with Cabergoline, uncontrolled diabetes was managed and was referred to Neurosurgery service for further evaluation and tumor removal. Based on current literature, the incidence of acromegaly cases is low, more specifically when presenting with new onset diabetic ketoacidosis, insulin resistance and secondary to functioning macroadenomas. Medical awareness should be promoted to assess for careful consideration of signs and symptoms, workup, management and treatment to assess and minimize further health complications and physical burdens acromegaly and pituitary adenomas could pose for affected individuals.

Neuroendocrinology and Pituitary

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Discordance Between Prolactinoma Size and Prolactin Level: An Unusual Laboratory Finding

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Background: Prolactinomas are a common cause of hyperprolactinemia. Prolactin (PRL) level higher than 250 mcg/L is associated with a prolactinoma and serum prolactin levels generally correlate with tumor size. It is unusual to find a PRL level that is markedly elevated out of proportion to prolactinoma size. We present the case of a 32-year-old man who was referred to Endocrinology Clinic with fatigue and low testosterone, found to have a PRL level of 1302 mcg/L with a 9 x 8 x 9 mm microprolactinoma. **Clinical Case:** A 32-year-old man with past medical history of migraines reported fatigue, weight gain, low libido and erectile dysfunction and was referred to Endocrinology Clinic due to PRL elevation. His medications included a multivitamin and chasteberry herbal supplement. Physical exam was unremarkable and no visual field abnormalities were detected. Baseline lab results showed PRL: >1000 mcg/L and 1302 mcg/L after serial dilutions, FSH: 1.8 mIU/mL [0.7- 10.8 mIU/ml], LH:0.9 L [1.2- 10.6 mIU/ml], total testosterone 136 ng/dL [250-1100 ng/dl], free testosterone: 32 [35- 155 pg/ml], with normal cortisol, ACTH, IGF-1, TSH and Free T4 levels. MRI pituitary revealed a 9 mm x 8 mm x 9 mm microadenoma on the right side of the pituitary gland without optic chiasm compression. He was diagnosed with microprolactinoma, with very high PRL level causing secondary hypogonadism. Cabergoline 0.25 mg twice weekly resulted in significant improvement in PRL level. With dose increment to 0.5 mg twice weekly, PRL level improved further along with improvement in symptoms related to hypogonadism. Five months after initiation of treatment, total PRL was 58 ng/mL with monomeric PRL of 41 mcg/L, indicating only trace contribution of macroprolactin to the total PRL level. **Review of the Literature:** In a retrospective study by Colao et al. (2003), men with hyperprolactinemia due to micro-prolactinoma had average pre-treatment PRL levels of 187.7 mcg/L (SD 51.8 mcg/L) and average pre-treatment diameter of 8.0 mm (SD 1.4 mm). A retrospective study of 1234 patients by Vilar et al. (2008) reported similar findings: microprolactinomas had average baseline PRL of 165.6 mcg/L (SD 255.1 mcg/L) and the highest reported level of PRL due to microprolactinoma was 525 mcg/L. **Conclusion:** Our case illustrates that assumptions about prolactinoma size should not be made based on laboratory findings alone. **References:** Melmed S. et al, Diagnosis and Treatment of Hyperprolactinemia: An Endocrine Society Clinical Practice Guideline. *J Clin Endocrinol Metab*. 96: 273-288, 2011.Colao A. et al. Gender differences in the prevalence, clinical features and response to cabergoline in hyperprolactinemia. *European Journal of Endocrinology* 148: 325-331.Vilar L. et al. Diagnosis and Management of Hyperprolactinemia: Results of a Brazilian Multicenter Study with 1234 Patients. *J Endocrinol Invest* 31:436-444.

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NEUROENDOCRINOLOGY AND PITUITARY CASE REPORTS

Diverse Manifestation of Acromegaly With Suspicion of Ectopic GHRH Secretion. Report of Two Cases

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Introduction: Acromegaly can rarely be caused by an ectopic production of GH or GHRH by various neoplasms, most commonly neuroendocrine tumors of pancreatic or pulmonary origin. Squamous cell carcinoma of the lung has not been associated with ectopic GHRH secretion yet. The authors present two cases of acromegaly with suspicion of GHRH ectopy. **Presentation of the Cases:** Case 1. A male born in 1945 presenting with typical morphologic features of acromegaly. Pituitary MRI revealed a cuneatic-shaped, hypointense focal lesion (8x7x6 mm) in the right posterior part of the anterior pituitary lobe, radiologically interpreted as either hyperplasia of the intermediate lobe or atypical adenoma in a normal-sized pituitary. IGF-1 and no suppression of GH secretion after oral glucose load were observed. Furthermore, a polycyclic tumor in the 2nd segment of the right lung with pathological metabolic activity in 18FDG- PET/CT was discovered. Ga^{68}- DOTA TATE PET/CT revealed no pathological accumulation of the tracer. After upper right lobectomy, squamous cell carcinoma, non-keratinizing, G3, with a negative immunohistochemical reaction for GH was confirmed. IHC for GHRH and serum GHRH have been scheduled. After surgery and chemotherapy, no biochemical remission of acromegaly was observed and the pituitary MRI showed stable radiological image of the pituitary tumor, suggesting rather a possible

metastasis to the pituitary. Due to unfavorable prognosis, the patient was disqualified from neurosurgical resection of the pituitary tumor. Case 2. a male born in 1948 with typical symptoms of acromegaly, elevation of IGF-1 and no suppression of GH in OGTT. Due to MRI contraindications, only CT of the head was performed- it revealed partially empty sella, compressed pituitary with maximal diameter 3 mm and no focal lesions. Treatment with somatostatin analogue was introduced, however, only partial biochemical control was achieved. Ga^{68}- DOTA TATE PET/CT performed after 8 years showed pathological expression of somatostatin receptors in the pancreatic tail. Abdominal CT confirmed a focal lesion in this location, 14x9 mm, with a strong enhancement after contrast administration, suggesting a neuroendocrine tumor. The patient refused to undergo any invasive procedures and remains treated with SRL. Serum GHRH has been scheduled. **Conclusion:** The authors report two cases of ectopic acromegaly suspicion, with an ambiguous clinical and radiological presentation. In unclear cases of acromegaly, ectopic production of GHRH should be taken into consideration.

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Does Pituitary Dysfunction Always Occur Following Penetrating Head Trauma?

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Background: Pituitary dysfunction and panhypopituitarism remain underdiagnosed in penetrating and blunt head trauma and can occur in both acute and chronic settings. **Case:** A 56 years old male with no significant PMH was admitted with a gunshot wound to the left T9 rib paraspinally with bullet deflection cranially along the left lung, left sternocleidomastoid, and resting anterior to the suprasellar cistern just above the midline of the sphenoid sinus close to the pituitary gland. Moderate volume pneumocephalus, chest hemopneumothorax, and sudden loss of right-sided vision required neurosurgical and pulmonary intervention. Endocrinology was consulted to evaluate pituitary function in the context of the bullet within the cranium. Physical examination showed intact mental status, non-focal exam, right-sided blindness, and foley catheter with normal urine output. Laboratory hormonal assessment for hypothalamic-pituitary axis (HPA) was performed consistent with normal sodium, potassium, FSH of 14.2 mIU/ml (1.0-13.0 mIU/ml), LH of 4.5 mIU/ml (1.0-9.0 mIU/ml), AM cortisol of 13.6 ug/dl (5-25 ug/dl), free cortisol of 2.06, ACTH of 10 pg/ml (10-60 pg/ml), IGF-1 of 80 ng/ml (78-220 ng/ml), TSH 1.93 mIU/L (0.5-5.0 mIU/L), FT4 1.05 ng/dl (0.8-1.8 ng/dl), Prolactin of 14.2 ng/ml (4-23 ng/ml) and HbA1c 5.1%. He reported no symptoms of adrenal insufficiency and remained hemodynamically stable. He was monitored for symptoms of pituitary insufficiency and suppression of the HPA axis along with urine output which remained normal and reassuring for the absence of central DI. The patient will continue outpatient endocrine surveillance. **Discussion:** The development of hormone deficiencies is directly related to the severity of head trauma. Mild traumatic brain injury (TBI) patients discharged from the ED, without loss of consciousness or post-traumatic amnesia less than 30 minutes do not require endocrine surveillance. Pituitary dysfunction occurs in 20-40% of patients with moderate to severe TBI. Pituitary ischemia leads to pituitary injury, due to changes in cerebral blood flow, cerebral hypoxia, and increased intracranial pressure. Compressive effects on the stalk from increased intracranial pressure is another indirect mechanism for pituitary dysfunction. Hospitalizations longer than 48 hours following TBI, require pituitary screening at 3-6 months. Chronic hypopituitarism develops in 15-20% of patients within 2-3 years with ACTH and GH deficiencies. Other changes in LH, FSH, TSH, and development of central diabetes insipidus can occur. However, despite severe TBI, acute pituitary hormonal involvement may not always occur, as in our patient. **References:** Tan CL, Alavi SA, Baldeweg SE, et al. The screening and management of pituitary dysfunction following traumatic brain injury in adults: British Neurotrauma Group guidance. J Neurol Neurosurg Psychiatry. 2017 Nov;88(11):971-981.

Neuroendocrinology and Pituitary

NEUROENDOCRINOLOGY AND PITUITARY CASE REPORTS

Giant Pituitary Adenoma Presenting With Disinhibition: A Case Report of a 25-Year-Old Male

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Background: Giant growth hormone secreting pituitary adenomas (defined by a diameter >4cm) are rare and difficult to treat (1). These typically invade surrounding structures, making surgery challenging or impossible (1). This report highlights a giant mammosomatotrophinoma with an unusual clinical presentation. **Clinical Case:** A 25-year-old male presented with personality changes, disinhibition and executive dysfunction progressing over a 3 to 4-year period. Further enquiry elicited a history of increasing headaches and significant visual loss, on the background of unilateral childhood visual loss in the contralateral eye. He was noted to have clinical features of acromegaly and complete temporal visual field loss in the left eye. On initial testing plasma IGF-1 was 137 nmol/L (reference interval [RI] 13-43nmol/L), GH >100 ug/L and prolactin 23,900 mIU/L (RI 0-400mIU/L). GH remained >100ug/L at 120 minutes after a 75g oral glucose load. He has hypogonadotropic hypogonadism, however thyrotropic and corticotropic function remained normal. MRI of the brain with contrast revealed an 8.0 x 7.1 x 7.4 cm mass arising from the pituitary fossa, extending into the suprasellar region and