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