immunohistochemically by positive chromogranin, synaptophysin and BCl2 and negative for calretinin and S100.

Discussion and Conclusion

The etiology of hypoglycemia after resection of unilateral pheochromocytoma can be explained by impaired glucagon secretion and decreased gluconeogenesis due to the suppression from higher catecholamine levels in the blood pre-operatively. The second mechanism is rebound insulin secretion from the pancreas due to sudden withdrawal of catecholamines.

In our patient, the transient hypocortisolemia could be another reason. The lack of immunohistochemical evidence in post-surgical pathology report excluded cortisol- secreting tumor. Another rare situation, ACTH-secreting pheochromocytoma, has been reported but was not checked in the case.

In a word, hypoglycemia is common after surgical removal of unilateral pheochromocytoma. Careful monitoring of patients' glucose level in immediate post-resection period is essential to prevent transient hypoglycemia

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Diabetes Mellitus and Glucose Metabolism

GESTATIONAL DIABETES, DIABETES IN PREGNANCY, AND IN UTERO EXPOSURES

Gaps in Quality of Delivery of Post-Partum Care in Preconception Counselling for Pregnant Women with Pre-Existing Diabetes at a Large Academic Tertiary Centre.

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BACKGROUND: Women with pre-existing diabetes are at increased risk of serious adverse pregnancy outcomes compared with the general maternity population including congenital anomaly, stillbirth and neonatal death. The 2018 Diabetes Canada Clinical Practice Guidelines (DC CPG) on Diabetes in Pregnancy recommend that women with pre-existing diabetes are provided with postpartum and preconception counseling by their diabetes healthcare team, as this is associated with improved maternal and fetal outcomes.

OBJECTIVE: To evaluate the quality of physican counselling of post-partum management and pre conception advice for women with pre-existing diabetes who receive their intrapartum care at Mount Sinai Hospital Diabetes in Pregnancy Clinic in Toronto Ontario.

METHODS: Eligible patients were pregnant women with pre-existing Type 1 and Type 2 diabetes who were followed in pregnancy until their 6 weeks postpartum clinic visit. Consecutive baseline chart review of patients between June 2018 - June 2019 was performed to audit documentation of physician counselling of DC CPG recommendations at the 6 week post-partum visit. Key components of the recommendations included: 1) targeting an HbA1c of <7% pre-pregnancy, 2) folic acid supplementation and neural tube defect prevention, 3) weight management and optimization of BMI, 4) contraceptive measures and family planning, 5) information regarding outcomes and risks for mother and baby 6) yearly retinal exam.

RESULTS: Results of our chart review found that 42% (n=50) of women with pre-existing diabetes who received their intrapartum care at our clinic returned for their 6 week postpartum visit between June 2018-June 2019. Audit of the 6 week post-partum clinic note found that less than 20% of women had physician documentation of counselling on two or more key components of the DC CPG recommendations (1-6).

CONCLUSION: There is a large gap in women attending postpartum appointments and there are significant gaps in physician documentation of counselling among women with pre-existing diabetes. Further analysis will be conducted in order to determine if there is a patient knowledge gap regarding counselling recommendations and a quality improvement project will be undertaken to close this gap.

Neuroendocrinology and Pituitary

PITUITARY TUMORS I

Muscarinic and Adrenergic Receptor Cooperativity in a Human Adrenocortical Carcinoma Cell Line

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The role of autonomic receptors in the regulation of the adrenal cortex is poorly understood. We recently showed that activation of M_3 muscarinic receptors stimulates intracellular calcium oscillations, aldosterone production, and expression of *CYP11B2* (1). The present study explores the relationship between muscarinic and adrenergic receptors in corticosteroid production. Using live-cell fluorescence imaging of HAC15 adrenocortical cells with the calcium-sensitive probe Fluo-4, we have shown that stimulation of adrenergic receptors with the endogenous, non-selective adrenergic agonist norepinephrine (10μM) enhances intracellular Ca^{2+} oscillations caused by the cholinergic agonist carbachol (1μM). However, Ca^{2+} is not affected by norepinephrine alone. Adrenergic enhancement of carbachol-induced Ca^{2+} oscillations is blocked by the α adrenergic receptor antagonist phentolamine, but not by the β adrenergic receptor antagonist propanolol. Specifically, α2 and β2 antagonists (such as yohimbine and butoxamine, respectively) significantly suppressed the norepinephrine effect, but α1 and β1 antagonists (such as tamsulosin and metoprolol, respectively) had no effect. RT qPCR identified α2A receptors as the most abundant adrenergic receptor