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Pituitary Adenoma Presenting as Cranial Nerve III Palsy and Hemifacial Pain

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INTRODUCTION

Pituitary adenomas are common, slow growing tumors originating from the anterior pituitary. Though common, its etiology is still poorly understood. In some instances, certain genetic mutations have been deemed to be contributory, these include multiple endocrine neoplasia gene family, carney complex, and a few others. These mutations all vary, including cyclin-dependent kinase inhibitors, tumor suppressor genes, and few more.²

Functional adenomas present based on the hormone secreted i.e., prolactin, growth hormone, ACTH, and TSH with their respective clinical syndromes. Non-functioning adenomas albeit slow growing like most pituitary adenomas, may present due to their mass effect on neighboring structures.² The pituitary gland sits within the sella turcica of the sphenoid bone, near the center at the base of the cranium. It is bound anteroinferiorly by the sphenoid sinus, and laterally by the cavernous sinus. The optic chiasm lies anterosuperior to the gland. These anatomical boundaries have clinical and surgical significance, and the mass effects of a macroadenoma reflects these boundaries.⁵ (Figure **1).** Of these mass effects, an overwhelming majority of the reported symptoms were visual field defects at almost 70%, and headaches at over 40%.³ This is mostly due to a superior expansion and hence chiasmal compression. In rare instances, inferior expansion, and or cranial nerve palsies have been reported.⁴ Here, we present a case of a pituitary macroadenoma with cavernous sinus extension presenting with a pupil involving CN III palsy, as well as hemifacial pain.



Figure 1. Anatomy of the pituitary gland with surrounding structures.⁶

Pituitary Adenoma Presenting as Cranial Nerve III Palsy and Hemifacial Pain

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An 80-year-old African American female with past medical history of congestive heart failure, hypertension, cerebral vascular accident, diabetes mellitus type 2, atrial fibrillation on Eliquis, hypothyroidism and anxiety presented to the ED with complaints of double vision, inability to open her right eye, and right eye and face pain. Symptoms started abruptly a day before. Of note, the patient was incidentally found to have a pituitary adenoma following a visit to the ED a month ago after a witnessed fall. All other history was non-contributory.

On ophthalmologic examination, vision without correction was 20/70 in the right eye, and 20/40 in the left. Pupils were 4mm fixed in the right eye, and 3mm, round and reactive in the left eye. Intraocular pressure was 14 mmHg in the right eye, and 13 mmHg in the left eye. Color vision was 7/8 in the right eye, and 8/8 in the left via Ishihara. Extraocular muscle movement in the right eye revealed exotropia with 70, 80, and 50% deficits in supraduction, infraduction, and adduction respectively, the left eye was full. Confrontational visual field test revealed gross superior constriction in the right eye, and full in the left eye. Anterior exam was pertinent for a complete right sided ptosis, and bilateral 3+ NS. Posterior exam was essentially normal.

CT head without IV contrast was pertinent for a 1.6 cm (16 mm) right sellar heterogeneous soft tissue mass with sellar expansion, and suspected early extension into the right cavernous sinus (Figure 2). There was no evidence of ischemia, hemorrhage, or other masses.



Figure 2. (A: Sagittal view, B: Coronal view). CT Head without IV contrast displaying mass with extension into cavernous sinus.

CASE REPORT

Pituitary adenomas are very common, a recent study suggests pituitary adenomas to be the second most common brain tumors, another estimates 10-15% of all intracranial tumors to be pituitary adenomas.⁷ Despite the high incidence, these tumors are still poorly understood. Also as illustrated in this case, a large percentage of these tumors are found incidentally, and most of these are asymptomatic.⁸ However, a subset of these tumors can be symptomatic with clinical symptoms mirroring its classification. Secreting tumors present based on their hormonal effects, and nonsecreting tumors present based on mass effect.⁹

Our patient presented with a CN III palsy, as well as hemifacial pain. This is most likely due to a parasellar expansion with extension into the cavernous sinus. This is confirmed by the CT findings, as well a review of an MRI a few weeks prior from an outside institution which reported a finding of a pituitary macroadenoma (1.5 cm) extending into the right cavernous sinus. Our patient's clinical presentation mirrors the imaging findings, the complete ptosis, and pupillary findings correspond to a CN III compression in the CS, her hemifacial pain can be rightly attributed to a compression of the CN (branch 1 and 2) in the CS.

Given the above findings, urgent neurosurgical intervention was recommended to avoid further structural damage and or apoplexy which can be catastrophic.

REFERENCES



DISCUSSION

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