the emergency room where he was treated for pituitary apoplexy with steroids and surgery. Vision improved the next day. Despite uncomplicated post-operative course, patient developed proximal muscle weakness and exam notable for diffuse motor deficit in bilateral lower extremities with hyperreflexia. Endocrinology workup was negative for hypercortisolism and ophthalmology diagnosed him with optic neuropathy. Neurology evaluation led to a diagnosis of multiple sclerosis (MS). Patient was started on natalizumab with complete resolution of all visual and muscle symptoms.

Clinical lesson Our patient presented with complaints of fatigue, decreased libido and work up that showed a macroprolactinoma without MRI evidence of optic chiasm impingement. During treatment, he developed acute visual deficits that were attributed to pituitary apoplexy. This visual disturbance improved after surgery and use of high dose IV steroids, with the latter likely treating what had been an MS flare. In hindsight, ophthalmologic evaluation before surgery had shown new color blindness, a sign of optic neuropathy. Despite temporary relief, patient progressed to develop new muscle weakness and recurrent visual disturbance which led to the diagnosis of MS. Since being diagnosed and treated for MS, he has had complete resolution of his symptoms. This case stresses the importance of considering other etiologies for visual defects in patients with pituitary adenomas.

Neuroendocrinology and Pituitary

CASE REPORTS IN CLASSICAL AND UNUSUAL CAUSES OF HYPOPITUITARISM II

Histologically Proven Lymphocytic Hypophysitis with Marked Improvement on Glucocorticoid Therapy

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Background: Lymphocytic hypophysitis (LH) is a rare autoimmune disorder characterized by lymphocytic infiltration of the pituitary gland. The disease predominantly affects women, with >50% of cases presenting during pregnancy or postpartum.[1] LH is often associated with other autoimmune conditions, primarily thyroiditis, and adrenalitis.[2]

Clinical case: A 27-year-old female presented with secondary amenorrhea for eight months. Workup revealed hyperprolactinemia (PRL 65 ng/mL) and a heterogenous pituitary mass measuring 3.3 cm in the largest dimension. Cabergoline was initiated for a presumed prolactinoma. Laboratory evaluation was significant for hypogonadotropic hypogonadism (estradiol <50 pg/mL, progesterone <1 ng/mL, FSH 2.9 mIU/mL, LH 0.45 mIU/mL) despite normalization in prolactin. She was also found to have Hashimoto's thyroiditis (FT4 0.7 ng/dL, TSH 8.2 uU/mL with positive TPO antibodies) and was started on levothyroxine.

Repeat imaging demonstrated a 2.4 cm heterogenous expanding sellar mass with soft tissue extension to the dorsum sella concerning for a meningioma. Visual field testing was intact without evidence of chiasmal compression. She underwent trans-sphenoidal pituitary decompression surgery which was terminated prematurely due to the presence of extensive fibrous tissue in the sella. Pathology was consistent with LH. Immunohistochemical staining was positive for lymphocytic markers CD3 and CD20, confirming marked infiltration of inflammatory B-cells and T-cells. Her postoperative course was notable for panhypopituitarism. In view of the pathological findings of LH, she was started on a high dose of 40mg of prednisone daily. Within two months, sellar magnetic resonance imaging revealed a homogenous normal-appearing pituitary with a reduction in soft tissue mass in the sellar and suprasellar region. Oral contraceptive therapy was initiated for sex hormone replacement with the resumption of menses. Prednisone was gradually tapered to 5mg/day, and she was subsequently transitioned to maintenance hydrocortisone for central adrenal insufficiency.

Discussion: LH is a rare chronic inflammatory disease that should be considered in the differential diagnosis of a non-secreting pituitary mass, especially if occurring in young women presenting during pregnancy or postpartum. The condition is associated with preferential destruction of corticotroph and thyrotroph cells.[3] Appropriate management remains controversial. High dose glucocorticoid therapy, to which our patient responded to dramatically, has been shown to be beneficial in reducing mass effect. Optimal treatment involves surgical resection of the pituitary mass to decompress surrounding structures.[3]

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Adrenal

ADRENAL CASE REPORTS I

Recurrent Co-Driver Mutation in CTNNB1-Mutant Aldosterone-producing Adenomas (APA), Causing Reversible Hypertension in Puberty, Pregnancy or Menopause

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