

A Case of Nonapoplectic Pituitary Adenoma Presenting with Isolated Oculomotor Nerve Palsy

Abstract

We report a rare case of nonapoplectic pituitary adenoma that did not invade the cavernous sinus and was associated with isolated oculomotor nerve palsy. A 61-year-old male was admitted to our hospital due to diplopia that had gradually worsened from 6 months to presentation. He was diagnosed with right oculomotor nerve palsy, and brain magnetic resonance imaging (MRI) showed a mass lesion within the sella. The tumor was homogeneously enhanced on contrast-enhanced MRI. However, no findings suggestive of pituitary apoplexy were found. Brain computed tomography revealed the tumor to have eroded the right side of the posterior clinoid process by gradual expansion. Endoscopic transsphenoidal surgery was used for complete resection of the tumor. Intraoperative findings showed that the tumor did not invade the cavernous sinus. The histological diagnosis was pituitary adenoma, and symptom improvement was observed from the early postoperative stage onward. Surgical treatment is essential because oculomotor nerve palsy caused by the enlargement of pituitary adenoma is not expected to resolve if treated conservatively, unlike that caused by pituitary apoplexy.

Keywords: *Isolated oculomotor nerve palsy, microendoscopic transsphenoidal surgery, nonapoplexy pituitary adenoma*

Introduction

Oculomotor neuropathy is often observed in patients with pituitary adenoma when the onset of pituitary apoplexy is observed. In addition to most oculomotor nerves, optic nerves, trochlear nerves, and abducens nerves are affected simultaneously.^[1,2] However, there are rare instances in which only the oculomotor nerve is damaged in patients with pituitary adenoma. All reported patients developed symptoms associated with pituitary apoplexy.^[3-6] In our case, the gradual increase in intratumor pressure may have only damaged the oculomotor nerve. Here, we detail our experience with a patient with nonapoplectic pituitary adenoma that did not invade the cavernous sinus and only oculomotor nerve damage progressed during a period of 6 months. There have been no reports of isolated oculomotor nerve palsy in patients with pituitary adenoma without pituitary apoplexy.

Case Report

Approximately 6 months prior to presentation, a 61-year-old male became

aware of his diplopia in left gaze. The Hess chart revealed restricted movements of supraduction and adduction of the right eye [Figure 1a], and the patient was diagnosed with oculomotor nerve palsy. The patient did not experience headache, vomiting, or visual field defects. Other symptoms such as anisocoria, abnormal pupillary light reflex, and ptosis were also not observed. Magnetic resonance imaging (MRI) showed a neoplastic lesion within the sella [Figure 2]. The tumor was homogeneously isointense on T1-MRI [Figure 2a], with no evidence of hemorrhage, and the tumor was homogeneously enhanced on contrast-enhanced MRI [Figure 2b-d]. Computed tomography (CT) revealed erosion on the right side of the posterior clinoid process, and the tumor progressed above and behind the right cavernous sinus [Figure 2e]. Endocrine function test revealed mild hypothyroidism and adrenal gland dysfunction (TSH 0.298 μ U/ml, F-T4 0.79 ng/dl, ACTH 6.8 pg/dl). Intraoperatively, after removing the tumor and pseudocapsule,

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Access this article online

Website: www.asianjns.org

DOI: 10.4103/ajns.AJNS_397_20

Quick Response Code:



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How to cite this article: Yokoyama K, Ikeda N, Sugie A, Yamada M, Tanaka H, Ito Y, *et al.* A case of nonapoplectic pituitary adenoma presenting with isolated oculomotor nerve palsy. *Asian J Neurosurg* 2021;16:391-3.

Submitted: 14-Aug-2020

Revised: 25-Nov-2020

Accepted: 28-Mar-2021

Published: 28-May-2021

the medial wall of the cavernous sinus seemed smooth and venous bleeding from the cavernous sinus was not observed with the angled scope. This may indicate that the tumor did not invade into the cavernous sinus. The tumor was completely removed endoscopically [Figure 2f], and histological diagnosis was pituitary adenoma with no malignant component [Figure 3]. There were no issues in the postoperative course; diplopia clearly reduced from the immediate postoperative stage [Figure 1b].

Discussion

If pituitary apoplexy subsequently occurs, the cranial nerve system that runs into the sinus venosus can be easily damaged by the rapid increase in intracavernous sinus pressure.^[1,2,7]

Instances wherein only the oculomotor nerve is damaged in patients with pituitary adenoma are rare. The following three hypotheses can explain the mechanisms by which the oculomotor nerve is selectively damaged. First, the damage can occur when tumors that invade the cavernous sinus with pituitary apoplexy penetrate the lateral wall of the sinus and progress into the oculomotor cistern.^[2,8] Second, damage can occur when pituitary tumors erode the lateral part of the posterior clinoid process, resulting in a sudden increase in intratumor pressure due to pituitary apoplexy and incarceration of the tumor at the entrance of the oculomotor nerve.^[3] Third, oculomotor nerve damage alone can be caused by abutment of the cavernous sinus due to the sudden increase in mass effect with the pituitary apoplexy, even if the tumor does not invade the cavernous sinus.^[6] In all of these hypotheses, pituitary apoplexy is closely related to oculomotor nerve damage only.

In this patient, the onset of pituitary apoplexy was ruled out; diplopia gradually progressed during 6 months but was not associated with symptoms such as acute headache. In addition, findings such as pituitary hemorrhage were not observed on imaging. Because the tumor did not invade the cavernous sinus based on intraoperative findings and the posterolateral progression of the tumor was observed with right-sided posterior clinoid process erosion on imaging, the pathogenesis of oculomotor nerve palsy in this patient seemed to fall under the second hypothesis.^[3] The tumor was incarcerated in a weak area of the dura at the entrance of the oculomotor nerve and seemed to compress only the oculomotor nerve. In the anatomical study, there are weak points of the cavernous sinus wall against tumor invasion.^[9] The meningeal pocket of the oculomotor nerve is one of the weak points of the cavernous sinus wall. In the pocket, the dural layer is extremely thin or missing, and the tumor can easily extend into the subdural

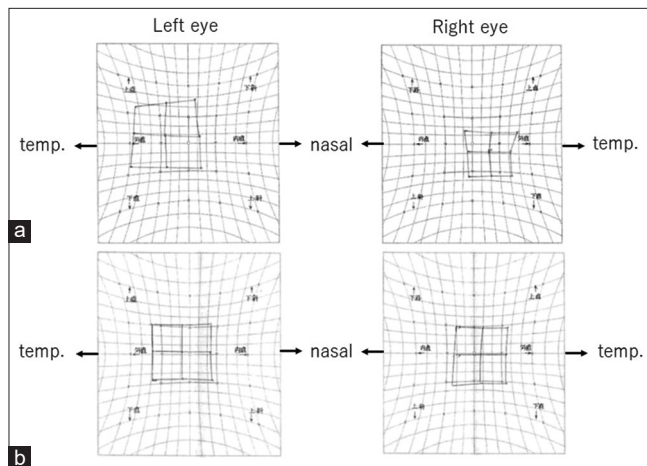


Figure 1: (a) Preoperative Hess chart showing features of a right third nerve palsy. (b) Postoperative Hess chart showing the improvement of ocular movement (14 days after surgery)

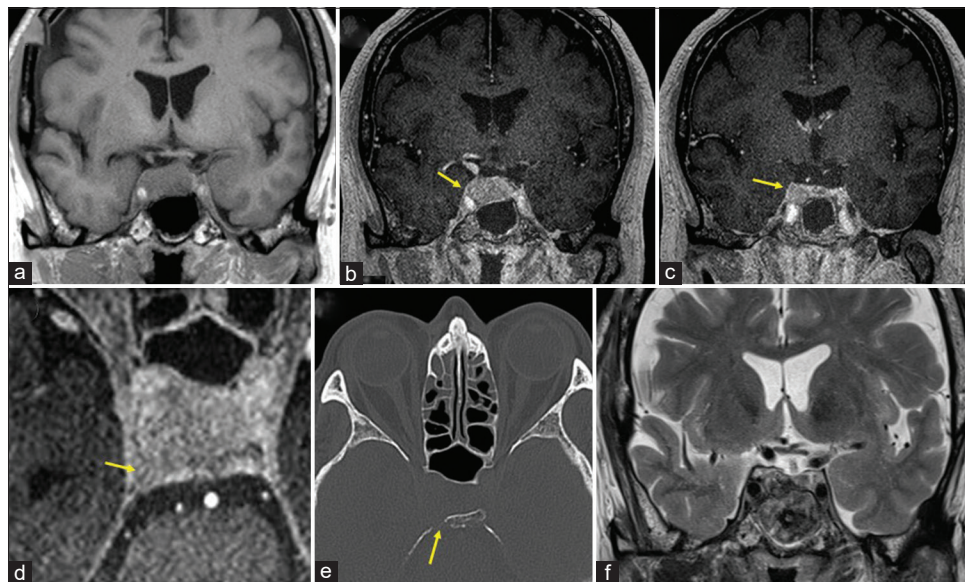


Figure 2: Pre and postoperative images of the patient. (a) T1-weighted magnetic resonance imaging demonstrated a homogeneous pituitary mass that represents nonpituitary apoplexy. (b-d) Gd enhanced T1-weighted magnetic resonance imaging demonstrated the protrusion of the pituitary tumor latero-posteriorly into the right cavernous sinus (yellow arrow). (e) Bone window CT demonstrated the erosion of the right posterior clinoid process (yellow arrow). (f) Postoperative T2-weighted magnetic resonance imaging demonstrated the near-total resection of the pituitary tumor

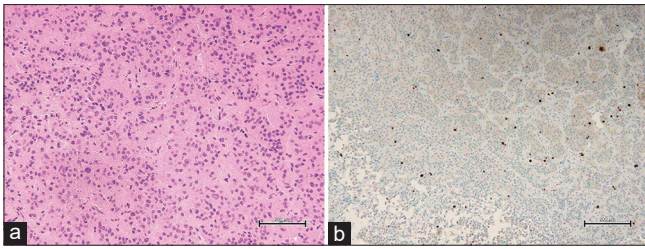


Figure 3: Photomicrographs of hematoxylin-eosin and immunohistochemical staining. (a) Hematoxylin and eosin staining show pituitary adenoma with no evidence of malignancy. (b) Ki67-labeling index was <3%

space. Oculomotor nerve is vulnerable as a result of being kinked or encased by the bulging of the tumor at this point, and it alone can be damaged if the posterior clinoid process is eroded by gradually elevated intratumor pressure without pituitary apoplexy and if the tumor is incarcerated at the entrance of the oculomotor nerve.

The indication of surgical treatment for pituitary apoplexy presenting with oculomotor nerve palsy is controversial, and a consensus has not been obtained yet.^[7,8,10] Many have reported that symptom resolution can be expected even with conservative treatment if the mass effect is reduced.^[8,10] However, in this patient, improvement in oculomotor nerve palsy was not observed over 6 months. Surgical treatment is essential for oculomotor damage caused by enlarging tumors without pituitary apoplexy because spontaneous remission cannot be expected.

Conclusion

We report a rare case of nonapoplectic pituitary adenoma that was associated with isolated oculomotor nerve palsy. If a tumor grows posterior to the lateral side of the cavernous sinus and erodes the posterior clinoid process, the tumor might damage only the oculomotor nerve without apoplexy. Surgical treatment is essential because oculomotor nerve palsy caused by enlarging tumors is not expected to resolve with conservative treatment.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and

other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Hage R, Eshraghi SR, Oyesiku NM, Ioachimescu AG, Newman NJ, Biousse V, *et al.* Third, fourth, and sixth cranial nerve palsies in pituitary apoplexy. *World Neurosurg* 2016;94:447-52.
2. Hoang N, Tran DK, Herde R, Couldwell GC, Osborn AG, Couldwell WT. Pituitary macroadenomas with oculomotor cistern extension and tracking: Implications for surgical management. *J Neurosurg* 2016;125:315-22.
3. Kobayashi H, Kawabori M, Terasaka S, Murata J, Houkin K. A possible mechanism of isolated oculomotor nerve palsy by apoplexy of pituitary adenoma without cavernous sinus invasion: A report of two cases. *Acta Neurochir (Wien)* 2011;153:2453-6.
4. Komurcu HF, Ayberk G, Ozveren MF, Anlar O. Pituitary adenoma apoplexy presenting with bilateral third nerve palsy and bilateral proptosis: A case report. *Med Princ Pract* 2012;21:285-7.
5. Lau KK, Joshi SM, Ellamushi H, Afshar F. Isolated bilateral oculomotor nerve palsy in pituitary apoplexy: Case report and review. *Br J Neurosurg* 2007;21:399-402.
6. Rossitch E Jr., Carrazana EJ, Black PM. Isolated oculomotor nerve palsy following apoplexy of a pituitary adenoma. *J Neurosurg Sci* 1992;36:103-5.
7. Rutkowski MJ, Kunwar S, Blevins L, Aghi MK. Surgical intervention for pituitary apoplexy: An analysis of functional outcomes. *J Neurosurg* 2018;129:417-24.
8. Fan Y, Bao X, Wang R. Conservative treatment cures an elderly pituitary apoplexy patient with oculomotor paralysis and optic nerve compression: A case report and systematic review of the literature. *Clin Interv Aging* 2018;13:1981-5.
9. Kawase T, van Loveren H, Keller JT, Tew JM. Meningeal architecture of the cavernous sinus: Clinical and surgical implications. *Neurosurgery* 1996;39:527-34.
10. Seo Y, Kim YH, Dho YS, Kim JH, Kim JW, Park CK. The outcomes of pituitary apoplexy with conservative treatment: Experiences at a single institution. *World Neurosurg* 2018;115:E703-10.