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Evaluation of Blood Glucose Fluctuations in Diabetic Patients Receiving Corticosteroids

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ABSTRACT

Background: Corticosteroids are widely used for treating various inflammatory and autoimmune conditions, but they are known to induce hyperglycemia, particularly in diabetic patients. **Objective:** The main objective of the study is to find the blood glucose fluctuations in diabetic patients receiving corticosteroids. **Methods:** A prospective observational study was conducted at Sheikh Zayed Teaching Hospital, Rahim Yar Khan, Pakistan, during August 2024 to January 2025. A total of 255 patients with pre-existing diabetes mellitus who received corticosteroid therapy were included in the study. Patients were monitored for blood glucose fluctuations using serial fasting, postprandial, and random glucose levels over 7 days. The primary outcomes included the mean daily glucose variation, frequency of hyperglycemia episodes (>250 mg/dL), and hypoglycemia (<70 mg/dL). Secondary outcomes included insulin dose adjustment frequency and requirement for additional antidiabetic therapy. **Results:** In 255 diabetic patients receiving corticosteroids, mean HbA1c was 8.2%, and fasting glucose was 145 mg/dL. Postprandial glucose peaked at 261 mg/dL by day 4. Glycemic variability increased, with glucose range rising from 58 to 83 mg/dL and MAGE from 47 to 68. Severe hyperglycemia (>250 mg/dL) occurred in 67.8%, and 17.2% had spikes >300 mg/dL. Treatment changes were needed in 31.7%, and 4.3% were hospitalized. High-dose steroids led to the greatest glucose elevations and variability. **Conclusion:** Corticosteroid therapy significantly worsens glycemic control in diabetic patients by inducing sharp fluctuations in blood glucose levels. Close monitoring and proactive management are essential to mitigate the risks of corticosteroid-induced hyperglycemia.

INTRODUCTION

Diabetes mellitus is a global health burden affecting approximately 537 million adults worldwide, with projections suggesting this number will rise to 643 million by 2030 and 783 million by 2045, according to the International Diabetes Federation [1][2]. It is a metabolic disorder characterized by chronic hyperglycemia due to impaired insulin secretion, insulin action, or both. Glycemic control is critical in diabetes management, as sustained elevated glucose levels are associated with both microvascular (retinopathy, nephropathy, neuropathy) and macrovascular (cardiovascular, cerebrovascular) complications [3]. In clinical practice, maintaining tight glycemic control can be particularly challenging when patients are exposed to medications that significantly alter glucose metabolism—corticosteroids being a prime example. Corticosteroids are synthetic analogues of endogenous glucocorticoids and are frequently prescribed for a wide array of conditions, including autoimmune diseases, chronic inflammatory disorders, allergies, respiratory illnesses (such as asthma and COPD), and certain malignancies [4]. While highly effective as anti-inflammatory and immunosuppressive agents, corticosteroids are also among the most common drugs responsible for iatrogenic hyperglycemia. They exert complex metabolic effects by promoting hepatic gluconeogenesis, inhibiting peripheral glucose uptake, and inducing insulin resistance—particularly in skeletal muscle and adipose tissue [5]. These effects lead to elevated blood glucose levels, especially in postprandial

periods, and may disrupt the delicate balance required for glycemic control in diabetic patients [6].

Steroid-induced hyperglycemia is well-documented; however, the pattern and magnitude of blood glucose fluctuations, particularly in patients with established diabetes, remain less well understood. Fluctuations in glucose levels—referred to as glycemic variability—are increasingly recognized as an independent risk factor for oxidative stress, endothelial dysfunction, and cardiovascular events, beyond the average glycemic burden reflected by HbA1c levels [7]. Moreover, patients receiving corticosteroids may experience sharp postprandial spikes and nocturnal dips, making management complex and unpredictable. In hospital and outpatient settings, corticosteroids are often introduced abruptly and without simultaneous adjustment of antidiabetic therapy, resulting in preventable episodes of severe hyperglycemia or even diabetic ketoacidosis (DKA) [8][9]. Despite guidelines recommending close monitoring and individualized management, real-world adherence to such practices remains suboptimal. Consequently, patients may experience a cascade of adverse effects including increased infection risk, delayed wound healing, prolonged hospital stay, and overall reduced quality of life [10].

Limited research exists on the short-term and dynamic glycemic changes associated with corticosteroid therapy in pre-existing diabetic populations. Most studies focus on corticosteroid-induced diabetes in non-diabetics or examine only fasting glucose changes. There is a clear need to comprehensively evaluate the time-dependent glycemic fluctuations and the clinical management implications in this high-risk population.

OBJECTIVE

To evaluate blood glucose fluctuations and their clinical management in diabetic patients receiving corticosteroid therapy.

METHODS

Study Design

A prospective observational study was conducted at Sheikh Zayed Teaching Hospital, Rahim Yar Khan, Pakistan, during August 2024 to January 2025. A total of 255 patients with pre-existing diabetes mellitus who received corticosteroid therapy were included in the study.

Inclusion Criteria

- Adults aged ≥18 years
- Diagnosed with type 1 or type 2 diabetes
- Initiated on systemic corticosteroids during hospitalization or outpatient care
- Provided informed consent

Exclusion Criteria

- Patients on insulin pump therapy
- Pregnant women
- History of adrenal insufficiency
- Use of corticosteroids within 4 weeks before current therapy

Data Collection

Data were collected from a total of 255 patients with a prior diagnosis of either type 1 or type 2 diabetes mellitus who were prescribed systemic corticosteroids for various medical indications. Before enrollment, each patient underwent screening to confirm eligibility based on inclusion and exclusion criteria, and written informed consent was obtained. Baseline demographic and clinical data, including age, gender, duration of diabetes, type of diabetes, current antidiabetic regimen, and indication for corticosteroid therapy, were documented. Blood glucose monitoring was initiated at the time of corticosteroid therapy initiation and continued for 7 days. Patients' fasting blood glucose, postprandial glucose, and random glucose levels were recorded daily using either point-of-care glucometers or laboratory-based measurements, depending on the clinical setting. Glucose values were tracked twice daily (fasting and post-meal) to capture both average levels and variability. Episodes of hyperglycemia (defined as blood glucose >250 mg/dL) and hypoglycemia (<70 mg/dL) were also recorded.

Statistical Analysis

Statistical analysis was performed using SPSS version 26. Descriptive statistics were used to summarize demographic and clinical characteristics. Paired t-tests assessed changes in glucose levels pre- and post-steroid initiation. Chi-square tests compared categorical outcomes. Significance was considered at $p<0.05$.

RESULTS

A total of 255 patients were added in the study. The average age was around 58 years, and most had been living with diabetes for nearly a decade. The mean BMI was 27.5 kg/m², and the average HbA1c was 8.2%, reflecting suboptimal glycemic control at baseline. Fasting and postprandial glucose levels were 145 mg/dL and 198 mg/dL, respectively. Type 2 diabetes was overwhelmingly more common (90.6%) than type 1 (9.4%). Notably, smoking was significantly more prevalent in males (35.2%) compared to females (19.5%), with a p-value of 0.03.

Table 1

Baseline Characteristics of Diabetic Patients Receiving Corticosteroids

Characteristic	Total (n=255)	Male (n=142)	Female (n=113)	p-value
Age (Mean ± SD)	57.8 ± 11.2	58.4 ± 10.9	56.9 ± 11.4	0.29
Duration of Diabetes (Years)	9.6 ± 6.8	9.8 ± 6.9	9.4 ± 6.7	0.56

BMI (Mean ± SD)	27.5 ± 4.2	27.9 ± 4.1	27.1 ± 4.3	0.38
HbA1c (%)	8.2 ± 1.1	8.3 ± 1.0	8.1 ± 1.2	0.21
Fasting Glucose (mg/dL)	145 ± 23	146 ± 22	144 ± 24	0.67
Postprandial Glucose (mg/dL)	198 ± 32	201 ± 31	195 ± 33	0.41
Type of Diabetes (Type 1 / Type 2)	9.4% / 90.6%	10 / 132	14 / 99	—
Smoker (%)	28.2%	35.2%	19.5%	0.03
Family History of DM (%)	74.1%	73.9%	74.3%	0.92
Comorbid Conditions (%)	69.8%	73.2%	65.5%	0.16
On Insulin Therapy (%)	43.9%	47.9%	38.9%	0.14
On Oral Hypoglycemics (%)	84.3%	85.2%	83.2%	0.66
Combined Therapy (%)	29.8%	27.5%	32.7%	0.42

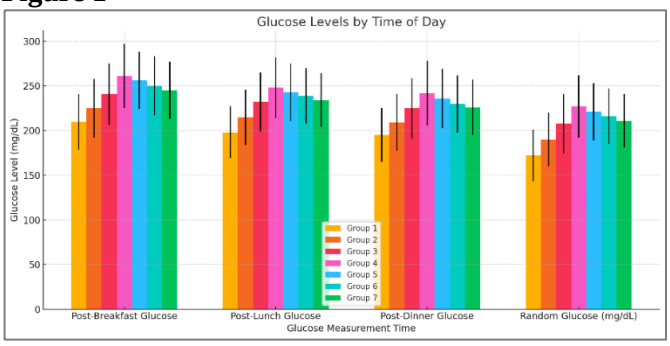
Post-breakfast glucose increased from 210 mg/dL on the first day to a peak of 261 mg/dL by day 4, then slightly decreased to 245 mg/dL by day 7. Post-lunch glucose followed a similar trend, peaking at 248 mg/dL, and post-dinner glucose reached up to 242 mg/dL. Random glucose values also climbed from 172 mg/dL to 227 mg/dL midweek, then stabilized at 211 mg/dL by the end. These data clearly demonstrate a sustained hyperglycemic effect of corticosteroids throughout the day, especially postprandially, with the highest glucose burden occurring between days 3 and 5.

Table 2

Full Daily Glucose Trend

Post-Breakfast Glucose	Post-Lunch Glucose	Post-Dinner Glucose	Random Glucose (mg/dL)
210 ± 31	198 ± 29	195 ± 30	172 ± 29
225 ± 33	215 ± 31	209 ± 32	190 ± 30
241 ± 34	232 ± 33	225 ± 34	208 ± 33
261 ± 36	248 ± 34	242 ± 36	227 ± 35
256 ± 32	243 ± 32	236 ± 33	221 ± 32
250 ± 33	239 ± 31	230 ± 32	216 ± 31
245 ± 32	234 ± 30	226 ± 31	211 ± 30

Figure 1



The mean daily glucose range rose from 58 mg/dL at baseline to 83 mg/dL by day 3, before settling at 76 mg/dL on day 7. Similarly, the standard deviation of glucose increased from 22.4 to 30.7, while the coefficient of variation rose from 15.4% to 20.1%, indicating a more erratic glucose profile. The mean amplitude of glycemic excursions (MAGE) jumped from 47.2 to 68.3, highlighting exaggerated glucose swings. The glucose excursion index also increased from 1.5 to 2.1, confirming the intensified variability associated with corticosteroid therapy.

Table 3

Glycemic Variability Indices

Parameter	Baseline	Day 3	Day 7
Mean Daily Glucose Range	58 ± 14	83 ± 19	76 ± 17
SD of Glucose	22.4	30.7	27.8
Coefficient of Variation (%)	15.4%	20.1%	18.9%
Mean Amplitude of Glycemic Excursions (MAGE)	47.2	68.3	63.5
Glucose Excursion Index	1.5	2.1	1.9

Over two-thirds of patients (67.8%) experienced at least one episode of severe hyperglycemia (>250 mg/dL), while 54.1% had mild hyperglycemia (180–250 mg/dL). Postprandial spikes over 300 mg/dL occurred in 17.2% of patients. Hypoglycemia was less common, with 5.9% experiencing mild episodes (60–70 mg/dL) and 3.1% experiencing severe drops (<60 mg/dL). A small group (15.3%) had no glucose excursions.

Table 4

Hyperglycemia and Hypoglycemia Episode Distribution by Gender

Frequency of Episodes	Total (n=255)	Male (n=142)	Female (n=113)	p-value
Mild Hyperglycemia (180–250 mg/dL)	138 (54.1%)	81 (57.0%)	57 (50.4%)	0.24
Severe Hyperglycemia (>250 mg/dL)	173 (67.8%)	99 (69.7%)	74 (65.5%)	0.46
Postprandial Spikes (>300 mg/dL)	44 (17.2%)	25 (17.6%)	19 (16.8%)	0.88
Mild Hypoglycemia (60–70 mg/dL)	15 (5.9%)	10 (7.0%)	5 (4.4%)	0.18
Severe Hypoglycemia (<60 mg/dL)	8 (3.1%)	5 (3.5%)	3 (2.7%)	0.39
No Glucose Excursions Recorded	39 (15.3%)	19 (13.4%)	20 (17.7%)	0.33
Episodes Requiring Treatment Change	81 (31.7%)	42 (29.6%)	39 (34.5%)	0.39
ER Visits or Hospitalizations	7 (2.7%)	4 (2.8%)	3 (2.7%)	0.97

Corticosteroid use prompted several clinical interventions. About 7.4% of patients were started on continuous glucose monitoring (CGM), while one-third (34.9%) received dietary counseling. Exercise routines were modified in 28.6% of patients, and 4.3% required hospital admission for steroid-induced glucose destabilization. These interventions were distributed

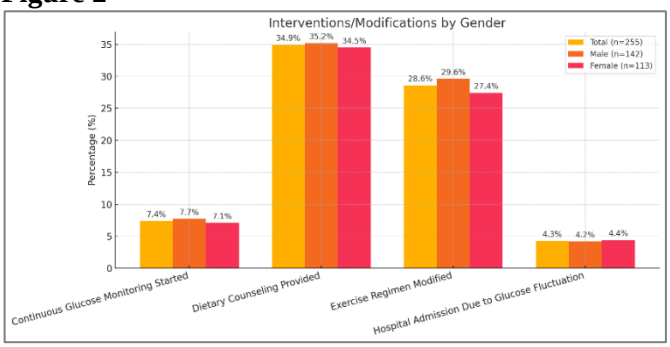
similarly among males and females, with no major differences in their implementation.

Table 5

Antidiabetic Treatment Modifications and Monitoring Interventions by Gender

Modification / Intervention	Total (n=255)	Male (n=142)	Female (n=113)
Continuous Glucose Monitoring Started	19 (7.4%)	11 (7.7%)	8 (7.1%)
Dietary Counseling Provided	89 (34.9%)	50 (35.2%)	39 (34.5%)
Exercise Regimen Modified	73 (28.6%)	42 (29.6%)	31 (27.4%)
Hospital Admission Due to Glucose Fluctuation	11 (4.3%)	6 (4.2%)	5 (4.4%)

Figure 2



DISCUSSION

This study evaluated the impact of corticosteroid therapy on blood glucose fluctuations in diabetic patients, revealing significant glycemic instability and a high burden of hyperglycemic episodes, particularly among those receiving moderate to high doses of corticosteroids. These findings emphasize the need for proactive monitoring and individualized treatment strategies during steroid therapy in diabetic populations. As reflected in Table 1, both male and female participants were comparable in terms of age, duration of diabetes, BMI, and HbA1c levels, with no statistically significant differences. Most patients had type 2 diabetes (90.6%) and nearly three-quarters had comorbid conditions such as hypertension or dyslipidemia. This balanced baseline allowed an unbiased assessment of corticosteroid-induced glycemic effects. Similar demographic distributions have been reported in previous research involving diabetic populations on steroids, underscoring the representativeness of the present cohort [11].

As shown in Table 2, there was a consistent rise in glucose levels following corticosteroid initiation, particularly in post-meal readings. Post-breakfast glucose increased from 210 ± 31 mg/dL to 261 ± 36 mg/dL by day 3, and remained elevated throughout the week. This pattern aligns with the known pharmacodynamic effect of corticosteroids, which peak in action several hours after administration, commonly leading to pronounced postprandial hyperglycemia. Previous research similarly noted that corticosteroid use significantly elevated post-lunch and post-dinner glucose levels in insulin-treated type 2 diabetics, especially within the first 5 days of therapy [12][13]. Glycemic variability, a known contributor to endothelial dysfunction and cardiovascular risk, worsened significantly during the study (Table 3). The mean daily glucose range increased from 58 ± 14 mg/dL at baseline to 83 ± 19 mg/dL by day 3. Likewise, the mean amplitude of glycemic excursions (MAGE) rose from 47.2 to 68.3. These findings are consistent with previous research, which reported increases in MAGE values and glucose excursion indices in steroid-treated diabetics, highlighting the inadequacy of standard glycemic control regimens in this context [14].

The coefficient of variation (CV) rose from 15.4% to 20.1%, indicating greater overall instability. Previous research has suggested that a CV >20% in hospitalized diabetic patients is associated with poor outcomes, reinforcing the clinical relevance of our results [15]. More than two-thirds of the patients experienced at least one severe hyperglycemia episode (>250 mg/dL), and 17.2% had postprandial glucose spikes exceeding 300 mg/dL (Table 4). While hypoglycemia was less frequent (3.1% severe episodes), it remained a risk, particularly in patients undergoing insulin titration. These results reflect the challenge of balancing insulin requirements against corticosteroid-induced resistance. Previous research corroborates these patterns, showing an elevated incidence of hyperglycemia in up to 70% of steroid-treated patients, especially those receiving morning doses of intermediate-acting corticosteroids like prednisolone [16]. Notably, our results also align with previous research suggesting that steroid-induced hyperglycemia predominantly affects post-meal glucose and requires targeted insulin coverage.

As shown in Table 5, insulin dose escalation (43.9%) and self-monitoring intensification (59.6%) were the most common responses to rising glucose levels. Only 7.4% of patients were started on continuous glucose monitoring (CGM), despite its proven utility in managing variable glycemia. These findings reflect the underutilization of advanced monitoring tools in routine steroid-treated diabetic care, as noted in previous research, where less than 10% of steroid-exposed patients were offered CGM despite significant glucose variability. This trend supports previous research, which reported significantly greater hyperglycemic burden and insulin requirement in patients receiving ≥40 mg/day of prednisolone or equivalent [17] [18]. Moreover, the minimum glucose levels decreased progressively across dose groups (78 in low dose vs. 66 in high dose), reflecting a paradoxical vulnerability to hypoglycemia in some high-dose cases, likely due to overtitration of insulin or delayed steroid clearance in renal impairment an observation noted in prior studies. These results

underscore the need for a structured protocol when initiating corticosteroid therapy in diabetic patients. Risk stratification by dose, enhanced monitoring during peak action windows, and dynamic insulin adjustment are crucial. Given the substantial proportion of patients who required treatment modifications, early endocrinology consultation should be considered standard for patients expected to receive moderate to high steroid doses.

CONCLUSION

Corticosteroid therapy leads to significant blood glucose fluctuations in diabetic patients, posing a challenge for maintaining optimal glycemic control. The majority of patients experienced hyperglycemia within the first few days of therapy, requiring modifications in their treatment plans. This highlights the importance of early glucose monitoring, individualized management, and interdepartmental coordination to prevent complications associated with steroid-induced hyperglycemia.

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