arranged to look for conditions associated with pituitary hyperplasia resulting in GHRH production, including the genetic tests for inherited conditions. Her calcitonin level was normal. She had an ultrasound scan guided fine needle aspiration of the thyroid nodule, this showed Thy3f oncocytic nodule with no features of medullary thyroid carcinoma. She had an NMGa68DOTATATE whole body PET CTand the scan showed a large DOTATAE avid mass from the right adrenal gland compatible with Pheochromocytoma. Her 24 hours total urinary metadrenaline and normetadrenalin was high. Her genetic test for MEN1, CDKN1B, and MEN2 are negative. We have requested the GHRH measurement. After a pituitary surgery, her GH suppressed adequately on OGTT. Her IGF-1 and prolactin is low and her hypogonadism is resolved. **Conclusion:** Excess production GHRH can result from neuroendocrine tumors of the lung, pancreas, thyroid (medullary thyroid cancer), or pheochromocytomas and hypothalamic gangliocytomas.
Several familial syndromes, multiple endocrine neoplasia type 1 (MEN1) and 4 (MEN4), familial isolated pituitary adenoma (FIPA), Carney complex, and sporadic germline mosaic disorder McCune-Albright disease predispose to pituitary hyperplasia. GHRH acromegaly should be suspected in a patient with biochemical/clinical features of acromegaly in the presence of co-existing neuroendocrine tumors when there is diffuse pituitary enlargement on imaging or resolution of acromegaly after the surgical resection of the primary neuroendocrine tumour or persistent acromegaly after surgery if there is histological evidence of somatotroph hyperplasia. **References:** Akirov A, ASA SL, AMER L Shimon I and Ezzat S. The clinicopathological spectrum of acromegaly**. J. Clin. Med.** 8(11), 1962 (2019) *J. Clin. Med.* 2019; *8:* 11, 1962

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Acute Ischaemic Stroke With Hyperprolactinemia:A Case Report

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Background: Macroincidentalomas were reported in 0.2% of patient underwent imaging (CT scans) for central nervous symptoms (1). In acute ischaemic stroke with hyperprolactinemia, the diagnosis of a double pathology of ischemic stroke and sellar tumour especially prolactinoma need to be considered. Hyperprolactinemia itself may be considered as a risk factor for ischemic stroke due to its thrombogenic effect (3).
Clinical Case: A 47-year old man underlying hypertension and diabetes mellitus for 5 years presented with sudden onset of right sided body weakness associated with facial asymmetry and aphasia. No history of fever or trauma. Asymptomatic of hyperprolactinemia previously. On general examinations Glasgow Coma Scale 11/15 Eye 4 Verbal 1 Motor 6, blood pressure was unstable with readings of systolic 244mmHg and diastolic 142mmHg. Neurological examinations showed expressive aphasia, right hemianopia, right facial nerve palsy and absence of gag reflex. Cerebellar signs were negative. Motor function examinations of right upper and lower limbs showed hypertonia, reduce power of 2/5, normal reflexes and up going plantar response. Sensory functions of right upper and lower limbs were reduced. Clinically diagnosed as stroke with hypertensive emergency. CT brain showed multiple hypodensities due to recent infarct and incidental finding of an aggressive sellar mass. MRI brain showed left Middle Cerebral Artery territory infarct and an aggressive sphenoid sinus mass with suprasellar and bilateral cavernous sinus extension possibility of a macroadenoma. Serum prolactin level showed markedly hyperprolactinemia (21146 ng/ml, n 4.04 – 15.2 ng/ml) which level of 500ng/ml or greater is diagnostic of a macroprolactinoma (2). FSH level (0.929 IU/L, n 1.5-12.4 IU/L) and LH level (1.11 IU/L, n 1.7-8.6 IU/L) were low in this patient due to suppression of GnRH secretion from hypothalamus by prolactin. Testosterone level (0.15 nmol/L, n 8.64-29.0 nmol/L) was low secondary to low LH. Serum cortisol, growth hormone and TSH were normal. Platelet count and coagulation profiles were normal. The patient was treated conservatively in ward for acute ischaemic stroke and later was started on dopamine agonist cabergoline for hyperprolactinemia.
Conclusion: This is a case report of acute ischaemic stroke with markedly hyperprolactinemia secondary to incidentaloma macroprolactinoma.
Reference: (1)Freda PU, Beckers AM, Katznelson L, et al. Pituitary incidentaloma: an endocrine society clinical practice guideline. J Clin Endocrinol Metab. 2011; 96 (4): 894-904.(2)Abha Majumdar and Nisha Sharma Mangal. Hyperprolactinemia. J Hum Reprod Sci. 2013 Jul-Sep; 6(3): 168–175.(3)Sankalp Kumar Tripathi, Pallavi Kamble, M.G. Muddeshwar. Serum Prolactin Level in Patients of Ischemic stroke. International Journal of Contemporary Medical Research 2016; 3(12): 3459-3460.

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Acute Myeloid Leukemia Leading to Central Diabetes Insipidus

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Background: Central diabetes insipidus (CDI) as a complication of acute myeloid leukemia (AML) is rare, occurring in less than 0.6% of AML cases. The mechanism is thought to involve leukemic infiltration in or around the pituitary gland, not always seen on imaging. In one study, as many as 61.4% of patients with CDI due to AML had no abnormalities on MRI, and at autopsy 46% of AML patients had perihypohyseal leukemic infiltration in the absence of overt CDI. CDI is also associated with AML in cases that involve monosomy 7 and inversion 3q21q26, both of which result in ectopic viral integration site 1 (EVI-1) overexpression. It is postulated that EVI-1 overexpression interferes with hypothalamic secretion of antidiuretic hormone (ADH) or may lead to its inactivation.

We present a case of adipsic CDI due to AML in a patient with monosomy 7.

Case: A 70-year-old female presented for routine follow-up and was found to have a white blood cell count of 2.6 K/μL with 29% blasts, anemia (Hgb 10.3 g/dL) and normal platelets (300 K/μL). She was diagnosed with AML and molecular evaluation showed del (3)(q21),-7,add(17(p13) consistent with monosomy 7. She was admitted for induction chemotherapy with cytarabine, daunorubicin and intrathecal methotrexate. She denied thirst. On physical exam she was euvolemic and visual fields were full on confrontation. Her admission sodium was 146 mmol/L, urine osmolality was 149 mOsm/kg H_2O, urine sodium 14 mmol/L. Urine output was 5.1 L over the first 24 hours. She underwent a 6 hour water deprivation test, during which her urine output averaged 250 cc/hr. Her sodium increased to 158 mmol/L, serum osmolality 331 mOsm/kg H_2O, urine osmolality 146 mOsm/kg H_2O. She was then administered 100 μg of DDAVP PO and her serum sodium and osmolality decreased to 155 mmol/L and 326 mOsm/kg H_2O, respectively, while her urine osmolality nearly doubled to 292 mOsm/kg H_2O. Urine output decreased to 50-100 cc/hr. At no point during her testing did she report thirst. The patient's pituitary laboratory profile did not show any other abnormalities. Her pituitary MRI revealed subtle thickening of the proximal infundibulum and hypothalamus but no definitive intra-sellar pathology. She was discharged on twice daily DDAVP with a sodium of 142. Unfortunately, her AML was refractory to treatment. She was transitioned to comfort care and died peacefully.

Conclusion: CDI as a complication of AML is very rare and is a poor prognostic marker. Based on her MRI findings, the most likely mechanism in this case was infundibular/hypothalamic infiltration. Her adypsia is interesting and may point to more generalized hypothalamic involvement including thirst center. Her monosomy 7 mutation may have also played a role. We present this case to bring awareness to this etiology of DI and its proposed mechanisms.

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Acute Transient Central Diabetes Insipidus: A Rare Case of DI Secondary to Vasopressin Withdrawal

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Introduction: Transient Central Diabetes Insipidus (tCDI) induced by vasopressin withdrawal is a rare condition which is possibly under recognized. It occurs in very sick, often critically ill patients and usually complicates an already complex clinical picture, so early recognition and treatment are critical to reduce morbidity.

Case Description: A 47-y/o male with a PMH of Coffin Lowry syndrome, atrial fibrillation, and GERD presented with abdominal pain and flu like symptoms. His home medications were metoprolol, loratadine and colestipol. Work up revealed bowel perforation for which he was taken to the OR for repair. Intraoperatively he developed septic shock requiring pressor support with norepinephrine and vasopressin. He was weaned off norepinephrine on post-op day (POD)1, and vasopressin on POD 2. Approximately three hours after withdrawal of vasopressin support his urine output increased dramatically up to a peak of 350 cc/hour with a recorded 24hr urine volume of >5L. Concurrently, his serum sodium was found to have increased from 147 mmol/L to 173 mmol/L (n 135-145) over the course of 13 hours. Clinically, he became increasingly lethargic with abnormal eye movements. His sodium did not improve with fluid management with D5W. His other laboratory values included a urine osmolality of 141 mOsm/kg, urine sodium of 60 mmol/L and a peak serum sodium of 177mmol/L. He was administered 1mcg desmopressin and his D5W rate was increased. His urine output dropped gradually to ~150cc/hr, his serum sodium level started to trend down to 168 mmol/L and his urine osmolality increased to 439 mOsm/kg five hours after desmopressin administration, with improvement in mental status. The patient received a total of two doses of desmopressin and continued support with IV fluids. His sodium eventually normalized, and his polyuria did not return.

Discussion: This patient's clinical picture is consistent with tCDI secondary to discontinuation of vasopressin. Transient Central diabetes insipidus due to vasopressin withdrawal is a phenomenon that is not well understood, but there is a strong male preponderance, and it tends to occur more commonly in patients with underlying neurological conditions, as did our patient. Current proposed mechanisms include decreased production and release of exogenous ADH due to negative feedback, down regulation of the V2 receptors, and hypoperfusion to the posterior pituitary. This condition deserves more investigation to better understand the incidence, risk factors and pathophysiological mechanisms.

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An Atypical Case of TSH-Secreting Pituitary Adenoma in a Patient Presenting With Low Libido and Insomnia

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Background: TSH-secreting pituitary adenoma is an incredibly rare cause of hyperthyroidism. Most patients with TSHomas present with clinical hyperthyroidism, but some patients may have atypical signs. Here we present the case of a patient who had been suffering from low libido and insomnia who was found to have secondary hyperthyroidism from a TSH-secreting pituitary adenoma.

Clinical Case: A 47-year-old man presented with complaints of progressive headache, fatigue, depression, insomnia, low libido and erectile dysfunction. After questioning, he noted the presence of intentional tremor for more than 5 years. He denied anxiety, palpitations, heat intolerance, hyper defecation, proximal muscle weakness, local compressive symptoms of the neck and ocular symptoms. Physical exam was notable for fine tremor and mild thyromegaly, but otherwise patient had no evidence of anxiety, exophthalmos, hyperreflexia or diaphoresis. The patient was found to have a normal free testosterone (46