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Case Report : Etiologies and Management of Sixth Cranial Nerve Palsy
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Etiologies and Management of Sixth Cranial Nerve Palsy

Abstract

Objective: to report two cases of sixth nerve palsy

Case report:

Case 1: A 44 year old woman reported a 2 weeks history of horizontal diplopia that was worse at distance. She had a left sixth cranial nerve palsy. The patient had history of stoke for several times. The palsy was resolved gradually in 8 weeeks.

Case 2: A 20 year old woman came to Cicendo Eye Hospital on January 25th 2012 with a chief complaint of blurred and double vision since five days before the admission. She had history of appendicitis surgery and ovarii cyst 2 weeks before. She had left sixth nerve paresis. After giving steroid, the palsy resolved in 4 weeks.

Conclusion: A lesion anywhere along the course of the nerve, from the pons to the orbit, can cause a paresis or palsy. After ruling out trauma and non-neurological problems, cases should be classified into neurologically isolated or non-neurologically isolated cases. Neurologically isolated sixth nerve palsies are associated most commonly with vascular disease. A sixth nerve palsy of vascular or undetermined causes typically resolves within 6 to 8 weeks. Inflammation can be endurance as well as a cause of sixth nerve palsy.

I. Introduction

The sixth cranial nerve, also kwon as abduscens nerve, innervates the ipsilateral lateral rectus, which function to abduct the ipsilateral eye. An abduction deficit, which may be complete (palsy) or incomplete (paresis), results in esotropia and ipsilateral abduction deficiency. Patients will report diplopia that is worse at distance and when looking toward the affected muscle.[1,2,3]

A lesion anywhere along the CN VI course, from the pons to the orbit, can cause a paresis or palsy. Prompt and correct diagnosis by an eye care practitioner is critical in determining the cause and, therefore, the proper evaluation, follow-up, and treatment. Chronic inflammation may cause ipsilateral abduscens palsy. In patients older than 50 years, vascular disease or unknown causes are most common. The following case presents patients with sixth nerve palsy with associated dyslipidemia and inflammation.[4]

II. Case 1

A 44 year old woman came to Cicendo Eye Hospital on January 21th 2012 with a chief complaint of head turn to the left with diplopia since 2 weeks. Separation of objects was only horizontal. She had a history of stroke for four times with asymmetric face since four years ago. There was no history of pain on eye movement, headache, nausea and vomiting. History of trauma was denied. Ophthalmology examination revealed her visual acuity were 0.1 with 0.3 pinhole of both eyes, esotropia, and limitation of movement in abduction on left eye about -4. Intra ocular pressure was within normal limit. Anterior and posterior segment was within normal limit. Laboratoium examination performed 171 of cholesterol LDL number. Head MRI was generally within normal limit, no signs of cerebral infaction, intacerebral haemorrhage, inflammation and SOL. The diagnosis was left sixth cranial nerve palsy caused by suspected ischemic. She was given methilprednisolone oral 1 mg/Kg body weight once a day for two weeks, citicholine 500mg twice a day and roborantia. The progression of eye movement was showed in 8 weeks.

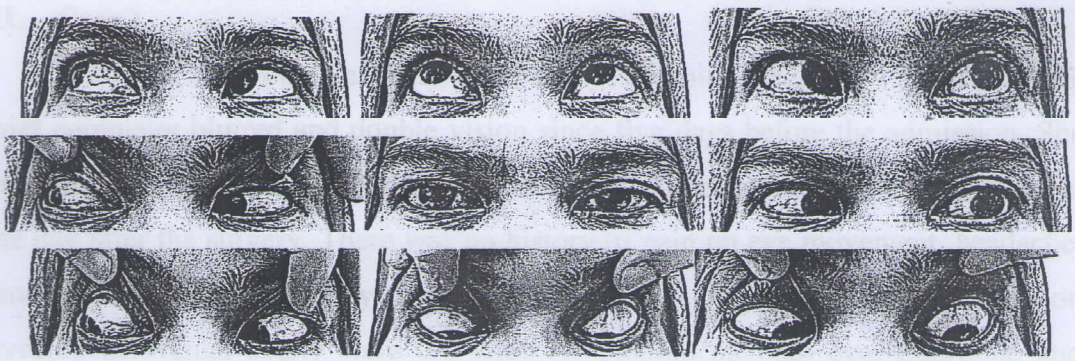

Figure 1. Limitation on abduction movement on left eye on January 27th 2012

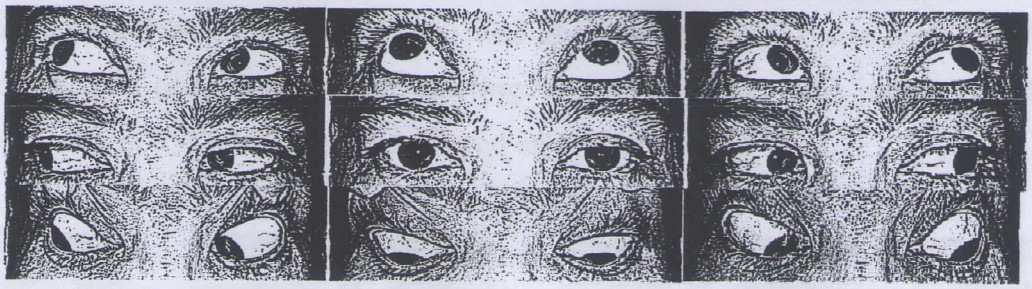

Figure 2. Progression of limitation on abduction movement on left eye on March 16th 2012

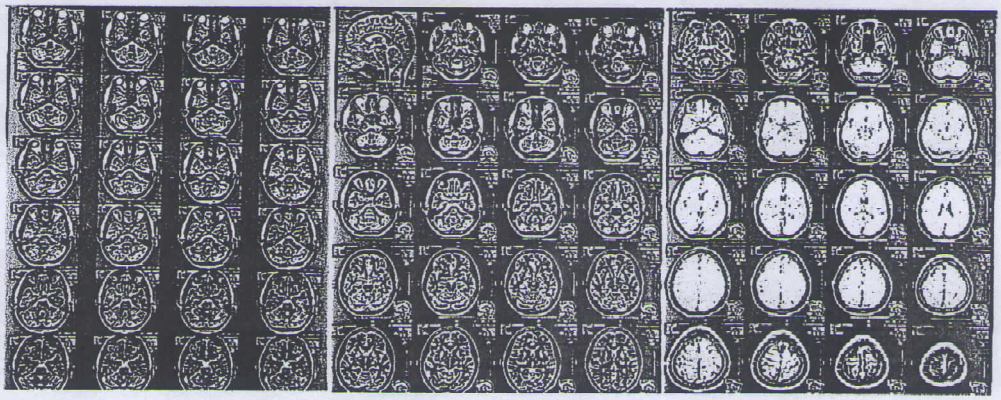

Figure 3. MRI within normal limit

III. Case 2

A 20 year old woman came to Cicendo Eye Hospital on January 25th 2012 with chief complaint blurred and double vision since five days before the admission. She had history of appendicitis surgery and ovarii cyst 2 weeks before. Patient got fever one day after the surgery. There was no history of pain on eye movement, headache, nausea and vomiting. History of trauma was denied. Ophthalmology examination revealed her visual acuity were 0.8 with 1.0 pinhole on both eye, esotropia, and limitation of movement in abduction on right eye about -1. Intra ocular pressure was within normal limit. Anterior and posterior segment was within normal limit. Laboratoium examination performed leukositosis with 16.800 of leukosit. CT Scan was generally within normal limit, no signs of cerebral infaction, intacerebral haemorrhage, inflammation and SOL. The diagnosis was left sixth cranial nerve

paresis caused by inflammation. She was given methilprednisolone oral 1 mg/Kg body weight once a day with tapering after two weeks and roborantia. The progression of eye movement was showed on 4 weeks.

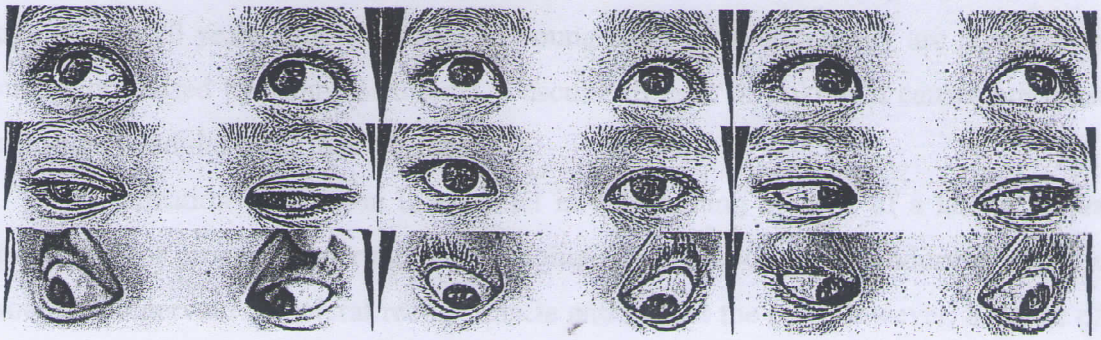

Figure 4. Limitation on abduction movement on left eye, February 1st 2012

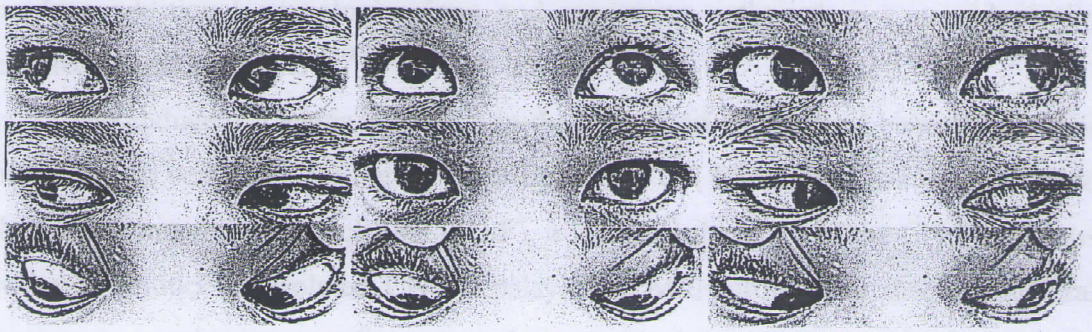

Figure 5. Full abduction movement on left eye, February 22th 2012

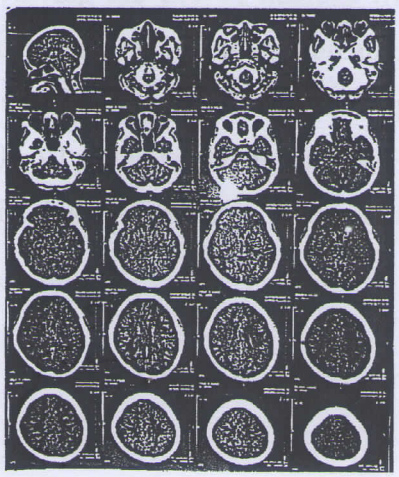

Figure 6. CT scan within normal limit

IV. Discussion

Sixth cranial nerve palsy can occur in all age group, its estimated incidence being 2.5 cases per 100,000 population. Causes of acquired CN VI palsies differ depending on the age of the patient. The peak incidence of CN VI palsy is between age 60 and 70 years. CN VI palsies in young adult most commonly are postviral in nature or caused by tumor or trauma. Vascular disease or unknown causes are most common in adults older than 50 years.[4]

Localization of a lesion is essential in determining a cause of a lateral rectus palsy and for properly ordering neuroimaging. The function of the abducens nerve is solely to innervate the lateral rectus muscle and abduct the eye. However, because of its close proximity to other nerves throughout its cranial course, lesions are likely to produce additional neurologic signs and symptoms rather than a neurologically isolated lateral rectus palsy.[4]

The abducens nucleus is located in the lower part of the pons beneath the floor of the fourth ventricle (*see* Figure 7). Because the CN VI nucleus has projections to the medial longitudinal fasciculus (MLF), a lesion at the level of the CN VI nucleus will not produce an isolated CN VI palsy. The MLF projects to the contralateral oculomotor nucleus to coordinate lateral gaze with the medial rectus in the opposite eye. Therefore, a lesion involving the CN VI nucleus will cause an ipsilateral gaze palsy with sparing of vergence movements rather than an isolated abduction deficit. A lesion near the nucleus can also cause an ipsilateral facial palsy. The facial nerve (CN VII) courses around the CN VI nucleus and is in close proximity to the abducens fascicle (*see* Figure 7). This close relationship accounts for the frequent concurrent damage.[4,5]

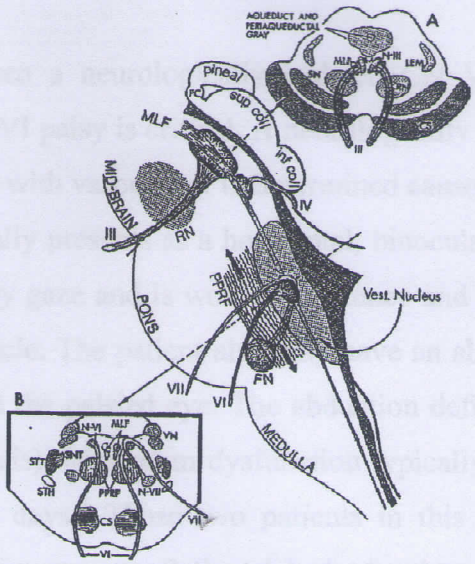

Figure 7. The relationship of CN VI nucleus to the MLF and CN VII.
Sumber: Glaser JS, Siatkowski RM. Neuro-ophthalmology, 3rd ed.[5]

Axons of CN VI emerge from the brainstem in the groove between the pons and the medulla oblongata (*see* Figure 8). The nerve continues forward in the subarachnoid space taking a sharp bend over the petrous portion of the temporal bone before piercing the dura and entering the cavernous sinus. Lesions between the brainstem and cavernous sinus may produce either unilateral or bilaterallateral rectus palsy. Depending on the cause, many other neurologic symptoms may be present including papilledema, facial pain or numbness, or facial nerve palsy.[4]

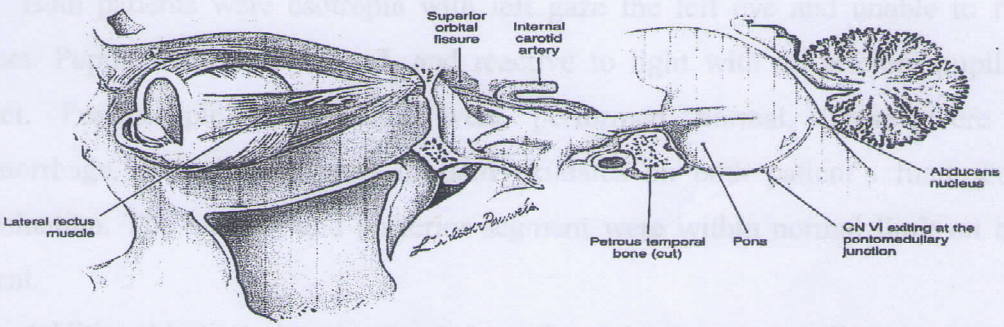

Figure 8. The route of CN VI from the pons to the lateral rectus muscle.

Differentiation between a neurologically isolated CN VI palsy and a non-neurologically isolated CN VI palsy is critical. A neurologically isolated CN VI palsy is predominantly associated with vascular or undetermined causes.[4,6]

A CN VI palsy typically presents as a horizontal, binocular diplopia. Esotropia is usually present in primary gaze and is worse at distance and in lateral gaze in the direction of the paretic muscle. The patient also may have an abnormal head posture with the face turned toward the palsied eye. The abduction deficit may be complete (palsy) or incomplete (paresis). Maximum dysfunction typically occurs at onset but may progress over several days.[4] Those two patients in this case have binocular diplopia and esotropia in primary gaze. Patient 1 had a head turn to the left or to the palsied eye.

Forced ductions are helpful to rule out mechanical limitations of eye movements such as muscle fibrosis, muscle entrapment, tumor, muscle trauma, or inflammation. Resistance, or a positive result with forced duction testing, indicates that the damage does not have a neurogenic origin.[4]

A dilated fundus examination including evaluation of the optic nerve head is required for all patients who present with a CN VI palsy. Increased intracranial pressure can produce papilledema. Retinal vascular changes associated with hypertension and diabetes include a change in blood vessel size, retinal hemorrhages, cotton-wool spots, and hard exudates.[1,4]

Both patients were esotropia with left gaze the left eye and unable to fully abduct. Pupils were equal, round, and reactive to light with no afferent pupillary defect. Funduscopic examination were performed normal. There were no haemorrhage, cotton wool spots or hard exudates on both patient's funduscopic examination. The anterior and posterior segment were within normal limit on both patient.

Additional indicated testing depends on the patient's age as well as the presence of associated neurologic findings. The cause of CN VI palsy in patients abaut 50

years old is most often vascular in nature. Therefore, a complete patient history regarding hypertension and diabetes is necessary. Blood pressure measurements as well as a fasting glucose level or glycosylated hemoglobin should be obtained. If signs of GCA such as headache, scalp tenderness, jaw claudication, fever, weight loss, or malaise are present, especially in patients older than 55 years, an erythrocyte sedimentation rate and C-reactive protein level should be obtained and a temporal artery biopsy should be done. Studies indicate that this testing is not necessary if the ocular motor defect is well defined and the patient is otherwise asymptomatic. A careful history and examination will help differentiate patients that can be observed and those who need neuroimaging. An MRI is recommended for older adults if the CN VI palsy does not resolve within 3 to 6 months, the esotropia is progressing after 2 weeks from its onset, other neurologic signs or symptoms are present, or if the patient has a previous history of malignancy. Patients younger than 45 years with a CN VI palsy require a neurologic workup even if the palsy is neurologically isolated. Cerebrospinal fluid analysis may also be necessary in younger patients if neuroimaging results are inconclusive. A bilateral CN VI palsy should never be considered vascular in origin. MRI and cerebrospinal fluid analysis are required for these patients. [1,4,7]

On this cases report, both patients had laboratory examination, CT Scan or MRI. The laboratoty result of patient 1 performed higher number of cholesterolemia, while leukositosis found on patient 2. The MRI and CT Scan result of both patient within normal limit.

Not all investigators agree with the approach to postpone neuroimaging. Following a prospective study of 43 patients with isolated CN VI palsy referred to a tertiary care center, Bendszus et al. recommended that an MRI be performed on all patients with acute CN VI palsy. Goodwin et al. reported a lesion causing the CN VI palsy was identified in 63% of patients. However, only 15% of these patients were known to have vascular disease, and the average age of these patients was 43 years.

Following established recommendations, imaging would have been performed on patients in this age group especially in the absence of vascular disease.[7]

On patient 1, 44 year old woman with history of stoke and asymmetric face for four times. From physical examination showed limitation on abduction movement and hipercholesterolemia on laboratory examination, we considered patient had sixth cranial nerve palsy caused by ischemic. While on patient 2, 20 year old woman, she had history of appendicitis surgery and ovarii cyst 2 weeks before. She also had fever one day after the surgery. Limitation on abduction movement showed on physical examination with leukositosis on laboratory examination. Based on the age, history, physical examination and laboratory examination, we were considered the sixth cranial nerve paresis caused by inflammation on case 2.

Treatment of an abducens palsy involves treating the underlying medical condition causing the palsy. Treatment of symptoms associated with CN VI palsy includes patching, prism, injection of botulinum toxin (Botox) into the medial rectus, or extraocular muscle surgery if the esotropia is longstanding. Surgery should be considered when the deviation has been stable for at least 6 months. Approximately half of all CN VI palsies recover spontaneously approximately 3 months after onset.[7]

Neuroprotection has been a popular subject of research for optic nerve disease, as a way of preventing the death of retinal ganglion cells. Most studies of neuroprotection on animals use as an outcome measure the number of surviving retinal ganglion cells. Citicholine has shown the greatest promise as a neuroprotective and neuroreparative agent.[8]

In case 1, she was given citicholine 500mg twice a day as a neuroprotective agent for preventing the death of retinal ganglion cells. The progression showed gradually on 8 weeks. On case 2, she given methylprednisolone 1 mg/Kg body weight once a day with tapering. The steroid given to reduce the edema caused by the inflammation. The progression showed on 4 weeks. The prognosis of those patient were quo ad vitam ad bonam and quo ad functionam ad bonam.

Daftar Pustaka

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