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Determinants of neonatal hypoglycemia after antenatal administration of corticosteroids (ACS) for lung maturation: Data from two referral centers and review of the literature

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ABSTRACT

Background: A correlation between ACS and neonatal hypoglycemia has been recently demonstrated.
Aims: The aim of the study was to evaluate the determinants of neonatal hypoglycemia in women exposed to ACS for respiratory distress syndrome prevention.
Material and methods: Retrospective, multicenter, cohort study conducted in two Tertiary University Units. All fetuses delivered from 2016 to 2017 after ACS (two doses i.m. of Betamethasone 12 mg 24 h apart) were considered eligible for the study purpose. The primary outcome was the incidence of hypoglycemia, defined as a glycemic value ≤ 45 mg/dl within the first 48 h of neonatal life. The effect on neonatal glycaemia due to timing (interval from exposure to delivery) and type (single completed, single partial or repeated course) of ACS administration was also assessed.
Results: Overall, 99 neonates met the inclusion criteria. Hypoglycemia occurred in 38/99 (38.4%) of the included newborns. Compared to normoglycemic neonates, those with hypoglycemia had lower gestational age at delivery (33.06 ± 3.37 vs. 35.94 ± 3.17 g; $p < 0.0001$). Lower birthweight (1747.28 ± 815.29 vs. 2499.24 ± 780.51 g; $p < 0.0001$), a shorter interval time from administration to delivery (1.85 ± 2.59 vs. 3.34 ± 3.39 weeks; $p = 0.02$) and a higher incidence of single partial course (23.7 vs. 8.72%; $p = 0.03$). Multivariate logistic regression found that only birthweight was significantly associated with neonatal hypoglycemia (OR 0.4 95% CI −1.16/−0.04; $p < 0.038$).
Conclusion: Hypoglycemia occurs in a large proportion of fetuses exposed to ACS independently from the type of exposure (single partial/single completed) and from the time interval between ACS administration and delivery. Birthweight seems to be the strongest determinant for the occurrence neonatal hypoglycemia after antenatal administration of steroids for lung maturation.

1. Introduction

Antenatal corticosteroids administration (ACS) is the accepted standard of care for women at risk of preterm delivery due to their beneficial effects on neonatal morbidity and mortality [1]. Following the demonstration of their clinical usefulness among the preterm neonates delivered within 2 to 7 days from maternal administration [2], the use of steroids has started to be widely offered to all women deemed to be at risk of impending premature delivery. In our daily practice in fact the use of steroids turns out to be mostly inappropriate as the vast majority of fetuses remain undelivered for weeks after the drug exposure [3].

A single course of ACS is generally considered to be safe for the fetus. However, concerns have been expressed regarding their short and long-term side effects on the fetus [4] including reduced size and head circumference, increased and prolonged dysregulation of the adrenal axis [5], cognitive and motor impairment [6]. Moreover, a correlation between ACS and neonatal hypoglycemia has been recently

Abbreviations: ACS, antenatal corticosteroids; BMI, body mass index; IUGR, intrauterine growth restriction

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demonstrated [7,8]. Although neonatal hypoglycemia is amenable of successful treatment by care providers, its long-term metabolic and neurological consequences are still matter of debate [9,10].

The aim of this study was to evaluate the determinants of neonatal hypoglycemia in women exposed to ACS for the induction of fetal lung maturation.

2. Materials and methods

2.1. Study design and participants

This is a retrospective multicenter cohort study. Clinical records of all consecutive neonates delivered from 2016 to 2017 at two tertiary care university Hospitals in Italy (University of Parma; University of Naples Federico II) were included in a dedicated merged database. Only neonates exposed to ACS were included. Women at risk for spontaneous or iatrogenic preterm delivery between 24 0/7 and 33 6/7 or those undergoing to late preterm or early term cesarean between 34 0/7 and 37 6/7 weeks were considered eligible for the study. Indication for planned cesarean delivery < 38 weeks included placenta previa and/or accreta, severe pregnancy hypertensive disorders, severe and early fetal growth restriction. In the two institutions participating to this study, ACS included two doses of Betamethasone 12 mg administered intramuscularly 24 h apart [11].

Exclusion criteria were neonatal congenital anomalies, multiple pregnancy, neonatal hypoxic ischemic encephalopathy or sepsis, maternal diabetes and maternal steroids treatment due to other medical indications.

We defined as early preterm those newborns delivered between 24 0/7 and 33 6/7 weeks while late preterm infants were those delivered between 34 0/7 and 36 6/7 weeks. Optimal ACS administration was defined as an administration between 24 and 7 days before delivery; suboptimal administration was defined as an administration < 24 h or > 7 days before delivery.

All the neonates included in the present study underwent a strict monitoring protocol. Neonatal blood glucose concentrations were measured on capillary blood samples taken by heel-prick lance and analyzed on a blood gas analyzer using the glucose oxidase method. Samples were taken at 1 h after birth regardless of feeds, then before feeds 3–4 hourly in the first 24 h, then 3–8 hourly for the next 24 h. Measurements were discontinued if there were 3 normal blood glucose concentration measurements and no clinical concerns about feeding.

Neonatal hypoglycemia was defined as a glycemic value ≤45 mg/dl within the first 48 h of neonatal life [12]. The lowest glycemic value found during this interval-time was considered for the purpose of the study.

2.2. Outcomes

The primary outcome was the overall incidence of neonatal hypoglycemia in a cohort of women exposed to ACS for the prevention of respiratory distress syndrome. The secondary outcome was to compare demographics, clinical features and pregnancy characteristics between cases with hypoglycemic vs normoglycemic neonates.

Population birthweight percentiles were retrospectively calculated using Italian data and gestation calculator [13]. According to these national neonatal charts, small for gestational age (SGA) neonates were defined as those whose birthweight was below the 10th centile, while large for gestational age (LGA) infants were defined as those whose birthweight was above the 90th centile [13].

2.3. Statistical analysis

Statistical analysis was performed using Statistical Package for Social Sciences (SPSS) v. 21.0 (IBM Inc., Armonk, NY, USA). Data are shown as means ± standard deviation (SD), or as medians (range), or as numbers (percentage). Univariate comparisons of dichotomous data were performed with the use of the chi-square with continuity correction. Comparisons between groups were performed with the use of the *t*-test to test group means with SD by assuming equal within-group variances.

To assess the independent predictors of neonatal hypoglycemia baseline characteristics associated with a $P < 0.05$ in univariate analyses were implemented into a multivariable backward-stepwise regression model with removal criteria of $P > 0.1$. After testing collinearity, correlated variables (VIF > 3) were not used simultaneously in the same model (e.g. Gestational age at delivery and birthweight). We calculated two-sided *p*-values. A *p*-value < 0.05 was considered to indicate statistical significance. This study was reported following the STROBE guidelines [14].

This study has been approved by the local ethics committee (976/2018/OSS/UNIPR).

3. Results

Over the study period, a total of 99 neonates met the inclusion criteria. Overall, a mean glycemic value of 52.24 ± 32.17 mg/dl was reported while the incidence of neonatal hypoglycemia was 38.4% (38/99). Neonatal characteristics and main indications to ACS administration are summarized in Table 1. The incidence of hypoglycemia was significantly different among early preterm, late preterm and term-delivered neonates (60.5% vs. 38.5% vs. 14.3% respectively; $p = 0.002$).

An overall rate of optimal ACS administration of 40.4% was reported with 52.5% of deliveries occurring > 7 days from ACS administration and 7.1% of them within 24 h from ACS course.

On Table 2 maternal demographics, pregnancy characteristics and type of ACS exposure between hypo- vs normoglycemic neonates are illustrated. As shown on Table 2, neonatal hypoglycemia was more frequent in cases with lower gestational age at delivery, lower birthweight, single partial course and shorter time interval from exposure to delivery.

At multivariable backward stepwise regression including the relevant covariates selected by univariate analysis only the birthweight remained significantly associated with neonatal hypoglycemia (Table 3); in addition, a positive correlation between the neonatal glycaemia (mg/dl) and birthweight (g) was demonstrated by using linear regression ($y = 5.6639x + 1914.7$; $R^2 = 0.044$; $p = 0.038$).

Table 1
Neonatal outcome, characteristics and main indications for antenatal corticosteroids (ACS) course.

	n = 99
Neonatal outcome	
GA at ACS course (weeks)	32.06 ± 3.33
GA at delivery (weeks)	34.83 ± 3.53
Birthweight (g)	2210.61 ± 871.22
Birthweight percentile	38.15 ± 27.41
Neonatal Hypoglycemia	38(38.4)
Mean glycemic value (m/dl)	52.24 ± 32.17
Characteristics of ACS course	
Complete ACS course	70(70.7)
Interval from exposure to delivery (weeks)	2.77 ± 3.18
Repeated ACS course	13(13.1%)
Optimal ACS course	34(34.3)
Main indications for ACS course	
Preeclampsia/IUGR	32(32.4)
p-PROM	12(12.1)
Preterm labor	30(30.3)
Placenta previa	19(19.2)
Vaginal bleeding	3(3.0)
Elective caesarean section	3(3.0)

GA = gestational age, p-PROM = preterm premature rupture of membranes; IUGR = intra-uterine growth restriction.

Table 2
Secondary outcome.

	Hypoglycemia (n = 38)	Normoglycemia (n = 61)	*p*-Value
Maternal age	31.94 ± 6.53	29.85 ± 7.10	0.14
Caucasian	26(68.4%)	47(77.0%)	0.34
Gestational age at delivery	33.06 ± 3.37	35.94 ± 3.17	< 0.0001
Fetal gender (M:F)	19:19	30:31	0.93
Caesarean section	9 (23.7%)	26(42.6%)	0.06
Neonatal weight	1747.28 ± 815.29	2499.24 ± 780.51	< 0.0001
Birthweight percentile	32.00 ± 26.08	40.55 ± 27.35	0.17
Preterm delivery (< 37 gestational weeks)	33(86.8%)	31(50.8)	< 0.001
Small for gestational age (SGA)	12(31.6%)	11(18.0%)	0.12
Large for gestational age (LGA)	0	3(4.9%)	0.16
Gestational age at I dose	31.21 ± 3.21	32.59 ± 3.32	0.04
Interval time from administration to delivery	1.85 ± 2.59	3.34 ± 3.39	0.02
Single partial course	9(23.7%)	5(8.2%)	0.03
Repeated course	6(15.9%)	7(11.5%)	0.53
Optimal course	13(34.21%)	21(34.42%)	0.98

Table 3
Multivariate backward stepwise regression analysis for predictors of neonatal hypoglycemia.

Variable	OR (95% CI)	*p*-Value
Birthweight (kg)	0.4 (−1.16/−0.04)	0.038
Time interval from exposure to delivery (weeks)	1.00 (−0.20–0.20)	0.99
Single partial course	2.00 (−0.62–2.00)	0.30
Preterm delivery (< 37 gestational weeks)	1.97 (−0.77–2.13)	0.36

The multivariate regression model including gestational age at delivery did not demonstrate a significant association between this variable and neonatal hypoglycemia (Supplement 1).

4. Discussion

4.1. Principal findings

Our study confirmed that hypoglycemia during the first 48 h of neonatal life occurs in a large proportion of fetuses exposed to ACS for respiratory distress syndrome (RDS) prevention; in our series a glycemic value below the threshold of 45 mg/dl was noted in more than one third of the neonates with a higher prevalence in preterm newborns.

Furthermore, we found that birthweight is the strongest determinant for neonatal hypoglycemia among a population of women exposed to antenatal steroids while type of ACS exposure (single partial/single completed) and interval between ACS administration and delivery does not seem to affect the occurrence of neonatal hypoglycemia.

4.2. Interpretation

There are two main mechanisms potentially implicated in neonatal hypoglycemia due to ACS exposure. The first one is corticosteroid-induced hyperglycemia in mothers leading to fetal and neonatal hyperinsulinemia and hypoglycemia due to hyperplasia of fetal pancreatic beta cells [15]; the second one, as suggested by some, is mediated by fetal adrenal suppression [16].

On the other hand, previous experimental studies demonstrated that antenatal exposure to corticosteroids may produce the induction of fetal hepatic enzymes involved in the regulation of glucose metabolism [17].

These studies, together with our findings may support the concept that antenatal exposure to steroids can promote substantial changes on the fetal glucose metabolism. A higher insulin resistance in adults exposed to corticosteroids during fetal life has been recently shown in a large RCT [18]. On this basis, it is plausible to speculate that in some cases neonatal hypoglycemia or possibly adult hyperglycemia may be considered as a long-term side-effect of ACS administration, possibly mediated by “fetal programming” mechanisms.

Our own observation that a lower birthweight is the strongest determinant of hypoglycemia among neonates exposed to antenatal steroids seems to confirm that the occurrence of hypoglycemia is more likely if the fetal storage of glycogen is absent or reduced [19]. Fetal growth restriction rather than prematurity per se has been actually reported as the major risk factor for neonatal hypoglycemia, with an incidence ranging from 17 to 61% [20] and this may indicate that the body weight composition may affect the risk of neonatal hypoglycemia independently and more importantly than gestational age at delivery. A recent study found that low body fat is the strongest risk factor for neonatal hypoglycemia even in neonates with an appropriate birthweight [21].

In most of the existing studies on the association between prematurity and neonatal hypoglycemia, the net contribution of maternal steroids on the occurrence of neonatal hypoglycemia is difficult to quantify. In women at risk of delivery before 34 gestational weeks in fact ACS are routinely administrated, and this mostly precludes to assess the incidence of hypoglycemia among preterm newborns whose mothers were not exposed to steroids [1]. Also, in our study including a cohort of women at risk of preterm delivery in whom the antenatal administration of steroids was clinically indicated we lack a control group of women unexposed to betamethasone.

A European cohort study of 4594 infants found that only 14% of early preterm infants were not exposed to antenatal ACS [22]. For this reason, existing literature on metabolic effects of ACS on early preterm infants is mostly based on small retrospective studies with a bias due to a more severe clinical condition requiring delivery before ACS course.

A summary of the studies which have compared the incidence of neonatal hypoglycemia among neonates exposed to ACS compared to non-exposed neonates is provided on Table 4 [8,23–25]. According to this data a higher incidence of neonatal hypoglycemia has been found in neonates whose mothers has been exposed to ACS.

4.3. Clinical implications

Our results confirm the need of a strict glycemic control for neonates exposed to ACS, in particular for those of low birthweight. In our series a higher incidence of hypoglycemia among both early and late preterm neonates was found compared with previous data, probably due to the higher cut-off used in our study for the definition of hypoglycemia. On the other hand, we noted a not negligible incidence of hypoglycemia even in full-term neonates. Among them, strong figures on the incidence of hypoglycemia are lacking as routine blood glucose assessment in asymptomatic newborns is not recommended. No definitive consensus has been actually reached on how to define and when

Table 4
Summary of existing studies on neonatal hypoglycemia after antenatal corticosteroid (ACS) administration.

Study, year	Study type	Neonates exposed to ACS (n)	Definition of hypoglycemia	Hypoglycemia in exposed neonates (%)	Hypoglycemia in non-exposed neonates (%)
Early preterm (24 + 0–33 + 6)					
Kuper et al. [23], 2017	Retrospective cohort study	604	< 40 mg/dl within the first 48 h of life	23.0	16.1
Late preterm (34 + 0–36 + 6)					
Porto et al. [24], 2011	Double blind RCT	143	Not stated	10.5	7.0
Gyamfi-Bannerman et al. [8], 2016	Double blind RCT	1427	< 40 mg/dl at any time	24.0	15.0
Ramadan et al. [25], 2016	Prospective cohort study	64	< 40 mg/dl within 1 h of life	20.3	10.9

to look for hypoglycemia among full-term neonates. A study by Bromiker et al. [26], analyzed the incidence of neonatal hypoglycemia among unselected healthy full-term neonates reporting an incidence ranging between 3% and 12% according to the cut-off used for hypoglycemia (< 40 mg/dl and < 47 mg/dl, respectively). No data are available regarding the incidence of hypoglycemia in neonates born after 37 weeks of gestation whose mothers were antenatally exposed to corticosteroids [7].

These observations may suggest a more intensive metabolic surveillance of neonates exposed to ACS, before the occurrence of symptoms of hypoglycemia.

Neonatal hypoglycemia can be considered as a minor side effect if compared to the beneficial effects of ACS administration on neonatal outcome especially for infants born before 34 gestational weeks. After this gestational age, the available evidences show a modest clinical benefit of steroids mainly on morbidity but not on mortality rate compared to the potential risks, including hypoglycemia [2]. While hypoglycemia can be easily managed especially in high-intensity care settings, its long-term consequences are still undetermined. The recent CHYLD study ("Children with Hypoglycemia and Their Later Development") has shown a dose-dependent increased risk of poor executive function and visual motor function at 4 years of age in children who experienced neonatal hypoglycemia [13]. However, since hypoglycemia was mostly documented in preterm neonates and since prematurity itself is a major risk factor of poor neurological outcome, the true association between hypoglycemia and neurodevelopmental delay remains matter of investigation [27].

4.4. Strengths and limitations

The main limitations of this study are its retrospective design and the small number of neonates included. Moreover, the lack of a control group not exposed to steroids does not allow to assess the net contribution of steroids to the occurrence of neonatal hypoglycemia. However, this was not the main goal of our work. The objective of this study was rather to establish which factors may affect the incidence of neonatal hypoglycemia among women exposed to ACS for the prevention of respiratory distress syndrome.

The main strength of this work is that we have thoroughly examined a list of pregnancy characteristics and variables associated with ACS administration to work out if and how steroids may affect the incidence of hypoglycemia among both preterm and full-term neonates.

In conclusion, our results demonstrated that hypoglycemia after ACS is more likely to occur in neonates with a lower birthweight irrespectively from the interval between the drug exposure and the gestational age at delivery.

On this basis due to the uncertain long-term effects of neonatal hypoglycemia the antenatal administration of steroids should be reserved only to the women at risk of impending preterm delivery where the good of the drug exposure for the offspring clearly outweighs the bad or the unknown [3]. Moreover, glycemic control should be carried out in all neonates exposed to ACS particularly in low birthweight neonates and independently from gestational age at delivery and from the time interval between exposure and delivery.

Finally, despite the small size of our study we believe that our findings are worthy to be reported and reassessed on a larger population. Additional investigations and continuous promotion of clinical guidelines are certainly warranted to optimize the administration of antenatal corticosteroids and to minimize the harmful effects on the exposed neonates.

Supplementary data to this article can be found online at https://doi.org/10.1016/j.earlhumdev.2020.104984.

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Declaration of competing interest

The authors report no conflict of interest.

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