to be down-regulated in adrenals from PA patients and spontaneously hypertensive rat adrenals compared to control adrenals[a,b]. *RORA* encodes for the protein retinoic acid receptor (RAR)-related orphan receptor alpha, a member of the NR1 subfamily of nuclear hormone receptors (NR1F1). Interestingly, adrenal is the second organ to skin with the highest expression of *RORA* and treatment of angiotensin II in the adrenocortical cell line H295R increases *RORA* expression[c,d]. Taken together, this pilot GWAS highlights *RORA* as a potential nuclear hormone receptor that regulates aldosterone production.

References
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Neuroendocrinology and Pituitary
CASE REPORTS IN SECRETORY PITUITARY PATHOLOGIES, THEIR TREATMENTS AND OUTCOMES

Tension Pneumothorax Following Cabergoline Initiation for Macroprolactinoma

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SAT-LB47

Background Pneumocephalus is a rare and life-threatening complication of dopamine agonists (DA) for the treatment of invasive giant prolactinomas. Here we present a catastrophic case of pneumocephalus following cabergoline therapy for invasive macroprolactinoma. *Clinical Case* A 49-year-old man presented with transient left-sided facial weakness for one day. MRI Brain showed no acute infarcts but revealed a 3.7cm pituitary macroadenoma extending into the sphenoid sinus and left cavernous sinus, encasing the left internal carotid artery with scattered hemorrhagic foci. He was discharged on cabergoline 0.25mg twice weekly for hyperprolactinemia of 7640 ng/mL (N 2.64-13.13 ng/mL). Four weeks later, he was readmitted for altered sensorium, clear rhinorrhea and positional headache. Work-up showed a prolactin of 204 ng/mL confirmed on dilution testing, and a random cortisol of 8.3 mcg/dL. MRI Brain revealed mass involution measuring 2.8cm with the central component replaced by air, and extensive pneumocephalus overlying bilateral cerebral hemispheres, within lateral ventricles and basal cisterns. Further DA therapy was held, and the patient was started on stress dose steroids. He underwent emergent surgical repair of the CSF leak, partial tumor resection and lumbar drain placement. Pathology confirmed pituitary adenoma staining positive for prolactin. Two weeks later, prolactin was 7157 ng/mL. Subsequent attempts to restart DA therapy was complicated by recurrent CSF leaks requiring two additional surgical repairs. After a complicated hospital course requiring prolonged intubation, tracheostomy and PEG tube placement, he was discharged to an acute rehabilitation center on low dose bromocriptine 2.5mg daily as well as maintenance hydrocortisone 10mg twice daily. *Discussion* CSF leak with pneumocephalus is a rare complication of DA therapy for invasive macroprolactinomas. It occurs due to disruption of the dura with an osseous defect of the skull base. Rapid volume reduction by DA leads to exposure of previously created pathologic opening in the skull base originally plugged by tumor itself until then. Out of 60 patients from 1980 to 2017 who developed DA therapy-induced CSF leak, more than half (57%) were on bromocriptine. Median initial prolactin was 5460 ng/ml and median time from therapy initiation to presence of rhinorrhea was 6 weeks, although it can occur as late as 2 years. The recommended definitive management of DA-induced rhinorrhea is surgical repair. Subsequently, there is no consensus on how to restart DA post-repair. *Conclusion* This case illustrates the importance of watchful monitoring of response after DA therapy initiation in invasive macroprolactinomas. Although data is sparse, there may be benefit in lower and less frequent dosing initially. Patient education regarding the risk of complications and signs/symptoms to watch out for with DA therapy is also crucial.

Neuroendocrinology and Pituitary
PITUITARY TUMORS II

Thyrotropinoma and Pregnancy.

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MON-LB46

Thyrotropinomas (TSHomas) are rare pituitary tumours, comprising 1-2% of all pituitary adenomas. Thyrotropinomas in pregnancy are exceedingly rare and management of these in pregnancy can be challenging due to the potential for maternal and foetal harm. We report the case of a 35 year old woman who was found to have a pituitary macroadenoma on imaging whilst being evaluated for headaches and sinusitis. She had felt more stressed than usual but no other overt thyrotoxic symptoms. There were no visual field abnormalities or symptoms to suggest other endocrine hypo or hypersecretion. Pituitary MRI revealed a macroadenoma and biochemistry demonstrated raised free T4 24 pmol/L and free T3 6.8 pmol/L and inappropriately elevated TSH of 4.2 mIU/L, in keeping with secondary hyperthyroidism. She was scheduled for transsphenoidal (TSA) pituitary surgery, however on review she had naturally fallen pregnant. After a multi-disciplinary discussion, it was decided that surgery should be deferred and close observation be undertaken under the care of a multidisciplinary team. During the first half of pregnancy she suffered hyperemesis gravidarum with ongoing thyrotoxicosis but declined carbimazole. Her visual fields were normal throughout pregnancy. She delivered vaginally at 38 weeks, weight 3.395kg and had no malformations. Post birth was complicated by post-partum

haemorrhage requiring multiple blood transfusions and intensive care. One week later after recovering, she was able to commence breastfeeding. She went on to TSA at 6months post partum with complete tumour resection. This case demonstrates the complexity of managing TSHomas in pregnancy and the potential cross reactivity of the early hCG rises with the already elevated TSH levels, likely exacerbating her hyperemesis gravidarum.

Adrenal

ADRENAL CASE REPORTS III

Pheochromocytoma - Illusive Myriad of Symptoms

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MON-LB041

Background: Pheochromocytoma is a rare neuroendocrine neoplasm arising from the adrenal medullary chromaffin cells. It contributes to 80-85% of catecholamine secreting tumors. The annual incidence of pheochromocytoma is 0.8 cases per 100,000 person-years. It can be both sporadic or familial. The classic triad of headache, palpitations and diaphoresis is seen in only 4% of cases. Rare presentations include cardiomyopathy, stroke, diabetes mellitus, ventricular arrhythmias and myocardial infarction. Our patient presented concurrently with dilated cardiomyopathy, hypertensive emergency and new-onset diabetes mellitus (DM) followed by ischemic stroke within a week. Heart failure can present as takotsubo or dilated cardiomyopathy with an incidence of 10%. The underlying pathophysiology is catecholamine mediated myocardial stunning, diffuse coronary vasospasm, microvasculature dysfunction and fibrosis. DM is seen in 23% of pheochromocytoma and is due to catecholamine-induced impaired glucose tolerance, and insulin resistance. Cerebral ischemia has an incidence of 3%, and is secondary to severe hypertension and cerebral vasospasm. Clinical Case: A 47-year-old African American woman presented with a 1-week duration of worsening dyspnea, orthopnea, dizziness, and palpitations. Past medical history includes HTN, non-ischemic cardiomyopathy on carvedilol and pravastatin. Physical exam: BP 182/119, HR 120, mild pulmonary crackles. Labs: proBNP 3533 (1-150 pg/ml), HbA1c 13.1%. Echocardiogram: moderate global hypokinesis with left ventricle ejection fraction (LVEF) of 30-35%. On imaging, CT of chest revealed an incidental finding of 4.7 cm right adrenal adenoma. MRI abdomen confirmed heterogeneously enhancing 4.3 cm right adrenal mass. Further testing showed high plasma catecholamines of 9963 (242-1125 pg/ml), 24-hour urine catecholamines of 2125 (50-100 mcg), thus confirming the diagnosis of pheochromocytoma. Further, her clinical course was complicated by left-sided weakness. MRI brain reported an acute infarct in right corona radiata. Pre-operative blockade was done with phenoxybenzamine and metoprolol. Subsequently, she underwent right adrenalectomy and tissue diagnosis confirmed pheochromocytoma. On follow up visit, her symptoms resolved. Also noted normalized catecholamine levels, HbA1c of 5.4% and LVEF of 40- 45%. Conclusion: Pheochromocytoma can rarely present with multi-organ failure. It warrants a high index of suspicion in non-ischemic cardiomyopathy. As per recent Mayo Clinic criteria, diagnosis of takotsubo cardiomyopathy mandates ruling out pheochromocytoma. As seen in our patient, it is a reversible cause of left ventricular dysfunction, focal weakness and DM. Based on our knowledge, this is the only contingently diagnosed pheochromocytoma with varied clinical presentations. It has been aptly described as "The Great Masquerader".

Genetics and Development (including Gene Regulation)

ENDOCRINE DISRUPTING CHEMICALS

Tamoxifen Affects MiRNA Expression in Uterus and Breast Tamoxifen Affects MiRNA Expression in Uterus and Breast Tamoxifen Affects MiRNA Expression in Uterus and Breast

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SAT-LB130

In addition to genetic factors, environmental factors and lifestyle can play a significant role in the development of hormone-dependent tumors, such as endometrial cancer (EC) and breast cancer (BC). The discovery of microRNAs (miRs) involved in the post-transcriptional regulation of many genes, including those of hormonal carcinogenesis, namely, steroid receptors and their target genes, strengthened the epigenetic direction in the study of carcinogenesis mechanisms. A critical event in the development of hormone-dependent human tumors is violation in the metabolism of steroid hormones, primarily estradiol. An interesting aspect of the problem of ERα inhibition is the use of tamoxifen (TAM) in clinical practice in the treatment of hormone-dependent BC. A well-known side effect of TAM is increased proliferation in the endometrium and an elevated risk of EC. One of the mechanisms explaining such differences in the effects of TAM is formation of DNA adducts in endometrial cells, but this mechanism has not yet been substantiated. Therefore, the problem of carcinogenesis of the uterus with this drug remains unresolved and requires further research. The aim of our study was to evaluate the expression of miRs and target genes for hormonal carcinogenesis in the uterus and mammary gland under the exposure with TAM. As an object of study, we used female rats, primary human cell cultures and tissues of TAM-induced human endometrial hyperplasia. The results showed that estradiol enhances the expression of oncogenic microRNAs miR-21-, 221, -222 by three-ten times, both in the rat mammary gland and endometrium, which confirms its oncogenic properties. In the rat endometrium, TAM, to a greater extent than estradiol, increased the expression