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Neonatal hypoglycaemia in early preterm infants exposed to antenatal steroid therapy in pregnant mothers with diabetes: a retrospective cohort study

Sarah Al-Nafisee ,[1] Russell Miller,[1] Eve Overton,[1] Qi Yan,[2] Cynthia Gyamfi-Bannerman[3]

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For numbered affiliations see end of article.

Correspondence to
Dr Sarah Al-Nafisee;
sa3797@caa.columbia.edu

ABSTRACT

Objective To compare the incidence of neonatal hypoglycaemia (NH) in early preterm infants born to mothers with diabetes who were exposed to antenatal corticosteroids (ACS) and those born to mothers without diabetes who were also exposed to ACS.

Study Methods A retrospective cohort study of pregnant mothers who delivered between 28 0/7 and 33 6/7 weeks of gestation who had received ACS. All deliveries were at Columbia University Medical Centre, a tertiary care centre in the city of New York. ACS doses and timing of administration prior to delivery were compared between primary exposure groups, mothers diagnosed with diabetes versus mothers without diabetes. The primary outcome was NH (<40 mg/dL) within the first 48 hours of life. Associations were tested using χ^2 test and logistic regression.

Results Of 212 eligible patients, neonates of mothers with diabetes demonstrated a higher rate of NH (39.4% vs 19.6%, p=0.01). Other risk factors of NH included maternal body mass index and maternal age at delivery. Steroid dose and timing of administration were not determinant factors in developing NH.

Conclusions Early preterm neonates of mothers with diabetes who received ACS have a higher risk of NH. However, due to the lack of a control group of unexposed infants, the direct impact of ACS on NH cannot be conclusively determined.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ While the majority of current studies indicate an increased risk of neonatal hypoglycaemia (NH) following antenatal corticosteroid (ACS) administration in the late preterm period, studies on early preterm infants exposed to ACS have yielded inconsistent results. Some report an increased risk, while others show no significant difference in neonatal outcomes.

WHAT THIS STUDY ADDS

⇒ Our study focused on pregnancies complicated by diabetes, all of which received ACS treatment, and found a higher incidence of NH in early preterm infants born to mothers with diabetes compared with those born to mothers without diabetes.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Due to the lack of a control group of unexposed infants, we are unable to make definitive conclusions regarding the specific role of ACS in NH. Future research, particularly with appropriate control groups and prospective designs, is needed to better assess the relationship between maternal glycaemic control, ACS and NH outcomes in early preterm infants.

INTRODUCTION

Antenatal corticosteroid (ACS) administration is a well-established means of improving fetal lung maturity and decreasing neonatal morbidity in preterm neonates.[1] The American College of Obstetricians and Gynecologists recommends ACS administration between 24 0/7 and 33 6/7 weeks of gestation in cases at risk of preterm delivery within 7 days.[2] Diabetes in pregnancy has been described as a risk factor for preterm delivery,[3] and the International Federation of Gynecology and Obstetrics has recommended ACS therapy in mothers with pregestational and gestational diabetes who are at risk of a preterm delivery. This is in addition to increasing insulin therapy and continuing close monitoring during pregnancy.[4] It is, however, important to note that the use of ACS therapy has not been recommended in late preterm pregnancies complicated with pregestational diabetes due to the increased risk of worsening neonatal hypoglycaemia (NH).[5]

Some studies that evaluated neonates exposed to ACS therapy have shown that the risk of hypoglycaemia is increased with ACS administration, but only in the late preterm

period (≥34 weeks of gestation).[6] Some of these studies have also included pregnancies complicated by diabetes.[7] However, the data appear to be inconsistent when evaluating ACS therapy in early preterm deliveries (<34 weeks of gestation), some of which have not included pregnant mothers with diabetes[8] while others have.[9]

Our objective is to evaluate the incidence of hypoglycaemia in neonates exposed to ACS therapy in the early preterm period (<34 weeks of gestation) in pregnancies complicated with diabetes. We will also be discussing the dose of ACS administered and the timing of administration prior to delivery.

MATERIALS AND METHODS

Our study is a retrospective cohort study that included patients with non-anomalous singleton gestations who received ACS for threatened preterm delivery. We included eligible pregnancies with preterm deliveries between 28+0 and 33+6 weeks of gestation, who delivered between January 2018 and September 2019, all of which resulted in a live birth. We excluded multifetal pregnancies, patients who did not receive ACS therapy, deliveries (<28 weeks and ≥34 weeks), fetal genetic and structural abnormalities and stillbirths.

Subjects satisfying all inclusion criteria were each categorised into one of two exposure groups based on maternal diabetes mellitus status. For the purposes of this study, women with either pregestational diabetes or gestational diabetes (A1 Gestational Diabetes Mellitus (diet-controlled) and A2 Gestational Diabetes Mellitus (medication-controlled)) were defined as 'diagnosed with diabetes'. The comparison group was composed of women at risk for preterm delivery without gestational or pregestational diabetes. All eligible participants had received ACS for threatened preterm delivery. Data were collected for eligible patients who delivered between January 2018 and September 2019, and all deliveries took place at Columbia University Medical Center, a tertiary care centre in the city of New York.

We compared baseline demographics between both exposure groups and compared the proportion of neonates diagnosed with hypoglycaemia between groups. The primary outcome was NH defined as a heel stick glucose level measurement (<40 mg/dL) within the first 48 hours of life. We also evaluated the number of betamethasone courses each patient received (full course (12 mg × 2 doses 24 hours apart) or partial course (12 mg × one dose) in addition to any rescue courses that were administered during the admission period. The timing of steroid administration was evaluated and was defined as the number of days from the time of ACS administration to the time of delivery. Categorical variables were analysed using χ^2 or Fisher's exact test, and continuous variables were assessed using either the t-test or Wilcoxon rank sum test. All hypothesis tests were 2-sided, and significance was set at a p-value <0.05. The association of NH with a maternal diagnosis of diabetes was tested by a χ^2 test and a further logistic regression with adjustment for maternal race, maternal ethnicity, maternal body mass index (BMI) at delivery, steroid dose, additional rescue dose administration, neonatal gestational age at delivery, birth weight and timing of ACS administration prior to delivery that may have affected our outcome.

All study procedures and protocols were approved by the institutional review boards (IRBs) at our tertiary care centre (IRB number: IRB-AAAS7551) on 3 June 2020. Data collection was confidential, with access only available to IRB-approved research personnel. Patients or the public were not involved in the design, conduct, reporting or dissemination plans of our research. Informed consent was waived for this retrospective study, as approved by the IRB. The study involved the use of deidentified, coded medical data, and no direct contact was made with the patients. The study met all criteria for a waiver of consent under 45 CFR 46.116, including minimal risk to participants, no adverse effect on their rights and welfare, and the inability to practicably conduct the study without the waiver. Patient confidentiality was ensured by securely storing data and removing identifiers after data collection.

RESULTS

During our study period, a total of 642 patient charts were reviewed for threatened preterm delivery. Of those patients, n=430 patients were excluded in the following sequential order: multiple gestation pregnancy n=152, fetal genetic or structural anomaly n=52, stillborn n=18, gestational age (<28 weeks or ≥34 weeks) n=162, out of state third trimester termination n=1, neonatal demise (<24 hours of life) n=1, did not receive ACS (medication order was placed but not administered) n=9, no data/unavailable record n=7, duplicate chart: n=28.

A total of 212 participants met the study inclusion criteria (not diagnosed with diabetes n=179 (84.4%) and diagnosed with diabetes n=33 (15.6%)). Baseline characteristics and demographics were compared between both the exposure groups. Results were adjusted in our regression model for maternal age, ethnicity, race, maternal BMI at delivery, steroid dose, rescue dose administration, timing of ACS administration prior to delivery and neonatal gestational age at delivery. Concerns arose regarding multicollinearity in our regression model when looking at two independent variables, birth weight and gestational age at delivery, and we assessed for any correlation between these two variables. The correlation coefficient was 0.67, which we considered significant, and we only included one of these variables in our model. Our output in table 1 included our regression model with only one of these two variables (gestational age at delivery), and a second model, which only included the birth weight variable, demonstrated a similar output (please refer to the online supplemental Material Section). Our multiregression model indicated that maternal diabetes, maternal BMI and maternal age at delivery were all significant variables that contributed to NH (table 1). Additional

Table 1 Multivariable analysis for the association between the risk of neonatal hypoglycaemia and maternal diabetes in pregnancy (the model was adjusted for maternal age, race, maternal ethnicity, maternal BMI at delivery, steroid dose, rescue dose, gestational age and timing of ACS (excluding birth weight))

Variable	OR	Lower 95% CI	Upper 95% CI	P-value
Maternal age	1.08	1.004	1.17	0.04
Race				
Black/African American	2.47	0.99	6.33	0.05
Asian	0.89	0.11	5.15	0.91
Ethnicity Hispanic/Latino	1.33	0.53	3.38	0.54
Maternal BMI at delivery	0.92	0.85	0.99	0.03
Mother with diabetes	3.41	1.16	10.31	0.03
Dosing steroids				
Partial	0.31	0.06	1.40	0.13
Rescue dose	0.50	0.17	1.33	0.17
Gestational age at delivery	0.98	0.95	1.02	0.39
Timing ACS				
1–7 days	1.46	0.45	5.20	0.53
8–14 days	1.03	0.19	5.17	0.97
>14 days	0.15	0.001	1.80	0.15

ACS, antenatal corticosteroid; BMI, body mass index.

information on neonatal gender, birth weight, diagnosis of respiratory distress syndrome and intubation data was also included in our analysis and did not demonstrate any significant difference between both groups, as detailed in (table 2). Maternal demographics and outcomes were also described in (table 3) and demonstrated no significance in the type of labour or delivery.

Neonates born to mothers diagnosed with diabetes had a higher rate of hypoglycaemia in the first 48-hours of life compared with neonates of mothers without a diagnosis of diabetes (39.4% vs 19.6%, p=0.01). This significant association between maternal diabetes and NH persisted on multivariable logistic regression (OR 3.41 (95% CI 1.16 to 10.31), p=0.03). Although neonates of mothers diagnosed with diabetes had a higher risk of developing hypoglycaemia, the median glucose level of the hypoglycaemic neonates in the first 48 hours of life was not significantly different when measured between the two groups: neonates born to mothers diagnosed with diabetes and neonates born to mothers without diabetes (median (IQR): 34 (7) mg/dL vs 35 (11) mg/dL, p=0.69). Unlike adult hypoglycaemia, NH does not have specific categories of severity (ie, mild, moderate, severe). For this reason, we decided to divide our NH values into three categories: (<28 mg/dL, 28–34 mg/dL and 35–39 mg/dL). Neonates of both groups had a higher number of cases in the lowest hypoglycaemic category (<28 mg/dL), with similar distributions across both groups (neonates born to mothers diagnosed with diabetes vs neonates born to mothers without diabetes).

When evaluating the number of steroid doses administered (full course vs partial course vs full course+rescue dose), we found that there was no significant difference. The latency of ACS administration (administration <24 hours, 1–7 days, 8–14 days, >14 days) also did not affect the development of NH in our study participants.

DISCUSSION

Early preterm neonates who received ACS therapy have a higher risk of developing hypoglycaemia in the postpartum period when born to mothers diagnosed with diabetes.

One of the common side effects of steroids in pregnancy is the tendency to cause maternal hyperglycaemia due to an increase in gluconeogenesis and an increase in insulin resistance. ACS alters pancreatic beta-cells (the insulin-producing cells) in the mother, thus reducing insulin synthesis and has also been suggested to cause beta cell apoptosis. ACS treatment also decreases the glucose uptake peripherally in the muscles and adipose

Table 2 Neonatal outcomes of exposure groups

	Mothers with diabetes (n=33)	Mothers without diabetes (n=179)	P-value (mothers with vs without diabetes)
Neonatal gender n (%)			
Male Female	16 (48.5) 17 (51.5)	102 (57.0) 77 (43.0)	0.37
Gestational age at birth, days, median (IQR)	225 (20)	224 (19.5)	0.65
Neonatal birth weight, grams, median (IQR)	1795 (910)	1635 (707.5)	0.38
Neonatal hypoglycaemia n (%)	13 (39.4)	35 (19.6)	0.01
Diagnosis of RDS at birth n (%)	26 (78.8)	161 (89.9)	0.08
CPAP administration at birth n (%)	25 (75.8)	158 (88.3)	0.09
Intubation at birth n (%)	2 (6.1)	3 (1.7)	0.17

CPAP, continuous positive airway pressure therapy; RDS, respiratory distress syndrome.

Table 3 Maternal demographics and outcomes of exposure groups

	Mothers with diabetes (n=33)	Mothers without diabetes (n=179)	P-value (mothers with vs without diabetes)
Maternal race n (%)			
Black/African American	7 (21.2)	42 (23.5)	0.7182
White/Caucasian	17 (51.5)	78 (43.6)	
Asian	2 (6.1)	6 (3.4)	
Native Hawaiian/Other Pacific Islander	0	1 (0.6)	
Not reported	7 (21.2)	52 (29.1)	
Maternal ethnicity n (%)			
Hispanic/Latino origin	10 (30.3)	83 (46.4)	0.0454
Not Hispanic or Latino	19 (57.6)	63 (35.2)	
Not reported	4 (12.1)	33 (18.4)	
Dosing of steroids n (%)			
Full course without rescue course*	13 (39.4)	99 (55.3)	0.0607
Full course with rescue course*	14 (42.4)	41 (22.9)	
Partial course *	6 (18.2)	39 (21.8)	
Timing of ACS (interval between time of administration & delivery) n (%)			
<24 hours	13 (39.4)	57 (31.8)	0.4873
1–7 days	17 (51.5)	93 (52.0)	
8–14 days	1 (3.0)	20 (11.2)	
>14 days	2 (6.1)	9 (5.0)	
Type of labour n (%)			
Spontaneous	9 (27.3)	61 (34.1)	0.89
Induced	4 (12.1)	22 (12.3)	
Spontaneous augmented	2 (6.1)	12 (6.7)	
No labour	18 (54.5)	84 (46.9)	
Type of delivery n (%)			
Vaginal delivery	5 (15.2)	56 (31.3)	0.06
LSCS	28 (84.8)	123 (68.7)	

*Full course (12 mg x2 doses 24 hours apart), Partial course (12 mg x 1 dose), Rescue Course (12 mg).
ACS, antenatal corticosteroid; LSCS, lower segment caesarean section.

tissues, hence contributing to the maternal hyperglycaemic state.[10] Steroid-induced maternal hyperglycaemia may precipitate NH due to a compensatory fetal hyperinsulinaemia. Several studies looking at NH after ACS administration in early and late preterm births have defined it as a blood glucose <40 mg/dL in the first 48 hours of life.[8 11] Neonates diagnosed with hypoglycaemia have a higher risk of developing hypothermia, apnoea, hypotonia, feeding difficulties and seizures,[12] all of which may result in the admission to the neonatal intensive care unit. NH also increases the risk of brain injury with subsequent developmental delay.[13 14]

Several studies have evaluated the risk of NH secondary to ACS administration[6 8 11 15 16] and have concluded that neonates exposed to antenatal steroid therapy had a higher rate of hypoglycaemia in the postpartum. One study that evaluated antenatal steroid administration in pregnancies that resulted in early preterm births (<34 weeks gestation) indicated that the incidence of NH in the first 48 hours of life may be slightly higher in mothers who had received ACS versus those who had not (23.0% vs 16.1%, adjusted OR 1.3, 95% CI 0.5 to 3.6), though the difference was not statistically significant. This study included 635 participants but did not distinguish pregnant mothers diagnosed with diabetes from pregnant mothers without diabetes.[8] Another study that included a larger cohort of patients (n=4429) had included pregnancies complicated with diabetes (n=510) where 439 (86.1%) had received ACS therapy. Although this study found no difference in maternal diabetes when evaluating the neonatal effect of ACS therapy, it also did not include information on NH. Data were available on neonatal morbidities that included respiratory distress syndrome, necrotising enterocolitis, grade 3 or 4 intraventricular haemorrhage, sepsis and neonatal death.[9] Current literature states that neonates of mothers diagnosed with diabetes have an increased risk of developing hypoglycaemia after birth.[17 18] These studies have also noted no difference in the risk of NH when comparing GDM pregnancies with pre-GDM.[19 20]

The current paediatric guidelines direct the management of hypoglycaemia in high-risk neonates, which include neonates of mothers diagnosed with diabetes, but do not specify pregnancies complicated with diabetes that

received ACS treatment for fetal lung maturity.[21 22] NH, if not promptly identified and treated, can have significant short- and long-term health implications for the newborn. In the short term, infants with severe hypoglycaemia may experience neurological symptoms such as seizures, apnoea and poor feeding. Long-term consequences, although less well understood, may include an increased risk of developmental delays and cognitive impairments.[23]

Early recognition of symptoms of NH (which include tremor, apnoea and hypotonia), and the initiation of oral or parenteral feeding in the first hour of life is necessary in decreasing neonatal morbidity in the postpartum. While a cut-off threshold for hypoglycaemia leading to neurologic sequelae is not established, the aim of treatment is to establish a glucose concentration greater than 50–60 mg/dL in these neonates and to avoid any neurologic sequelae.[24–26]

Our findings regarding the incidence of NH in neonates born to mothers with diabetes are consistent with those reported in the existing literature. Specifically, the rate of NH observed in our study (39.4%) aligns closely with the findings of Harding *et al* (2024), who reported an approximately 40% incidence of NH in neonates born to mothers with diabetes.[27] This reinforces the validity of our results and suggests that the association between maternal diabetes and an increased risk of NH is well-established. Our study supports this body of evidence and highlights the importance of vigilant screening and management of NH in this high-risk population.

Future studies are needed to evaluate the effects of maternal glycaemic control during pregnancy and NH, both for preterm and term deliveries. Studies evaluating the maternal glucose levels at different stages prior to delivery are also necessary in order to assess any correlation with developing NH. While it is known that hypoglycaemia is common in late preterm infants and more common after ACS administration, it is not known whether maternal glucose control can mitigate these effects.

The strengths of our current study represent a retrospective cohort of high-risk women receiving similar treatment for a high likelihood of preterm delivery at a single centre. Additionally, neonates had similar management plans and evaluation for hypoglycaemia. Our study supports the current literature and evaluates a major risk factor for NH, which is maternal diabetes in pregnancy.

One limitation of our study is the relatively small sample size within the diabetes group (n=33), which may have limited the statistical power to detect significant associations in subgroup analyses, particularly regarding the dose and timing of ACS in relation to NH. While our findings provide preliminary insights, larger studies with expanded diabetic cohorts are needed to validate these results and explore dose-response relationships more robustly.

In interpreting the findings, it is important to acknowledge the limitations of the study, particularly regarding sample representativeness and potential biases. The data used in this study were derived from a single tertiary medical centre with a patient population that may not be representative of all racial and ethnic groups. While the sample size of 212 patients is relatively large, the study population primarily reflects the demographic characteristics of the local population, which may not fully represent the broader national or global population. Notably, the racial and ethnic distribution of our sample is predominantly Caucasian, African American and Hispanic patients. Given that maternal diabetes and NH may present differently across racial and ethnic groups, the results of this study should be interpreted with caution when considering populations with different demographic profiles. Future studies should aim to include a more diverse cohort to better understand how different demographic factors, such as race, ethnicity and socioeconomic status, may influence the incidence of NH in mothers diagnosed with diabetes.

We also acknowledge potential sources of bias, including selection bias, given the use of a single tertiary medical centre and potential limitations in the representativeness of the sample. To mitigate this bias, we employed strict inclusion and exclusion criteria and ensured systematic data collection from medical records. Efforts were made to reduce missing data by using complete and verified records for all included patients. Nonetheless, residual confounding cannot be entirely ruled out, and prospective studies with more diverse patient populations would be essential to confirm the findings and improve generalisability. Additionally, information bias may have occurred due to inconsistencies or inaccuracies in medical record documentation, such as errors in the classification of diabetes or NH. These biases were minimised by using strict inclusion criteria and validating data where possible, though residual confounding remains a limitation. Further prospective cohort studies or randomised controlled trials would be valuable in confirming the causal relationships suggested by our findings. Such studies would also help address the generalisability of our results and allow for more comprehensive evaluations of the effects of prenatal corticosteroid treatment in different patient populations.

A noteworthy limitation of our study is the availability of information pertaining to the maternal glucose control prior to delivery. This could not be consistently found in the medical record, limiting our ability to correlate maternal glycaemia with NH. There was also no ability to compare blood glucose measurements of mothers diagnosed with diabetes who received steroids with mothers diagnosed with diabetes who did not receive antenatal steroid treatment, as they were likely at differential risk of preterm delivery. In addition, a lack of detailed maternal glycaemic control data, such as haemoglobin A1c levels and insulin usage, may influence the risk of NH and could have helped further clarify the relationship between maternal diabetes and neonatal outcomes. However, this information was inconsistently documented in the

medical records and was therefore unavailable for analysis. Future studies should aim to incorporate comprehensive glycaemic control metrics and should aim to investigate the relationship between maternal blood sugar levels before delivery and NH with more comprehensive data collection, including detailed maternal glucose monitoring during labour and delivery, to better assess their impact on NH. Studies using prospective, multicentre designs and randomised controlled trials would help to validate these findings and provide stronger evidence for clinical recommendations. Such studies would allow for more controlled data collection, help establish causal relationships and explore the long-term effects and neonatal outcomes.

While our study demonstrates that early preterm neonates of mothers diagnosed with diabetes who received ACS have a higher risk of NH, our study lacks a control group of neonates born to mothers with diabetes who did not receive ACS treatment. As such, we cannot directly attribute the increased risk of NH solely to ACS administration, as the observed association may be influenced by other factors, such as maternal hyperglycaemia or the underlying diabetes condition. The absence of a non-ACS-exposed group restricts the ability to conclusively determine whether ACS itself is the cause of the higher NH risk or if this is a confounding effect due to the maternal diabetes. We acknowledge that the conclusion about the role of ACS in NH in early preterm neonates of mothers with diabetes should be interpreted with caution, and further research—including studies with a comparison group—would be required to more definitively assess the role of ACS exposure.

Given that healthcare providers already closely monitor both mothers diagnosed with diabetes and their neonates, our findings further emphasise the importance of continued vigilance in this population. We recommend reinforcing current practices by focusing on the careful management of maternal blood glucose levels in the perinatal period to mitigate the risk of NH. Additionally, early screening and timely intervention for NH remain essential for these infants. By continuing to prioritise these measures, healthcare providers can further improve maternal and neonatal outcomes. We also suggest that future research investigate additional strategies to optimise care, including prospective studies and randomised controlled trials to refine management approaches. Data on maternal blood glucose concentrations and maternal glycaemic control during pregnancy are important next steps to further interpret our findings.

In conclusion, ACS administration is important in pregnancies threatened with a preterm delivery. Neonates of mothers diagnosed with diabetes who received ACS therapy are at an increased risk of developing hypoglycaemia, thus requiring a higher level of attention and care following delivery. Due to the lack of a control group of unexposed infants, the direct impact of ACS on NH cannot be conclusively determined and further studies are required to evaluate this.

Author affiliations
[1]Department of Obstetrics and Gynecology, Division of Maternal-Fetal Medicine, Columbia University in the City of New York, New York, New York, USA
[2]Department of Obstetrics and Gynecology, Columbia University Irving Medical Center, Columbia University in the City of New York, New York, New York, USA
[3]Department of Obstetrics, Gynecology & Reproductive Sciences, Division of Maternal-Fetal Medicine, University of California San Diego School of Medicine, San Diego, California, USA

Contributors SA-N designed the study and wrote the project proposal, was responsible for all IRB submissions, collected and analysed data, drafted, edited and revised the paper. RM analysed, edited and revised the paper. EO analysed, edited and revised the paper. QY wrote the statistical analysis plan and analysed the data. CG-B designed and supervised the study, analysed, drafted, edited and revised the paper. SA-N is the guarantor and corresponding author for this manuscript.

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Data availability statement The data supporting this study are not publicly available due to confidentiality agreements but may be available from the corresponding author upon reasonable request and with permission from the Department of Obstetrics and Gynecology at Columbia University.

ORCID iD
Sarah Al-Nafisee https://orcid.org/0009-0008-4305-7433

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