Clinical Case: A 48 year old male with history of chronic hyponatremia of unknown cause, fatty liver, hypertension, was in the hospital post operatively after resection of a meningioma along dura. Endocrine was consulted for management of his chronic hyponatremia. Had chronic hyponatremia for over 20 years and was always asymptomatic. Normally drank 6-7 L of water at home, mostly at night. Also found to have a spinal compression fracture of unknown cause. Both his father and brother had chronic hyponatremia of unknown cause as well, suggesting possible familial component. His baseline sodium levels were 129-133 mmol/L. In the hospital, serum sodium levels decreased to the 120s. TSH was 0.307mcunit/mL (0.27-4.2). Was also placed on 1.5 L fluid restriction. Urine osmolality was 900 mOsm/kg (500-800) with sodium of 123 mmol/L (136-145), consistent with SIADH. A rare inherited disorder, nephrogenic SIADH (NSIADH), was considered. However, it has an X-linked inheritance pattern. Fluid restriction was removed, then did fluid load with 2L of water and obtained urine sodium, serum sodium, urine osmolality, serum osmolality, Copeptin (pro-AVP) before fluid load and 1 hour after fluid load. Serum sodium level went from 127mmol/L before to 125 mmol/L after. Urine osmolality improved from 984 mOsm/kg prior to 575 mOsm/kg after. Urine sodium went from 183 mmol/L prior to 91 mmol/L after. Serum osmolality went from 278 mOsm/kg (270-310) to 268 mOsm/kg after. His co-peptin pro-AVP levels were 16.4 pmol/L (ref. <13.1). They are found to be low in NSIADH. It was decided that his chronic hyponatremia was likely due to reset osmostat. After discharge and follow up, his serum sodium was rechecked and was 128 mmol/L. It would have been challenging, but useful, to try a vaptan for diagnostic purposes and possibly to increase serum sodium. However, there are complications from overcorrection. Since patient had long standing asymptomatic chronic hyponatremia with family history, it was decided not to pursue aggressive measures just to "normalize" serum sodium. Otherwise, it would have been an example of treating the numbers and not the patient.

Conclusions: Case demonstrates the importance of keeping the patient, their symptoms, and clinical picture in mind, and to not just follow numbers, as difficult as it may be, especially when managing conditions in which diagnosis may be uncertain or unclear. Sometimes no intervention is needed at all, however tempting it may be to do one, it is important to keep the former option in mind. An asymptomatic patient with longstanding chronic hyponatremia due to reset osmostat is an example of that.

Neuroendocrinology and Pituitary

NEUROENDOCRINOLOGY AND PITUITARY CASE REPORTS

Change in Character of Headache in a Young Female Patient With Prolactin Secreting Pituitary Adenoma Complicated With Mild Hemorrhage

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Background: Prolactin (PRL) secreting tumors are the most common functional neoplasms of the pituitary and are commonly subdivided into microprolactinomas (<10 mm) and macroprolactinomas (≥10 mm) according to their baseline diameter. The diagnosis of macroadenomas is usually straightforward and these large tumors may be associated with mass effects such as severe headache, nerve palsies or visual changes. Hyperprolactinemia can be due to other causes like pregnancy, drugs, hypothyroidism, and pituitary stalk effect should be considered in the differential.

Clinical Case: A 30 year female having symptoms of prolactinoma showing amenorrhea of 3 years, galactorrhea, decrease libido who presented to the clinic for headache recently associated with anxiety. Headache initially frequently with 2-3 times in a week, moderate intensity lasting few hours, on one side of head and sometimes on the forehead, associated with blurry vision. Patient was on oral contraception taking on and off, but not on any medication currently. Initial work up consistent with hyperprolactinemia: elevated prolactin level 737.6 ng/ml (n 3.0-30 ng/ml), however other pituitary hormones are within normal limit, ACTH 32 pg/ml (n 6-50 pg/ml),FSH 3.2 mid/ml, LH 0.3miu/ml, total testosterone 13 ng/dl (n 2-35 ng/dl),TSH 1.6 mid/l (N 0.35-3.7 mid/l), FT4 0.92 ng/dl (N 0.76-1.46 ng/dl), human growth hormone <0.1ng/ml (N <=7.1 ng/dl), random cortisol 14.6 ug/dl (N 4.3-22.4 ug/dl). Pregnancy test is also negative. MRI Brain with and without contrast revealed a mass within the pituitary fossa producing smooth depression of the floor and suprasellar extension just the right of midline producing mild impingement on the undersurface of the optic chiasm, craniocaudal dimension of the tumor approximately 1.85 cm by 1.8 cm mediolateral by 1.4 cm anteroposterior without encasement or diminution of carotid arteries flow. Within the sella there is a spherical collection of T1 high signal displaying a fluid level strongly concerning for hemorrhage within the pre-existing tumor. There is no prior CT Head or MRI Brain available. Ophthalmology evaluation showed normal dilated retinal exam and no visual field defects. Medical treatment with Cabergoline 0.25 mg PO twice weekly was given on the visit, however patient did not start it yet and repeat prolactin level with 629 ng/ml. Patient endorsed that headache is better with less frequent about once in two weeks but still with absence of menstruation.

Conclusion: This is the case of prolactin secreting pituitary macroadenoma with the change in character of headache due to hemorrhage in the tumor, having gradual improvement of headache which may be due to spontaneous resolving of hemorrhage from the pre-existing pituitary mass. It is very important to inform the patient with pituitary tumor go to ED if there is change in headache or vision.

Neuroendocrinology and Pituitary

NEUROENDOCRINOLOGY AND PITUITARY CASE REPORTS

Clinically Functioning FSH-Secreting Pituitary Adenoma

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Background: Gonadotroph adenomas are the most common type of non-functioning pituitary adenomas.

However functioning gonadotroph adenomas are rare with only a few cases reported in literature.
Clinical Case: A 42-year old man was admitted as a case of COVID-19 when an incidental finding of a pituitary gland mass was detected. Upon evaluation, the patient was found to have blurred vision and dizziness since few years, however no history of headache, seizures, visual field deficit were reported, nor clear symptoms related to anterior pituitary hypo- or hyperfunction. Pituitary MRI revealed a pituitary gland mass measuring 30x23x22 mm expanding the pituitary fossa and extending to suprasellar cistern. Initial laboratory investigations revealed low free T3 (1.8 pmol/L, normal: 3.1-6.8 pmol/L) and low free T4 (6.6 pmol/L, normal: 12-22 pmol/L) with normal TSH (3.54 mIU/L, normal: 0.27-4.2 mIU/L), high FSH (31.4 IU/L, normal: 1-18 IU/L), and high total testosterone (50.31 nmol/L, normal 8.3-32.9 nmol/L). Testicular ultrasound showed bilateral enlarged testicles (right testis 38.14 cm^3 and left testis 33.38 cm^3). Visual Field testing revealed monocular superior temporal defect affecting only the left eye. Patient was treated medically with levothyroxine and hydrocortisone and surgically by minimally invasive transsphenoidal resection of pituitary adenoma. Histopathology assessment confirmed the diagnosis of gonadotroph secreting adenoma, with positive immunostain for FSH. At 6 weeks postoperatively, insulin tolerance test was performed after which hydrocortisone was only prescribed during periods of stress. Anterior pituitary profile was repeated which revealed normalization of free T4 (14.3 pmol/L) and FSH (1.2 IU/L) and decrease in testosterone (0.66 nmol/L). Assessment of visual field revealed marked improvement of previous findings. Pituitary MRI was also repeated with findings of enhancing soft tissue remnants. At 12 weeks postoperatively, anterior pituitary profile revealed normalization of testosterone (11.3 nmol/L). Testicular ultrasound was repeated and showed a marked decrease in testicular volume (right testis 17.93 cm^3 and left testis 21.13 cm^3).
Conclusion: Functional gonadotroph adenomas are a rare subtype of pituitary adenomas which pose a diagnostic challenge and require meticulous clinical evaluation and multidisciplinary therapeutic approach.

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NEUROENDOCRINOLOGY AND PITUITARY CASE REPORTS

Co Existence of a Corticotroph Adenoma and a Multifunctional Pituitary Cyst

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The co-existence of a corticotroph adenoma and a pituitary cyst is very unusual. We present the case of a 50-year-old female who presented with a Cushingoid phenotype, severe hypokalaemia, hyperglycaemia and hypertension. Urinary free cortisol was markedly elevated at 50 fold. ACTH levels were elevated at 121.4 pg/ml. She failed both the low and high dose dexamethasone suppression tests. The CRF test did not show a satisfactory rise in ACTH levels but the Inferior petrosal sinus sampling revealed a central to peripheral ACTH gradient highly suggestive of Pituitary dependant Cushing's. MRI pituitary revealed a large cystic lesion with a small solid component. Computed Tomography (CT) of thorax abdomen and pelvis was normal. A trans-sphenoidal hypophysectomy was performed, during which a fine needle aspiration of the intra-cystic fluid was obtained. This showed markedly elevated pituitary hormone levels of ACTH (1399pg/ml), prolactin (353,084mIU/L), TSH (217IU/L) FSH (205mIU/ml) and GH (519 ng/ml) consistent with a multifunctional pituitary cyst. Neuropathology of the solid component confirmed a corticotroph adenoma. Post-operative am cortisol levels were persistently suppressed to less than 50 nmol/l with marked improvement in clinical features. This case highlights the challenges in the work up of Cushing's syndrome and the limitations of diagnostic tests. The coexistence of a corticotroph adenoma and a multifunctional pituitary cyst is very unusual and to our knowledge has not been reported before.

Neuroendocrinology and Pituitary

NEUROENDOCRINOLOGY AND PITUITARY CASE REPORTS

Corticosteroids Replacement in a Patient With Panhypopituitarism and COVID-19 Infection

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Background: Hypopituitarism refers to complete or partial insufficiency of pituitary hormone secretion and patients require lifelong hormone replacement. Those with ACTH deficiency rely on exogenous glucocorticoids and at times of intercurrent illness require stress doses to prevent an adrenal crisis. The benefits and adverse effects of corticosteroids for treatment of COVID-19 pneumonia are currently under investigation. We report our experience in a patient with COVID-19 pneumonia who received high dose corticosteroids for panhypopituitarism.
Clinical Case: A 51-year-old man presented with one-week history of fever and generalized weakness. He had been diagnosed with a non-functional pituitary macroadenoma causing panhypopituitarism 1 year ago when he developed generalized tonic-clonic seizures. He underwent trans-sphenoidal resection of the pituitary adenoma. However, he then discontinued his hormonal therapy and was lost to follow up. He had postural hypotension but was not tachypneic or hypoxemic. He tested positive for COVID-19 and chest x-ray showed prominent bilateral bronchovascular markings; he was hospitalized as mild COVID-19 pneumonia. Laboratory investigations revealed secondary adrenal insufficiency, secondary hypothyroidism and hypogonadotropic hypogonadism. MRI of the pituitary now showed persistence of the pituitary macroadenoma, measuring 3.5 x 3.7 x 2.4 cm in dimensions, causing sellar obliteration and left cavernous sinus invasion. Treatment with stress dose steroids, Hydrocortisone 50 mg 4 times daily was initiated, followed by thyroid hormone replacement