68Ga-DOTATATE PET-CT scan which revealed an avid 7 mm lesion (SUVmax 14.5) in the pancreatic tail suspicious for a pancreatic NET - the presumed ectopic source of ACTH. Given the patient’s poor functional status she was not a candidate for tumoral resection via distal pancreatectomy. Ketoconazole was initiated for treatment of hypercortisolism with good biochemical control, but the patient developed liver toxicity. The decision was made to pursue endoscopic ultrasound guided biopsy and radiofrequency ablation of the lesion. Pathology confirmed ACTH immunoreactive low-grade pancreatic NET. Post-procedure, morning ACTH was 8 pg/ml and cortisol was 9.1 mcg/dL, and the patient required steroids for relative adrenal insufficiency. Further follow up with repeat POMC, cortisol, ACTH levels, 24-hour UFC is required to monitor response.

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**Null Cell Adenoma With Low Ki-67 Presenting as Recurrent Pituitary Mass**

Leo Tiu, M.D. and Carolyn Montano, MD

**Background:** Pituitary adenomas account for 10% to 15% of all intracranial masses. Among these, approximately one-third are identified as nonfunctioning pituitary adenomas (NFPA) which have a heterogeneous profile and an increased potential for relapse one to five years after pituitary surgery. In a retrospective analysis by Almeida et al., multiple surgical resections, elevated ki-67 and cavernous sinus invasion were predictive of recurrence. They typically present with symptoms of mass effect and most are macroadenomas at time of diagnosis.

**Clinical Case:** A 65-year-old female initially presented with bitemporal hemianopsia, galactorrhea and amenorrhea 28 years prior. She subsequently underwent transsphenoidal hypophysectomy with resolution of symptoms post operatively. She was clinically stable until 12 years after the surgery when she noted new onset peripheral visual field loss. Upon reevaluation, she was diagnosed with non-functioning pituitary macroadenoma for which she was advised gamma knife surgery but she opted a repeat transsphenoidal pituitary surgery. Her vision improved.
and she remained asymptomatic thereafter. Sixteen years after her second surgery, she developed blurring of vision. Cranial MRI was done which showed lobulated, heterogeneous hyperdense sellar-suprasellar mass measuring 55×36×47 mm extending to the bilateral sphenoid and ethmoid sinuses, left pterygopalatine fossa, left superior orbital fissure, probably the left foramen lacerum, and left cavernous sinus with encasement of the left internal carotid artery and associated erosion of the adjacent osseous structures. Visual field testing showed mild reduction in field sensitivity with consideration of media opacity and/or uncorrected error of refraction. Baseline hormonal work up was unremarkable. Hence, endoscopic endonasal transphenoidal, transethmoidal parasellar excision of sellar mass, with reconstruction via Hadad Flap was done. There were no intraoperative nor postoperative complications. The specimen was sent for histopathology with provisional anatomic diagnosis of pituitary adenoma. Immunohistochemistry stained negative for chromogranin and any of the pituitary hormones which was consistent with null cell adenoma. The Ki-67, an independent marker of tumor progression and recurrence, was low at less than 1%. However, after 1 month, repeat MRI showed no significant change in the lobulated, heterogeneously enhancing mass centered in the sellar-suprasellar region. Thus, she received adjuvant radiotherapy with total dose of 5040 cGy divided in 28 fractions and was advised close monitoring of pituitary MRI and/or development of any new symptoms.

**Conclusion:** In this case, although with low Ki-67, the presence of multiple surgeries and high Knosp grade were recognized as risk factors for its recurrence. Treatment of recurrent NFPA is multimodal which includes re-operation, radiosurgery and radiation therapy. A multidisciplinary team approach is required for its comprehensive management and long-term follow-up.

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