adenoma. Laboratory workup did not reveal any hormonal deficiencies. The patient was managed conservatively with close neurological follow up and empiric high dose dexamethasone. Headaches improved significantly after treatment and eventually resolved. After clinical improvement, the patient was discharged home on physiologic replacement of glucocorticoids with outpatient follow up and plans for re-evaluation of hormonal axis.

Conclusion: Oral anticoagulants can increase the risk of PA, even in the absence of a pre-existing pituitary adenoma, as other case reports have shown. Management is controversial, and although there are agents for reversal of Apixaban effects (recombinant factor Xa), their use in PA has not been described. This case was managed conservatively with excellent results. Although we cannot exclude a pre-existing pituitary adenoma in this patient, this case shows that Apixaban increases the risk of PA.

Neuroendocrinology and Pituitary

NEUROENDOCRINOLOGY AND PITUITARY CASE REPORTS

Pituitary Hyperplasia: To Operate or Not to Operate, That Is the Question

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Pituitary hyperplasia is defined as an absolute increase in the number of one or more adenohypophyseal cell subtypes, manifesting radiologically as pituitary enlargement beyond what is considered normal. It has been noted in certain physiological conditions like pregnancy however can also be seen in pathological conditions with end organ insufficiency like severe hypothyroidism.

21- year old female with a past medical history of Primary Hypothyroidism secondary to Hashimoto's thyroiditis presented initially for evaluation of worsening headache and blurry vision. She was diagnosed with hypothyroidism at 10 years of age and had an extensive family history of hypothyroidism. At the time of presentation, she was 11 months post- partum and had been on and off her levothyroxine supplementation, having stopped it completely for a few months after delivery. MRI brain showed an 18 mm homogeneously enhancing intrasellar mass with suprasellar extension. She was referred to Neurosurgery for further evaluation. Workup revealed a TSH >100 (0.27 - 4.2 mIU/L) and FT4 <0.4 (0.8 - 2 ng/dL). In the context of severe untreated hypothyroidism and MRI findings consistent with pituitary hyperplasia with abutment but no mass effect on the optic apparatus, initial plan was to treat the hypothyroidism medically and observe closely. Patient was started on levothyroxine supplementation. Her TSH improved to 3.367 (0.550 - 4.780 uIU/mL) and FT4 to 2.00 (0.89 - 1.76 ng/dL), ηοωεπερ she continued to have worsening of visual symptoms. Surgery was considered to decompress the optic nerve, but pre-operative MRI showed a significant decrease in size of the pituitary gland with decreased suprasellar bulging and no mass effect on the optic chiasm. Surgery was subsequently cancelled.

Prolonged primary hypothyroidism leads to pituitary hyperplasia due to loss of negative feedback from lack of circulating T4 and T3, leading to excessive TRH secretion from the hypothalamus. The high TRH can lead to thyrotroph as well as lactotroph hyperplasia. Subsequently patients can present with headache, vision changes along with signs and symptoms of hypothyroidism and increased prolactin secretion. It is important to differentiate hyperplasia from other sellar lesions like pituitary macroadenoma or hypophysitis. Patients with hypothyroidism, who have pituitary enlargement diagnosed on brain imaging, should be promptly diagnosed and treated with thyroid hormone replacement. With a higher frequency and improved quality of imaging techniques, we are increasingly coming across scenarios of abnormal findings on imaging. Correlation of radiographic imaging results with a thorough history and biochemical testing is essential prior to proceeding with surgical intervention.

Neuroendocrinology and Pituitary

NEUROENDOCRINOLOGY AND PITUITARY CASE REPORTS

Pituitary Hyperplasia Secondary to Primary Hypothyroidism Mimicking a Macroadenoma With Optical Chiasm Compression

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Background: A long term untreated primary hypothyroidism can stimulate thyrotropes proliferation, leading to pituitary hyperplasia. This condition is known as pituitary hyperplasia secondary to primary hypothyroidism (PHPH). It is a rare condition that mimics pituitary adenoma and can achieve large proportions with optic chiasm compression. A misdiagnosis may be catastrophic, and a pituitary resection wrongly performed. **Clinical Case:** A 25-year-old woman with a medical history of delayed neuro psychomotor development and epilepsy due to congenital CNS malformation had a Brain MRI performed for epilepsy follow-up three years earlier. At that time, neuroimaging had shown a pituitary enlargement, and laboratory data were diagnostic of severe primary hypothyroidism with TSH of 290.6 uUI/mL (normal range 0.4 to 4.5 uUI/mL) and T4L <0.23 ng/dL (normal range: 0.9 to 1.8 ng/dL). She then had received a 75 mcg levothyroxine prescription. However, the patient missed medical follow-up and returned three years later, when sella turcica MRI showed a 0.9x1.0x1,4 cm pituitary lesion, consistent with a macroadenoma with suprasellar extent near the optic chiasm. Because of the cognitive impairment, the patient was not able to complete the visual field test. Neurology service referred the patient to endocrinology evaluation for surgical treatment. Hypothyroidism was still uncontrolled with TSH 157.1 uUI/mL and T4L 0.28 ng/dL. We had adjusted the levothyroxine dose to 125 mcg and advised adherence. Subsequent thyroid function tests had shown TSH 6.91 uUI/mL and T4L 1.15 ng/dL. After thyroid function stabilization, the patient performed a new sella turcica MRI, which had not evidenced pituitary lesion. Pituitary hyperplasia secondary to primary hypothyroidism was her final diagnosis. **Conclusion:** This case report illustrates the importance of the correct diagnosis