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Original Research Article

Antenatal Betamethasone and Maternal Dysglycemia: A Hidden Metabolic Concern in Preterm Pregnancy

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Abstract: **Background:** Antenatal administration betamethasone (BM) during pregnancy lowers the frequency and intensity of neonatal respiratory distress syndrome. Pregnancyrelated placental insulin resistance and its diabetogenic potential cause a brief rise in blood glucose levels. The effects of steroids on glucose homeostasis in pregnant women without diabetes are poorly understood. **Objective:** To characterize the glycaemic response to antenatal betamethasone therapy in normoglycaemic pregnant women at risk of preterm birth and to assess the magnitude of steroid induced maternal dysglycaemia. **Patients and Methods:** This was cross sectional study and conducted in Gynaecology department, JPMC Karachi from July 2025 to March 2026. Total 214 nondiabetic pregnant women, whose deliveries were planned before 34+6 weeks of gestation and had received either a single course or two doses of betamethasone according to the clinical indication. Maternal glycaemic status was carefully observed throughout the study period using Bedside Diagnostic Testing and monitoring, with approximately 3–4 readings recorded daily for each participant. To minimize dietary variations that could influence glucose levels, all enrolled women were provided with a standardized balanced diet during the monitoring period. **Results:** A significant increase in blood glucose levels was observed following betamethasone administration. After the first dose, mean fasting glucose levels were 98.71±7.38 mg/ dl (morning) and 96.27±6.24 mg/dl (night), while mean random glucose levels were 145.28±9.27 mg/dl (morning) and 139.98±8.40 mg/dl (night) ($p<0.001$). Following the second dose, glucose levels increased further, with mean fasting glucose levels of 101.26±9.77 mg/dl (morning) and 99.78±5.59 mg/dl (night), and mean random glucose levels of 165.01±10.74 mg/dl (morning) and 157.29±7.82 mg/dl (night) ($p<0.001$), indicating a significant glycaemic response to betamethasone treatment. However, no significant association was identified between glycaemic fluctuations and maternal characteristics such as age, parity, gestational age, or other studied variables. **Conclusion:** This study emphasises the importance of careful and individualized glucose monitoring in pregnant women receiving betamethasone therapy. Since corticosteroid administration can significantly influence maternal blood sugar levels, timely identification and management of hyperglycaemia are essential to reduce potential maternal and fetal complications.

Keywords: Betamethasone, Neonatal Respiratory Distress Syndrome, Pre & Postprandial Glucose.

INTRODUCTION

Globally, approximately 9.9% of live births occur preterm, corresponding to an estimated 13.4 million preterm births annually. Southern Asia bears a disproportionate burden, accounting for nearly two thirds of global preterm births, while Pakistan has one of the highest preterm birth rates in the region, estimated at 14.3%, making prematurity a leading cause of neonatal morbidity and mortality [1].

Complications of prematurity are the leading cause of death among children under five years of age and are associated with substantial neonatal morbidity, including respiratory distress syndrome (RDS), intraventricular hemorrhage (IVH), necrotizing enterocolitis (NEC), sepsis, broncho-pulmonary dysplasia, and long term neurodevelopmental impairment. The burden of preterm birth is particularly high in South Asia and sub Saharan Africa, accounting for more than 65% of all preterm births globally [12]. Current evidence demonstrates that ACS administration significantly reduces neonatal mortality, respiratory distress syndrome, intraventricular hemorrhage, and severe neonatal morbidity among preterm infants. Consequently, international guidelines recommend a single course of antenatal corticosteroids for women at risk of delivery before 34 weeks gestation, with selected use extending into the late preterm period [3]. Prenatal corticosteroid was first used in 1972 by Liggins and Howie to treat preterm delivery. They showed that it could lower the risk of neonatal RDS from 25.8% to 9.0% and the neonatal mortality rate from 15.0% to 3.2% [3-4].

The World Health Organization (WHO) recommends a single course of antenatal corticosteroids for pregnant women at high risk of preterm delivery within 7 days between 24+0 and 34+0 weeks of gestation, as this intervention significantly improves neonatal outcomes associated with prematurity [2-5]. Two intramuscular doses of 12 mg betamethasone spaced 24 hours apart or four intramuscular doses of 6 mg dexamethasone spaced 12 hours apart are part of the standard regimen. Prenatal corticosteroids are recommended by the Royal College of Obstetricians and Gynaecologists (RCOG) for women who are at risk of preterm birth and are between 24+0 and 34+6 weeks of gestation [5]. Nevertheless, corticosteroid treatment has certain negative effects on mothers, such as decreased glucose tolerance and adrenal suppression [4]. Pregnant women experience a brief rise in blood glucose levels due to the well known diabetogenic potential of betamethasone and placental insulin resistance [6]. There aren't many studies in the literature on how betamethasone affects pregnant women's glucose homeostasis. Small sample sizes may prevent them from drawing conclusions that are statistically sound [6-7].

Hyperglycaemia may occur in cases of gestational diabetes or diabetes mellitus with controlled blood glucose levels, requiring the start of temporary insulin therapy. In the event of a preterm birth, maternal hyperglycaemia may result in severe hypoglycaemia in the newborn [8-9]. Little research has been done on the timing and severity of glucose increases, and little is known about how steroids affect glucose homeostasis in pregnant women who do not have diabetes. The current study was conducted to shed light on the negative effects of betamethasone while taking the aforementioned factors into consideration [10]. This study evaluates how betamethasone given during preterm gestation affects the pre-meal and postprandial blood glucose levels of pregnant women with normoglycemia.

METHODS

This cross sectional study was carried out over a period from July 2025 to March 2026 in the Department of Gynaecology at Jinnah Postgraduate Medical Centre. A total of 214 pregnant women aged between 18 and 40 years with singleton pregnancies ranging from 24+0 to 33+6 weeks of gestation were enrolled in the study. These women were admitted for obstetric indications requiring planned termination or anticipated preterm delivery. Depending upon the clinical judgment of the attending obstetrician, participants received either a single course or two doses of intramuscular betamethasone for fetal lung maturation.

Only women with previously confirmed normal glucose tolerance were included. Normal glycaemic status was established through 75 gms oral glucose tolerance test (OGTT) performed between 24 and 28 weeks of gestation. Pregnant women diagnosed with overt diabetes mellitus or gestational diabetes mellitus were excluded from the study. Additional exclusion criteria included multiple pregnancies, prior or ongoing corticosteroid therapy, associated maternal medical disorders such as systemic infections or renal disease, administration of intravenous dextrosecontaining fluids, prior betamethasone exposure outside the study hospital, active labor, and refusal to provide informed consent. Baseline maternal characteristics including age, body mass index (BMI), blood pressure, and OGTT findings were documented at enrolment. The study focused on evaluating glycaemic fluctuations among hospitalized women with high risk pregnancies, including conditions such as pre-labor rupture of membranes, oligohydramnios, preeclampsia, and antepartum haemorrhage. Ethical approval was obtained from the Institutional Ethics Committee, and written informed consent was secured from all participants prior to inclusion in the study.

The standard corticosteroid regimen consisted of two intramuscular doses of betamethasone 12 mg administered 24 hours apart. Maternal blood glucose levels were serially assessed using point of care capillary glucose measurements obtained through finger prick testing with a calibrated glucometer with approximately five to six readings recorded daily. Hyperglycaemia was defined as fasting or pre-meal blood glucose levels >90 mg/dl and postprandial blood glucose levels measured one hour after meals >140 mg/dl. Blood glucose measurements were recorded at predetermined morning and nighttime intervals, and any elevated reading was considered

indicative of maternal hyperglycaemia. To minimize dietary variability, all participants received a standardized balanced diet throughout hospitalization.

The collected data were analysed using Microsoft Excel and presented as frequencies, percentages, and mean ± SD. Statistical comparison of glycaemic values before and after corticosteroid administration was performed using the paired ttest, with statistical significance assessed accordingly.

RESULTS

Current study had total 214 patients, out of which this study population 93 were delivered per vagina and rest of the other 121 were by c-section (Figure 01). Those patients who received 2 doses of betamethasone were 163 (76.16%), while 51 (23.8%) were received only single dose because they gave birth before the 2nd dose (Figure 02). In the majority of cases, there were significant changes, amongst all, 176 patients (83.4%) have shown escalation pattern of hyperglycaemia during fasting and random levels i.e. 98.34±8.26 mg/dl and 151.78±8.92 mg/dl respectively and remaining 35 individuals (16.5%) had without any modification in glycaemic index (Table 02).

After the 1st dose of betamethasone fasting glucose levels were 96.27±6.24 mg/dl at night while in morning it was much higher i.e. 98.71±7.38 mg/dl ($p < 0.001$) which were significant statistically. Meanwhile, 145.28± 9.27 mg/dl was the random glucose levels in morning, and at night it was 139.98±8.4 mg/dl ($p < 0.001$) and considered significant statistically (Table 02). There was increasing trend in hyperglycaemia after receiving 1st dose which was statistically significant. After following the 1st dose, there escalating pattern in glucose levels which was significant statistically. Additionally, 132 patients were received 2 doses of betamethasone had shown hyperglycaemia and the results were significant statistically. After receiving single 1st dose mean fasting and random glucose levels were 97.22±6.73 mg/dl and 147.92±8.12 mg/dl respectively. Subsequent to 2nd dose mean fasting and random glucose levels were 99.38±9.44 mg/dl ($p <0.03$) and 151.09±10.16 mg/dl (Table 03) ($p <0.001$) respectively which were significant statistically. An incremental rise in blood glucose levels was noted after the 2nd dose of betamethasone relative to the 1st, supporting a possible additive glycaemic effect (Table 04). These findings suggest a dose dependent relationship between betamethasone exposure and maternal hyperglycaemia.

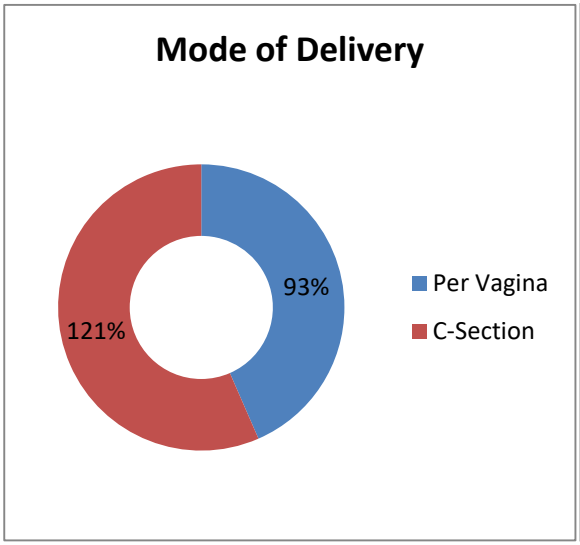

Figure 01: Mode of Delivery

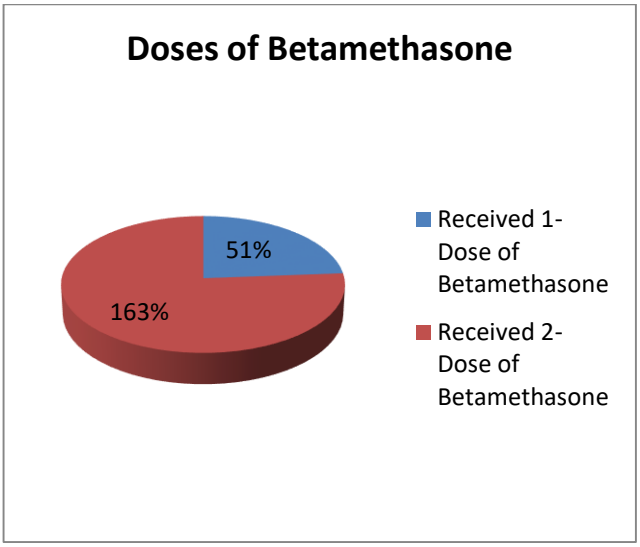

Figure 02: Percentage of patients received doses of Betamethasone

Table 1: Protocol for Blood Glucose Monitoring During Betamethasone Therapy: Day 1 And 2

Days	Monitoring Time Point	Procedure
Day 1	Predose (09:00 AM)	Baseline blood glucose assessment
	09:00 AM	Administration of first dose of betamethasone
	1 hour postdose (10:00 AM)	Fasting blood glucose measurement
	2 hours postdose (11:00 AM)	Random blood glucose measurement
	10:00 PM	Blood glucose monitoring
	11:00 PM	Blood glucose monitoring
Day 2	Predose (08:00 AM)	Blood glucose assessment before second dose
	09:00 AM	Administration of second dose of betamethasone

	1 hour postdose (10:00 AM)	Fasting blood glucose measurement
	2 hours postdose (11:00 AM)	Random blood glucose measurement
	10:00 PM	Blood glucose monitoring
	11:00 PM	Blood glucose monitoring

Table 2: Comparison of Fasting & Random Blood Glucose Levels before & After the 1st Dose of Betamethasone

	Fasting Glucose Levels		Random Glucose Levels		p-value
	Morning	Night	Morning	Night	
1st Dose	98.71±7.38	96.27±6.24	145.28±9.27	139.98±8.40	0.00*
*paired t-test; p value is significant 0.05					

Table 3: Comparison of Fasting & Random Blood Glucose Levels before & After the 2nd Dose of Betamethasone

	Fasting Glucose Levels		Random Glucose Levels		p-value
	Morning	Night	Morning	Night	
2nd Dose	101.26±9.77	99.78±5.59	165.01±10.74	157.29±7.82	0.00*
*paired t-test; p value is significant 0.05					

Table 4: Association of Glucose level after 1st and 2nd Dose of Betamethasone

	Fasting Glucose Reading	Random Glucose Reading	p-value
1st Dose	97.22±6.73	147.92±8.12	0.00*
2nd Dose	99.38±9.44	151.09±10.16	0.00*
*unpaired t-test; p value is significant 0.05			

DISCUSSION

The present study demonstrated a significant increase in both fasting and random blood glucose levels following antenatal betamethasone administration, with a more pronounced glycaemic response observed after the 2nd dose. These findings suggest that betamethasone induces transient maternal dysglycaemia even in women without previously diagnosed diabetes, highlighting the metabolic impact of antenatal corticosteroid therapy. As anticipated, alterations in the glycaemic profile were noted after betamethasone treatment in the majority of pregnant women (77.14%) exhibiting hyperglycaemia, which was similar to studies by Shaikh et al. [11] and Karim et al. [13]. The number of betamethasone doses was linked to the glycemia, but not other factors like age, BMI, parity, gestational age, and preterm labor causes, among others[14].

Our findings are consistent with those reported by Wali et al.,[9] Fatima et al., [10] and Hiersch et al., [14] who observed significant elevations in maternal glucose levels following betamethasone administration and emphasized the need for close glucose surveillance during the first 48–72 hours after treatment. Similarly, Jones et al., [15] reported substantial glucose excursions after antenatal betamethasone, particularly among women with impaired glucose tolerance, supporting the observation that corticosteroid exposure can markedly disrupt maternal glycaemic control.

The higher glucose values observed after the second dose in our study are in agreement with the concept of a cumulative corticosteroid effect. Rizzo et al., [16] demonstrated alterations in materno placenta fetal glucose homeostasis following antenatal betamethasone exposure, suggesting that repeated steroid administration may further amplify metabolic disturbances. Likewise, Jones et al. reported that antenatal betamethasone induced hyperglycaemia often necessitated intensified glucose monitoring and insulin adjustment, particularly after sequential doses. Our results also parallel those of McDonnell el al., [17], Khan et al., [18] and Dow et al., [19] who identified significant changes in maternal glycaemic profiles following antenatal corticosteroid administration. Although the magnitude of glucose elevation varied across studies due to differences in study populations, diabetic status, corticosteroid regimens, and monitoring protocols [20], the overall trend consistently indicates a transient but clinically relevant increase in maternal blood glucose levels after steroid exposure.
The pathophysiological basis of these findings may be attributed to the well established diabetogenic effects of glucocorticoids, which promote hepatic gluconeogenesis, increase peripheral insulin resistance, and impair glucose utilization. During pregnancy, when physiological insulin resistance is already enhanced by placental hormones, the additional metabolic burden imposed by betamethasone may further compromise glycaemic regulation.

Taken together, the findings of the present study reinforce growing evidence that antenatal betamethasone, while indispensable for fetal lung

maturation, is associated with significant short term maternal dysglycaemia [21]. Therefore, routine glucose monitoring should be considered following corticosteroid administration to facilitate early identification and management of steroid induced hyperglycaemia and to optimize maternal and fetal outcomes.

Our findings support existing evidence that antenatal betamethasone may induce transient maternal dysglycaemia, emphasizing the need for routine glycaemic surveillance in high risk pregnancies. Further well designed multicenter studies are needed to confirm these observations and optimize monitoring strategies.

CONCLUSION

The present study demonstrated that antenatal betamethasone administration in normoglycemic pregnant women during preterm gestation is associated with significant transient elevations in maternal blood glucose levels. A considerable proportion of participants developed increased pre-prandial and postprandial glycaemic values following corticosteroid therapy, with the degree of hyperglycaemia showing a direct relationship with the number of betamethasone doses administered. However, maternal demographic and obstetric variables such as age, parity, and gestational age were not significantly associated with glycaemic fluctuations. These findings highlight the diabetogenic potential of betamethasone even in women without pre-existing diabetes and underscore the necessity for vigilant glucose surveillance during corticosteroid therapy in pregnancy. Early detection and prompt management of steroid induced hyperglycaemia may help minimize adverse maternal and fetal outcomes while preserving the established neonatal benefits of antenatal corticosteroid administration.

Recommendations

Current study recommends that routine maternal glucose monitoring should be considered for all pregnant women receiving antenatal betamethasone, regardless of pre-existing diabetic status. Close glycaemic surveillance, including bedside point of care glucose testing, is particularly warranted during the first 48–72 hours following corticosteroid administration, when transient hyperglycaemia is most likely to occur. Women receiving repeated or multiple doses of betamethasone may require intensified monitoring due to the potential dose dependent increase in blood glucose levels. Furthermore, large scale multicenter prospective studies are needed to better define the duration, severity, and maternal fetal implications of betamethasone induced dysglycaemia, while future research should explore the comparative glycaemic effects of different corticosteroid regimens and evaluate effective preventive and therapeutic strategies.

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