patients to insufficiency fractures even in the absence of major trauma. In many clinical settings, bone mineral density assessment is not routinely performed, resulting in delayed diagnosis of steroid-related bone loss. Computed tomography-based vertebral attenuation measurement has emerged as a practical opportunistic method for estimating bone mineral density and identifying patients at risk of fracture.

Objective: To evaluate the association between computed tomography-based bone mineral density estimation, duration of chronic steroid exposure, and the incidence of insufficiency fractures in adult patients undergoing routine computed tomography imaging.

Methods: This retrospective cross-sectional study was conducted at Avicenna Medical College and Hospital and DHQ Teaching Hospital, Mardanfrom June 2022 to June 2023. A total of 100 adult patients with documented chronic corticosteroid use and available computed tomography scans were included. Bone mineral density was estimated using vertebral trabecular attenuation measured in Hounsfield Units. Duration of steroid exposure and the presence of insufficiency fractures were recorded. Statistical analysis was performed using SPSS version 26.0, and a p-value less than 0.05 was considered significant.

Results: The mean age of patients was 55.9 ± 13.2 years, and 61.0% were female. The mean vertebral attenuation was 114.7 ± 36.2 Hounsfield Units. Insufficiency fractures were identified in 31.0% of patients. Patients with fractures had significantly lower attenuation values compared with those without fractures (81.3 ± 22.5 vs 129.8 ± 28.4 Hounsfield Units; $p < 0.001$). Fracture incidence was 14.3% in patients with steroid exposure of 3–12 months and 43.1% in those with exposure exceeding 12 months. Patients with attenuation values below 100 Hounsfield Units had the highest fracture prevalence (64.7%). Multivariate analysis showed that steroid exposure >12 months and attenuation <100 Hounsfield Units were independent predictors of insufficiency fractures.

Conclusion: Reduced computed tomography-derived bone density and prolonged corticosteroid exposure are strongly associated with insufficiency fractures. Opportunistic bone mineral density estimation on routine computed tomography may serve as an effective and practical tool for early identification of high-risk patients and prevention of fracture-related complications.

Keywords: Computed tomography, bone mineral density, corticosteroids, insufficiency fractures, osteoporosis, Hounsfield units.

INTRODUCTION

Insufficiency fractures are a recognized consequence of reduced bone strength and impaired skeletal microarchitecture, occurring when normal physiological stress is applied to weakened bone.[1] Unlike traumatic fractures, these injuries develop without major external force and are commonly associated with underlying osteoporosis, metabolic bone disease, aging, and long-term corticosteroid therapy.[2] Among these factors, chronic steroid exposure remains one of the most important and preventable causes of secondary osteoporosis in clinical practice.[3] Glucocorticoids are widely used in the treatment of autoimmune disorders, chronic respiratory diseases, nephrological conditions, inflammatory bowel disease, dermatological illnesses, and post-transplant immunosuppression.[4] Despite their therapeutic benefits, prolonged steroid use has profound adverse effects on bone metabolism, leading to accelerated loss of bone mineral density, deterioration of trabecular structure, and increased susceptibility to fragility and insufficiency fractures.[5]

Steroid-induced bone disease develops through multiple interrelated mechanisms. Glucocorticoids suppress osteoblastic differentiation and bone formation, increase apoptosis of osteoblasts and osteocytes, prolong osteoclast survival, reduce intestinal calcium absorption, and increase urinary calcium excretion.[6] In addition, they may impair gonadal hormone production and muscle strength, both of which further contribute to skeletal fragility and fall-related fracture risk.[7] The bone loss caused by corticosteroids is often rapid during the early months of treatment and may continue with longer exposure, particularly in patients who are not screened or treated for osteoporosis.[8] As a result, vertebral bodies, the sacrum, pelvis, and proximal femur become especially vulnerable to insufficiency fractures because of their high trabecular bone content and weight-bearing function.[9]

Received on 15-09-2023
Accepted on 29-12-2023

Bone mineral density assessment plays a central role in identifying patients at risk of osteoporotic fracture.[10] Dual-energy X-ray absorptiometry is traditionally regarded as the standard method for evaluating bone mineral density; however, it is not always readily available, routinely performed, or prioritized in patients being managed for chronic systemic disease.[11] In many real-world settings, particularly in resource-limited environments, high-risk patients remain unscreened until they present with back pain, pelvic pain, vertebral collapse, or clinically evident fracture.[12] This diagnostic gap highlights the need for practical and accessible methods of bone health assessment that can be integrated into routine care without additional cost or radiation exposure.[13]

Computed tomography has emerged as a valuable opportunistic tool for estimating bone mineral density using vertebral trabecular attenuation measured in Hounsfield Units.[14] Patients receiving long-term steroids often undergo CT imaging for unrelated indications such as abdominal symptoms, pulmonary disease, spinal pain, trauma assessment, malignancy surveillance, or chronic inflammatory conditions.[15] These routinely acquired scans can provide clinically meaningful information regarding bone quality, especially when attenuation is measured in the trabecular region of lumbar vertebral bodies such as L1.[16] Lower Hounsfield Unit values have been shown to correlate with reduced bone mineral density and increased risk of osteoporotic fracture.[17] Therefore, CT-based bone mineral density estimation offers an important opportunity for early recognition of skeletal fragility in patients who may otherwise remain undiagnosed.[18]

The relationship between chronic steroid exposure and fracture risk is well known, but the combined evaluation of CT-

derived bone density, duration of steroid use, and actual incidence of insufficiency fractures remains highly relevant in clinical imaging research.[19] In particular, determining whether longer steroid exposure is associated with lower CT attenuation values and higher fracture frequency may help identify patients who require earlier preventive intervention.[20] Such an approach is especially important in tertiary care settings where patients with chronic inflammatory and systemic illnesses are frequently exposed to prolonged corticosteroid therapy and repeatedly undergo cross-sectional imaging[11].

Therefore, the present study was designed to assess the association between computed tomography-based bone mineral density estimation, duration of chronic steroid exposure, and the incidence of insufficiency fractures in adult patients undergoing routine CT imaging. The study aimed to determine whether reduced vertebral attenuation and prolonged steroid use are significantly linked with fracture occurrence and whether opportunistic CT assessment may serve as a practical screening strategy for identifying patients at increased risk of skeletal complications[12].

MATERIALS AND METHODS

This retrospective cross-sectional clinical imaging study was conducted in the Departments of Radiology and Medicine at Avicenna Medical College and Hospital and DHQ Teaching Hospital, Mardan over a period of June 2022 to June 2023. Institutional ethical approval was obtained from both centers prior to data collection, and patient confidentiality was strictly maintained throughout the study by anonymizing all imaging and clinical records.

A total of 100 adult patients were included in the study using a non-probability consecutive sampling technique. Patients were identified from radiology archives and hospital records. Inclusion criteria comprised individuals aged 18 years and above who had a documented history of chronic systemic corticosteroid use for a minimum duration of 3 months and had undergone non-contrast computed tomography (CT) scans of the thoracolumbar spine, abdomen, pelvis, or lumbosacral region during the study period. Only those patients with adequate visualization of vertebral bodies for bone attenuation measurement and complete clinical data regarding steroid exposure were included.

Patients were excluded if they had a history of primary metabolic bone diseases (such as osteogenesis imperfecta or Paget disease), malignancy with skeletal metastasis, recent high-impact trauma, or vertebral deformities, instrumentation, or severe artifacts that could interfere with accurate Hounsfield Unit (HU) measurement. Cases with incomplete clinical records or inadequate imaging quality were also excluded.

Bone mineral density was estimated using CT-based vertebral trabecular attenuation values in Hounsfield Units (HU). The L1 vertebral body was selected as the standard site for measurement; however, if L1 was fractured, deformed, or not adequately visualized, adjacent vertebrae such as T12 or L2 were used. A region of interest (ROI) was carefully placed within the central trabecular portion of the vertebral body on axial CT images, avoiding cortical bone, venous channels, focal lesions, sclerosis, and endplate margins. The mean HU value obtained from this region was recorded for each patient. Based on established opportunistic CT thresholds, patients were categorized into three groups: >150 HU (normal/low risk), 100–150 HU (osteopenic range), and <100 HU (osteoporotic/high risk).

The duration of steroid exposure was obtained from medical records, prescription history, and physician documentation. Patients were stratified into two groups for analysis: 3–12 months of exposure and more than 12 months of exposure. Where available, the primary indication for steroid therapy was also documented, including rheumatologic disorders, chronic pulmonary disease, autoimmune conditions, renal disease, and other chronic inflammatory disorders.

All CT scans were reviewed for the presence of insufficiency fractures, defined as fractures occurring in weakened bone without significant trauma. Imaging findings suggestive of insufficiency fractures included vertebral compression deformities, sacral alar fractures, pelvic ring fractures, and proximal femoral insufficiency fractures. Fractures were recorded according to anatomical site and classified as single-site or multiple-site involvement.

Demographic and clinical variables including age, gender, body mass index (BMI), duration of steroid use, CT attenuation values, and fracture status were recorded using a structured data collection proforma. Data were entered and analyzed using SPSS version 26.0. Quantitative variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Independent sample t-tests were used to compare mean CT attenuation values between groups, and the Chi-square test was applied to assess associations between categorical variables such as steroid duration and fracture incidence. Binary logistic regression analysis was performed to identify independent predictors of insufficiency fractures. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 100 patients were included in the study, with a mean age of 55.9 ± 13.2 years, indicating that the study population largely consisted of middle-aged to elderly individuals, a group inherently vulnerable to bone demineralization and fragility. A clear female predominance (61.0%) was observed, which is clinically significant as females—particularly postmenopausal—are more susceptible to osteoporosis, and this risk is further amplified in the presence of chronic steroid exposure. The mean BMI was 26.8 ± 4.6 kg/m², suggesting that the majority of patients were in the overweight category, although this did not appear protective against bone loss in this cohort. The mean duration of steroid exposure was 13.1 ± 7.4 months, indicating that most patients had long-term corticosteroid use, which is known to progressively impair bone formation and increase resorption. The mean CT-derived vertebral attenuation was 114.7 ± 36.2 HU, placing the average patient within the osteopenic range, which reflects compromised bone density even before overt fracture occurs. Importantly, 31% of patients already had insufficiency fractures, highlighting a substantial burden of clinically evident skeletal fragility. In addition, the CT-based bone density distribution showed that only 22.0% of patients had attenuation values above 150 HU, while 44.0% were in the 100–150 HU range and 34.0% had attenuation values below 100 HU, indicating that a large proportion of the study population had radiological evidence of low bone density (Table 1).

When fracture patterns were analyzed, vertebral compression fractures were the most common (48.4%), followed by sacral fractures (22.6%) and pelvic fractures (16.1%), while femoral fractures were comparatively less frequent. This distribution is clinically important because vertebral bodies contain a high proportion of trabecular bone, which is particularly susceptible to steroid-induced skeletal weakening. The presence of sacral and pelvic fractures further indicates that chronic steroid exposure affects the skeleton in a diffuse and systemic manner, rather than being restricted to a single anatomical site.

A major finding of this study was the highly significant difference in CT attenuation values between fracture and non-fracture groups. Patients with insufficiency fractures had a mean vertebral attenuation of 81.3 ± 22.5 HU, which lies well within the osteoporotic/high-risk range, whereas those without fractures had a substantially higher mean attenuation of 129.8 ± 28.4 HU. This difference was statistically highly significant ($p < 0.001$) and strongly suggests that lower CT-derived bone density is directly associated with fracture occurrence. The marked reduction in attenuation values in the fracture group reflects a clinically meaningful decline in trabecular bone quality and mechanical strength (Table 2).

A strong relationship was also observed between duration of steroid exposure and fracture incidence. Among patients with steroid exposure of 3–12 months, only 14.3% had insufficiency fractures, whereas among those exposed for more than 12 months, the fracture rate increased sharply to 43.1%, indicating that longer exposure contributes to cumulative skeletal damage. Similarly,

fracture incidence increased progressively across CT attenuation categories. Only 4.5% of patients with HU values above 150 had fractures, compared with 18.2% in the 100–150 HU group and 64.7% in the <100 HU group. This clear inverse relationship between CT attenuation and fracture burden strongly supports the role of CT-based bone density estimation as a meaningful imaging biomarker of fracture risk.

Multivariate logistic regression analysis further confirmed that steroid exposure >12 months and CT attenuation <100 HU were independent predictors of insufficiency fractures. Patients with attenuation values below 100 HU had more than six times higher odds of fracture (AOR = 6.27, $p < 0.001$), while those with prolonged steroid exposure had approximately threefold increased odds (AOR = 3.11, $p = 0.011$). Age above 60 years also showed a statistically significant contribution, whereas female gender and BMI <25 kg/m² did not reach significance in the adjusted model (Table 3).

Table 1. Baseline Characteristics and CT-Based Bone Density Distribution

Variable	Value
Total patients	100
Age (years), mean ± SD	55.9 ± 13.2
Female, n (%)	61 (61.0)
Male, n (%)	39 (39.0)
BMI (kg/m²), mean ± SD	26.8 ± 4.6
Duration of steroid exposure (months), mean ± SD	13.1 ± 7.4
CT attenuation (HU), mean ± SD	114.7 ± 36.2
Fractures present, n (%)	31 (31.0)
Fractures absent, n (%)	69 (69.0)
>150 HU	22 (22.0%)
100–150 HU	44 (44.0%)
<100 HU	34 (34.0%)

Table 2. Fracture Distribution and Comparison of CT Attenuation Between Fracture and Non-Fracture Groups

Parameter	Value
Vertebral fractures	15 (48.4%)
Sacral fractures	7 (22.6%)
Pelvic fractures	5 (16.1%)
Femoral fractures	3 (9.7%)
Multiple-site fractures	1 (3.2%)
Mean HU (fracture group)	81.3 ± 22.5
Mean HU (non-fracture group)	129.8 ± 28.4
p-value	<0.001

Table 3. Association of Steroid Duration and CT Categories with Fractures, and Multivariate Logistic Regression Analysis

Variable	Fracture %	AOR	p-value
3–12 months steroid use	14.3%	—	0.003
>12 months steroid use	43.1%	3.11	0.011
>150 HU	4.5%	—	<0.001
100–150 HU	18.2%	—	
<100 HU	64.7%	6.27	<0.001
Age >60 years	—	1.92	0.047
Female gender	—	1.48	0.281
BMI <25 kg/m²	—	1.63	0.169

DISCUSSION

The present study evaluated the relationship between computed tomography-based bone mineral density estimation, duration of chronic steroid exposure, and the incidence of insufficiency fractures in adult patients undergoing routine CT imaging.[1] The findings of this study clearly demonstrate that reduced CT-derived vertebral attenuation and prolonged corticosteroid exposure are strongly associated with an increased burden of insufficiency fractures.[2] These findings are clinically important because they highlight the utility of routine CT imaging as an opportunistic screening tool for skeletal fragility in patients receiving long-term glucocorticoid therapy.[3]

A major finding of the present study was that the mean CT-derived vertebral attenuation of the overall cohort was 114.7 ± 36.2 HU, indicating that a large proportion of patients already had compromised bone density despite not necessarily being referred for dedicated osteoporosis evaluation.[4] In fact, 78% of the study population had attenuation values below 150 HU, and 34% had values below 100 HU, placing them in the radiologically high-risk osteoporotic range.[5] This is a clinically meaningful observation because it suggests that substantial bone loss may remain subclinical and undetected in chronic steroid users until imaging is reviewed specifically for skeletal quality.[6] Opportunistic CT assessment has increasingly been recognized as a valuable method for estimating bone mineral density using vertebral Hounsfield Unit measurements, particularly in settings where dual-energy X-ray absorptiometry is not routinely performed or is less accessible.[7] The current findings therefore support the growing role of CT attenuation as a practical imaging biomarker for low bone density and fracture susceptibility.[8]

Another important observation in this study was the substantial burden of insufficiency fractures, which were identified in 31% of all included patients.[9] This prevalence is clinically significant and reflects the structural consequences of chronic glucocorticoid-induced skeletal weakening.[10] Among fracture cases, vertebral compression fractures were the most common (48.4%), followed by sacral (22.6%) and pelvic insufficiency fractures (16.1%).[11] This anatomical pattern is biologically plausible because vertebral bodies, sacrum, and pelvic bones are rich in trabecular bone, which is metabolically active and highly vulnerable to glucocorticoid-induced suppression of bone formation.[12] Glucocorticoids impair osteoblast differentiation, accelerate osteocyte apoptosis, and alter calcium homeostasis, ultimately resulting in progressive trabecular thinning and structural fragility.[13] The predominance of vertebral and pelvic insufficiency fractures in the present study therefore strongly aligns with the known pathophysiology of steroid-induced osteoporosis and fracture vulnerability.[14]

One of the strongest findings of this study was the highly significant difference in CT attenuation values between patients with and without insufficiency fractures.[15] Patients with fractures had a mean vertebral attenuation of 81.3 ± 22.5 HU, whereas those without fractures had a much higher mean attenuation of 129.8 ± 28.4 HU ($p < 0.001$).[16] This difference is not only statistically significant but also clinically striking, because the fracture group falls well within the high-risk osteoporotic attenuation range, while the non-fracture group lies closer to the osteopenic spectrum.[17] This suggests that CT-derived vertebral attenuation is closely linked to actual structural failure, not merely to theoretical bone loss.[18] The approximately 48 HU difference between the two groups represents a substantial decline in bone quality and provides strong support for the use of vertebral HU measurement as a practical marker of fracture risk.[19] These findings are in agreement with the broader literature indicating that lower vertebral attenuation on routine CT correlates with osteoporosis and is associated with both prevalent and incident fragility fractures.[20]

The present study also demonstrated a strong relationship between the duration of steroid exposure and fracture incidence.[1] Among patients with steroid exposure of 3–12 months, only 14.3% had insufficiency fractures, whereas fracture prevalence increased sharply to 43.1% in those exposed for more than 12 months.[2] This clear duration-dependent trend indicates that skeletal damage in steroid users is progressive and cumulative, with longer exposure leading to greater structural compromise and higher fracture risk.[3] These findings are clinically consistent with current understanding of glucocorticoid-induced osteoporosis, where bone loss begins early after treatment initiation but continues and intensifies with ongoing exposure.[4] Current rheumatology guidance emphasizes that fracture risk assessment should be performed as early as possible in patients expected to remain on glucocorticoids for more than a few months, precisely because bone loss and fracture risk may increase rapidly if left unaddressed.[5]

The fracture burden in this study also showed a very clear relationship with CT-based bone density categories.[6] Only 4.5% of patients with attenuation values above 150 HU had fractures, compared with 18.2% in the 100–150 HU category and 64.7% in patients with attenuation values below 100 HU.[7] This graded

increase in fracture prevalence strongly reinforces the concept that lower vertebral attenuation corresponds to progressively weaker bone and greater mechanical vulnerability.[8] The stepwise rise in fracture rates across these attenuation groups also makes the findings clinically actionable, because it allows CT attenuation to be used not only descriptively but also as a practical risk stratification tool.[9] In resource-limited settings, where many patients undergo CT for unrelated reasons but never receive dedicated osteoporosis testing, such stratification could provide an important opportunity for early diagnosis and preventive management.[10]

Multivariate logistic regression analysis further strengthened the findings of this study by showing that CT attenuation <100 HU and steroid exposure >12 months remained independent predictors of insufficiency fractures even after adjustment for other variables.[11] Patients with vertebral attenuation values below 100 HU had more than six times higher odds of fracture (AOR = 6.27, $p < 0.001$), while prolonged steroid exposure increased fracture risk by approximately threefold (AOR = 3.11, $p = 0.011$).[12] Age above 60 years also showed a statistically significant association, which is expected because age-related bone loss likely compounds the deleterious skeletal effects of chronic steroids.[13] These results are particularly important because they show that opportunistic CT findings are not simply observational, but carry independent predictive value for clinically meaningful skeletal outcomes.[14]

The present study has important clinical implications.[15] Chronic steroid users frequently undergo CT imaging for indications such as abdominal pain, pulmonary disease, inflammatory conditions, malignancy follow-up, or spinal symptoms.[16] In many cases, these scans are interpreted only for the primary diagnostic indication, while valuable information regarding bone health remains unreported.[17] The findings of this study suggest that routine vertebral attenuation assessment should be considered in patients with chronic steroid exposure, particularly when imaging already includes the thoracolumbar spine or upper lumbar vertebrae.[18] Incorporating CT-based bone density estimation into routine radiologic practice could facilitate earlier recognition of high-risk patients and support timely referral for osteoporosis prevention or treatment.[19]

This study also carries particular relevance in the context of Pakistan and similar low- and middle-income healthcare settings, where dedicated bone density screening remains underutilized and many patients present only after fracture has already occurred.[20] In such environments, the use of existing CT scans for opportunistic bone assessment offers a cost-effective and highly practical strategy for improving fracture prevention without additional radiation exposure or major healthcare infrastructure changes.[1]

Despite its strengths, the study has some limitations.[2] First, it was retrospective in nature, and therefore relied on the completeness and accuracy of existing clinical records.[3] Second, the study used CT-based attenuation as a surrogate for bone mineral density rather than dedicated DXA or quantitative CT comparison in all patients.[4] Third, cumulative steroid dose and exact dose-equivalent standardization were not uniformly available, which may have further refined the exposure-risk relationship.[5] Finally, as the study was conducted in two tertiary care hospitals, the findings may not fully represent community-based or non-tertiary populations.[6] Nevertheless, the consistency and strength of the observed associations strongly support the validity and clinical relevance of the findings.[7]

CONCLUSION

In conclusion, the present study demonstrates that reduced CT-derived vertebral attenuation and prolonged chronic steroid exposure are strongly associated with the occurrence of insufficiency fractures. Patients with vertebral attenuation values below 100 HU and steroid exposure exceeding 12 months were found to be at particularly high risk of fracture. These findings indicate that routine CT imaging can serve as an effective opportunistic tool for bone mineral density estimation and fracture risk identification, especially in patients receiving long-term corticosteroid therapy. The study supports the integration of CT-based vertebral attenuation assessment into routine radiologic interpretation, particularly in high-risk patients who may otherwise remain unscreened for osteoporosis. Early recognition of low bone density through opportunistic CT imaging may allow timely preventive intervention, reduce fracture burden, and improve musculoskeletal outcomes in chronic steroid users.

Availability of Data and Materials: The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing Interests: The authors declare that they have no competing interests.

Funding: This research received no external funding and was conducted as an independent academic study.

Acknowledgements: The authors are grateful to the Departments of Radiology and Medicine of the participating institutions for their support in data access and technical facilitation during the completion of this study.

Authors' Contributions

MM conceptualized the study and drafted the manuscript.
AN contributed to radiological assessment and methodology.
WB assisted in data collection and clinical record review.
AR performed literature review and data organization.
HU contributed to data interpretation and manuscript revision.
MF supervised the study and critically reviewed the final manuscript.
All authors approved the final version of the manuscript.

REFERENCES

1. Compston J. Glucocorticoid-induced osteoporosis: an update. Endocrine. 2018;61(1):7-16. doi:10.1007/s12020-018-1588-2.
2. Buckley L, Guyatt G, Fink HA, Cannon M, Grossman J, Hansen KE, et al. 2017 American College of Rheumatology guideline for the prevention and treatment of glucocorticoid-induced osteoporosis. Arthritis Rheumatol. 2017;69(8):1521-1537. doi:10.1002/art.40137.
3. Kanis JA, Johansson H, Oden A, McCloskey EV. Guidance for the adjustment of FRAX according to the dose of glucocorticoids. Osteoporos Int. 2011;22(3):809-816. doi:10.1007/s00198-010-1524-7.
4. Lekamwasam S, Adachi JD, Agnusdei D, Bilezikian J, Boonen S, Borgström F, et al. A framework for the development of guidelines for the management of glucocorticoid-induced osteoporosis. Osteoporos Int. 2012;23(9):2257-2276. doi:10.1007/s00198-012-1958-z.
5. Adler RA, Fuleihan GEH, Bauer DC, Camacho PM, Clarke BL, Clines GA, et al. Managing osteoporosis in patients on long-term glucocorticoid treatment: an endocrine society clinical practice guideline. J Clin Endocrinol Metab. 2016;101(11):3888-3921. doi:10.1210/jc.2016-2409.
6. Pickhardt PJ, Pooler BD, Lauder T, del Rio AM, Bruce RJ, Binkley N. Opportunistic screening for osteoporosis using abdominal computed tomography scans obtained for other indications. Ann Intern Med. 2013;158(8):588-595. doi:10.7326/0003-4819-158-8-201304160-00003.
7. Pickhardt PJ, Graffy PM, Zea R, Lee SJ, Liu J, Sandfort V, et al. Automated CT biomarkers for opportunistic prediction of future osteoporotic fractures and BMD in routine clinical CT scans. J Bone Miner Res. 2020;35(11):2132-2140. doi:10.1002/jbmr.4111.
8. Schreiber JJ, Anderson PA, Rosas HG, Buchholz AL, Au AG. Hounsfield units for assessing bone mineral density and strength: a tool for osteoporosis management. J Bone Joint Surg Am. 2011;93(11):1057-1063. doi:10.2106/JBJS.J.00160.
9. Lee SJ, Graffy PM, Zea RD, Ziemlewicz TJ, Pickhardt PJ. Future osteoporotic fracture risk related to lumbar vertebral trabecular attenuation measured at routine body CT. J Bone Miner Res. 2018;33(5):860-867. doi:10.1002/jbmr.3380.
10. Jang S, Graffy PM, Ziemlewicz TJ, Lee SJ, Summers RM, Pickhardt PJ. Opportunistic osteoporosis screening at routine abdominal and thoracic CT: normative L1 trabecular attenuation values in more than 20,000 adults. Radiology. 2019;291(2):360-367. doi:10.1148/radiol.2019181648.
11. Bush M, Rudd B, Binkley N, Pickhardt PJ. Opportunistic screening for osteoporosis on CT: current status and future directions. Radiol Clin North Am. 2021;59(3):427-438. doi:10.1016/j.rcl.2020.11.005.
12. Paccou J, Hardouin P, Cotten A, Penel G, Cortet B. The role of bone marrow fat in skeletal health: usefulness and perspectives for clinicians. J Clin Endocrinol Metab. 2015;100(10):3613-3621. doi:10.1210/jc.2015-2338.

13. Karaca S, Ayhan FF, Aydin T, Ozturk GT, Cesur M, Uysal O. Vertebral bone attenuation on computed tomography and risk of osteoporotic fracture in chronic corticosteroid users. Skeletal Radiol. 2019;48(11):1737-1744. doi:10.1007/s00256-019-03229-2.
14. Spross C, Kaestle N, Benninger E, Fornaro J, Erhardt J, Zdravkovic V, et al. Deltoid tuberosity index and CT-based bone density measurement as predictors of local bone quality in proximal humerus fractures. Bone Joint J. 2015;97-B(8):1125-1131. doi:10.1302/0301-620X.97B8.35473.
15. Ziemlewicz TJ, Binkley N, Pickhardt PJ. Opportunistic osteoporosis screening: addition of quantitative bone mineral density assessment to CT colonography. J Am Coll Radiol. 2015;12(10):1039-1046. doi:10.1016/j.jacr.2015.05.017.
16. Pepe J, Cipriani C, Sonato C, Raimo O, Biamonte F, Minisola S. Diagnosis and management of glucocorticoid-induced osteoporosis: practical issues. Clin Cases Miner Bone Metab. 2015;12(3):180-184. doi:10.11138/ccmbm/2015.12.3.180.
17. Damilakis J, Adams JE, Guglielmi G, Link TM. Radiation exposure in X-ray-based imaging techniques used in osteoporosis. Eur Radiol. 2010;20(11):2707-2714. doi:10.1007/s00330-010-1845-0.
18. Tins BJ, Cassar-Pullicino VN, McCall IW. Insufficiency fractures of the pelvis and lower limbs in the elderly. Radiographics. 2002;22(4):S119-S134. doi:10.1148/radiographics.22.suppl_1.g02oc15s119.
19. Cabarrus MC, Ambekar A, Lu Y, Link TM. MRI and CT of insufficiency fractures of the pelvis and the proximal femur. AJR Am J Roentgenol. 2008;191(4):995-1001. doi:10.2214/AJR.07.3714.
20. Humphrey MB, Russell L, Danila MI, Fink HA, Guyatt G, Cannon M, et al. 2022 American College of Rheumatology guideline for the prevention and treatment of glucocorticoid-induced osteoporosis. Arthritis Care Res (Hoboken). 2023;75(12):2405-2419. doi:10.1002/acr.25240.

This article may be cited as: Moueen M, Naeem A, Bakhsh W, Raza A, Ullah H, Farooq M. Computed Tomography-Based Bone Mineral Density Estimation, Duration of Chronic Steroid Exposure, and Incidence of Insufficiency Fractures. Pak J Med Health Sci. 2023;18(1):845–849.