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Short-Term Effects of Corticosteroid Therapy on Blood Glucose Fluctuations in Hospitalized Patients

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Abstract

Background: Corticosteroids are extensively used among hospitalized patients due to their anti-inflammatory and immunosuppressive properties, yet have been demonstrated to cause hyperglycemia. Fluctuations in short-term glucose during steroid therapy may enhance morbidity, especially among patients with pre-existing diabetes.

Objective: To assess the immediate impacts of corticosteroid treatment on blood glucose variability in hospitalized clients.

Methods: The length of this prospective observational study was six months from January 2025 to July 2025. Consecutive sampling was used to enroll 216 patients who were undergoing systemic corticosteroids ≥48 hours. Capillary glucose levels were assessed both at fasting and post-prandial rates for five days following the initiation of corticosteroids. Standard deviation (SD) and mean amplitude of glycemic excursions (MAGE) were used to measure glycemic variability. ANOVA, independent t-tests, Mann-Whitney U, and chi-square tests were used to compare diabetic and non-diabetic patients. The significance level was set to a p-value less than 0.05.

Results: The level of blood glucose rose markedly in both control and diabetic patients under corticosteroid treatment ($p<0.001$). The patients with diabetes had an increased glycemic variability (SD: 20.3 ±5.6 vs 12.8 ± 4.2 mg/dL, $p<0.001$; MAGE: 45.6 ±10.2 vs 28.7 ±7.4 mg/dL, $p<0.001$) and a higher frequency of hyperglycemic episodes ($p<0.001$).
Conclusion: In hospitalized patients, corticosteroid therapy in the short-term causes severe glucose variations, especially in the post-prandial period and in diabetic patients. The metabolic complications of corticosteroid therapy should be reduced by early glucose monitoring and individualized glycemic management.

Introduction

The most common drugs used in hospitalized patients are the corticosteroids because they possess great anti-inflammatory, immunosuppressive, and anti-allergic properties.[1] They are usually applied in the treatment of acute and chronic diseases like asthma, chronic obstructive pulmonary disease, autoimmune diseases, cerebral edema, septic shock, and post-transplant immunosuppression.[2, 3] Although corticosteroids are undoubtedly considered a beneficial therapeutic option, they are also infamous for eliciting massive metabolic side effects, and glucose homeostasis disruption is especially crucial in an inpatient population.[4]

The hyperglycemia induced by steroids is due to the increased gluconeogenesis in the liver, decreased peripheral glucose uptake, and insulin secretion and sensitivity.[4] They can be experienced in a very short period of time, even in patients who have pre-existing diabetes mellitus, and even in patients who have no history of glucose intolerance.[5] Acute changes in the level of blood glucose in hospitalized patients are a clinical concern because they are linked to the risk of infection, wound healing, and longer hospitalization, higher admission to intensive care, as well as morbidity and mortality.[6]

According to epidemiological data, hyperglycemia caused by corticosteroids is not a rare condition, but it is often underdiagnosed. Research indicates that about 30%-50% of patients admitted to hospitals who are taking systemic corticosteroids develop hyperglycemia, and 20%-40% of patients with unknown diabetes may develop new-onset steroid-induced hyperglycemia.[4, 7] In addition, it has been demonstrated that short- and intermediate-acting corticosteroids induce high post-prandial and afternoon glucose excursions, which add to high glycemic variability, but not to sustained fasting hyperglycemia.[8] These variations have become more acknowledged as autonomous predictors of unfavorable clinical results in hospitalized patients.

Although corticosteroids are widely used in acute care, limited local and regional data are available with a specific emphasis on the short-term outcomes of corticosteroid therapy on blood glucose variability during hospitalization.[9, 10] The majority of existing research considers long-term intake of steroids, or they target only diabetic groups, thus ignoring temporary but clinically significant glycemic imbalances in non-diabetic patients.[11, 12] This knowledge of these short-term glucose alterations is critical in the prompt diagnosis, prompt observation, and correct glycemic interventions in hospitalized patients under corticosteroid therapy.

Since corticosteroid use is quite high among hospitalized patients, and acute glucose variability may have a significant adverse effect on patient outcomes, there is a strong need to determine the short-term glycemic impact of corticosteroid treatment. This research was thus aimed at determining the immediate impact of corticosteroid treatment on the glucose fluctuation in hospital patients. The purpose of the research is to compare the blood glucose levels before and after the corticosteroids and to assess the rate and trend of glucose patterns during the short course of corticosteroid use in the hospital environment.

Methodology

This was a prospective observational cohort study that assessed short-term outcomes of systemic corticosteroid treatment on blood glucose variability in patients in the hospital. The study was carried out in Tertiary Care Hospital of Lahore from January 2025 to July 2025.

The sample size was estimated through the help of OpenEpi (Version 3.01) basing on the findings of the previous research that reported the incidence of steroid-induced hyperglycemia in hospitalized patients to be about 34% in the same environment in the tertiary care hospitals.[13] The confidence level of 95%, an anticipated proportion (p) of 0.34, and a desired level of 7% yielded a required minimum sample size of approximately 195 patients. The final target population was 216 pieces after the consideration of a possible 10% loss to follow-up or incomplete data.

The method used was a consecutive sampling technique, in which all the eligible patients admitted in the study wards who met the inclusion criteria within the study period were invited and included until the required sample size was attained. This methodology has been selected to reduce selection bias, as well as provide representation of actual world clinical practice in the hospital environment.

The patients were to be considered included when they were adults (age 18 years and above) and were admitted to the study wards and received systemic corticosteroid therapy (oral or intravenous) for at least 48 hours during the period of hospitalization. Differential glucose responses were evaluated by including both patients diagnosed previously with diabetes mellitus and those without any diagnosis of the disease. Patients were also excluded when they had a known endocrine condition that could lead to glucose metabolism (other than diabetes mellitus), when continuous parenteral nutrition was used, when the pancreas was pathologized, when pregnant, or when the medical records of blood glucose monitoring were incomplete.

Baseline demographic and clinical data were gathered after institutional ethical approval and informed written consent of participants in the form of age, sex, body mass index, comorbidities, and type, dose, and duration of corticosteroid use based on the medical record of each participant. The standardized point-of-care glucose meters were used to measure capillary blood glucose levels. Glucose samples were collected on the initial five days of corticosteroid treatment, daily: fasting (pre-breakfast) and post-prandial (2 hours after eating), and subsequently on 48-hour intervals until the steroid treatment was stopped in case treatment was longer than five days. Glycemic variability scales, such as standard deviation (SD) of glucose values, mean amplitude of glycemic excursions (MAGE), and hyperglycemic events (glucose >180 mg/dL) and hypoglycemic events (glucose <70 mg/dL), were measured. The trained research assistants entered all the data into a pre-designed standardized proforma.

Data collected was then analyzed using the IBM SPSS Statistics (version 26). Continuous variables were measured in terms of mean and standard deviation (SD) or median and interquartile range (IQR) values, respectively. Frequencies and percentages were used as the presentation of categorical variables. Repeated measures ANOVA was used to compare mean glucose levels with time. Independent t-tests (continuous variables) and chi-square tests (categorical data) were used to perform subgroup analyses between patients with and without preexisting diabetes on the effects of glycemic variability. A p-value less than 0.05 was regarded as statistically significant.

Results

The study incorporated 216 patients, of whom 69.4% were non-diabetic, and 30.6% had pre-existing diabetes mellitus. The average age of the respondents was 52.3 ± 14.1 years, with diabetic subjects being marginally older as compared to non-diabetic individuals (p=0.045). Most of the respondents were men 57.4%, and the body mass index (BMI) was also significantly higher in diabetic patients (p=0.002). The most

prescribed corticosteroid was prednisolone 59.3%, with dexamethasone being the one used by 40.7% of respondents. The average length of steroid treatment was 5.80 ± 0.12 days of steroid medications without any statistical difference between the two groups (p=0.18) (Table 1).

Table 1. Baseline Characteristics of Study Participants (n = 216)

Characteristic	Total (n=216)	Non-Diabetic (n=150)	Diabetic (n=66)	p-value
Age, mean ± SD (years)	52.3 ± 14.1	50.7 ± 13.8	55.2 ± 14.5	0.045*
Male, n (%)	124 (57.4)	86 (57.3)	38 (57.6)	0.97
BMI, mean ± SD (kg/m²)	27.5 ± 4.6	26.8 ± 4.3	29.2 ± 5.1	0.002*
Corticosteroid type, n (%)				0.32
Prednisolone oral	128 (59.3)	92 (61.3)	36 (54.5)	
Dexamethasone IV	88 (40.7)	58 (38.7)	30 (45.5)	
Duration of steroid therapy, days, mean ± SD	5.8 ± 1.2	5.7 ± 1.1	6.0 ± 1.3	0.18
*Independent t-test for continuous variables, chi-square for categorical variables; *p<0.05 considered significant.				

After the administration of corticosteroids, the level of blood glucose rose in both non-diabetic and diabetic individuals. Fasting glucose in non-diabetic subjects increased in the group, with a baseline of 96.2 ± 8.5mg/dl, and on day 5, 115.4 ± 12.5mg/dl, and post-prandial glucose baseline was 136.7 ± 18.3mg/dl on day 1, and 138.8 ± 17.5mg/dl on day 5. The patients with diabetes revealed greater glucose excursions, and the fasting glucose levels grew to 179.3 ± 26.8mg/dL and post-prandial levels to 218.6 ± 29.5mg/dL in the same period. The glucose increased significantly over time in both groups (p<0.001), and glucose levels were significantly higher in diabetic patients than in non-diabetic patients at all-time points (p<0.001) (Table 2).

Table 2. Blood Glucose Levels Over 5 Days of Corticosteroid Therapy

Time Point	Non-Diabetic (mg/dL), mean ± SD	Diabetic (mg/dL), mean ± SD	p-value (between groups)	p-value (over time, repeated measures ANOVA)
Baseline (pre-steroid)	96.2 ± 8.5	148.4 ± 22.7	<0.001*	–
Day 1 Fasting	112.5 ± 12.1	178.9 ± 25.4	<0.001*	<0.001*
Day 1 Post-Prandial	136.7 ± 18.3	215.3 ± 28.6	<0.001*	<0.001*
Day 3 Fasting	118.9 ± 13.2	182.6 ± 27.1	<0.001*	<0.001*
Day 3 Post-Prandial	142.1 ± 19.0	222.5 ± 30.2	<0.001*	<0.001*
Day 5 Fasting	115.4 ± 12.5	179.3 ± 26.8	<0.001*	<0.001*
Day 5 Post-Prandial	138.8 ± 17.5	218.6 ± 29.5	<0.001*	<0.001*
*Independent t-test for comparison between groups; repeated measures ANOVA for change over time; *p<0.05 considered significant.				

Glycemic variability (standard deviation, SD, and mean amplitude of glycemic excursions, MAGES) was significantly higher in diabetic patients than non-diabetic patients (SD: 20.3± 5.6 vs 12.8 ± 4.2 mg/dL, p= 0.001; MAGE: 45.6 ±10.2 vs 28.7± 7.4 mg/dL, p= 0.001). The relative rates of hyperglycemic events (>180 mg/dL) were significantly greater in patients with diabetes (p<0.001), and there were no differences in the relative rates of hypoglycemic events (<70 mg/dL) between the groups (p=0.64) (Table 3).

Table 3. Glycemic Variability and Hyperglycemic Episodes

Parameter	**Non-Diabetic (n=150)**	**Diabetic (n=66)**	**p-value**
Mean SD of glucose (mg/dL)	12.8 ± 4.2	20.3 ± 5.6	<0.001*
Mean MAGE (mg/dL)	28.7 ± 7.4	45.6 ± 10.2	<0.001*
Patients with ≥1 hyperglycemic episode (>180 mg/dL), n (%)	32 (21.3%)	58 (87.9%)	<0.001*
Patients with ≥1 hypoglycemic episode (<70 mg/dL), n (%)	5 (3.3%)	3 (4.5%)	0.64
*Independent t-test for continuous variables; chi-square for categorical variables; *p<0.05 considered significant.			

Both groups had more post-prandial hyperglycemia than fasting hyperglycemia. The prevalence of post-prandial hyperglycemia was 21.3versus 12% fasting hyperglycemia in patients who were not diabetic (p=0.03). Post-prandial hyperglycemia was found to be 87.9% in diabetes patients, which is significantly higher than fasting hyperglycemia (p=0.01) (Table 4).

Table 4. Comparison of Fasting vs Post-Prandial Hyperglycemia

Patient Group	**Fasting Hyperglycemia n(%)**	**Post-Prandial Hyperglycemia n(%)**	**p-value**
Non-Diabetic (n=150)	18 (12%)	32 (21.3%)	0.03*
Diabetic (n=66)	44 (66.7%)	58 (87.9%)	0.01*
*Chi-square test for paired categorical comparisons; *p<0.05 considered sisignificant.			

Discussion

In our prospective observational cohort study on hospitalized patients who received short-term corticosteroid therapy, we found that the level of blood glucose in diabetic patients significantly increased during the therapy period, and the glycemic variability and the number of hyperglycemic episodes were greater in diabetic patients than in non-diabetic patients. The urgent effect of corticosteroids on glycemic regulation is our finding and consistent with the emerging literature that indicates the clinical significance of terrestrial administration of glucocorticoids in inciting clinically significant blood glucose variables in diabetic and non-diabetic people.

The identified trend of glucose rising shortly after the beginning of corticosteroid treatment is in line with past studies. The same acute glucose variability was reported in a prospective study by Pakistan in the case of diabetic patients on corticosteroid therapy, which found that there was a great deal of post-prandial peaks and more demand on the modulation of therapy, and this supports our observation that corticosteroid therapy is a deterrent to glycemic control, particularly in individuals with a preexisting diabetes diagnosis.[14] In addition, the high rates of steroid therapy with elevated blood glucose in our cohort are consistent with the estimates of a Saudi Arabia-based study that indicated that around one-third of the hospitalized

corticosteroid users developed hyperglycemia.[13] These results support the thesis that steroid-induced hyperglycemia is a common and relevant clinical issue in the inpatient environment.

Several large-scale observational studies have also supported the finding that there is an association between systemic glucocorticoid treatment and hyperglycemic outcome. Matched cohort studies of over 450,000 adults hospitalized revealed that exposure to systemic corticosteroids increased the risk of new onset hyperglycemia more than twofold over non-exposed patients, and greater cumulative steroid doses, and in older adults and heavier patients.[15] Even though this study mainly dealt with new onset hyperglycemia, its findings are compatible with ours that the use of corticosteroids is strongly related to the marked disruption of the glucose homeostasis process in the hospitalized period.

Our result on the extent of glycemic variability, especially in diabetic individuals, is reminiscent of the findings of a systematic review and meta-analysis that highlighted the commonness and clinical importance of glucocorticoid-induced hyperglycemia and the difficulties encountered in the quest to achieve optimal glycemic control in the absence of specific therapeutic recommendations.[16] Another finding of this meta-analysis is that intensive glucose-lowering interventions can lead to superior glycemic control, which can be applied to interpret our findings of a higher MAGE and glucose standard deviation in the diabetic subgroup.

Additional evidence supporting this can be found in the research conducted on the nature and the strength of steroid regimens. A retrospective study published in 2023 found dexamethasone and methylprednisolone to be associated with an increased overall hyperglycemia relative to prednisolone or hydrocortisone, which could also apply to the glucose excursions found in our cohort, since mixed steroid type was used.[17] These findings indicate that decision and dosing of corticosteroid preparations may affect the extent of glycemic derailment, which might also explain the variation in postprandial and fasting glucose responses that we reported.

Besides quantitative data on glucose variability, our findings are also consistent with clinical findings on how challenging glycemic control can be in steroid therapy. A previous investigation conducted on patients with lung disease showed that it was hard to achieve good control of steroid-induced hyperglycemia, and most patients demonstrated long-term glucose level elevation, and only a part of them reached the target glycemic levels, which is similar to the high variability and frequent hyperglycemia observed in our population.[18]

Although the majority of research addresses diabetic patients, our subgroup results that the non-diabetic patients had significant post prandial hyperglycemia were consistent with the general literature that the steroid treatment has an impact on glucose metabolism in non-diabetic patients. There is considerable cohort evidence that corticosteroid therapy is highly associated with the risk of hyperglycemia among non-diabetic hospitalized patients, even though the likelihood of this tendency is lower when compared to diabetic patients.[15]

Certain studies have indicated subtle results on steroid effects. Indicatively, in a multicenter cross-sectional study, corticosteroid treatment did not have significant correlations with the lack of good glycemic regulation or acute hyperglycemic complications in some hospitalized diabetic patients, but such results might be mediated by variations in monitoring habits or the composition of patient populations.[19] This underscores the variability of clinical protocols and patient characteristics that may be used to modulate observed outcomes.

Our work contributes to the existing literature by measuring not only the existence of hyperglycemia but also the trend and the variability of glucose oscillations during the short-term corticosteroid exposure. Notably, our findings highlight the necessity of active glucose monitoring and personalized management plans in the context of corticosteroid use, which is also supported by the guideline reports that suggest

systematic glucose monitoring in steroid-receiving patients to reduce the risk of acute complications.[20]

Although our findings are consistent with the current evidence, we have limitations. The variability in the study designs, population, corticosteroid regimens, and glucose monitoring structures in the literature complicates the direct comparisons. Furthermore, although our findings indicate that there are distinct trends of steroid-induced glycemic change, the causal effects of certain steroid doses and metabolic variables need to be studied in randomized controlled conditions.

Conclusively, the findings of this study are in agreement with the vast body of evidence that corticosteroid medication has a highly significant effect on glucose metabolism in hospitalized patients, with high glycemia variability and hyperglycemia during short treatment periods. These trends reflect the trends of recent clinical and epidemiological studies and contribute to the use of alert glucose surveillance and active treatment measures in clinical practice.

The results of this research reveal the paramount significance of active glucose monitoring in hospitalized patients under corticosteroid treatment. Corticosteroids, even when used in the short term, may result in profound hyperglycemia and glycemic variability, especially in patients with underlying diabetes, though it may also happen in non-diabetic people. Post-prandial glucose spikes are another issue that should be observed by clinicians, and they were more severe than fasting hyperglycemia in our cohort. Standardized glucose surveillance measures, insulin or oral hypoglycemic modifications, and patient-specific counseling should be implemented to alleviate these issues of acute complications, minimize the risk of infection, enhance the healing process of a wound, and possibly decrease hospital stay. Besides, these results specify the importance of a special choice of corticosteroid type and dose, particularly in patients with a high risk of hyperglycemia, to find the balance between the therapeutic effects and the metabolic safety.

This research has a number of limitations. To begin with, it was conducted in one tertiary care hospital, which might restrict the applicability of the results in other healthcare facilities. Second, we only studied glucose fluctuations during the short-term period of steroid therapy, but not the metabolic effects in the long-term. Third, the research failed to consider other drugs and dietary differences that might affect the blood glucose, despite trying to standardize the monitoring processes. Also, the diabetic and non-diabetic subgroups were rather small in size, and the research was based on the point-of-care glucose measurements, which might cause slight fluctuations when compared to laboratory tests. Lastly, causality between corticosteroid therapy and glucose changes cannot be conclusively established since the study is observational in nature.

Conclusion

Acute corticosteroid treatment of hospitalized patients results in substantial rises of blood glucose levels and intense glycemic fluctuations, where post-prandial hyperglycemia is the most notable. The effect is more drastic in diabetic patients, although non-diabetic individuals are vulnerable as well, and universal vigilance is crucial. The results support the need to monitor glucose at the earliest times, provide personalized care, and select corticosteroids with caution to reduce the metabolic risks of the therapy. Clinicians will be able to improve patient outcomes, decrease complications, and provide better overall quality inpatient care by proactively managing steroid-related hyperglycemia.

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