Retrospective Audit of Pre-gestational and Gestational diabetes women receiving betamethasone at Bankstown-Lidcombe Hospital.

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Introduction: Antenatal corticosteroids are used to accelerate lung maturity for several obstetric indications, including pre-term labour[1]. Betamethasone has the propensity to cause significant hyperglycemia, particularly in women with pre-gestational or gestational diabetes mellitus (GDM). This highlights the need for glycemic monitoring and involvement of the endocrine team to determine and institute appropriate glycaemic management strategies to prevent maternal hyperglycaemia.

Aim: We sought to evaluate current practice at Bankstown-Lidcombe Hospital with the aim of developing appropriate guidelines around betamethasone administration in pregnancy to ensure best practice.

Methods: We performed a retrospective audit of maternal records from May 2017 to May 2018 in consecutive women with pre-gestational diabetes or GDM receiving antenatal betamethasone. An equal number of pregnant women with normal glucose tolerance (also receiving betamethasone) acted as controls. Women requiring urgent betamethasone before an emergency caesarean section were excluded.

Results: There were 23 women in each group, with baseline characteristics shown in Table 1.

Table 1 Baseline Characteristics

	Pre-gestational DM/GDM n=23 mean ± SD	Control n=23 mean ± SD	P value
Maternal Age (years)	29.8 ± 6.0	30.5 ± 5.0	NS
Maternal Weight (kg)	84.6 ± 14.2	71.3 ± 16.6	<0.01
Gestational Age at betamethasone administration (weeks)	36.6 ± 2.1	36.3 ± 2.4	NS
Gestational Age at Delivery (weeks)	37.6 ± 0.5	37.4 ± 1.4	NS

Identified areas for improvement were: timing of betamethasone administration, initiation of glucose monitoring and early involvement of the endocrine team prior to betamethasone administration. Significant hyperglycaemia was experienced in 22/23 pre-gestational DM/GDM women (95.7%), and severe hyperglycaemia (postprandial >10.0 mmol/L) in 4/23 women (17.4%). The median time between betamethasone administration and delivery was 2.8days (Range 0.0-67.2days). Table 2 includes other findings.

Table 2 Current practice and betamethasone administration.

	Number of cases n=23 (%)
Was the patient admitted for betamethasone?	19 (82.6)
Was betamethasone administered before 9am?	2 (8.7)
Was the endocrine team contracted before betamethasone administration?	10 (43.4)

Was blood glucose tested before betamethasone administration?	8 (34.8)
Was the endocrine team contacted at all?	19 (82.6)
Was the glucose lowering therapy adjusted?	18 (78.3)
Was hyperglycaemia experienced (>3 above target)?	22 (95.7)
Was severe hyperglycaemia experienced (>10mmol/L)?	4 (17.4)
Was blood glucose monitored 4-8 hours post betamethasone administration?	20 (87.0)
Were blood glucose levels checked at least 4x daily	17 (73.9)
Was blood glucose levels monitored for at least 48 hours after the last betamethasone dose.	18 (78.3)

All but one Pre-gestational diabetes/GDM woman had delivered at the time of data collection. Neonatal hypoglycaemia was more common in Pre-gestational diabetes/GDM women, compared to NGT women 54.5% vs 17.4% (OR 5.7, 95% CI 1.5–22.2), despite no significant difference in birthweight or macrosomia rates. Results are summarised in Table 3.

Table 3 Outcomes

	Pre-gestational DM/GDM n=22 Mean ± SD or Cases (%)	Control n=23 Mean ± SD or Cases (%)	p-value
Birth Weight (grams)	3266 ± 559	2922 ± 582	NS
Macrosomia (>4000g)	2 (9.1)	0 (0.0)	NS
Low birth weight (<2500g)	2 (9.1)	5 (21.7)	NS
Premature Delivery (<37 weeks)	2 (9.1)	4 (17.4)	NS
Neonatal hypoglycaemia	**12 (54.5)**	**4 (17.4)**	**<0.01**
NICU/SCN admission	10 (45.5)	7 (30.4)	NS

Conclusion:
Our audit demonstrated that a structured guideline of glycaemic monitoring and management is required for pre-gestational DM and GDM women having betamethasone therapy. These women are at high risk of hyperglycaemia following betamethasone administration and their infants at high risk of neonatal hypoglycaemia.

References:

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