Effects of Antenatal Steroids in Late Preterm Infants

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Introduction
Antenatal-corticosteroid therapy for women at risk for preterm delivery (<34 weeks gestational age [GA]) significantly improves survival/respiratory outcomes. Initial recommendations for antenatal-steroid administration from the American College of Obstetricians and Gynecologists (ACOG) did not include late-preterm infants (34–36 weeks GA) due to lower mortality rates; however, research continues to show late-preterm infants have higher morbidities compared to term counterparts, including respiratory distress, hypoglycemia, hypothermia, hyperbilirubinemia, and feeding difficulties.

Gyamfi-Bannerman et al. demonstrated antenatal-steroids decreased severe respiratory complications in late-preterm infants, but increased rates of hypoglycemia. Based on these findings, ACOG extended their recommendations for antenatal-steroids to include women at risk for late-preterm delivery in an attempt to improve respiratory outcomes in late-preterm infants. However, respiratory and glycemic effects of antenatal-steroids on this gestation are still not clear.

Objective
To determine whether antenatal-steroids mitigate respiratory morbidities, but become a new risk factor for hypoglycemia in late-preterm neonates.

Design/Methods
This single center study includes mother-baby dyads delivered at 34 0/7–36 6/7 weeks GA between 01/2016–12/2016. Respiratory effects of antenatal-steroids were assessed by diagnosis of respiratory distress in first 24 hours-of-life and total days of respiratory support in patients with respiratory distress. Glycemic effects were assessed by lowest serum glucose levels in first 24 hours-of-life. Effects on outcomes were evaluated using t-tests, chi-squares, and generalized estimating equations (GEE) to account for multiple gestations and adjust for confounding variables.

Results
Study included 500-neonates and 426-mothers. 59.8% neonates (N = 299) were exposed to steroids. To account for potential confounders, maternal/neonatal risk factors (p < 0.10) were included in the analysis (GA, preterm premature rupture-of-membranes, gestational diabetes, Apgars, birth-weight, large for gestation [LGA]).

Among the entire group, there were no differences in rate of respiratory distress, duration of respiratory support, or lowest glucose levels. Considering effects of GA on respiratory/glycemic outcomes, the cohort was divided into subsets based on GA for further analysis.

In the 34 week GA-group, antenatal-steroids did not affect rate of respiratory distress or lowest glucose levels, but significantly decreased the duration of respiratory support (2.7 ± 0.5 days steroid-group vs. 4.7 ± 0.8 days non-steroid group, p = 0.03). In the 35 week GA-group, steroids didn’t affect rate of respiratory distress, duration of respiratory support, or lowest glucoses. In the 36 week GA-group, steroids didn’t affect rate of respiratory distress or duration of respiratory support, but significantly decreased lowest glucose levels (35.2 ± 2.4 vs. 38.6 ± 1.9 mg/dL, p = 0.04). This effect was additive when combined with other risk factors (gestational diabetes, LGA).

Conclusion
Antenatal-steroid exposure shortened duration of respiratory therapy only in infants born <35 weeks GA, and increased severity of hypoglycemia in infants born ≥36 weeks GA. (Graphs 1 and 2). Findings suggest current ACOG recommendations for late preterm antenatal-steroid administration may not be beneficial for infants born ≥36 weeks.
Graph 1: Duration of Respiratory Support in Infants with Respiratory Distress by Gestational Age and Antenatal Corticosteroids.

Graph 2: Lowest Glucose Level by Gestational Age and Antenatal Corticosteroids.