Neurology Asia 2024; 29(1) : 273 – 276

A case of pituitary apoplexy presented with isolated complete oculomotor nerve palsy

[1]Sumonthip Leelawai *MD*, [2]Nuttha Sanghan *MD*, [1]Suwanna Setthawatcharawanich *MD*, [1]Pat Korathanakhun *MD*, [1]Thanyalak Amornpojnimman *MD*, [1]Pornchai Sathirapanya *MD*

[1]Department of Medicine, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkhla, Thailand; [2]Department of Radiology, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkhla, Thailand

Abstract

The oculomotor nerve (CN3) innervates four extra-ocular muscles and contains parasympathetic fibers controlling pupillary light reflex (PLR). CN3 palsy with impaired PLR or complete CN3 palsy usually suggests a compressive lesion against the CN3 because the parasympathetic fibers located superficially on the cranial nerve trunk are affected. A cerebral aneurysm originated from posterior communicating artery, posterior cerebral artery or superior cerebellar artery is a common cause of isolated complete CN 3 palsy. Here, we reported a less common intracranial lesion causing isolated complete CN3 palsy for which the preceding recurrent temporal headaches before the CN3 palsy assisted the neurological localization.

Keywords: Midbrain; oculomotor nerve; pituitary apoplexy; pupillary reflex

INTRODUCTION

The parasympathetic fibers controlling pupillary light reflex originating from Edinger-Westphal nucleus (EWN) are located superficially at dorso-lateral part of oculomotor nerve (CN3) trunk. Therefore, CN3 palsy associated with impaired pupillary light reflex (iPLR) or complete CN3 palsy is usually caused by a compressive lesion on the CN3. For example, temporal lobe tumor, uncal herniation, or cerebral aneurysm originated from the vessels forming circle of Willis, particularly posterior communicating artery aneurysm (PComA) is frequently diagnosed in this clinical condition. Because of an intracranial compressive cause, the patient presents with complete CN3 palsy usually requires an emergent neurosurgical treatment. However, we report a case of pituitary apoplexy presented with isolated complete CN3 palsy who responded favorably to the conservative treatment.

CASE REPORT

A 58-year-old male with underlying diseases of well-controlled essential hypertension and hyperlipidemia experienced the first sudden and severe nocturnal left temporal headache which woke him up. The neurological examination was unremarkable on the initial evaluation, and the headache resolved a few days later. A week following the first headache, he had gradual left ptosis and binocular diplopia. The neuro-ophthalmological examination at this time revealed left complete ptosis, paralysis of all extraocular muscles innervated by CN3 and associated iPLR. A panel of routine blood tests showed unremarkable results. Thence, a magnetic resonance imaging of the brain (brain MRI) was done before transferring to our center. The brain MRI revealed acute and subacute hemorrhage in the pituitary adenoma, or pituitary apoplexy (Figure 1).

On the second admission day in our hospital, he experienced the second bout of more severe left temporal headache without further neuro-ophthalmological abnormalities were found. The recurrent left temporal headache slowly improved and completely resolved in two weeks following the conservative treatment and close neurological monitoring, while the isolated complete CN3 palsy remained stable. All pituitary hormone levels revealed normal results, so the patient was discharge from our center. Finally, the clinical presentation of isolated complete CN3

Address correspondence to: Pornchai Sathirapanya, M.D., Department of Medicine, Faculty of Medicine, Prince of Songkla University, 15 Kanchanawanich Road, Hat Yai, Songkhla 90110, Thailand. Tel 6674-451451-2, Email: sporncha@medicine.psu.ac.th

Date of Submission: 5 November 2023; Date of Acceptance: 4 December 2023

https://doi.org/10.54029/2024nzf

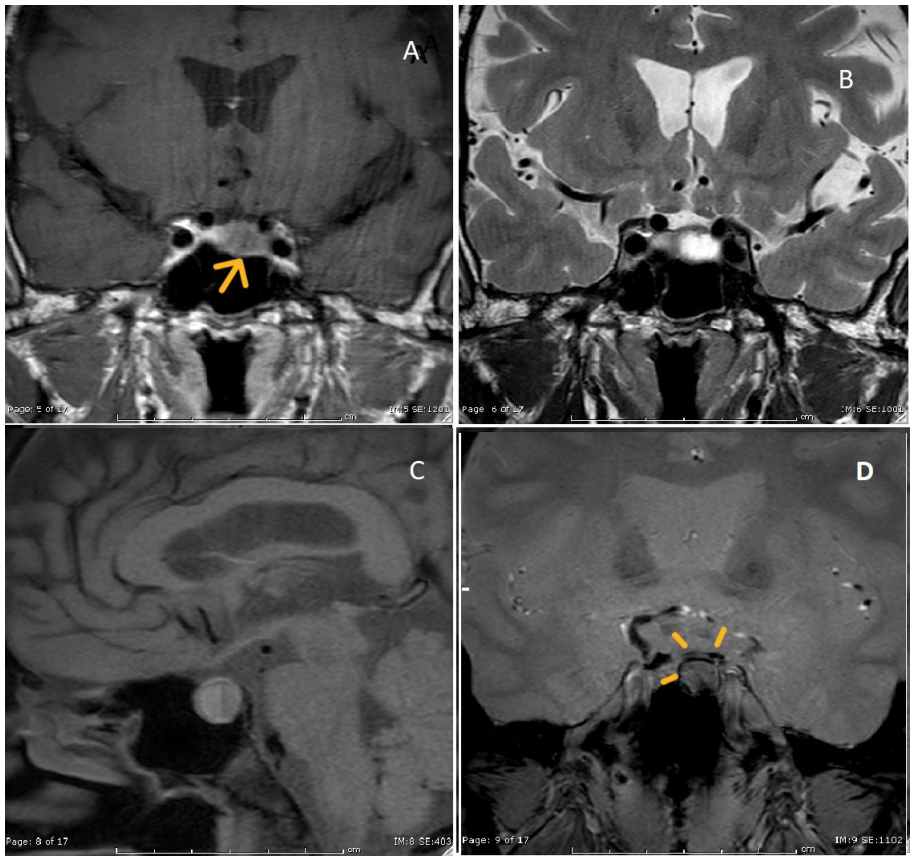

Figure 1. Magnetic resonance images of pituitary gland, (A) Coronal T1W with gadolinium enhancement demonstrated a slightly enhanced nodule at left side of the pituitary gland suggestive of pituitary adenoma (arrow). (B) Coronal T2W revealed hyperintense signal intensity in the adenoma. (C) Fluid-fluid level on sagittal T1W with fat suppression suggested different stage of internal hemorrhage in the adenoma, which the slightly hyperintense signal intensity was subacute hemorrhage, while the isointense signal was acute hemorrhage. (D) Coronal gradient echo image showed hypointense rim in the adenoma confirmed the presence of blood content (tips of the orange lines)

palsy completely resolved 4 months after the discharge. Also, the repeated brain MRI revealed complete resolution of the previous pituitary internal hematoma and he returned to his normal neurological condition as before.

DISCUSSION

The oculomotor nerves arise from oculomotor nuclear complex located at dorsal midbrain. The nuclear complex comprises a) Edinger-Westphal nuclei which provide preganglionic parasympathetic input to ciliary ganglions for mediating pupillary light reflex and accommodation of lens, and b) motor subnuclei which innervate superior rectus, inferior rectus, medial rectus and inferior oblique muscles. Both parasympathetic and oculomotor fibers form the oculomotor fascicles coursing dorsoventrally in the paramedian midbrain and exiting as CN3s from the ventral midbrain. The causes of complete CN3 palsy are mostly an extra-pontine compressive lesion against the CN3 trunk especially PComA, aneurysms of the other arteries forming circle of Willis, or a cerebral tumor at the medial temporal lobe. According to a population-based study of CN3 palsy, associated diabetes or essential hypertension is the most common etiology found (42%), followed by head trauma (12%), and temporal lobe tumor compression (11%), while pituitary apoplexy was found in only 2%.[1] Notably, the study found associated iPLR 16% in the patients with non-compressive, whereas 64% in compressive causes.[1]

The recurrent temporal headache in this patient was helpful in localizing a lesion in middle cranial fossa, especially at sella and parasella regions where pituitary gland is located. A lesion

located in middle cranial fossa usually produces a fronto-temporal or temporal headache and the laterality of the headache depends on the side of lesion location. As in our case, the hematoma was located on the left lobe of pituitary gland causing left temporal headaches. Because the common cerebral aneurysms are located more posteriorly near the posterior cranial fossa, it is unusual to cause temporal headache. Furthermore, when considering anatomical location of CN3, it is located at the top of cavernous sinus which lies along parasella region making it very close to pituitary gland. Hence, CN3 is highly susceptible to compression from an expanding pituitary adenoma or pituitary apoplexy like the presentation of this reported case.

Pituitary apoplexy is caused by internal hemorrhage, infarction or ischemic necrosis of a pre-existing pituitary adenoma. A few studies also suggested that the compression against internal carotid arteries by pituitary adenoma could result in pituitary infarction and apoplexy.[2-4] Fronto-temporal headache and isolated unilateral cranial nerve palsy either optic, oculomotor[3,5-8], or less frequently abducens nerve[9], were reported in pituitary apoplexy. Anecdotally, bilateral oculomotor or abducens nerve palsies were reported as well.[4,10-11] Moreover, a case of sudden diffused headache, rapidly depressed consciousness and isolated complete CN3 palsy mimicking a ruptured PComA was reported.[12]

There is no consensus regarding treatment for mild cases of pituitary apoplexy, particularly when no visual dysfunction is associated. One study reported a comparable outcome between pituitary surgical decompression and conservative medical treatment with close neuro-ophthalomological and endocrinological functions monitoring.[13] In the reported case, it is possible that the initial pituitary internal bleeding was self-limited for which the patient's first headache resolved, but rebleeding occurred subsequently producing the recurrent left temporal headache. This explanation is corresponding with the findings of the brain MRI showing different stages of internal pituitary bleeding.

Despite a less common cause of isolated complete CN3 palsy, pituitary apolplexy should be considered as a possible diagnosis, especially when fronto-temporal headache is associated.

ACKNOWLEDGEMENT

The autors thank David Patterson of the International Affairs Office, Faculty of Medicine, Prince of Songkla University for editing the English of this manuscript. We also thank Sakai Thongchuea of the Department of Radiology for his assisstance in technical adjustment of the selected MRI images to meet the publication requirements.

DISCLOSURE

Ethics: This manuscript was reviewed and approved by the Ethics Committee of the Faculty of Medicine, Prince of Songkla University (EC code no. 63-012-14-4). The consent for publication was obtained.

Financial support: None

Conflict of interest: None

REFERENCES

1. Fang C, Leavitt JA, Hodge DO, Holmes JM, Mohney BG, Chen JJ. Incidence and etiologies of acquired third nerve palsy using a population-based method. *JAMA Ophthalmol* 2017;135:23-8. doi: 10.1001/jamaophthalmol.2016.4456.
2. Cahill M, Bannigan J, Eustace P. Anatomy of the extra-neural blood supply to the intracranial oculomotor nerve. *Br J Ophthalmol* 1996;80:177-81. doi:10.1136/bjo.80.2.177.
3. Shrestha R, Bishokarma S, Rayamajhi S, *et al*. Pituitary apoplexy presenting as isolated third cranial nerve palsy: case series. *J Surg Case Rep* 2022;2022:rjac386. doi: 10.1093/jscr/rjac386.
4. Perotti V, Dexter M. Post-partum pituitary apoplexy with bilateral third nerve palsy and bilateral carotid occlusion. *J Clin Neurosci* 2010;17:1328-30. doi: 10.1016/j.jocn.2010.03.002.
5. Rosso M, Ramaswamy S, Sucharew H, Vagal A, Anziska Y, Levine SR. Isolated third cranial nerve palsy in pituitary apoplexy: Case report and systematic review. *J Stroke Cerebrovasc Dis* 2021;30:105969. doi: 10.1016/j.jstrokecerebrovasdis.2021.105969.
6. Sasagawa Y, Aburano H, Ooiso K, Oishi M, Hayashi Y, Nakada M. Oculomotor nerve palsy in pituitary apoplexy associated with pituitary adenoma: a radiological analysis with fast imaging employing with steady-state acquisition. *Acta Neurochir (Wien)* 2021;163:383-9. doi: 10.1007/s00701-020-04632-y.
7. 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. doi: 10.2147/CIA.S181109.
8. Gupta VK, Mamik HK, Gupta N, Bhagat RK. Pituitary apoplexy presenting as isolated unilateral third nerve palsy. *Neurol India* 2022;70:1689-91. doi: 10.4103/0028-3886.355153.
9. Syed SB, Mourra AA, Chatterjee T. Isolated unilateral abducens nerve palsy manifesting as a rare complication of idiopathic pituitary apoplexy: A case report. *Cureus* 2022;14:e22408. doi: 10.7759/cureus.22408.

10. Hage R, Eshraghi SR, Oyesiku NM, *et al*. Third, fourth, and sixth cranial nerve palsies in pituitary apoplexy. *World Neurosurg* 2016;94:447-52. doi: 10.1016/j.wneu.2016.07.026.
11. Singh A, Khurana M, Pal H, Azad S, Sihag RK, Kumar B. Bilateral sixth cranial nerve palsy, the first presenting feature of hemorrhagic apoplexy of pituitary macroadenoma: a case report. *Int J Surg Case Rep* 2022;98:107522. doi: 10.1016/j.ijscr.2022.107522.
12. Mehkri Y, Leone E, Sharaf R, *et al*. Pituitary apoplexy: delayed diagnosis of a rare clinical syndrome in a patient with a known pituitary adenoma. *Cureus* 2022;14:e31536. doi: 10.7759/cureus.31536.
13. Hanna V, Mednick Z, Micieli J. Rapid resolution of a third nerve palsy from pituitary apoplexy. *BMJ Case Rep* 2021;14:e241850. doi: 10.1136/bcr-2021-241850.