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*Case Report*

# Pituitary adenoma presenting as isolated oculomotor nerve palsy

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## ABSTRACT

**Background:** Isolated oculomotor nerve palsy is a relatively uncommon symptom of pituitary adenoma that usually occurs in association with pituitary apoplexy or cavernous sinus (CS) invasion.

**Case Description:** We report two cases of relatively small pituitary adenomas with neither apoplexy nor CS invasion presenting as isolated oculomotor nerve palsy. Both patients presented with gradually worsening diplopia, without headache or visual field defects. Magnetic resonance imaging (MRI) showed a pituitary tumor with no evidence of intratumoral hemorrhage. Computed tomography revealed a lateroposterior extension of the tumor with the erosion of the posterior clinoid process. Constructive interference in steady-state MRI revealed compression of the oculomotor nerve by the tumor at the oculomotor triangle. The patients underwent endoscopic transsphenoidal surgery, and the intraoperative findings showed that the tumors did not invade the CS. The tumors were completely resected, and the oculomotor palsies resolved fully.

**Conclusion:** These cases illustrate the need to consider isolated oculomotor nerve palsy as an initial manifestation of a relatively small pituitary adenoma with neither apoplexy nor CS invasion. Based on the characteristic radiological findings, early surgical treatment is recommended to preserve oculomotor function.

**Keywords:** Constructive interference in steady-state (CISS), Oculomotor nerve palsy, Pituitary adenoma

## INTRODUCTION

Visual field defects and endocrine dysfunction are the most common manifestations of pituitary adenoma. Oculomotor nerve palsy is less frequent and usually occurs in association with pituitary apoplexy or cavernous sinus (CS) invasion.[2,3] Here, we report two cases of isolated oculomotor nerve palsy as an initial manifestation of relatively small pituitary adenomas with neither apoplexy nor CS invasion and describe the possible mechanisms and radiological features.

## CASE PRESENTATION

### Case 1

A 65-year-old man presented with diplopia in the left gaze for the past 2 months. He did not experience headaches or vomiting. Ophthalmic testing revealed restricted supraduction and adduction of the right eye. Anisocoria, abnormal pupillary light reflex, or ptosis were not

observed. Visual acuity and field measurements were within the normal range. A neurological diagnosis of an isolated right oculomotor nerve palsy was established. Magnetic resonance imaging (MRI) revealed a 19 mm homogeneously enhanced pituitary tumor that extended latero-posteriorly on the right side [Figures 1a and b]. There was no evidence of intratumoral hemorrhage. Computed tomography (CT) revealed erosion of the right side of the posterior clinoid process [Figure 1c]. Pituitary hormone levels were within the normal range. The patient underwent endoscopic transsphenoidal surgery 3 months after the onset of symptoms. Intraoperatively, the medial wall of the CS was smooth, and venous bleeding from the CS was not observed, indicating no CS invasion of the tumor. The tumor was completely resected. The oculomotor nerve palsy resolved completely within 2 weeks. Postoperative constructive interference in steady-state (CISS) MRI revealed total resection of the tumor and the presence of the right oculomotor nerve at the oculomotor triangle [Figure 1d]. The histological diagnosis was a nonfunctioning pituitary adenoma without hemorrhage [Figure 2].

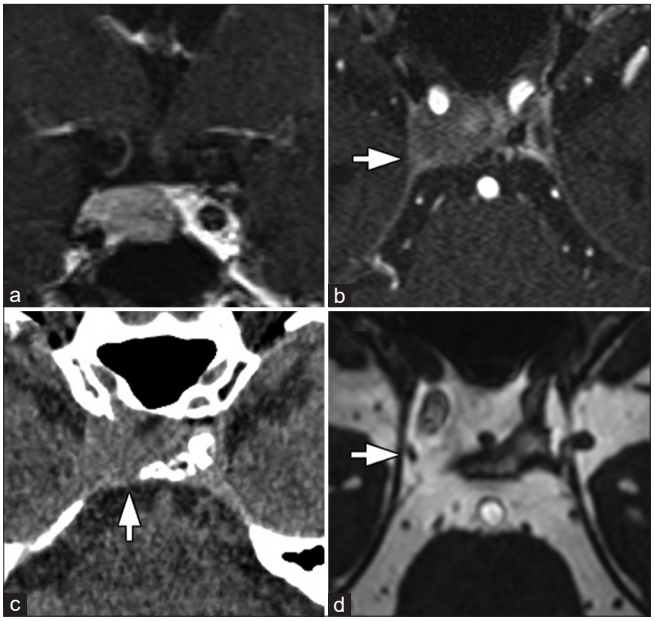


**Figure 1:** (a) Coronal gadolinium-enhanced T1-weighted image (Gd-T1WI) on magnetic resonance (MR) imaging showing homogeneously enhancing pituitary tumor with no evidence of intratumoral hemorrhage. (b) Axial Gd-T1WI showing lateroposterior tumor extension to the right cavernous sinus (arrow). (c) Computed tomography scan showing erosion of the right posterior clinoid process (arrow). (d) Postoperative constructive interference in steady-state MR image showing the right oculomotor nerve at the oculomotor triangle (arrow).

### Case 2

A 75-year-old man presented with diplopia and drooping of the left eyelid for 2 weeks. He did not experience headaches or vomiting. Examination of the patient revealed mild ptosis with limited supraduction, adduction, and infraduction of the left eye. Anisocoria or abnormal pupillary light reflexes were not observed. The neurological diagnosis was an isolated left oculomotor nerve palsy. MRI revealed an 18 mm homogeneously enhanced pituitary tumor that extended latero-posteriorly on the left side [Figure 3a]. There was no evidence of intratumoral hemorrhage. CT revealed an erosion of the left posterior clinoid process [Figure 3b]. CISS MRI revealed compression of the left oculomotor nerve by the tumor at the oculomotor triangle [Figure 3c]. The pituitary function was within the normal range.

Two months after the onset of symptoms, the patient underwent endoscopic transsphenoidal surgery, and the tumor was completely resected. Intraoperatively, there was no evidence of CS invasion by the tumor. The pathological diagnosis was a nonfunctioning pituitary adenoma with no evidence of hemorrhage or necrosis. Immediately after surgery, the patient's oculomotor nerve function recovered completely. Postoperative CISS MRI confirmed the presence of the left oculomotor nerve at the oculomotor triangle [Figure 3d]. The pathological diagnosis was a nonfunctioning pituitary adenoma with no evidence of hemorrhage or necrosis [Figure 4].

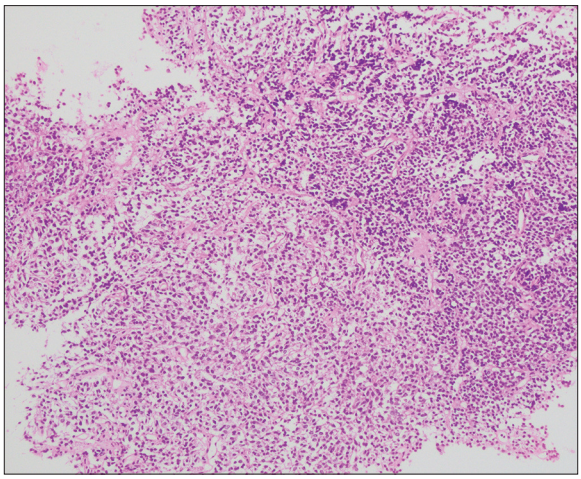

**Figure 2:** Photomicrograph of a histopathological specimen showing tumor cells with no evidence of hemorrhage or necrosis. Hematoxylin and eosin stain, ×100.

## DISCUSSION

Chuang *et al.*[2] reviewed 476 cases of patients with pituitary adenoma and found that 23 (4.8%) patients presented with oculomotor nerve palsy. Among these 23 patients, only 3 (0.6%) had nonapoplectic pituitary adenoma with a mean tumor size of 31 mm. In nonapoplectic cases, oculomotor nerve palsy is typically caused by a large tumor with CS invasion. When a relatively small pituitary tumor is detected in patients with isolated oculomotor nerve palsy, surgeons should ensure that the symptoms are caused by the tumor,

ruling out other diseases causing oculomotor nerve palsy, such as diabetic neuropathies, myasthenia gravis, brain stem infarction, inflammatory injury, and cerebral aneurysms.[1] If oculomotor nerve palsy is unrelated to the tumor, watchful waiting may be a treatment option.

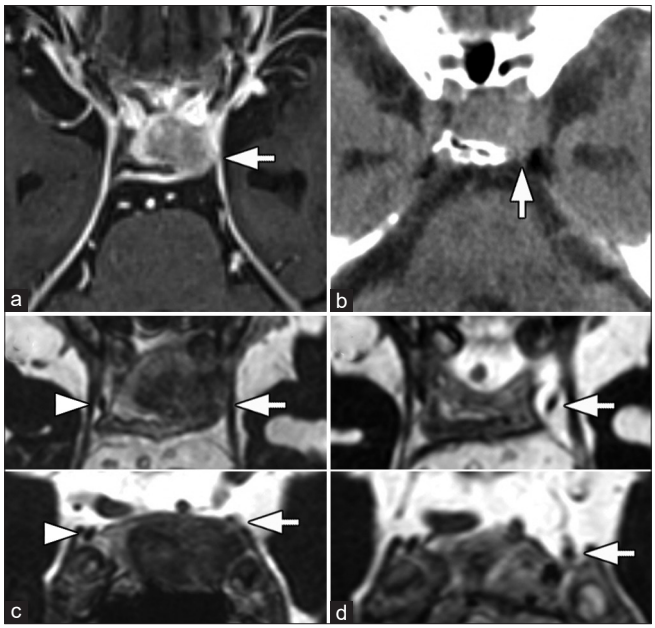


**Figure 3:** (a) Axial gadolinium-enhanced T1-weighted magnetic resonance (MR) image showing homogenously enhancing pituitary tumor protruding latero-posteriorly to the left cavernous sinus (arrow) with no apparent findings of pituitary apoplexy. (b) Computed tomography scan showing erosion of the left posterior clinoid process (arrow). (c) Preoperative constructive interference in steady-state (CISS) MR images (axial and coronal views) showing compression of the left oculomotor nerve by the tumor (arrow). The right oculomotor nerve is visible (arrowhead). (d) Postoperative CISS MR images (axial and coronal views) showing the left oculomotor nerve at the oculomotor triangle (arrow).

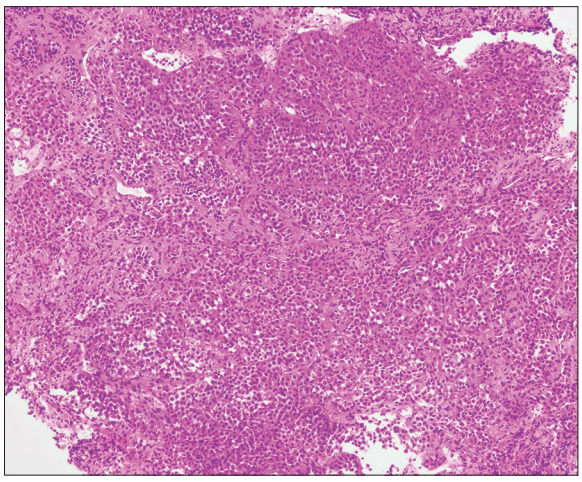

**Figure 4:** Photomicrograph of a histopathological specimen showing tumor cells with no evidence of hemorrhage or necrosis. Hematoxylin and eosin stain, ×100.

The oculomotor triangle at the entrance of the CS is considered the main site of isolated oculomotor nerve palsy in patients with pituitary adenomas with apoplexy.[5] The oculomotor nerve enters the roof of the CS through the oculomotor triangle, which is formed by the anterior petroclinoid, posterior petroclinoid, and interclinoid ligaments [Figure 5]. At the oculomotor triangle, the oculomotor nerve is accompanied by a dura-arachnoid cuff filled with cerebrospinal fluid (the oculomotor cistern).[6] At the meningeal pocket of the oculomotor nerve, the dural layer is thin and is a weak point in the CS wall,[4] making the oculomotor nerve more vulnerable to mechanical compression. The trochlear and abducens nerves are less affected because the trochlear nerve runs lateral to the anterior petroclinoid ligament, and the abducens nerve runs inferior to the posterior petroclinoid ligament. In our cases, the tumors exhibited lateroposterior extension with unilateral erosion of the posterior clinoid process and compression of the oculomotor nerve at the oculomotor triangle. These may be characteristic radiological findings in patients with pituitary adenomas with isolated oculomotor nerve palsy.[5,7]

The prognosis of patients with oculomotor nerve palsy mainly depends on: (1) the interval between the onset of palsy and operation and (2) the degree of preoperative deficit.[2] Compared to patients with apoplectic tumors, a considerable amount of time may be required until the treatment decision is made in patients with nonapoplectic tumors, due to the slow progressive nature of the symptoms.

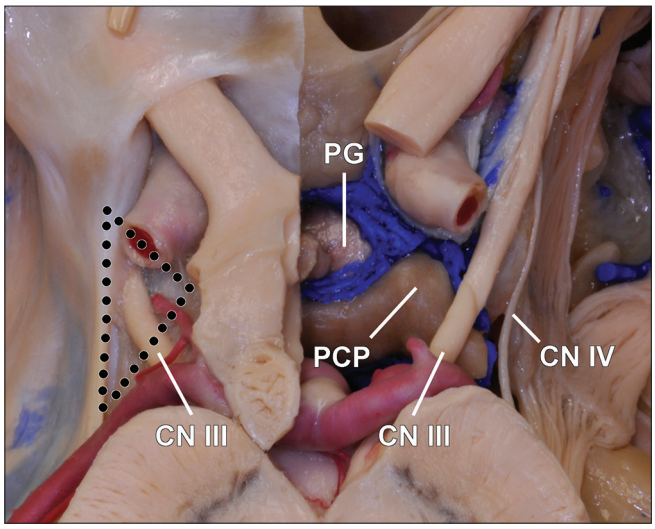


**Figure 5:** Photograph of a cadaveric specimen showing the normal anatomy of the oculomotor nerves. The left oculomotor nerve enters the roof of the cavernous sinus through the oculomotor triangle (dotted line). The right cavernous sinus roof has been removed to expose the entire course of the oculomotor nerve. CN: Cranial nerve, PCP: Posterior clinoid process, PG: Pituitary gland.

Recently, the clinical importance of pituitary tumor extension into the oculomotor cistern has been advocated.[3,8] Pituitary adenomas extending to the oculomotor cistern carry the risk of intraoperative injury to the oculomotor nerve and further extend into the interpeduncular cistern. Early diagnosis and tumor resection are the rationales for preserving good oculomotor nerve function.

## CONCLUSION

These two cases illustrate the need to consider isolated oculomotor nerve palsy as the initial manifestation of a relatively small pituitary adenoma with neither apoplexy nor CS invasion. Based on the characteristic radiological findings in patients with lateroposterior extension of the tumor and compression of the oculomotor nerve at the oculomotor triangle, early surgical treatment is recommended to preserve good oculomotor function.

### Declaration of patient consent

Patients' consent not required as patients' identities were not disclosed or compromised.

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Nil.

### Conflicts of interest

There are no conflicts of interest.

### Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The author(s) confirms that there was no use of Artificial Intelligence (AI)-Assisted Technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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