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COMPLETE RESOLUTION IN SIXTH CRANIAL NERVE PALSY AND PERIPHERAL SEVENTH CRANIAL NERVE PALSY IN A SIXTEEN YEAR-OLD BOY: A CASE REPORT

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ABSTRACT
Background: Sixth nerve palsy is the predominant cranial nerve palsy observed in children. Pediatric facial nerve palsy can manifest as either a congenital or acquired condition. The issue becomes more prominent due to both functional and aesthetic consequences.
Case Presentation: A 16-year-old boy patient was referred from the pediatric department with cerebral edema and meningoencephalitis. The patient presented with a headache that had been ongoing for one week prior to admission, along with double vision and recurring seizures that had started two days prior to admission. The visual acuity of both eyes was 5/5, indicating normal vision, and the intraocular pressure was within the normal range. The right eye had painless restriction of gaze in the temporal, superotemporal, and inferotemporal regions. The examination of the front part of the eye and the back part of the eye showed no abnormalities. The evaluation of ocular eye movement and facial expression revealed paralysis of the right abducens nerve (CN VI) and facial nerve (CN VII). The magnetic resonance imaging (MRI) brain with contrast evaluation revealed evidence of persistent vasculitis.
Conclusion: Close monitoring is necessary in children with sixth nerve palsy due to the potential for recurrence. Seventh nerve palsy is the most common type of facial paralysis observed in children. It is generally advisable to do a thorough diagnostic examination and differential diagnosis.
Keywords :Complete resolution, pediatric, peripheral seventh nerve palsy, sixth nerve palsy.

BACKGROUND

The prevalence of CN VI palsy is highest among adults and second highest among children among all types of ocular movement paralysis. CN VI palsy can manifest in pediatric patients either from birth or as a result of external factors. Additionally, it might occur as a result of an intracranial tumor. Secondary to open or closed head injuries, trauma can exert indirect pressure on the nerve, resulting in palsy. Increased or decreased intracranial pressure might result in elongation of the CN VI.[1,2] Meningitis is also a frequent cause of CN VI palsy. Risk factors for CN VI palsy include inflammatory and microvascular diseases.[1,3]

Esotropia of the afflicted eye, caused by the unopposed activation of the medial rectus muscle, is the main indicator of CN VI palsy. The esotropia is characterized by being incomitant, meaning that it varies in severity depending on the attempted abduction of the affected eye and the distance at which the eyes are fixated. The most prevalent initial symptom is diplopia. Patients will have horizontal double vision that is more pronounced while looking at distant objects compared to nearby objects. The diplopia is more severe when looking

towards the affected muscle and improves when looking in the opposite direction. The presence of other symptoms related to CN VI palsy is contingent upon the root cause.[3,4]

The identification of CN VI palsy is often established through a clinical examination. Following a clinical examination, the most often used diagnostic method is MRI of the brain and orbit. MRI is advised for all patients below the age of 50 who have non-isolated CN VI palsy, have a previous cancer diagnosis, or lack microvascular risk factors. A lumbar puncture may be considered if the results of the MRI are negative.[2,3]

CN VII palsy encompasses both full loss of function (paralysis) and weakening (paresis) of the CN VII. CN VII palsy can lead to a loss of any of these functions. In addition, individuals may exhibit unilateral or bilateral CN VII paralysis.[1,8] CN VII palsy can have various causes, including idiopathic, congenital, infectious, traumatic, inflammatory, neoplastic, and iatrogenic etiologies.[1,9]

The general population has an estimated overall prevalence of CN VII palsy of two to three occurrences per 10,000 people. CN VII palsy can occur in individuals of any gender, age, or ethnicity. Currently, there is no definitive agreement on whether males or females are more commonly affected. CN VII palsy primarily impacts individuals between the ages of 15 and 45.[1,9]

CASE PRESENTATION

A 16-year-old male was consulted from a pediatric department with brain oedema and meningoenchephalitis suspected for intracranial pressure signs. The patient complained of double vision on his right eye, especially when he glanced to the right, and has been felt since two days before admission. There was no decrease in vision on either his right or left eye. The patient also complained of headaches for one week before admission, followed by seizures of his entire body for two days. Before, there had been no history of fever, trauma, or intracranial tumor. There was also no history of ear infection. He denied having any muscle weakness. He never had hypertension or diabetic mellitus before.

From the general examination, the patient has normal blood pressure in the vital sign, which is 120/70 mmHg, with a heart rate of 50 times per minute, respiratory rate of 20 times per minute, and temperature of 36,8^{0}C. Bedside ophthalmology examination showed his visual acuity of right and left eye was >2/60. Intraocular pressure was within the normal limit in both eyes.

Anterior segment showed within the normal limit. There was neither nystagmus nor lagophthalmos in both eyes. There was neither edema nor spasm in both palpebras. No hyperemia in conjunctiva. Cornea was clear, and deep anterior chamber. Iris was radier in both eyes. Rounded pupils, isochoric, three mm in diameter, normal light reflex, and there was no relative afferent pupillary defect. The lens was also clear in both eyes. As shown in Figure 1, the primary position of the globe was within the normal range.

Figure 1. The patient's eye is in its primary position. Both eyes in central position (picture taken with patient's family consent). Courtesy of RSUD Dr. Soetomo.

From ocular motility examination, it was found that on his right eye there was a moderate limitation to the temporal, superotemporal, and inferotemporal sides, but there was no limitation on his left eye. There was no pain in ocular movement. Nine gaze examinations are revealed in Figure 2.

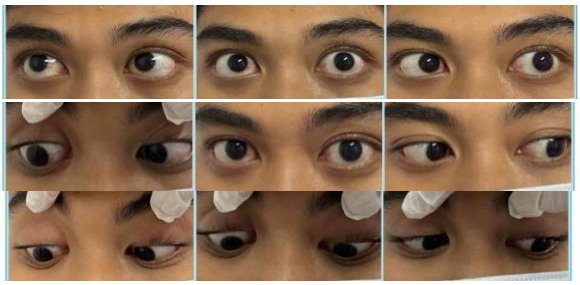

Figure 2. There were moderate limitations to temporal, superotemporal, and inferotemporal gaze on the right eye (picture taken with patient's family consent). Courtesy of RSUD Dr. Soetomo.

Posterior segment examination of both eyes showed optic nerve head normal color, sharp margin, and both cup-disc ratios were 0.3. There was no elevation or peripapillary hemorrhage. The ratio of arteries and veins was 2:3. There were no exudates, hemorrhages, venous beading, or neovascularization. Both eyes have a positive macular reflex as shown in Figure 3.

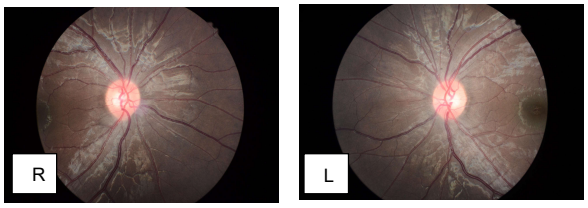

Figure 3. Fundus photography of the patient. Optic nerve head, retina, and macula within normal limits. (The picture was taken with the patient's family consent.) Courtesy of RSUD Dr. Soetomo.

The cranial nerve examination showed there were within the normal limit on the cranial nerve except for CN VI and CN VII. CN VII examination showed weakness on the right eye when lifting the eyebrow, facial asymmetry when smiling, and a hemiparesis lower motor neuron (LMN) type as shown in Figure 4.

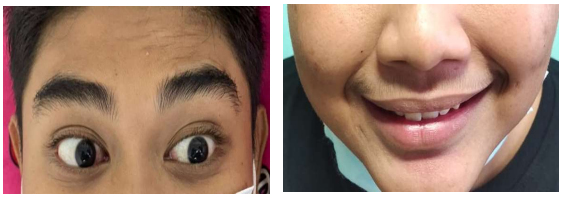

Figure 4. CN VII examination. (Left) Weakness on right side muscle when lifting eyebrow. (Right) Facial asymmetry when smiling. (Picture taken with patient's family consent.) Courtesy of RSUD Dr. Soetomo.

From a head computed tomography scan (CT scan) with contrast as shown in Figure 5, there was effacement sulci with ventricle and cysterna narrowing. There was no hypodense or hyperdense lesion in the brain parenchymal, and in contrast examination, there was no contrast enhancement. There was neither calcification nor midline deviation. The orbital, mastoid, and paranasal sinuses on the right and left were within normal limits. Calvaria was also normal, showing brain edema and no infarction, hemorrhage, infection, or mass in the parenchymal brain.

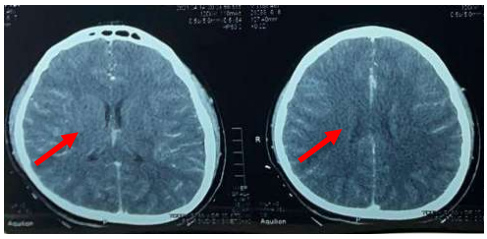

Figure 5. Head CT scan with contrast of the patient. Abnormalities were found in effacement sulci with ventricle and cysterna narrowing shown in red arrow. (courtesy of RSUD Dr. Soetomo).

This patient was assessed with right eye CN VI palsy, right eye peripheral CN VII palsy, brain edema, and space-occupying lesion. Planning therapy from the pediatric department for this patient includes oxygen

at two liters per minute nasally, D5 ¼ NS infusion 1500 ml/24 hours, manitol loading 20% 150 ml, and then maintenance at 75 mL/6 hours intravenous. Sulfas atropin 0,2 mg/kg weight if heart rate below 50 times/minute, dexamethasone loading 20 mg, then maintenance at 7.5 mg/8 hours intravenous.

An MRI with contrast was conducted to investigate a suspected case of meningoencephalitis. An MRI scan detected a hyperdense abnormality in the subcortical parietooccipital regions on both the right and left sides. A lacunar infarct has occurred in the subcortical parietal region on the left side. The sulci and gyrus were within the usual range. The ventricular system and cisterna appear to be in satisfactory condition. There was an absence of midline deviation. The pons and cerebellum appeared to be in decent condition. The hippocampal region exhibited no abnormalities. The mastoid, orbit, and right and left paranasal sinuses exhibited satisfactory conditions, as depicted in Figure 6. From MRI, the brain gave the impression of a chronic ischemic cerebral infarction at the right and left subcortical parietooccipital with chronic ischemic lacunar infarction at the left subcortical parietal, impressing vasculitis residual.

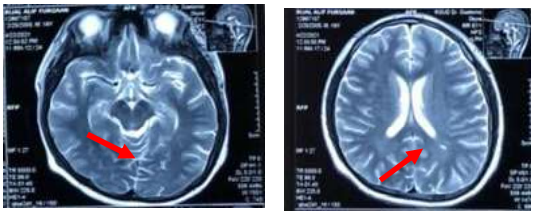

Figure 6. Brain MRI T2. Abnormalities found were in chronic ischemic cerebral infarction at right and left subcortical parietooccipital with chronic ischemic lacunar infarction at left subcortical parietal, impressing vasculitis residual, shown in red arrow. (Courtesy of RSUD Dr. Soetomo).

There was no aneurysm or vascular malformation from magnetic resonance angiography (MRA) circullus willisi within the normal limit.

The patient was monitored for a number of weeks and demonstrated a progressive improvement in their condition throughout the duration of the observation period. There was a notable improvement observed during the two-week follow-up. The patient's mobility was fully restored, allowing for a comprehensive assessment at an outpatient clinic. The patient no longer reported any complaints of double vision, indicating a resolution of the subjective symptoms. Upon inspection, ocular motility was found to be within the normal range. The function of CN VI and VII improved and returned to normal levels, as depicted in Figure 7.

Figure 7. Patient's condition after two weeks of follow-up. (Left) There was no weakness in the right side muscle when lifting the eyebrow. (Right) Facial symmetry when smiling. (The picture was taken with the patient's family consent.) Courtesy of RSUD Dr. Soetomo.

The visual acuity on both eyes was 5/5. Intraocular pressure on both eyes was 14.6 mmHg. The posterior segment of both eyes was within normal limits, as shown in Figure 8. From color examination using the Ishihara Book, both eyes were normal (38/38). From Hirschberg and Maddox rod examinations, there was orthoporia in both eyes. There were no suppressions in the worth four-dot test. Through confrontation

and the tangent screen test, the patient could see in all four quadrants.

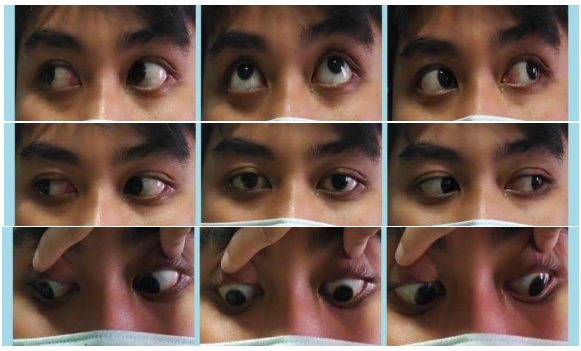

Figure 8. After two weeks of follow-up, the right eye's ocular movement has improved significantly and is now capable of moving in all directions. (Picture taken with patient's family consent.) Courtesy of RSUD Dr. Soetomo.

DISCUSSION

The CN VI nucleus is located in the middle level of the pons, positioned ventrally to the floor of the fourth ventricle. The fibers (fasciculus) exit the brainstem in a ventral direction at the junction between the pons and the medulla.[2,3] CN VI palsy is the predominant form of ocular movement paralysis in adults and the second most prevalent in children. Pediatric medicine encompasses both congenital and acquired CN VI palsies. Childhood cases of CN VI palsies can be caused by neoplasms, trauma, infections, inflammations, and idiopathic factors.[2, 16] Non-traumatic acquired CN VI palsies can result from various causes, including benign recurring CN VI palsy, pontine gliomas, increased or low intracranial pressure, and, in rare instances, acute lymphoblastic leukemia. Benign isolated CN VI palsy may manifest in childhood as a result of sinusitis, or as a consequence of an ear, throat, or viral infection.[3,4] This case presented a 16-year-old male with an acquired CN VI palsy. The reported incidence of CN VI palsy in children in the Asian population is 4.66 per 100,000 person years.[12,17]

CN VI palsy is characterized by horizontal diplopia, which becomes more severe when looking towards the same side as the affected eye. This is accompanied by a weakness in outward eye movement and inward eye deviation that intensifies while looking towards the affected side.[18,19] An ischemic mononeuropathy is the primary etiology for an isolated CN VI palsy. Lesions located at the cerebellopontine angle, particularly acoustic neuroma or meningioma, can affect the CN VI and other adjacent cranial nerves. This can result in reduced sensation in the face and cornea (CN V), facial paralysis (CN VII), and hearing loss accompanied by vestibular symptoms (CN VIII). CN VI palsy can be caused by the shearing forces of head trauma or increased intracranial pressure.[5,6, 13]

The brain and orbital MRI with gadolinium and fat suppression is the most frequent diagnostic method following a clinical examination. MRI is particularly valuable for pinpointing the location of CN VI lesions and excluding potentially severe underlying causes. In addition, MRI of the brain and orbits can occasionally aid in differentiating between elevated and reduced intracranial pressure.[6,7,13]

Treatment for CN VI palsy varies depending on the underlying cause. Typically, underlying or systemic diseases are primarily treated. The majority of individuals with a microvascular CN VI palsy are often monitored without intervention and typically experience a full recovery within a period of three to six months.[20,21] The diplopia caused by CN VI palsy can be effectively treated with prisms, occlusion, botulinum toxin, or surgery. It is recommended to use alternate patching in

the treatment of young children to prevent amblyopia.[7,22]

CN VII palsy include both full paralysis and partial weakening of the CN VII. The CN VII causes an automatic tearing response in the same eye.[23,24] Individuals may exhibit unilateral or bilateral CN VII paralysis. There are many potential causes of CN VII paralysis.[8,9]

The general population has an estimated overall prevalence of CN VII palsy of two to three occurrences per 10,000 people. CN VII palsy primarily impacts individuals between the ages of 15 to 45. It is important to mention that the most frequent reason for facial palsy is idiopathic CN VII palsy. Unilateral CN VII palsy often resolves spontaneously.[9,14]

Patients may present with ophthalmologic symptoms such as ocular erythema, lacrimation, ocular discomfort, and sense of a foreign object. Non-ophthalmic complaints may encompass several issues such as unevenness in facial appearance, simultaneous involuntary movements of the eye and mouth, general weakness or stiffness in facial muscles, challenges in chewing, difficulties in pronouncing specific words or sounds, heightened sensitivity to sound, changes in taste perception, and reduced production of saliva.[10,15]

The most prevalent indication during a physical examination is the presence of facial asymmetry. And whether the patient exhibits unilateral or bilateral involvement of CN VII. Additionally, the presence of the forehead and periocular region can help determine if the lesion is located at the higher motor neuron level (absence of forehead involvement) or the lower motor neuron level (involvement of the forehead).[11,15]

CN VII palsy resulting from an underlying cause may necessitate further investigation, such as the use of imaging techniques like CT or MRI, to uncover probable infection, tumor, fractures, or other factors that may be responsible for the involvement of the CN VII. Electrodiagnostic testing is used to stimulate the CN VII in order to evaluate the extent of damage to the CN VII. Conduct serologic testing to examine potential infectious causes. Conducting auditory assessments to ascertain the impact on the cochlear nerve or inner ear. Vestibular testing is conducted to ascertain the involvement of the vestibular nerve.[15,25]

The primary objective in the treatment of CN VII palsy is to preserve the individual's quality of life by safeguarding their vision and enhancing their overall well-being while minimizing any physical deformities. Patients with possibility for CN VII recovery are treated with conservative and medicinal management tactics.[8,10]

The outcome of CN VII palsy is influenced by various factors. The outcome of secondary CN VII palsy is contingent upon the effectiveness and handling of the underlying main condition. Idiopathic CN VII palsy, such as Bell's palsy, naturally recovers without treatment in around 70% of individuals during a period of six weeks.[10,15]

Peripheral lesions cause paralysis of certain facial muscles, such as the orbicularis oculi and frontalis muscles, resulting in face monoplegia. Supranuclear facial palsy refers to the condition where there is paralysis in the lower two-thirds of the face on the opposite side of the brain lesion. This weakness is accompanied by weakness in the orbicularis oculi muscle, but the ability to convey emotions is still intact.[8,23]

Supranuclear, nuclear, or infranuclear lesions can lead to facial weakness or paralysis. Supranuclear lesions refer to damage in the brain above the level of the cranial nerves. When a lesion occurs in the

frontal area of the precentral gyrus, it causes paralysis of voluntary facial movements on the opposite side of the body. This paralysis affects the lower part of the face more severely than the upper part. This type of paralysis is known as an upper motor neuron lesion. Facial movements related to emotions and reflexes, such as smiling and spontaneous blinking, are often maintained because they are regulated by extrapyramidal pathways. [8,20]

Peripheral lesions, namely peripheral or lower motor neuron lesions, can lead to facial weakness on the same side of the body. There are various potential explanations for this condition. Simultaneous dysfunction of CN V, VI, or VIII, or the presence of cerebellar symptoms, may suggest the presence of malignancies in the cerebellar pontine angle. Bell's palsy, the most prevalent form of facial neuropathy, primarily affects adults and is diagnosed by ruling out other possible causes. Bell's palsy is defined by the abrupt occurrence of facial paralysis. Pain might either occur before the palsy or happen at the same time. The process of recovery usually starts within three weeks after the deficiency appears and is fully completed within two to three months.[8,23]

CONCLUSION

Isolated CN VI palsy is a potentially harmless disorder. The occurrence is painless and recovery happens spontaneously within a period of six months. Peripheral CN VII palsy is less common in children compared to adults, occurring two to four times less frequently. The vast majority of children with CN VII palsy experience full recovery within a period of six months.

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