displacing both frontal lobes. The mass encased the basilar and right internal carotid arteries. It was deemed inoperable due to its exceptionally large size and encasement of vascular structures. Medical treatment was initiated with cabergoline (3.5mg per week in divided doses) and octreotide LAR, initially 30mg then increased to 60mg 4-weekly. A weaning course of steroids was initiated for oedema of his optic nerve. Within one month of treatment there was improvement of clinical symptoms of headaches, sweating, irritable mood and disinhibition with associated modest biochemical improvement (reduction of IGF-1 to 129 nmol/L and prolactin reduction to 2119 mIU/L). MRI of the brain revealed a reduction in the size of the adenoma to 7.8 x 7.1 x 6.3 cm, with a reduction in the mass effect on the frontal lobes. The patient continues medical management and close clinical monitoring with the aim of ongoing tumour shrinkage to allow reassessment for possible surgical debulking. **Conclusion:** This unique case of a GH and prolactin co-secreting giant pituitary adenoma posed a therapeutic challenge due to the significant surgical risk, limiting treatment to aggressive medical therapy. The cognitive and behavioral changes experienced due to tumour size and location added to the management complexity. Although early on in the treatment course, there is improvement of symptoms and tumour size on cabergoline and octreotide LAR injections. (1) Iglesias P, Rodríguez Berrocal V, Díez JJ. Giant pituitary adenoma: histological types, clinical features and therapeutic approaches. Endocrine. 2018 Sep;61(3):407-421.

Neuroendocrinology and Pituitary

NEUROENDOCRINOLOGY AND PITUITARY CASE REPORTS

Giant Prolactinoma Causing Proptosis and Stroke

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Background: Although suprasellar and cavernous sinus invasion are common in giant prolactinomas, intra-orbital extension is extremely uncommon [1]. Even less reported are cases of giant prolactinomas causing cerebral ischemia or death.
Clinical Case: A 51-year old woman presented to the ED with confusion, right-sided weakness and severe left eye proptosis with loss of vision. Five years prior, she underwent a partial transphenoidal resection for a macroprolactinoma due to acute vision changes with compression of the optic chiasm. Prior to surgery, prolactin level was elevated to 2,106 ng/mL (n 2.4-24.0 ng/mL). Post-operative MRI showed residual 2.7 x 3.1 x 2.6 cm mass. Thereafter she was prescribed cabergoline which she self-discontinued three years later. MRI of the brain at time of presentation demonstrated a 10.1 x 6.4 x 4.3 cm sellar/suprasellar mass extending into the left orbit causing severe proptosis and mass effect on the left frontal lobe, temporal lobe, midbrain, and basilar artery with encasement of the left cavernous internal carotid artery. A recent left striatocapsular infarct due to compression of the left middle cerebral artery was present. Prolactin level was elevated to 16,487 ng/mL. Neurosurgery was consulted and recommended medical management. Free thyroxine level was low and thyroid hormone replacement was started. Although the cosyntropin stimulation test showed an appropriate cortisol level peak of 21.5 mcg/dL, she was given stress dose glucocorticoids. Bromocriptine was initially started and titrated and later changed to cabergoline. Six weeks after discharge, she was readmitted with worsening confusion and seizure activity. On day 2 of admission, she decompensated. New hemorrhage inside the mass with increased vasogenic edema and a midline shift was discovered on a head CT. She underwent emergent craniotomy with surgical debulking of the tumor. Unfortunately, her mental status did not improve post-operatively. She was transitioned to hospice care and died 7 days after surgery. Surgical pathology showed a lactotroph adenoma with markedly elevated Ki67 proliferation index of 20-30%.
Conclusion: This case demonstrates an unusually aggressive macroprolactinoma causing severe proptosis, ischemic stroke and death and adds to the very few cases previously reported [2].
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Growth Hormone (GH) and Thyroid Stimulating Hormone (TSH) Co-Secreting Pituitary Macroadenoma

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Background: TSH secreting pituitary adenomas are rare and accounts for 0.5-3% of all pituitary adenomas. Only 20-25% of those adenomas co-secrete other hormones like growth hormone or prolactin. Mixed GH and TSH secreting adenomas present with symptoms from tumor growth and features of both acromegaly and hyperthyroidism.
Clinical Case: A 57 years old woman with a past medical history of chronic joint pains and bilateral knee swelling presented to her PCP with complaints of chronic fatigue. Evaluation revealed a normal TSH level and MNG and patient did not have any further work-up. She reported undergoing periodic knee arthrocentesis which gave her only temporary pain relief. Two years later, patient presented with complaints of unintentional weight loss, tremors and palpitations. She also reported enlargement in the size of hands, feet and forehead and no improvement in the knee swelling after multiple arthrocentesis. Lab evaluation revealed high free T4 2.31ng/dl (0.58-1.64), high free