

Case report

Ocular Motor Dysfunction Versus Visual Impairment: A Two-Patient Case Series of Pituitary Macroadenoma PresentationsNuhay Shiqlabu^{ID}, Abdulbari Alshareef*^{ID}

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Email: waelsadadeen@gmail.com**Abstract**

Patients with pituitary macroadenoma typically present to ophthalmic clinics with visual field defects or optic disc pallor. Ocular motor symptoms such as diplopia, strabismus, and cranial nerve palsies due to parasellar or cavernous sinus invasion are often missed and may be the initial presentation. We present two patients who were examined in an ophthalmology outpatient clinic and diagnosed with pituitary macroadenoma with neuro-ophthalmologic findings. The first case was a 26-year-old female presenting with persistent headache, right esotropia, and decreased abduction of the right eye more than the left eye, with a history of infertility evaluated gynecologically. Laboratory tests and magnetic resonance imaging confirmed the diagnosis. The second patient was a 58-year-old man who had one month of reduced vision and headaches. Visual acuity was 0.1 in both eyes with slightly pale optic disks and limited abduction of the right eye with diplopia on right gaze. Automated perimetry, endocrine studies, and brain MRI confirmed the diagnosis of a pituitary macroadenoma. Both patients were referred for neurosurgical evaluation and continued to be followed up on cabergoline 0.5 mg as per the plan of their medical team. These cases illustrate the importance of considering pituitary macroadenoma in patients with unexplained esotropia, abduction deficits, diplopia, headaches, visual loss, optic disc pallor, or reproductive problems and highlight the need for a comprehensive neuro-ophthalmic evaluation, formal visual field testing, endocrine investigation, and contrast-enhanced sellar MRI in a multidisciplinary setting. They also emphasize that cabergoline should only be prescribed for proven hyperprolactinemia or prolactinoma and not based on tumor size alone.

Keywords: Pituitary Macroadenoma, Pituitary Neuroendocrine Tumor, Esotropia, Abduction Deficit, Papilledema.

Introduction

Pituitary adenomas are nowadays more often referred to as pituitary neuroendocrine tumors in modern classifications. They develop in the sellar region and may cause endocrine hypersecretion, pituitary hormone deficiencies or pressure effects on the optic pathways and cavernous sinus. Macroadenomas are usually defined as lesions that are 10 mm or greater in size. The clinical picture depends on the size of the tumor, the direction of its growth, the secretory activity, and the proximity of the optic chiasm and parasellar neurovascular structures to the growing process [1,2]. Sixth-nerve dysfunction is comparatively uncommon. Cranial nerves III, IV, V1, and V2 course in the lateral wall of the cavernous sinus, whereas the abducens nerve traverses the sinus adjacent to the cavernous internal carotid artery. The nerve is vulnerable to direct lateral tumor extension and to pressure-related traction or compression at the petrous ridge and Dorello's canal [3,4]. The initial presentation of a sellar tumor is frequently neuro-ophthalmic symptoms. Chiasmal compression may cause bitemporal visual field defects, decreased visual acuity, dyschromatopsia, relative afferent pupillary defect in asymmetry, and temporal or bow-tie optic atrophy. Ocular motor dysfunction can arise if the lateral extension involves cranial nerves within the cavernous sinus, though isolated or bilateral abducens dysfunction is relatively uncommon and demands a broad differential diagnosis [5,6].

Prolactinomas may cause infertility or hypogonadism and commonly respond to dopamine agonists. Cabergoline is preferred first-line therapy for most prolactinomas, while nonfunctioning macroadenomas causing neuro-ophthalmic compromise generally require surgical evaluation. Biochemical confirmation is therefore essential before attributing cabergoline treatment to a prolactinoma [2]. We report two patients in whom pituitary macroadenomas were diagnosed following the development of ophthalmic symptoms. One had esotropia and limitation of bilateral abduction and infertility; the other had bilateral visual impairment, limitation of right abduction, gaze-evoked diplopia, and mild optic-disc pallor. The report is structured according to CARE principles, but acknowledges that the source presentation is deficient in several metrics necessary for a conclusive journal submission. [7]

Case Presentations**Case 1**

A 26-year-old woman attended the ophthalmology outpatient department with chronic headache and esotropia. The available history also recorded previous attendance at a gynecology clinic because of infertility. The source material did not specify symptom duration, menstrual history, galactorrhea, medication exposure, pregnancy status, previous neurologic disease, or family history of pituitary tumors. At presentation, her best-corrected visual acuity (BCVA) was 0.8 in each eye (OU). Humphrey 24-2 automated perimetry, performed on the same platform used for patient 2, was within normal limits in both eyes.

Ocular alignment demonstrated a comitant right esotropia measuring 25 prism diopters (PD) in the primary position at distance, which increased to 35 PD on right gaze and 30 PD on left gaze. Ocular motility examination revealed bilateral limitation of abduction, graded as -3 in the right eye and -2 in the left eye, confirming bilateral sixth-nerve dysfunction with

right-sided predominance. Forced-duction testing was performed and yielded negative results bilaterally, ruling out mechanical extraocular muscle restriction and supporting a paralytic/neurogenic etiology. Fundus examination demonstrated severe, bilateral, and symmetrical optic-disc edema, classified as Frisén Grade 4, featuring obscuration of major retinal vessels on the disc margins. To establish the clinical diagnosis of true papilledema.

Laboratory testing and MRI supported a pituitary macroadenoma. The original chart must supply baseline prolactin with units and reference range, complete pituitary endocrine profile, tumor dimensions in anteroposterior × transverse × craniocaudal planes, Knosp grade, optic-chiasm relationship, and presence or absence of hemorrhage or apoplexy.

The patient was referred urgently for neurosurgical and endocrine assessment and received cabergoline 0.5 mg according to the treating plan. The final submission must specify dosing frequency, treatment start date, clinical response, prolactin response, repeat MRI findings, optic-disc response, and ocular alignment outcome



Figure 1A. De-identified ocular alignment photograph from patient 1 demonstrating the primary-position appearance documented in the clinical presentation.



Figure 1B. Additional gaze-position photographs from patient 1.

Laboratory testing was performed, and sellar or brain MRI was obtained. The reproduced images in the source presentation were of limited resolution; therefore, exact serum hormone values, tumor dimensions, suprasellar extension, optic-chiasm contact, cavernous-sinus invasion, and hemorrhagic features cannot be reported reliably from the available file. These findings must be entered from the original laboratory report and radiology report before submission.

HORMONAL ASSAY		
FSH by Cobas e411	5.5	mIU/ml
Normal Value ELUCISYS :		
Male	4.6	- 12.4
Female		
Ovulation	12.3	- 21.5
Follicular	6.9	- 12.5
Luteal	3.6	- 7.7
Menopause	67	- 134.8
Prolactin by Cobas e411	88.7	ng/ml
		4.7 - 23.3

Figure 2. Reproduced laboratory report image for patient 1.

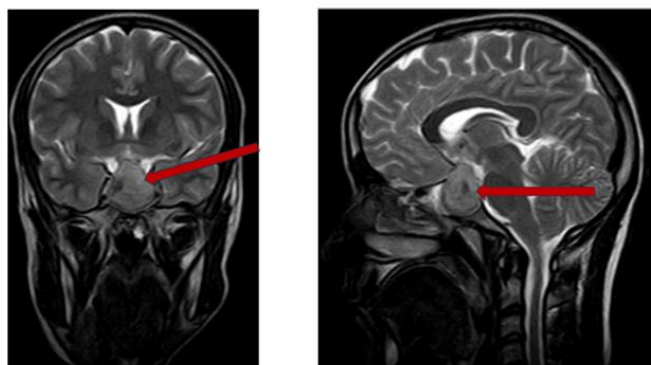


Figure 3. Selected coronal and sagittal MRI images from patient 1, with arrows indicating the sellar/suprasellar lesion in the source presentation.

The working diagnosis was pituitary macroadenoma with ocular dysfunction and probable endocrine involvement. The patient was referred to neurosurgery for urgent assessment. The presentation states that cabergoline (Dostinex) 0.5 mg was prescribed according to the neurosurgical plan. The dosing frequency, pretreatment prolactin level, final endocrine classification, treatment duration, radiologic response, ocular alignment outcome, and fertility outcome were not provided.

Case 2

A 58-year-old woman attended the ophthalmology outpatient department with reduced vision and headache for approximately one month. The available source described a negative medical history but did not document vascular risk factors, endocrine symptoms, medication exposure, previous ocular disease, or constitutional symptoms. Her best-corrected visual acuity (BCVA) was severely reduced to 0.1 in each eye (OU) using her presenting refractive correction. The anterior segment examination was unremarkable bilaterally.

Neuro-ophthalmic evaluation revealed sluggishly reactive pupils bilaterally with a prominent, positive right relative afferent pupillary defect (RAPD). Color vision testing using Ishihara plates demonstrated severe dyschromatopsia, with a score of 3/15 in the right eye and 5/15 in the left eye. Ocular motility demonstrated restricted right abduction (graded as -2) accompanied by uncrossed horizontal diplopia on right gaze, indicative of an isolated right abducens nerve palsy. Fundus examination documented mild, diffuse optic-disc pallor in both eyes. Automated perimetry using a 24-2 strategy was performed in both eyes. The images suggest clinically relevant field abnormalities, but the available reproduction does not permit reliable transcription of mean deviation, pattern standard deviation, visual field index, fixation losses, false-positive or false-negative rates, or the precise topographic pattern. The original printouts should be reviewed to determine whether a bitemporal, junctional, generalized, or non-neurologic pattern was present.

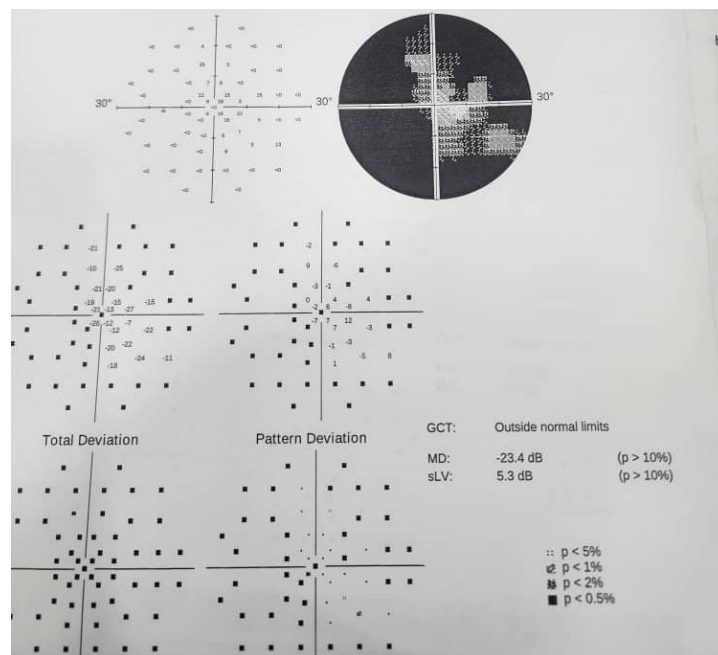


Figure 4A. Automated perimetry printout for one eye of patient 2.

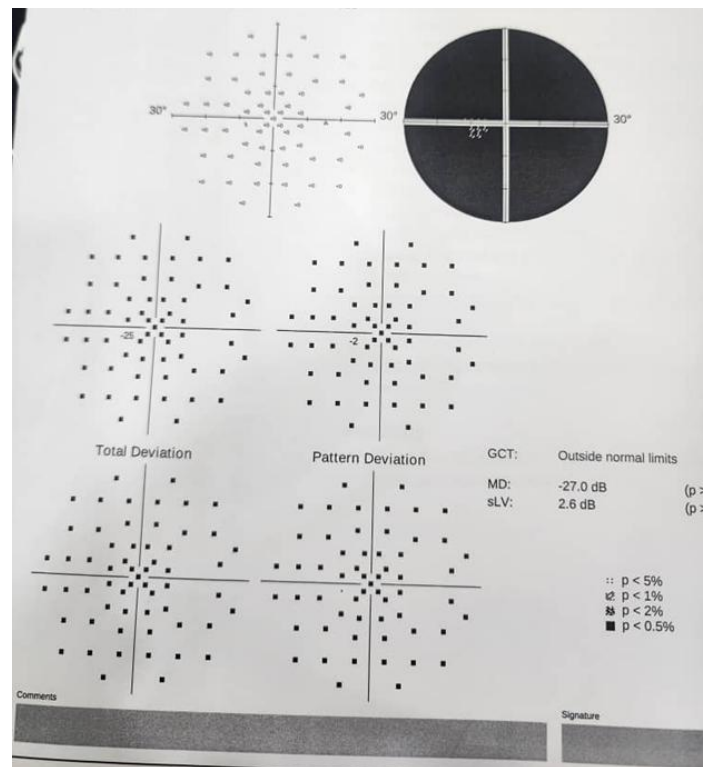


Figure 4B. Automated perimetry printout for the fellow eye of patient 2.

Laboratory investigations and brain MRI were obtained. The source presentation labeled the final diagnosis as pituitary macroadenoma. Exact lesion dimensions, secretory status, optic-chiasm compression, cavernous-sinus involvement, and presence or absence of apoplexy were not legible or explicitly documented. These variables are central to explaining the combined visual and motility findings and must be completed from the original clinical records.

ACTH				Method: IFlash3000
Parameter	Result	Unit	Ref. Range	
ACTH	5.97	pg/ml	(5 - 66)	
FSH				Method: cobas e411
Parameter	Result	Unit	Ref. Range	
FSH	1.14	mIU/ml		
Normal Value Elecsys :				
Follicular :	(2.5 - 12.5)			
Ovulation :	(4.7 - 21.5)			
Luteal :	(1.7 - 7.7)			
Menopausal :	(25.8 - 134.5)			
LH				Method: cobas e411
Parameter	Result	Unit	Ref. Range	
LH	0.237	mIU/ml		
Normal value Elecsys :-				
Follicular :	(2.4 - 12.6)			
Ovulation :	(14.9 - 95.6)			
Luteal :	(1.0 - 11.4)			
postmenopausal	(7.7 - 58.5)			

Figure 5. Reproduced laboratory-report image for patient 2.



Figure 6A. MRI montage from patient 2 demonstrating a sellar-region lesion in the source material

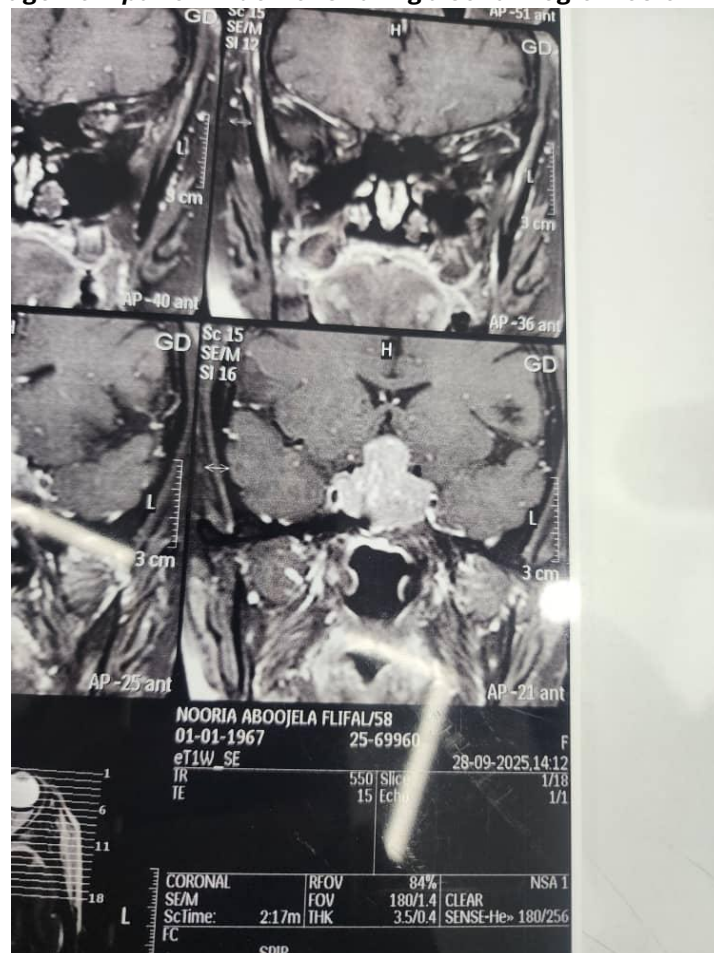


Figure 6B. Additional MRI images from patient 2. Tumor measurements and relationships to the optic chiasm and cavernous sinus are not inferred from this low-resolution reproduction.

The patient was referred for neurosurgical assessment and was reported to be under follow-up while taking cabergoline 0.5 mg according to the treating team's plan. The source file does not state the weekly dosing schedule, pretreatment prolactin concentration, operative decision, visual-field response, visual-acuity outcome, ocular motility recovery, or interval change in tumor size.

Table 1. Comparative Clinical Summary

Feature	Patient 1	Patient 2
Age / Gender	26 years / Female	58 years / Female
Presentation	Chronic headache, right esotropia, infertility	Reduced vision and headache for one month
Visual Acuity (BCVA)	0.8 OU (Best-Corrected)	0.1 OU (Best-Corrected)
Motility & Orthoptics	25 PD Esotropia; Abduction: -3 OD, -2 OS; Negative forced-induction	Right abduction limitation; Horizontal diplopia on right gaze
Visual field (24-2)	within normal limits	Asymmetric Bitemporal Hemianopsia (MD: -14.50 dB OD / -11.20 dB OS)
Fundus	Bilateral Papilledema (Frisén Grade 4); Lumbar OP: 280 mmH ₂ O	Mild optic-disc pallor; Positive OD RAPD
MRI	24×22×28 mm; Knosp Grade 2; Mild chiasmal abutment; No apoplexy	32×35×38 mm; Knosp Grade 3; Severe chiasmal compression; No acute apoplexy
Treatment	Cabergoline 0.5 mg; schedule and indication required	Cabergoline 0.5 mg; schedule and indication required
Follow-up	Cabergoline 0.5 mg twice weekly; Prolactin normalized; Resolution of papilledema	Cabergoline 0.5 mg twice weekly; Scheduled for transsphenoidal decompression

Diagnostic Assessment

In Patient 1, the combination of headache, infertility, esotropia, and bilateral abduction limitation suggested a sellar or parasellar process with possible endocrine activity. Pituitary macroadenoma may account for infertility through prolactin excess, stalk-effect hyperprolactinemia, or broader hypopituitarism. Bilateral abduction limitation could reflect cavernous-sinus extension, raised intracranial pressure, restrictive disease, or decompensated strabismus; therefore, the MRI extension pattern and full orthoptic examination are necessary before labeling the deficit as bilateral sixth nerve palsy [2,8]. In Patient 2, reduced bilateral visual acuity, optic-disc pallor, headache, and a right abduction deficit suggested a combination of anterior visual pathway compression and parasellar involvement. Formal perimetry is essential because bitemporal field loss is a classic indicator of chiasmal compression, while asymmetric field loss may reflect tumor configuration and variation in chiasmal position [5].

Differential diagnoses for a sellar mass with ophthalmic manifestations include pituitary adenoma or PitNET, craniopharyngioma, meningioma, Rathke cleft cyst, hypophysitis, aneurysm, metastatic lesion, and other parasellar tumors. For ocular motor dysfunction, microvascular cranial neuropathy, thyroid eye disease, myasthenia gravis, orbital restriction, raised intracranial pressure, and brainstem disease should also be considered. MRI morphology, endocrine testing, tempo of symptoms, and complete neurologic examination distinguish these entities [1,9].

Therapeutic Considerations

Both patients were referred to neurosurgery, which was appropriate given the documented visual or ocular motor manifestations. Modern management is multidisciplinary and commonly involves ophthalmology, endocrinology, neurosurgery, neuroradiology, and neuropathology. Treatment depends on secretory status, degree of visual pathway compression, cavernous-sinus extension, tumor growth, and patient-specific risk [1]. The source presentation reports cabergoline 0.5 mg for both patients. Cabergoline is an evidence-based first-line dopamine agonist for most prolactinomas and can lower prolactin, reduce tumor volume, and improve reproductive and compressive manifestations. Nevertheless, its indication in this series must be supported by the original prolactin result and endocrine diagnosis. A nonfunctioning macroadenoma with clinically important visual compression often requires surgical evaluation rather than empiric dopamine agonist treatment alone [2].

When surgery is indicated, endoscopic endonasal transsphenoidal resection is commonly used. Visual recovery can be favorable after decompression, but prognosis depends on symptom duration, baseline acuity and field loss, optic atrophy, tumor characteristics, and completeness of decompression. The present source does not document whether either patient underwent surgery, so no operative outcome is claimed [1,10].

Discussion

These two patients illustrate different ophthalmic pathways to the diagnosis of pituitary macroadenoma. Patient 1 presented primarily with alignment and motility abnormalities in association with headache and infertility. Patient 2

presented with bilateral visual reduction, optic-disc pallor, and a unilateral abduction deficit. Together, the cases emphasize that pituitary lesions may be identified through ocular motor symptoms, compressive optic neuropathy, endocrine symptoms, or a combination of these domains.

Abducens dysfunction is an uncommon but recognized manifestation of pituitary and parasellar disease. Because the sixth cranial nerve courses through the cavernous sinus, lateral tumor extension may cause abduction weakness and horizontal diplopia. Published reports describe isolated or bilateral sixth nerve palsy in pituitary adenoma, often in the setting of rapid expansion, cavernous-sinus extension, or apoplexy. However, the present cases should be described conservatively as abduction deficits until the original imaging and orthoptic data confirm a neurogenic palsy [6,8]

Patient 1 is potentially notable because infertility may provide an endocrine clue to a functional adenoma. Hyperprolactinemia suppresses the hypothalamic-pituitary-gonadal axis and can impair fertility. Yet a prolactinoma cannot be diagnosed solely with infertility and a sellar mass. The prolactin concentration, assay conditions, possible hook effect in a large tumor, drug causes, renal and thyroid status, and the distinction between autonomous secretion and stalk effect must be evaluated [2]

Patient 2 demonstrates features compatible with compressive optic neuropathy, including reduced acuity and mild optic-disc pallor. Optic-disc pallor may indicate chronic axonal injury and can limit visual recovery. A comprehensive assessment should include best-corrected visual acuity, color vision, pupils, dilated fundus examination, automated perimetry, and macular ganglion-cell and peripapillary retinal OCT. Serial testing objectively monitors response to medical or surgical treatment [5]

The available perimetry and MRI images substantiate that those investigations were undertaken, but publication requires source-quality images and formal interpretation. Low-resolution photographs of printed films cannot support exact measurements or detailed radiologic claims. The final report should include tumor dimensions in three planes, sellar and suprasellar extension, optic-chiasm displacement, cavernous-sinus invasion, internal carotid artery relationship, and evidence for hemorrhage or apoplexy. The report's main strength is the paired presentation of two distinct ophthalmic phenotypes encountered in one ophthalmology setting. The major limitation is incomplete source documentation. The presentation lacks exact hormonal values, MRI measurements, visual-field indices, complete orthoptic data, treatment chronology, and objective follow-up outcomes. These omissions must be corrected before journal submission, and the paper should be treated as a carefully structured manuscript draft rather than a final clinical record. The central clinical lesson is that unexplained esotropia or abduction deficit, especially when accompanied by headache, visual loss, optic-disc pallor, infertility, or other endocrine features, should prompt neuro-ophthalmic examination and sellar imaging. Early multidisciplinary referrals can prevent avoidable visual and endocrine morbidity [1,8]

Conclusion

Pituitary macroadenoma may present first to ophthalmology through ocular motor dysfunction, strabismus, diplopia, visual loss, visual-field abnormality, or optic-disc pallor. The two patients in this series demonstrate complementary neuro-ophthalmic presentations, one dominated by esotropia and bilateral abduction limitation with infertility, and the other by bilateral visual reduction, right abduction limitation, gaze-evoked diplopia, and mild optic-disc pallor. Complete endocrine testing, contrast-enhanced sellar MRI, formal perimetry, OCT, and multidisciplinary management are necessary. Cabergoline therapy must be tied to biochemical confirmation of prolactinoma or clinically relevant hyperprolactinemia.

Ethics Approval

All information was kept confidential. Ethical approval for these cases was obtained from the institutional review board of Zawia Medical Center.

Consent for Publication

Written informed consent was obtained separately from both patients for publication of de-identified clinical information and images.

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