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Pituitary Adenoma Hidden by Comorbid Cataract

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Pituitary Adenoma Hidden by Comorbid Cataract

Abstract

Background: A pituitary adenoma is an abnormal growth in the pituitary gland. Pituitary adenomas are the most common cause of sellar masses accounting for approximately 10% of all intracranial neoplasms. Early visual symptom of pituitary adenoma can be hidden by comorbid cataract in elderly patients. **Case Report:** A 74 y.o. male presented to the eye clinic with blurry vision at all distances and has trouble with glare from lights. His best corrected visual acuity (BCVA) was $20/20^{-1}$ OD, $20/25^{-2}$ OS, but subjectively of poor quality. Slit lamp exam confirmed mild nuclear sclerotic cataract OU and normal fundus exam. Patient underwent uncomplicated cataract extraction with intraocular implant, OS first then OD. Post-op course was unremarkable until one month later when patient stated that his vision OS was blurry to the left. BCVA was 20/25 OD, $20/25^{-2}$ OS. Post-op exam was still normal including FCF. Humphrey visual field (HVF) however showed a bitemporal hemianopia. Patient was referred for a brain imaging. Brain MRI confirmed a 24 x 22 x 28 mm T2 isointense enhancing mass of the sella with suprasellar extension consistent with a pituitary macroadenoma. Patient underwent endonasal transphenoidal craniectomy for debulking of the tumor subsequently by an ENT specialist. **Conclusion**: Clinicians need to be sure the cataract matches the visual complaint. When in doubt further ancillary testing such as comprehensive HVF is crucial to detect visual pathway disorder and brain tumors. Co-management with corresponding specialist is essential to preserve vision and save life.

Keywords

cataract, pituitary adenoma, space-occupying lesion, automated perimetry, magnetic resonance imaging

INTRODUCTION

A pituitary adenoma is an abnormal growth in the pituitary gland. Pituitary adenomas are the most common cause of masses on the sella (a depression in the sphenoid bone, containing the pituitary gland) accounting for approximately 10% of all intracranial neoplasms.[1] Adenomas are classified by their size and the cell of origin. Lesions smaller than 1 cm are classified as microadenomas, and lesions larger than 1 cm are classified as macroadenomas. Adenomas can arise from any type of cell of the anterior pituitary and may result in increased secretion of the hormone(s) produced by that cell, and/or decreased secretion of other hormones due to compression of other cell types.[2] Impaired vision is the most common symptom that leads a patient with a non-functioning or benign adenoma to seek medical attention.[2] A visual impairment is caused by suprasellar extension of the adenoma, leading to a compression of the optic chiasm. The most common complaint is diminished vision in the temporal fields.[2] However, other pathophysiologic conditions can also cause bitemporal visual field defects including, craniopharyngiomas, aneurysms of the anterior communicating artery, and tilted disc syndrome.[3] Papilledema is unusual with pituitary adenomas because the tumors tend to be detected before they get large enough to increase intracranial pressure. This case report presents an elderly patient with a pituitary adenoma that posed a diagnostic challenge because of comorbid bilateral cataracts.

CASE REPORT

A 74-year-old male presented to our optometry clinic with complaints of blurred vision with both eyes at all distances and difficulty with glare. He expressed interest in cataract surgery a year prior but was presurgical, yet still expressed a strong desire for cataract surgery at this visit. His medical history was positive for aortic valve stenosis, atrial fibrillation, and hypertension which were treated with atorvastatin, warfarin, metoprolol, and hydrochlorothiazide. His best corrected visual acuities were $20/20^{-1}$ right eye, $20/25^{-2}$ left eye, but subjectively of poor quality. The brightness acuity test (BAT) for glare disability and/or a test of contrast sensitivity may have added further evidence of visual impairment induced by cataract, but was not performed at this visit. His refractive errors were +1.00-1.00 x 095 right eye, +1.50-0.75 x 109 left eye; they were stable from the previous year. His pupils were equal and reactive to light without afferent pupillary defect. Extraocular movement was full without restriction and confrontation visual fields were full to finger counting. A cover test was orthophoric at near. Goldmann applanation tonometry was 13 mm Hg right eye,

11 mm Hg left eye at 11:15 in the morning. Color vision testing was not performed at this visit. Slit lamp examination confirmed mild nuclear sclerotic cataracts in both eyes. A dilated fundus examination revealed optic discs with distinct margins, without pallor (although disc pallor can be difficult to judge accurately with cataracts), and a cup-to-disc ratio of 0.20 in each eye. A fundus examination revealed no foveal reflexes and no early signs of macular degeneration. The patient was referred for cataract surgery because his daily activities were affected adversely by the cataracts, and he desired to have the surgery.

The patient underwent uncomplicated cataract extraction with an intraocular implant, left eye first then right eye, a few weeks after the initial visit. Post-operative findings were unremarkable, and the patient was satisfied with the results of less glare and better clarity until his one-month follow-up visit when he stated that vision with his left eye was getting blurrier to the left side. He confirmed that the blur was more noticeable after the cataract extraction, so the cataracts might have hidden some of his pre-existing visual impairment caused by the growing pituitary adenoma. Best corrected visual acuities were 20/25 OD, $20/25^{-2}$ OS.

Fundus evaluation and photos were normal and spectral domain optical coherence tomography (SD-OCT) was within normal limits as well **(Figure 1 a, b)**.

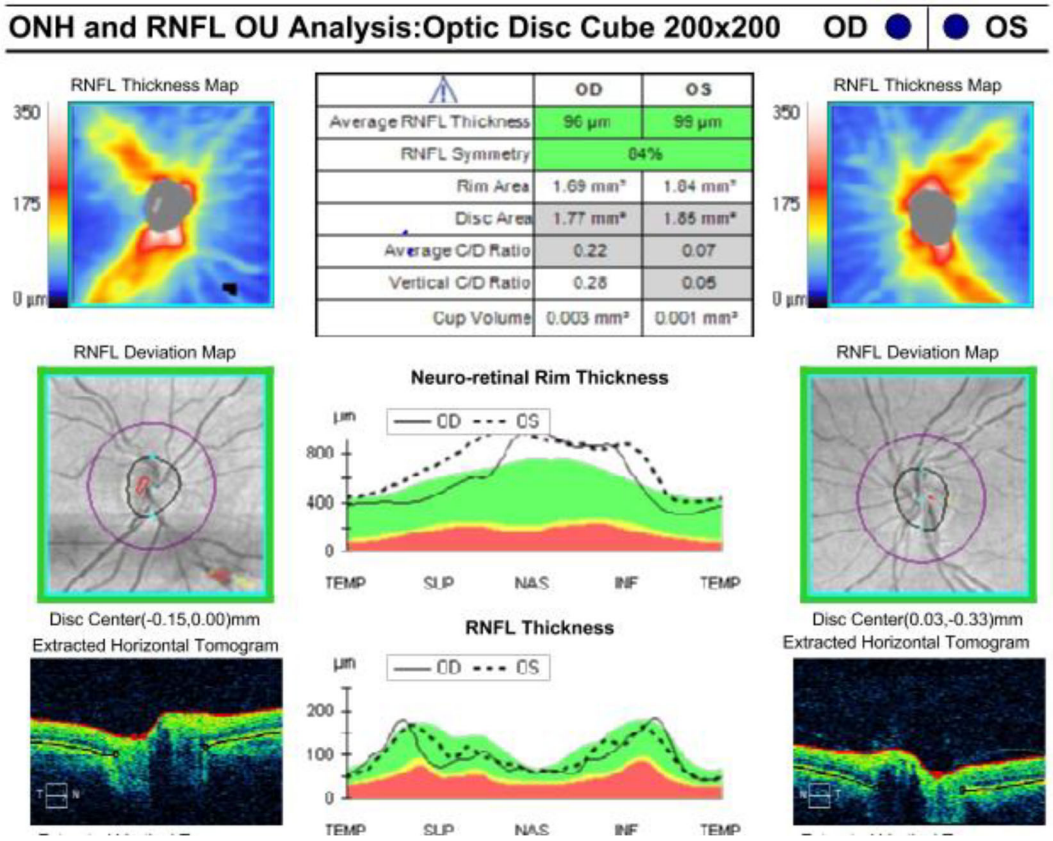


Figure 1 a: Optic Disc Cube SD-OCT scans show small optic nerve heads and normal retinal nerve fiber layers OU.

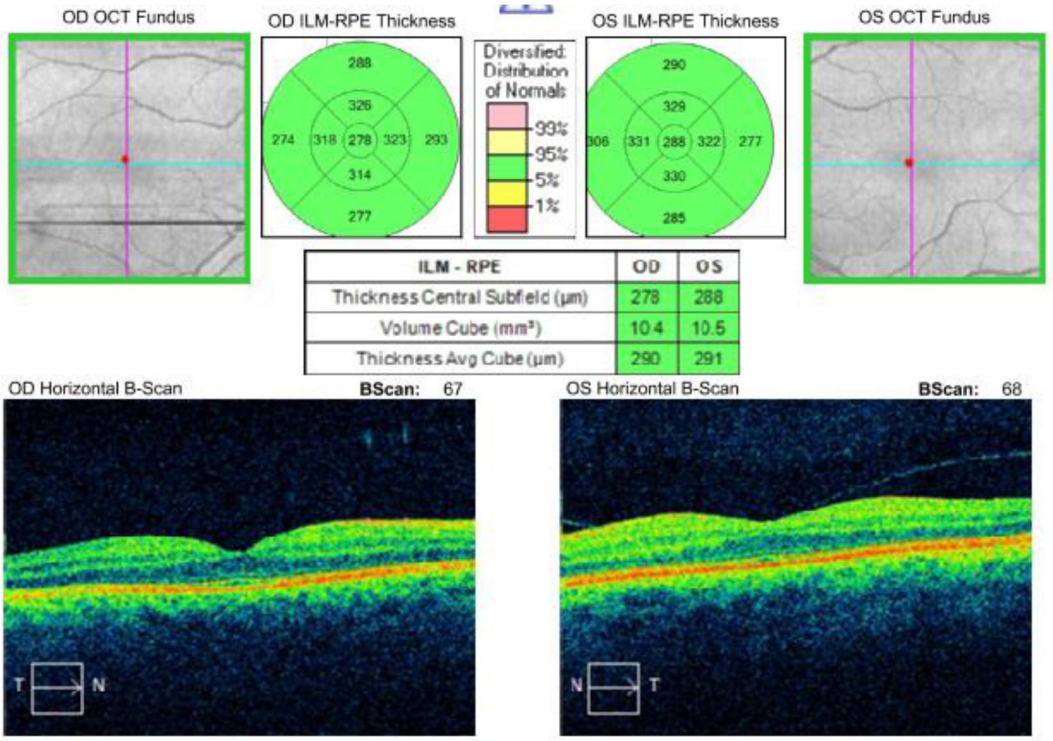


Figure 1 b: SD-OCT macular scans show normal macular thickness OU.

A 30-2 automated static threshold perimetry was ordered to rule out visual pathway disorders since his temporal view OS was blurry. Automated static threshold perimetry showed a bitemporal hemianopia **(Figure 2)**. The patient was educated on the possibility of a brain lesion and referred for brain imaging.

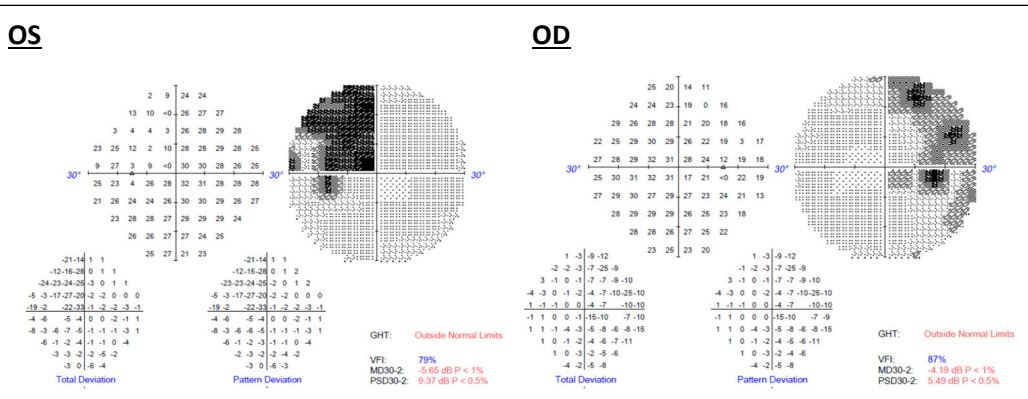
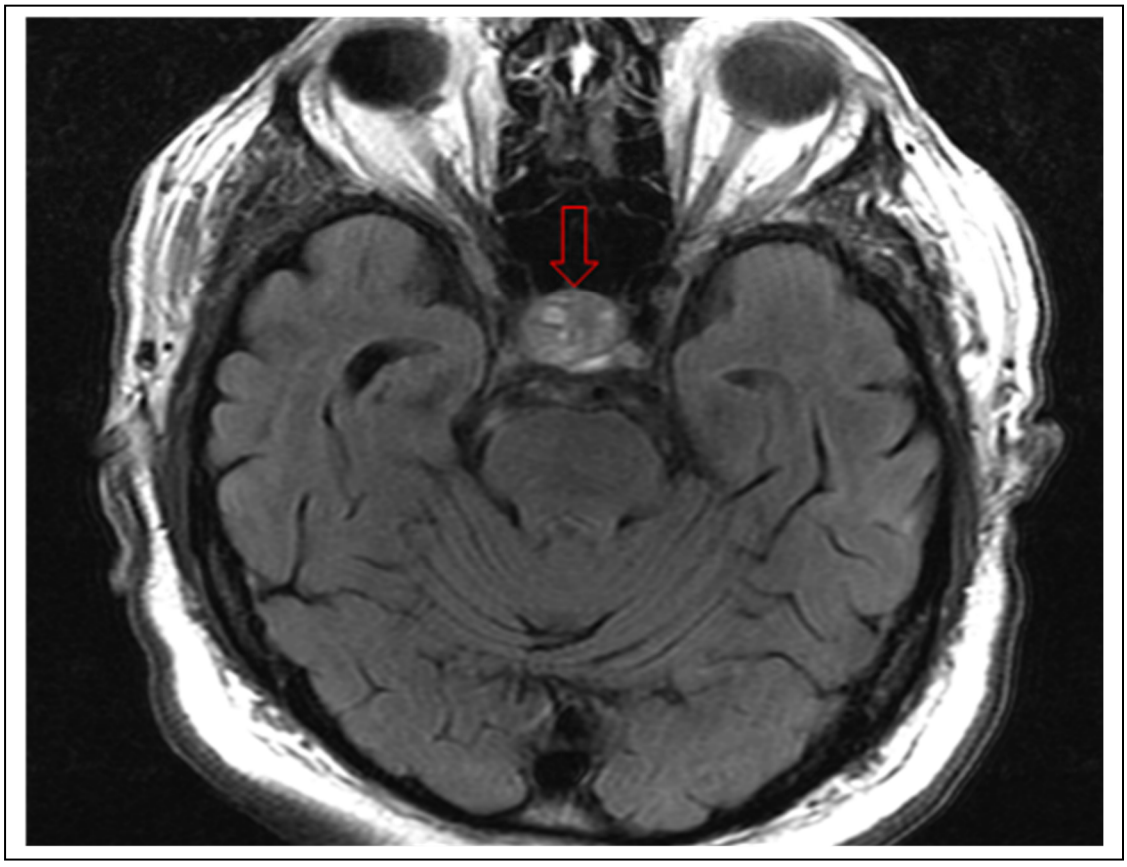


Figure 2: 30-2 threshold test automated perimetry shows a bitemporal heminanopia

A brain MRI was performed two weeks later and confirmed a large (24 x 22 x 28 mm) T2 isointense enhancing mass of the sella (a depression in the sphenoid bone, containing the pituitary gland) with suprasellar extension consistent with a pituitary macroadenoma causing a displacement of the optic chiasm. A representative T1 weighted axial section showed a pituitary adenoma impinging on the optic chiasm **(Figure 3)**. Laboratory work up was ordered to evaluate hormone levels. Cortisol, insulin-like growth, thyroid stimulating hormone and growth hormone were normal, except for an elevated prolactin level of 104.4 ng/mL (reference: 4-15.2 ng/mL).

Figure 3: A horizontal (transverse) MRI T1 weighted view of the brain from below shows the pituitary tumor (red arrow) applying pressure on the overlying optic chiasm.



The patient was referred to an ear, nose, and throat specialist for further management. A few weeks later, the patient underwent a successful endonasal

transphenoidal craniectomy for debulking the tumor by the specialist. His prolactin level returned to a normal level, 11.2 ng/mL a week after the resection. At a follow-up visit, the patient reported an improvement in vision and visual field **(Figure 4)**.

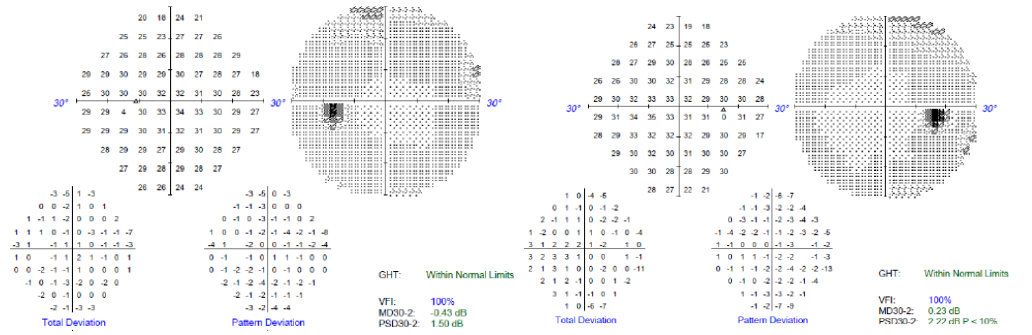


Figure 4: 30-2 threshold test automated perimetry shows resolving bitemporal heminanopia after tumor removal

DISCUSSION

The prevalence of pituitary adenomas is relatively high, at 10-20%, based on previous radiological and autopsy studies.[4] Although there are multiple causes of a sellar mass, pituitary adenomas are the most common **(Table 1)**.[5] In a clinical setting, microadenomas are over four-fold more common than macroadenomas.[6] Prolactinomas are the most common types of pituitary adenomas, accounting for approximately half of all pituitary tumors requiring medical attention.[6] Prolactinoma is observed five-to ten-fold more frequently in women than men, but this difference disappears after menopause.[7] The peak age of onset in women occurs at approximately 30 years and after age 50 in men as in this case .[8] Microadenomas are more common in premenopausal women with classic symptoms of prolactinomas including oligo- or amenorrhea, galactorrhea and infertility,[9] whereas macroprolactinomas are the predominant form in men, leading to a loss of libido, erectile dysfunction, gynecomastia, and infertility.[10]

Benign tumors Pituitary adenoma (most common sellar mass) Craniopharyngioma Meningiomas	**Pituitary hyperplasia** Lactotroph hyperplasia (during pregnancy) Thyrotroph and gonadotroph hyperplasia Somatotroph hyperplasia due to ectopic GHRH*
Cysts Rathke's cleft Arachnoid Dermoid	**Others** Pituitary abscess Lymphocytic hypophysitis Carotid arteriovenous fistula
Malignant tumors Primary Germ cell tumor (ectopic pinealoma) Sarcoma Chordoma Pituitary carcinoma (rare)	**Malignant tumors** Metastatic Lung Breast

Table 1: Causes of sellar masses.[4] *GHRH = Growth hormone-releasing hormone.

Macroadenomas may exert local mass effects on the chiasm, leading to bitemporal visual field defects. Further, headaches are a frequent symptom associated with the lateralization of the tumor. Hyperpituitarism may also result from direct pituitary compression, therefore, all patients with suspected pituitary adenoma should be evaluated for possible deficits in pituitary function.[11]

The diagnosis of hyperprolactinemia is determined by measuring basal prolactin levels. Virtually all patients with macroprolactinomas have levels greater than 100 ng/mL and most patients with microprolactinomas present levels ranging from 50 – 150 ng/mL.[12] The prolactin level in this case was elevated to 104.4 ng/mL. An MRI of the pituitary gland with and without contrast is necessary to detect and measure the size of the tumor.[13] Visual field testing is recommended in patients whose tumors are adjacent (<2 mm) to or impact on the optic chiasm, as visualized on an MRI scan.

Clinical management of prolactinomas includes surveillance, medical, and surgical treatment. Symptomatic eugonadal patients with microprolactinomas (<1 mm) do not require active therapy and can be monitored with regular measurements of PRL levels and visual field testing.[11] For patients with macroprolactinomas, therapy is usually needed because the tumor is likely to grow, particularly in males.[9] Dopamine agonists such as bromocriptine,

cabergoline, and quinagolide, are the primary therapy for almost all prolactinomas to decrease hyperprolactinemia and tumor volume.[14] They act on the dopaminergic D2 receptor to lower the synthesis and release of prolactin releasing factors. Neurosurgery is indicated for patients who are resistant or intolerant to dopamine agonists and acute complications such as pituitary apoplexy or cerebral spinal fluid leakage. Recent technological advances include endonasal endoscopy and intraoperative MRI and neuro-navigation, but the transsphenoidal surgical approach still represents the standard of care.[15]

Our senior male patient presented with blurry vision which was initially attributed to incipient cataracts, and he underwent uncomplicated cataract extraction. At his one-month post-op visit, his blurry vision on the left side worsened, so automated static perimetry was performed confirming a bitemporal hemianopia, despite a normal finger counting confrontation visual field. Subsequently, a brain MRI detected a large pituitary mass displacing the optic chiasm, and a transsphenoidal craniectomy was done to debulk the tumor. His vision returned to normal and so did his prolactin levels. The patient is being monitored closely with prolactin level measurement and visual field testing for any sign of recurrence and visual impairment.

Optical coherent tomography (SD-OCT) has been shown to identify retinal nerve fiber layer (RNFL) loss in eyes with chiasmal compression with good correlation, both in location and severity, between the degree of retinal nerve fiber layer thickness and visual field defects in comprehensive optic neuropathies[15]. In addition, patients with normal RNFL thickness showed an increased propensity for good visual recovery. For this case, although the patient's SD-OCT scans were largely unremarkable, his visual field results indicated a significant reduction in the sensitivity of the visual field temporally in both eyes, denser above than below the hemianopic line, indicating that the pathology was located below the chiasm. The deficits were denser for the left eye, correlating to his worse visual symptom on the left side.

Although not affecting the outcomes in this particular patient, a few limitations of this case analysis should be discussed. Firstly, a careful confrontation visual field may have detected early temporal defects. In addition, a Humphrey visual field SITA Faster could have been used to screen for early visual field defects. Brightness acuity testing could have been performed to gauge the severity of cataracts. If visual symptoms of cataracts do not match the examination findings, the clinician should look for other causes, including neurological origins. Another point to remember is that many patients with bitemporal loss may not notice the loss when both eyes are open, as the temporal

visual field loss OD is partially filled in with the nasal visual field OS. Furthermore, a patient's desire to have cataract surgery may occasionally mislead the clinical judgement and administration of proper testing to reach an appropriate diagnosis.

In conclusion, mild neurological symptoms can be difficult to detect in a patient with incipient cataracts because both the patient and the clinician may lean towards cataracts as the main cause. A quick finger counting visual field test may not be sensitive enough to detect early bitemporal visual field defects. A more elaborate confrontation visual field test and automated perimetric screening may be needed to identify early homonymous hemianopsia or bitemporal hemianopsia. Ancillary testing such as fundus photography and SD-OCT are also helpful in assessing the overall ocular health. Clinicians should ensure that cataract position and density match the visual complaint. When the physical examination does not correlate with the visual symptoms and ocular health, the clinician should search for causes, including a neurological etiology.

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