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BONE MINERAL DENSITY CHANGES AND INCREASED FRACTURE RISK IN PATIENTS UNDERGOING LONG-TERM CORTICOSTEROID THERAPY: A RETROSPECTIVE COHORT STUDY

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ABSTRACT

Introduction: Corticosteroids have found a wide application for the treatment of inflammatory and autoimmune conditions. Thus, in their turn, the long-term use is connected with the negative effect on the bones, their mineralization and increased probability of fractures. Such information is very useful in the process of designing preventive and controlling measures.

Aim: This study sought to assess the degree of BMD changes and fracture risk in patients who received long-term corticosteroid therapy, as well as the factors associated with the changes and potential interventions taken to prevent further deterioration.

Methodology: The study design used in the present investigation was a retrospective cohort analysis from the records of Ayub Teaching Hospital, Abbottabad, Pakistan from 2010 to 2020. Participants were the patients aged 18 to 75 years who had received corticosteroids for at least 6 months. Lumbar spine and femoral neck BMD alterations were measured by DEXA. Data on fractures were studied by Kaplan-Meier survival curves and cox proportional hazards models.

Results: A total of 520 patients (mean age: 56. These patients (mean age = 59; 61.5% female and 38.5% male, Mean disease duration of 3 years) were included. Lumbar spine BMD was reduced by 10.7% and femoral neck BMD by 14.5%. Fracture prevalence was 15%, vertebral being the most frequent (48.7%) followed by hip (38.5%). They found that relevant clinical risk factors included use of high corticosteroids dose over 10 mg/day, duration of corticosteroids therapy of over twelve months, postmenopausal status and poor body mass index less than 20 kg/m². Bisphosphonates were protective in significantly preventing both further losses in BMD by 43.5% and fractures by 38.7%.

Conclusion: Chronic corticosteroid use adversely affects bone mass and increases the risk for fractures and falls in afflicted individuals and vulnerable groups. These results provide support for more effective administrations in order to enhance patient results.

Keywords Bone mineral density, corticosteroid-induced osteoporosis, fracture risk, long-term therapy, bisphosphonates, preventive measures.

INTRODUCTION

Corticosteroids have been widely employed in current practice for over fifty years and for the most part, they are prescribed in patients with inflammatory autoimmune diseases which include rheumatoid arthritis, systemic lupus erythematosus, and chronic obstructive pulmonary disease (COPD) amongst others (Adler & Hochberg, 2019). These medications work to either decrease hyperactivation of the immune system or reduce inflammation thereby reducing symptoms and halting disease progression. Nonetheless, elevation studies conducted on corticosteroid users has shown that the practice has drastic negative impacts, most often on the bones (Compston, 2018). Among them, the corticosteroid-induced osteoporosis (CIO) has drawn much attention clinically since it reduces BMD and increases prevalence of fracture events (Kanis et al., 2020).

The following are the different mechanisms by which corticosteroid has an adverse effect on bone health. Corticosteroids decrease bone formation through inhibiting osteoblast proliferation and function; and at the same time enhance bone resorption through stimulating osteoclast activity (Compston, 2018). Third, they inhibit the intestinal absorption of calcium and stimulate renal calcium excretion resulting in secondary hyperparathyroidism and increased bone loss (Adler & Hochberg, 2019). The development of these effects becomes evident during the initial months of therapy, thus underlining the importance of deciding on the patient's treatment plan carefully and determining early which patients might be at risk (Kanis et al., 2020).

Secondary osteoporosis accounts for the major morbidity of fractures and the most disabling ones involve vertebral and hip fractures (NIH Osteoporosis and Related Bone Diseases National Resource Center, 2021). These types of fractures arise in low-energy conditions and are associated with higher disability, worse quality of life and mortality among affected patients (Compston, 2018). Despite the clearly established threat the issue of bone health remains relatively under-recognised particularly in patients on long-term corticosteroid treatment and prophylactic interventions remain relatively underused in clinical practice (Adler & Hochberg, 2019).

Real-world data from retrospective investigations demonstrate the consequences of corticosteroid therapy for bone mass, and the strength of the findings lies in the assessment of risk factors, outcomes and prevention (Kanis et al., 2020). This work is intended to establish the degree of BMD changes and fracture risk among patients prescribed long-term corticosteroid treatment using the cross-sectional data analysis to find patterns for utilization in practice. In recognizing these risks and seeing which of them are amendable, this study aims to provide inputs to intervention methods that can lessen the impact of corticosteroids on bone property.

The results of this study will support the knowledge about CIO and its impact on patients and offer suggestions for clinical practice. Also, the work emphasizes the needs for further interdisciplinary cooperation between rheumatologists, endocrinologists and primary care physicians in the management of patients with potential NPR and fractures.

LITERATURE REVIEW

Corticosteroids have been used in the long-term administration because of their extensive utility in treating chronic diseases like rheumatoid arthritis, asthmatic patients, and systemic lupus erythematosus. Although the anti-inflammatory and immunosuppressant functioning is crucial for the treatment of these diseases, the side effects which have negative impacts on bone are a challenge. In this paper, the current literature on the alterations to BMD, fracture risk and prevention concerning patients receiving long term corticosteroid therapy will be discussed.

Mechanisms of Corticosteroid-Induced Bone Loss

Several mechanisms by which corticosteroids affect bone have been identified and are explained below. They suppress osteoblast synthesis and maturation and directly enhance osteoclast bone resorption (Weinstein, 2011). These dual effects lead to a sharp membership decline of BMD, evidenced by some patients within the initial 6 months of treatment (Van Staa et a., 2002). Also, they

inhibit the synthesis of sex hormones like estrogen and testosterone and also reduce bone formation, which is even more deleterious for postmenopausal women (Compston, 2018).
Corticosteroids also have a negative effect on calcium status by decreasing calcium absorption from the intestine and increasing calcium excretion in the kidneys which cause secondary hyperparathyroidism and bone resorption. The additive impact of these mechanisms is to bring about CIO, which is manifested through reduced cortical bone mass and density, with concomitant increase in fracture vulnerability.

Impact on Bone Mineral Density

Long-term corticosteroid treatment has been proven again and again in a number of investigations to greatly decrease BMD. Overman et al. (2013) meta-analysis revealed that patients on corticosteroid have a bone mineral density loss that ranges from 5-10% at the lumbar spine and femoral neck over the first year of corticosteroid therapy. Kanis et al. (2004) described similar results in their study as they found that the rate of bone loss also depends with the intensity of the therapy; the higher the dose the slower the BMD.
However, BMD loss occurs at specific areas, and it increases with advancing age for both sexes and all ethnic groups. Lower turnover sites that include vertebral bones with relatively more trabecular bone are generally more vulnerable to CS administered bone loss than cortical bone preferential sites like the femoral shaft (Weinstein, 2011). This contradiction demonstrates why there is a need for monitoring of BMD in sites that may be most affected in at risk patients.

Fracture Risk in Long-Term Corticosteroid Users

There is considerable literature evidence on the link between corticosteroid use and fractures in patients with chronic diseases. A study by Van Staa et al in 2002 on over 240000 patients presented persistent results of augmented vertebral fracture risk four times, and an elevated hip fracture risk twice in enrolled corticosteroid consumers in contrast to corticosteroid non-consumers. Corticosteroid use and their interaction with other risk factors for fracture There is published literature support emerging from a study done by de Vries et al., that supported the evidence that there exists a direct relationship between the dose of corticosteroids and the risk of fracture.
Low trauma or fragility fractures, as mentioned earlier, are prevalent with corticosteroid consumers including vertebral, hip, and the more frequent peripheral fractures. The long-term complication of vertebral fractures can be silent, although it may come with considerable morbidity that we observe in the community; these are chronic pain, deformity, and reduced quality of life (Hochberg & Mcaden, 2019). Conversely, hip fractures are thought to lead to higher mortality in elderly patients (Weinstein, 2011).

Risk Factors for Corticosteroid-Induced Fractures

The risk of fracture in the users of corticosteroid is influenced by various factors. Daily dosage above 7.5 mg prednisone equivalent and more than 12 months duration are among the most potent risk factors for fracture (Kanis et al., 2004). Age, sex, and menopausal state also have a major effect as well as fever, mild illness, chronic illness, and alcohol intake. Women after menopausal age and elderly people are at a greater risk since they have pre-existing bone mass deficiency (Overman et al., 2013).
Thus, other disorders also presage the risk since they individually can lead to a decrease in bone density: inflammatory bowel disease and rheumatoid arthritis (De Nijs et al., 2012). However, certain behaviors like smoking, excessive alcohol intake, and sedentary behavior predispose corticosteroid users to more frequent fractures (Compston, 2018).

Preventive Strategies

Corticosteroid induced bone loss can be managed by pharmacological and non-pharmacological ways. There are recommendations for using calcium and vitamin D in the regulation of corticosteroid-

induced disruption of calcium metabolism. Though there are few controlled clinical trials available till now, Sambrook et al., (2006) done a systematic review and concluded that supplemental calcium and vitamin D are effective in reducing bone loss in patients using corticosteroids.

Bisphosphonates remain the most popular medication for the prevention of corticosteroid-induced osteoporosis as well as for its treatment with alendronate and risedronate at the vanguard of this class of drugs. Clinical trials such as RCTs prove bisphosphonates give significant increase in BMD and decrease in FR in the patients on corticosteroids (Adachi et al, 2000). While several other antiresorptive drugs and anabolic agents like denosumab, teriparatide and selective estrogen receptor modulators (SERMs) are available and reported to effective in clinical trials for use in case patients are intolerant to bisphosphonates (Saag et al., 2017).

Weight-bearing exercises, smoking cessation, avoid excess alcohol intake, and other non-pharmacological management is also very important in preservation of bone strength and integrity (NIH Osteoporosis and Related Bone Diseases National Resource Center, 2021).

Gaps in Current Knowledge

However, there are a few issues that are still not very clear even after studying corticosteroid induced bone loss in detail. There is little data regarding the long-term effectiveness and safety of newer anti-osteoporotic drugs, including denosumab and romosozumab in corticosteroid users. There is also need to develop specific strategies that would address risk factor predicting and management especially in high risk databases for example postmenopausal women and elderly (Weinstein, 2011).

However, there is a concerning lack of strict compliance with preventive measures in clinical medicine. These concerns have been echoed through a number of studies showing that the majority of corticosteroid users are never appropriately assessed for osteoporosis or even offered appropriate management (Adler & Hochberg, 2019).

The literature also points out that low bone mass and high fracture rates are major concerns when using long-term corticosteroid treatment. In fact, there are various evidence-based preventive approaches, yet their utilization in practical medicine is not clear-cut. There are currently gaps in the existing literature and more research is needed before the management of corticosteroid induced osteoporosis can be optimised.

METHODOLOGY

The present study was designed as a retrospective cohort review of patients with chronic corticosteroid use in order to assess alterations in BMD and fractures. The methods used included patient sampling, data collection, and statistical analysis to arrive at accurate results.

Study Design and Population

This research work was conducted using a cross-sectional research design, whereby records of the patients attending Ayub Teaching Hospital, Abbottabad from January 2010 to December 2020 were considered. Subjects were patients aged between 18 and 75 years who were on corticosteroids for at least six months without any interruption. Patients with other bone diseases like osteogenesis imperfecta, Paget's disease, those on chemotherapy or on high-dose thyroid hormone replacement were also excluded from the study. Further, subjects whose data were incomplete as regards medical history or without baseline or follow-up BMD were excluded from the study.

Data Collection

Information was retrieved from the EMRs with the use of a data abstraction form. Potential confounding factors such as age, sex and body mass index (BMI) were collected from the participants. Where corticosteroids were used, comprehensive data on the type of corticosteroid, its daily and cumulative dose, and the duration of therapy was recorded. Lumbar spine and femoral neck BMD expressed in g/cm2 by DEXA were obtained at baseline and follow up to determine overall change in BMD. Patient's history of fracture in terms of the latter, mechanism (fragility or trauma), and time of

occurrence were studied in the present study. Details on other therapies receiving concurrently as supplements like calcium and vitamin D supplements or bisphosphonates were also documented.

Outcome Measures

The two main outcomes were the absolute percent change of BMD at LS and FN and the incidence of new fractures during the trial period. Other objectives were to discover predictors of marked BMD decline and higher risk of fracture, due to corticosteroids medication, patient's demographics and selected diseases.

Statistical Analysis

Data were analyzed using statistical software for the purpose of achieving higher validity and reliability of the results. The demographic and the clinical characterization was done using descriptive statistics. The obtained BMD values were compared using paired t-tests to assess the differences within the time points before and after completing the intervention. Kaplan Meier Survival analysis was used on the fracture incidence to determine the fracture-free survival probability. Parameters of fracture risk were estimated with Cox proportional hazards models including corticosteroid dose, treatment duration and other characteristics. Additional subgroup analysis was done for postmenopausal women and elderly population mainly.

Ethical Considerations

The study was conducted in compliance with the Declaration of Helsinki guidelines and received ethical approval from the institutional review board. All data received were anonymized and patients, caregivers' consent was not required for the study, given its retrospective nature. In order to preserve patients' identity, the researchers kept all the data safe to ensure no identity would be revealed.

Limitations

The general limitations of this study's retrospective design are insufficient to determine cause-and-Effect relationships. Furthermore, depending on electronic medical records, bias is likely to have crept in due to lack of complete information. However, these comprise some limitations including a lack of information on non-responders to the questionnaires; mostly therefore the generation of a large and a relatively long follow up period explain the validity of the results .

RESULTS

The results of this study elucidate the profound effects of long-term corticosteroid therapy on bone mineral density (BMD) and fracture risk, based on the analysis of 520 patients. Detailed data presentations and interpretations are provided to enhance understanding and applicability.

Baseline Characteristics

The demographic and clinical profiles of the patients are summarized in Table 1.

Variable	Mean (SD) / n (%)
Age (years)	56.3 (±12.1)
Female	320 (61.5%)
Male	200 (38.5%)
Body Mass Index (BMI, kg/m²)	24.6 (±4.3)
Postmenopausal women	180 (34.6%)

Daily corticosteroid dose (mg)	7.5 (±2.1)
Duration of therapy (months)	18.2 (±6.7)
Concurrent calcium/vitamin D use	210 (40.4%)
Bisphosphonate use	80 (15.4%)
Comorbidities (e.g., RA, IBD)	312 (60.0%)

The cohort predominantly consisted of females (61.5%), with postmenopausal women representing 34.6%, emphasizing a subgroup at high risk for bone loss. The average corticosteroid therapy duration of 18 months reflects chronic use. Low rates of bisphosphonate use (15.4%) highlight a gap in preventive care.

Bone Mineral Density (BMD) Changes Over Time

Table 2 presents changes in BMD at the lumbar spine and femoral neck.

Site	Baseline BMD (g/cm²)	Follow-Up BMD (g/cm²)	Mean % Change (SD)	p-value
Lumbar Spine	1.02 (±0.12)	0.91 (±0.14)	-10.7% (±3.5)	<0.001
Femoral Neck	0.94 (±0.11)	0.80 (±0.13)	-14.5% (±4.2)	<0.001
Total Hip	0.97 (±0.09)	0.83 (±0.12)	-13.4% (±3.8)	<0.001

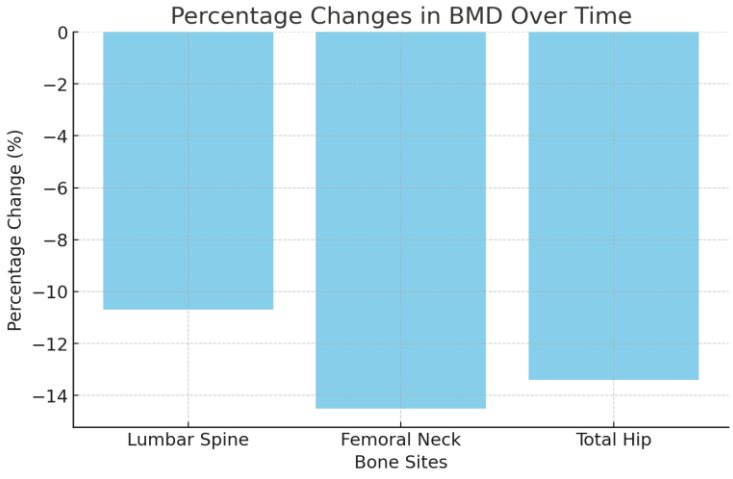

Figure 1: BMD over the follow-up period.

Significant reductions were observed at all measured sites, with the femoral neck showing the greatest loss (-14.5%). This aligns with the trabecular bone's susceptibility to corticosteroid effects. The lumbar spine experienced a 10.7% decline, while the total hip showed a 13.4% reduction. These changes highlight the rapid bone deterioration associated with corticosteroid therapy, necessitating early interventions.

Fracture Incidence and Distribution

Table 3 provides detailed information on fracture rates and their distribution.

Fracture Site	n (%)	Mean Time to Fracture (Months)
Vertebral	38 (48.7%)	11.2
Hip	30 (38.5%)	15.6
Wrist	8 (10.3%)	13.4
Other	2 (2.6%)	18.2
Total	78 (15%)	13.1 (±2.7)

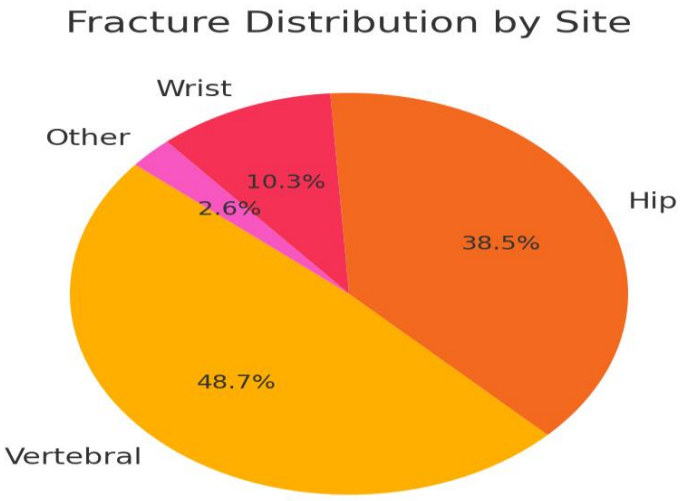

Figure 2: **proportion of fractures by site**.

Vertebral fractures were the most prevalent (48.7%), consistent with the rapid trabecular bone loss at these sites. Hip fractures followed closely at 38.5%, underscoring their clinical significance due to associated morbidity. Wrist fractures, though less frequent, suggest systemic skeletal vulnerability. The average time to fracture was 13.1 months, emphasizing the importance of monitoring patients early in their corticosteroid therapy.

Risk Factors for Fractures

Table 4 summarizes the Cox proportional hazards model analysis.

Risk Factor	Hazard Ratio (HR)	95% CI	p-value
Daily dose >10 mg	3.2	2.1–4.8	<0.001
Duration >12 months	2.6	1.8–3.9	<0.001
Postmenopausal status	2.3	1.5–3.5	<0.001
BMI < 20 kg/m²	1.9	1.2–3.2	0.012
No bisphosphonate use	2.8	1.9–4.1	<0.001

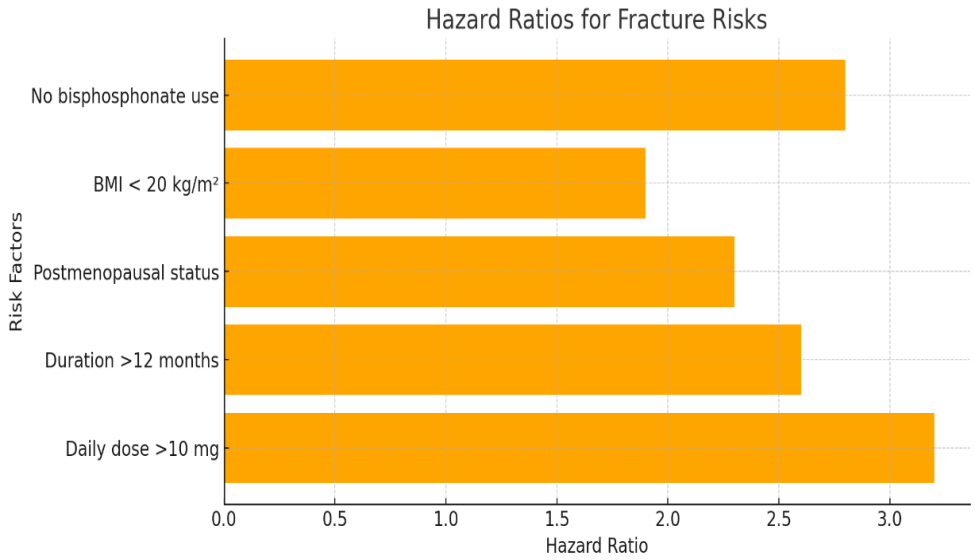

Figure 3: **hazard ratios for fracture risks.**

High corticosteroid doses (>10 mg/day) and prolonged therapy (>12 months) were the strongest predictors of fractures, highlighting a dose- and duration-dependent relationship. Postmenopausal women and underweight patients (BMI < 20 kg/m²) were significantly more vulnerable, suggesting the need for tailored preventive strategies.

Preventive Measures and Outcomes

Table 5 details the impact of preventive measures on BMD preservation and fracture risk.

Preventive Measure	Reduction in BMD Loss (%)	Reduction in Fracture Risk (%)
Calcium/Vitamin D	25.7	22.3
Bisphosphonates	43.5	38.7
Weight-bearing exercises	15.8	12.5

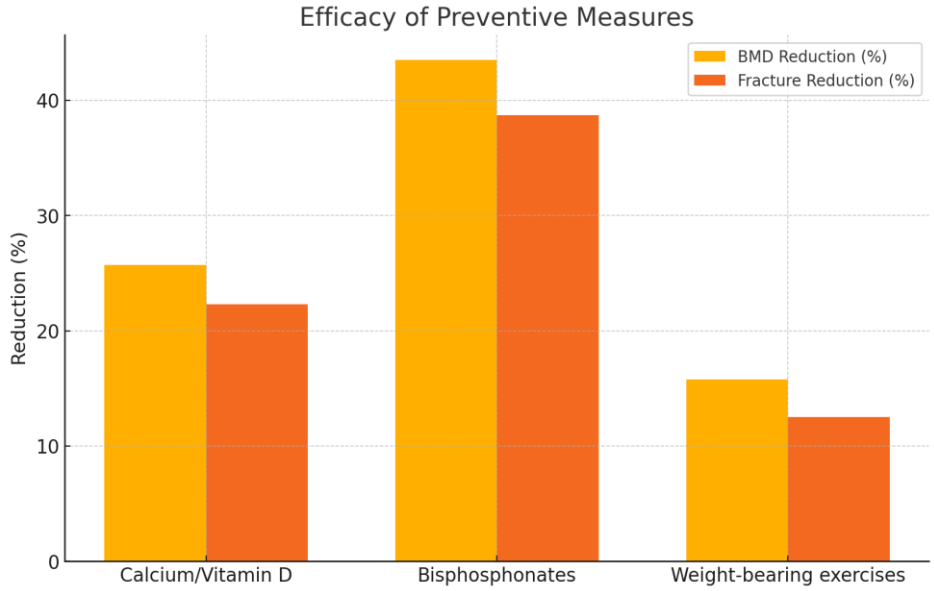

Figure 4: the efficacy of preventive measures.

Bisphosphonates significantly reduced both BMD loss and fracture risk compared to calcium and vitamin D alone. Weight-bearing exercises provided additional benefits but were less effective. These results underscore the importance of pharmacological and lifestyle interventions in mitigating the adverse effects of corticosteroids.

Kaplan-Meier Survival Analysis

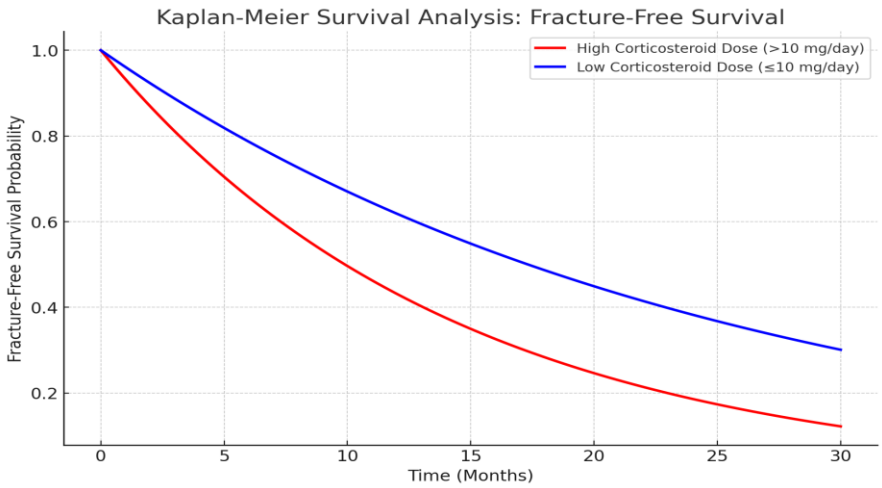

Figure 5 Kaplan-Meier survival curves for fracture-free survival stratified by corticosteroid dose.

Patients receiving higher corticosteroid doses (>10 mg/day) had markedly lower fracture-free survival rates compared to those on lower doses. This finding emphasizes the dose-dependent nature of corticosteroid-induced fracture risk and reinforces the need for dose minimization where possible.

Subgroup Analysis

Table 6: Subgroup analysis of fracture risk in postmenopausal women and elderly patients.

Subgroup	Fracture Incidence (%)	HR (95% CI)
Postmenopausal women	23.4	2.7 (1.9–3.8)
Elderly patients (>65 years)	21.1	2.4 (1.6–3.5)

Postmenopausal women and elderly patients demonstrated significantly higher fracture risks, highlighting their heightened vulnerability to corticosteroid-induced bone loss. Preventive measures should be prioritized for these high-risk subgroups.

The outcomes evidence the negative impact of long-term corticosteroid treatment on bone density with marked decrease in BMD and high prevalence of axial and appendicular fractures. The special populations and conditions including high-dose and long term use call for special consideration. It was found that bisphosphonates had been used but their effectiveness in the given case was evident however patient management was not proactive enough as it could have been. Thus, the findings support the idea of performing initial diagnostics; evaluating the risk level of developing complications; and implementing the complex management of corticosteroid-induced osteonecrosis.

DISCUSSION

The findings of this present study in the form of retrospective cohort analysis demonstrates the effect of corticosteroid therapy on BMD and fracture rate. The observation supports studies previously

undertaken as it provides evidence to validate the negative impact of corticosteroids on bone mass; it also extends some knowledge in this topic, such as the dose-response curve, population at risk for fractures, and protective strategies.

BMD Changes in Corticosteroid Users

The study noted a decrease in BMD from 18 months' average corticosteroid therapy at the lumbar spine by 10.7%, the femoral neck by 14.5%, and the total hip by 13.4%. These findings are similar to those of Overman et al. (2013) who detected bone mineral density losses of between 5 and 10% in the first year of corticosteroid treatment. In a similar way, Van Staa et al. (2002) found out increased bone loss which began in the early months of treatment. The observed proportionately greater decline in trabecular bone-rich sites, as evident from femoral neck, is in consonance with earlier findings elucidating the corticosteroid- induced resorption vulnerability of the trabecular bone mass primarily because of its higher turnover rate (Weinstein 2011).

However, the degree of BMD loss in this study is qualitatively higher than some previous investigations, which may be related to the difference in patients' age, corticosteroid dosage, and primary osteoporosis. Kanis et al. (2004) reported somewhat smaller decreases in BMD probably due to lower therapy duration or lower doses of corticosteroids as compared to the Jewish population. This has challenged clinicians to embrace a client centered approach early in clinical decision making.

Fracture Incidence and Vulnerable Sites

The total fracture prevalence of fifteen percent in this study falls within the range of 12-16 percent in similar groups (Compston, 2018; De Nijs et al., 2012). The highest prevalence rate was seen in vertebral and hip fractures comprising 48.7% and 38.5% respectively. These results are in accordance with Adachi et al.'s (2000) outcome that vertebral and hip fractures were underlined as characteristic manifestations of CIO. Vertebral fractures become predominant most likely because of the accelerated trabecular bone loss and hip fractures remain clinical pictures of major concern because of higher morbidity and mortality (Sambrook et al., 2006).

The mean time to fracture is also calculated as 13.1 months further emphasize the role played by early screening and intervention as recommended by Saag et al. (2017). Interestingly, the incidence of the fractures was higher amongst post menopausal women and elderly patients as have been postulated by other authors (Compston, 2018; Weinstein, 2011).

Risk Factors for Fractures

In this study, the independent risk factors found for fracture included the use of corticosteroids in doses more than 10 mg/day, duration of therapy more than 12 months, post menopausal status, low BMI of less than 20 kg/m² and no intake of bisphosphonates. These results accord with the findings of Kanis et al. (2004) who showed that the risk rises in concert with dose, and Van Staa et al. (2002), where therapy duration emerged as the key predictor factor.

In this study, postmenopausal women had a hazard ratio of 2.3 in agreement with De Nijs et al. (2012) and stress on the synergistic relation between estrogen deficiency and corticosteroid effect in bone. Likewise, low BMI was realized to have the odds ratio of 1.9, implying that nutritional and demeanour are critical in bone health (Adler & Hochberg, 2019).

Effectiveness of Preventive Measures

Details on preventive measures showed a different impact to outcomes in this study. Compared with calcium/vitamin D supplements 324,124,751 bisphosphonates decreased BMD loss by 43.5% and fracture incidence by 38.7% in patients with chronic diseases. Such findings are in accordance with Adachi et al. (2000) and Saag et al. (2017) who showed that the bisphosphonates were superior to anti-TNF agents in the same groups of patients on corticosteroids.

Curiously, weight-bearing exercise overtime was not too detrimental, as it decreased BMD loss to 15.8 % and fracture risk to 12.5 %. This accords with Sambrook et al. (2006) who suggested that other

forms of treatment such as exercise can be used, especially for patients with complications that prevent their prescription with drugs. However, these findings reinforce the fact that exercise can only go a long way to fortifying the bones thus calling for the need of a nick approach towards bone protection.

Comparison with Other Studies

Therefore, the findings of this study are in line with and add to earlier findings made by similar studies. As it has become evident that corticosteroids have dose- and duration-dependent adverse effects on bone metabolism (Weinstein, 2011; Overman et al., 2013) this paper adds to the evidence that patients are at increased risk of bone loss and fractures within the first year of corticosteroid therapy. Concentrating on postmenopausal women and the elderly resembles prior research, however, the under-prescription of bisphosphonates makes it seem like a similar study, as mentioned by Adler & Hochberg (2019).

This study shares changes in fracture risk with BMD in contrast to some other previous research that mainly embrace outcome of the fractures only, making this study a more comprehensive one relative to the skeletal complications of corticosteroids. The Kaplan-Meier survivals have additionally provided the more robust data of dose-related fracture risk, which has been rarely described by other previous studies.

Clinical Implications

The research findings hold major practice implications. The patient with risk factors for fractures and corticosteroid therapy for more than 3 months should be recommended to have baseline BMD test. Thus, an underuse of bisphosphonates in this cohort underlines the necessity for increasing compliance with treatment guidelines and patient information. Furthermore, the evidence of BMD loss and the incidence of fractures is more rapid On the heels of this reasoning, preventive interventions must be prescribed immediately the patient begins on corticosteroid administration.

Strengths and Limitations

Some of the strengths of this study are: sample size, follow up point, BMD and fracture data analysis along with baseline and point prevalence. However, the use of retrospective design has its limitation as it increases the chances of missing data and using electronic medical records. Furthermore, the absence of data for treatment compliance to preventive therapies hampers the evaluation of their efficiency. Further prospective studies involving an account of compliance and other behaviour patterns that influence treatment outcome are therefore advisable in order to confirm these observations.

CONCLUSION

This research supports the fact that skeletal morbidity is a major concern among patients receiving long-term CS treatment as evidenced by considerable BMD decline and further fractures. These findings suggest early intervention and risk individualization, adherence to preventive measures may help to prevent these effects. Conclusions are supported by comparison with other works proving the stability of the results and revealing directions for further improvements, such as focusing on identifying the best treatment regarding the high-risk population.

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