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COMPLETE UNILATERAL OCULOMOTOR NERVE PALSY WITH PUPILLARY INVOLVEMENT AS MANIFESTATION OF ANEURIYSM

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ABSTRACT
Background: Oculomotor nerve palsy is an ocular pathology resulting from damage to third cranial nerve and might be complete or partial, with or without pupillary involvement. Involvement of the pupillary function might indicate presence of life-threatening condition, especially associated with posterior communicating artery aneurysms, due to the anatomical proximity of the nerve to the aneurysmal wall.This case report is aimed to describe the best approach to find the associated etiology of unilateral complete oculomotor nerve palsy with pupillary involvement.
Case Presentation: A 74-year-old woman came with a chief complaint of drooping of the right eyelid since two weeks ago. Abnormal position and double vision of the eye was present in the past three weeks. The patient has hypertension and history of stroke one year ago. Right ocular motility showed reduction of motion to superior, inferior and medial with no pain on eye movement. Anterior segment of the right eye showed upper eyelid ptosis. Ocular motility, anterior segment of the left eye and posterior segments of both eyes were within normal limits. Computed Tomography (CT) angiography of head and orbits showed aneurysms at junction of posterior communicant artery with right internal carotid artery, anterior communicant artery with left anterior cerebral artery and branch of left medial cerebral artery.
Conclusion: Deep historical anamnesis, complete ocular evaluation along with appropriate imaging examination is needed to find the etiological factor of oculomotor nerve palsy.
Keywords*:* CT angiography, oculomotor palsy, pupillary involvement.

BACKGROUND

Oculomotor nerve palsy is an ocular pathology resulting from damage to third cranial nerve. The incidence of acquired oculomotor nerve palsy in patients aged older than 60 years old is 12.5 per 100.000 per year compared with 1.7 per 100.000 per year for younger patients. Disorders that produce dysfunction of oculomotor nerve might be located anywhere from the ocular motor nuclei to the termination of the nerves in the extraocular muscles within the orbit. Oculomotor nerve palsy might be complete or partial with or without pupillary involvement. Complete oculomotor nerve palsy shows downward and outward deviation of the eye; complete ptosis; and inability to adduct, infraduct, or supraduct the eye. Partial oculomotor nerve palsy presents with limitation of supraduction, infraduction, adduction.[1-4]

Involvement of the pupillary function might indicate presence of life-threatening condition such as intracerebral aneurism. Other causes are low-flow carotid-cavernous sinus fistula, tumour, or other compressive lesions.Complete and holistic evaluation is essential in determining the origin of the disease in order to find the definitive treatement.[1,2,5,6] This case report is aimed to describe the best approach to find the associated etiology of unilateral complete oculomotor nerve palsy with pupillary involvement.

CASE PRESENTATION

A 74-year-old woman came to Neuro Ophthalmology Unit with a chief complaint drooping of the right eyelid since two

weeks ago. Symptom was not relieved with rest nor worsen with sustained activity. Patient also complained abnormal position and double vision in the past three weeks and blurred vision since eight months ago. There was no headache, nausea and pain around the eyeball. There was no history of trauma, frequently chocking, shortness of breath, nor sudden muscle weakness. There was also no history of wearing glasses, surgery in both eyes, and routine consumption of certain drugs. Patient has uncontrolled hypertension and history of stroke on 2019.

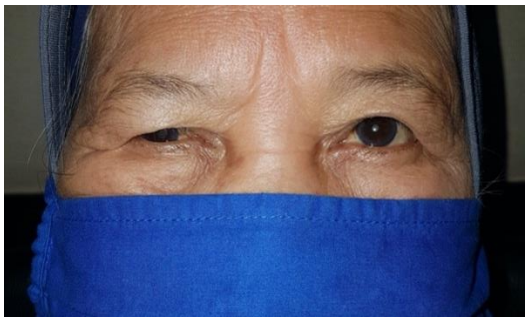

Figure 1 Primary Position of the eye

General status was within normal limits with blood pressure 160/100 mmHg. Visual acuity was cross face finger counting on the right eye and 0.2 on the left eye. Intraocular pressures with a noncontact tonometer (NCT) were 16 mmHg on the right eye and 14 mmHg on the left eye. The primary eye position was exotropia of 15 degree (Figure 1). Right ocular motility showed reduction of motion (-4) to superior, inferior and medial with no pain on eye movement (Figure 2). Left ocular motility showed full range of motion with no pain on eye movement.

Anterior segment of the right eye showed upper eyelid ptosis. Margin Reflex Index 1 (MRD 1) was -2 mm, MRD 2 was 5 mm, and Inter Palpebral Fissure (IPF) was 3 mm. The pupil of right eye was fixed and dilated with no reverse Relative Afferent Pupillary Defect (RAPD) on contralateral eye. There were lens opacity on both eyes (NO4NC4P4-5 OD and NO2NO3P1 OS).

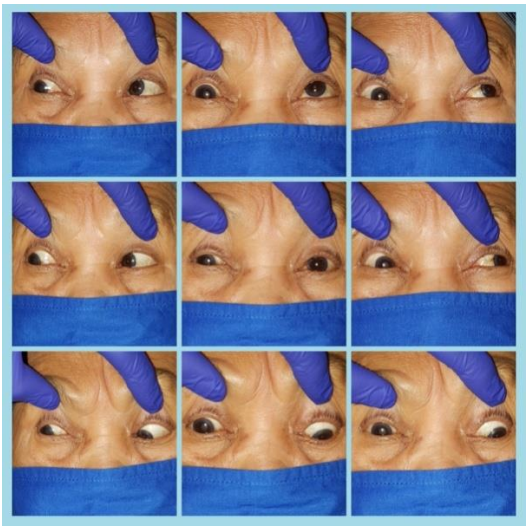

Figure 2 Restriction in eye movement to superior, inferior and medial

Other anterior examination of the left eye was within normal limit. Margin Reflex Index 1 (MRD 1) was 4 mm, MRD 2 was 6 mm, and Inter Palpebral Fissure (IPF) was 10 mm. The posterior segment showed normal optic disc with C/D ratio 0.3 – 0.4 on both eyes.

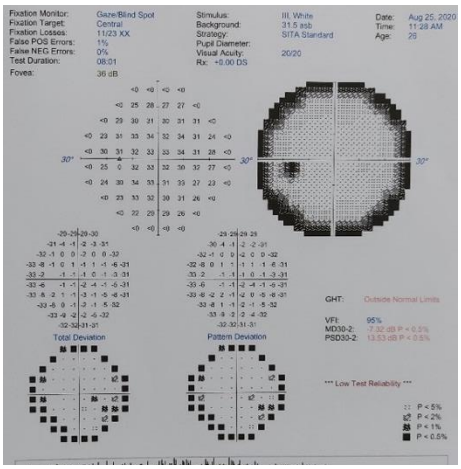

Figure 3 Humphry 24-2 visual field test of left eye

Cogan eye lid twitch test, stare test, and the ice pack test were negative. There was also no orbicularis muscle weakness. Neurological examination of other cranial nerves was within normal limits. Motor function of both extremities were strong (+5). Patient underwent Humphry 24-2 visual fields test, Optical Coherence Tomography (OCT) of optic disc.

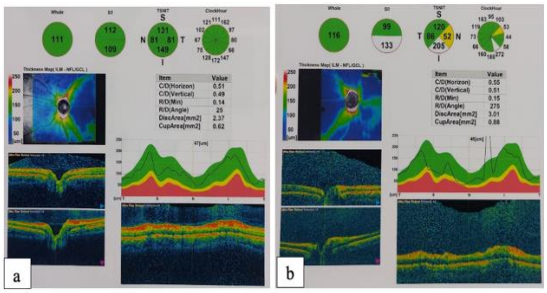

Figure 4 Optical Coherence Tomography (OCT) of optic disc: (a) left eye; (b) right eye

Examination of Humphry 24-2 visual field test of the right eye was not applicable due to limitation of visual acuitywhile normal visual field found on the left eye (Figure 3). OCT of the optic discs were within normal limits on both eyes (Figure 4).

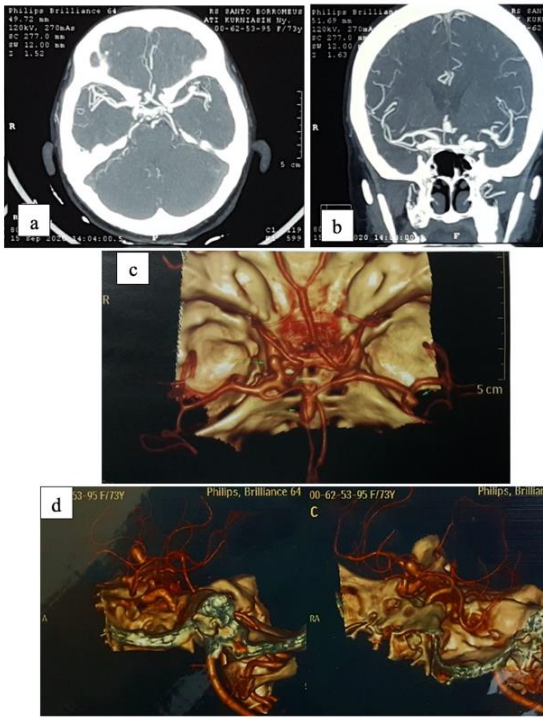

Figure 5 CT of Head – Orbits(a-b) and CT angiography (c-d)

Patient was diagnosed with complete oculomotor nerve palsy OD with pupillary involvement due to suspect aneurism with space occupying lesion and ocular myasthenia gravis as its differential diagnosis. Coherence Tomography (CT) scan of head and orbit was ordered along with CT Angiography.

CT of head and orbits showed mild cerebri and cerebelli atrophy. CT angiography showed aneurysms at junction of posterior communicant artery with right internal carotid artery, anterior communicant artery with left anterior cerebral artery and branch of left medial cerebral artery. (Figure 5).

Patient was diagnosed with complete oculomotor nerve palsy OD with pupillary involvement due multiple aneurysms. Patient were treated with citicoline 1x1000 mg orally and referred to neurosurgeon.

DISCUSSION

Oculomotor nerve palsy is an ocular pathology resulting from damage to third cranial nerve. Disorders that produce dysfunction of oculomotor nerve might cause both somatic extraocular muscle or autonomic pupillary sphincter and ciliary muscle dysfunction. CN III nucleus is located dorsally within the midbrain. The fascicles of CN III pass the red nucleus, substantia nigra, and corticospinal tract. It exits the cerebral peduncle and enter the subarachnoid space. It passes between Superior Cerebral Artery (SCA) and Posterior Cerebral Artery (PCA), lateral to Posterior Communicating Artery (PCoA). The pupillary fibres are found on the dorsomedial surface of the nerve, making them vulnerable to compression. At the back edge of dura of the clivus and cavernous sinus, the nerves enter Dural canal then separates into two division, superior and inferior. Both runs through the superior orbital fissure within annulus of Zinn. The superior division innervates the superior rectus and levator palpebrae

superioris muscle. The inferior division supply parasympathetic fibres and innervates medial rectus, inferior oblique, and inferior rectus muscle. Disorders may be located anywhere from the ocular motor nuclei to the termination of the nerves in the extraocular muscles within the orbit.[1,2,7,5]

Lesions on oculomotor nucleus are uncommon and produce bilateral defects in ocular motility eyelid position, or both. Lesion on fascicular oculomotor nerve produce both complete and incomplete palsies and difficult to differentiate clinically from lesions outside brainstem. Most lesion on fascicle may be isolated or associated with other neurologic manifestations which has characteristic syndromes.[1,2,5,8,9] In this case, unilateral defect was found with no other neurologic manifestation or other cranial nerves involvements. From these findings, we might assume that the lesion was not found on the nucleus nor the fasciculus of oculomotor nerve palsy.

Lesion in the subarachnoid space may produce complete or partial palsy with or without pupillary involvement. Complete oculomotor nerve palsy shows downward and outward deviation of the eye; complete ptosis; and inability to adduct, infraduct, or supraduct the eye. On the other hand, partial oculomotor nerve palsy presents with limitation of supraduction, infraduction and adduction. The pupil might be or might not be involved in both abnormalities.[1,2,5,8,9]

This patient was presented with right ocular motility showed reduction of motion (-4) to superior, inferior and medial with no pain on eye movement. Ptosis was also found on the right eye. These indicates complete involvement of oculomotor nerve palsy.

Compressive lesion of oculomotor nerve usually affect both the central somatomotorfibers and the peripheral superomedial pupil fibers, while ischemic lesions spare the latter. This anatomy is the basis for the ‘rule of the pupil’, which states that a complete motor third-nerve palsy with a normal pupil is most probably ischemic in origin and not compressive. Pupillary dysfunction with oculomotor nerve palsy result from parasympathetic input. The pupillary pathway starts in the mesencephalon with the visceral oculomotor nuclei, along to the oculomotor nerve to the ciliary ganglion and reaches the iris sphincter through short ciliary nerves. Lesion along this pupillary pathway might produce complete paralysis of pupillary constriction thus results in mid-dilated pupil that responds poorly to light. This combination of iridoplegia and cyclopegia often called internal ophthalmoplegia.[1,2,5,10]

Pupillary involvement was also found in this patient. Patient was presented with fixed and dilated right pupil with no reverse RAPD. This is a strong indication that the parasympathetic pathway was affected. Several aetiology might contributes to the pupillary involvement of oculomotor nerve palsy. One of the most life-threatening cause is aneurysm that arise at the junction of posterior communicating artery (PCoA) and Internal Carotid Artery (ICA). Aneurysm are adjacent to oculomotor nerve and might produce oculomotor nerve palsy as the initial manifestation of aneurismal expansion of rupture. Although other origins such as low-flow carotid-cavernous sinus fistula, tumour, and other compressive lesions might also contribute to pupillary involvement, until proven otherwise, nontraumatic oculomotor nerve palsy with pupillary involvement must be worked up secondary to an aneurism.[1,2,5,6,11-13]

Behaves on this reason, this patient was firstly diagnosed with complete oculomotor nerve palsy OD with pupillary involvement due to aneurism. Oculomotor nerve palsy with pupillary involvement is

one of a neuro-ophthalmology emergency. Thus, complete and immediate work up is needed.Complete oculomotor nerve palsy with pupillary involvement has the highest risk of compressive lesion as its aetiology thus best assessed through magnetic resonance imaging (MRI)/ magnetic resonance angiography (MRA) or computed tomography angiography (CTA). If normal result was found, further examination needs to be done through catheter angiography (Figure 6). Both MRA and CTA might reliably detect as small as 3 mm aneurism. However, between these two modalities, CTA is faster and provide images with slightly greater resolution. Catheter angiography is mostly used to confirm the diagnosis and definitive treatment of aneurism, but it is rarely used as diagnostic tool alone.[1-3,11] Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) might show better details of images but computed tomography (CT) and computed tomography angiography (CTA) gives faster results. Due to this reason, Computed Tomography of head and orbit along with CTA were ordered for this patient. Result showed aneurisms at junction of posterior communicant artery with right internal carotid artery, anterior communicant artery with left anterior cerebral artery and branch of left medial cerebral artery. Patient was then diagnosed with complete oculomotor nerve palsy OD with pupillary involvement due to aneurism.

Prognosis of acquired oculomotor nerve palsy may varied. Complete recovery may occur 1-2 weeks after onset of symptoms. Others complete within three months or even more. Prolonged recovery occurs especially after damage of fascicular portion of the nerve. The use of steroid has been reported but with inconsistent varying results. Patients with residual deficit might consider prism or strabismus surgery after 6-12 months of stability to maximize the surgical outcome. In case of chronic compression, infiltration of a tumour, or damaged by trauma, oculomotor nerve palsy might persist and completely unchanged.[1-3,14,15] Oculomotor nerve palsy in this patient was due to aneurysm thus complete recovery might be difficult. Patient was referred to neurosurgeon for further treatment.

CONCLUSION

Disorders located anywhere from the third cranial nerve nuclei to the termination of the nerves might resulted in oculomotor nerve palsy. Complete oculomotor nerve palsy with pupillary involvement is a neuro- ophthalmology emergency since its commonly caused by intracerebral aneurism. Deep historical anamnesis, complete ocular evaluation along with appropriate imaging examination are needed to find the etiological factor of this life- threatening condition.

REFFERENCE

1. Jameson JL, Kasper DL, Longo DL, S.Fauci A, L.Hauser S, Loscalzo J. Walsh and Hoyt's : Clinical Neuro-Ophthalmology. 2018.
2. AAO. Neuro-Ophthalmology. Vol. 5. American Academy of Ophthalmology; 2013. 2019–2020.
3. Colon-Acevedo B. Acquired Oculomotor Nerve Palsy [Internet]. Eyewiki. 2020 [cited 2020 Sep 9]. Available from: https://eyewiki.aao.org/Acquired_Oculomotor_Nerve_Palsy
4. Chengbo Fang, MD, Jacqueline A. Leavitt, MD, David O. Hodge, MS, Jonathan M. Holmes, BM , BCh, Brian G. Mohney, MD, and John J. Chen, MD P. Incidence and Etiologies of Acquired Third Nerve Palsy Using a Population-Based Method. Physiol Behav. 2016;176(1):139–48.
5. Bowling B. Kanski's Clinical Opthalmology. 8th ed. Sydney: Elsevier; 2016. 271, hlm 598–615.
6. Lin H Le, Hu T Te. Isolated third nerve palsy with pupillary involvement resulting from carotid-cavernous sinus fistula: A case report. Med (United States). 2019;98(6):9–12.
7. AAO. Basic Principles of Ophthalmic Surgery. Vol. 1. 2006.
8. Sikod A, Ahmed A, Mcmillan-persaud B, Kelsey-harris R. A Misdiagnosis of Partial Oculomotor Nerve Palsy : A Curious Case of Stroke. Sci Forschen J Neurol Neurobiol. 2016;3–5.
9. Bhatti MT, Eisenschenk S, Roper SN, Guy JR. Superior divisional third cranial nerve paresis: Clinical and anatomical observations of 2 unique cases. Arch Neurol. 2006;63(5):771–6.
10. Trobe JD. The Neurology of Vision. 2nd ed. New York: Oxford University Press, Inc.; 2001.
11. DeLengocky T, Bui CM. Complete ophthalmoplegia with pupillary involvement as an initial clinical presentation of herpes zoster ophthalmicus. J Am Osteopath Assoc. 2008;108(10):615–21.
12. Lee TY, Ting CY, Tsai MJ. Third nerve palsy and internal carotid aneurysm. Qjm. 2016;109(11):755–6.
13. Dhume K, Paul K. Incidence of pupillary involvement, course of anisocoria and ophthalmoplegia in diabetic oculomotor nerve palsy. Indian J Ophthalmol. 2013;61(1):13–7.
14. Masoud Aghsaei Fard, MD Elizabeth Montgomery, MD Neil R. Miller M. Complete, Pupil-Sparing Third Nerve Palsy in a Patient With a Malignant Peripheral Nerve Sheath Tumor. Arch Ophthalmology. 2019;129(6):813–4.
15. Menon S, Menon G, et al. Patterns and predictors of isolated oculomotor nerve palsy recovery following mild traumatic brain injury. Indian J Clin Exp Ophthalmol. 2019;5(3):343–7.