

CASE REPORT OLGU SUNUMU

DOI: 10.5336/ophthal.2024-101889

Traumatic Horner Syndrome Combine with Ipsilateral Peripheral Facial Paralysis, and Contralateral Abducens Nerve Paresis

İpsilateral Periferik Fasiyal Paralizi ve Kontralateral Abdusens Sinir Parezisi ile Kombine Travmatik Horner Sendromu

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ABSTRACT A 25-year-old male patient was admitted to Mersin University Hospital Ophthalmology Clinic five weeks after the traffic accident. There was a linear fracture in the bilateral temporal bone in the computed tomography taken in the emergency room on the day of the accident. It was learned that the patient was given 1 mg/kg methylprednisolone and methylprednisolone scheme treatment (5 days 60 mg-2 days 40 mg-2 days 10 mg-1 day 10 mg). Anisocoria and ptosis were detected in the left eyelid. Esotropia and -3 abduction limitation in the right eye were observed. There was peripheral facial paralysis on the left. The patient was diagnosed with traumatic Horner syndrome combined with ipsilateral peripheral facial paralysis and contralateral abducens nerve paresis. At the 3rd month follow-up after the trauma, the abduction restriction had disappeared and the signs of facial paralysis had alleviated.

Keywords: Traumatic Horner syndrome;
facial paralysis; abducens nerve paresis;
bilateral temporal bone fracture

ÖZET Yirmi beş yaşında erkek hasta, trafik kazasından 5 hafta sonra Mersin Üniversitesi Hastanesi Göz Hastalıkları Polikliniğine başvurdu. Kaza günü acil serviste çekilen bilgisayarlı tomografide temporal kemikte bilateral lineer fraktür hattı mevcuttu. Hastaya 1 mg/kg metilprednisolon ve metilprednisolon şeması tedavisi (5 gün 60 mg-2 gün 40 mg-2 gün 10 mg-1 gün 10 mg) verildiği öğrenildi. Muayenede sol gözde pupili yaklaşık 2 mm örten ptozis ve anizokori tespit edildi. Sağda ezotropya ve -3 abduksiyon kısıtlılığı mevcuttu. Solda periferik fasiyal paralizi mevcuttu. Hastada, bilateral temporal kemik fraktürüne bağlı ipsilateral periferik fasiyal paralizi ve kontralateral abducens sinir parezisiyle kombine travmatik Horner sendromu tanısı konuldu. Hastanın travma sonrası 3. ay kontrolünde abduksiyon kısıtlılığının kaybolduğu ve fasiyal paralizi kliniğinin hafiflediği görüldü.

Anahtar Kelimeler: Travmatik Horner sendromu;
fasiyal paralizi; abducens parezisi;
bilateral temporal kemik fraktürü

The cavernous sinus is a structure that contains the first two branches of the oculomotor, trochlear, and trigeminal nerves on its lateral wall. Within the cavernous sinus, contains the abducens and oculosympathetic nerve plexuses around the internal carotid artery. These anatomical structures, alone or in combination, cause various disorders of the cranial nerves and ultimately postganglionic Horner's syndrome.¹

While facial nerve palsy is common in ipsilateral temporal bone fractures, associated abducens palsy has rarely been reported. Cranial nerve paralysis is reversible depending on its type: motor nerve recovery is usually more satisfactory than sensory nerve recovery.²

We present a case of traumatic Horner syndrome combined with ipsilateral peripheral facial paralysis

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Peer review under responsibility of Türkiye Klinikleri Journal of Ophthalmology.

Received: 31 Jan 2024

Accepted: 13 Mar 2024

Available online: 19 Mar 2024

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and contralateral abducens nerve paresis, in a 25-year-old man after blunt trauma.

CASE REPORT

Five weeks after the traffic accident, a 25-year-old male patient was admitted to the ophthalmology clinic. It was learned that the patient was referred to Mersin University Hospital Emergency Service Hospital, where urgent evaluation and comprehensive imaging were performed. The patient's vitals were unstable and he was intubated and followed up in intensive care. In the computed tomography scan taken in the emergency room, there was a linear fracture line in the bilateral temporal bone, a linear fracture line was in the sphenoid sinus, the fracture line was extended to the carotid canal on the left and a linear fracture line was in the C2 vertebral body (Figure 1). As a result of the otorhinolaryngology consultation, the patient was treated with 1 mg/kg methylprednisolone and methylprednisolone scheme (5 days 60 mg-2 days 40 mg-2 days 10 mg-1 day 10 mg).

The best corrected visual acuity was 20/20 in both eyes. During the examination with a light source, anisocoria and ptosis were detected in the left

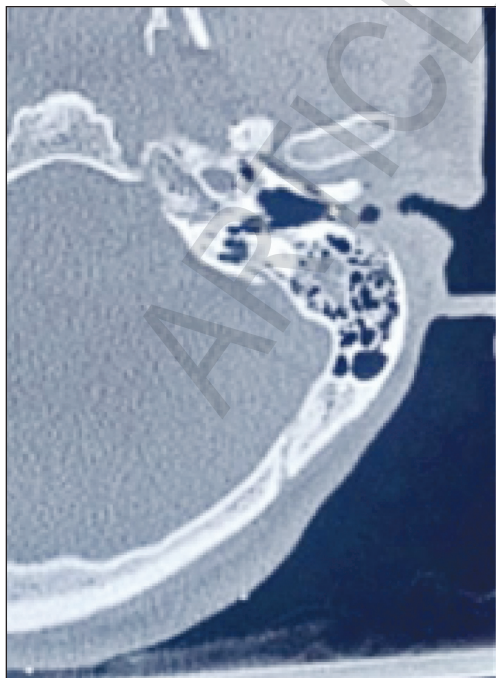


FIGURE 1: A linear fracture in the temporal bone on computed tomography.

eyelid, covering the pupil by approximately 2 mm. While direct and indirect light reactions were evaluated as positive in both eyes, no relative afferent pupillary defect was observed (Figure 2a). Esotropia in the right eye in the primary position and -3 abduction limitation in the right eye were observed when eye movements were assessed (Figure 2b). There was peripheral facial paralysis on the left. The optic disc, macula, and retinal structures were normal in the fundus examination of the patient, whose biomicroscopic examination and intraocular pressure measurements were within normal limits. The patient was diagnosed with traumatic Horner syndrome combined with ipsilateral peripheral facial paralysis, and contralateral 6th cranial nerve paresis. At the 3rd month follow-up after the trauma, the abduction restriction had disappeared and the signs of facial paralysis had alleviated (Figure 3).

Written informed consent for this case report was obtained from the patient.

DISCUSSION

The cavernous sinus is located close to the center of the base of the skull and lies medial to the two sphenoid sinuses on both sides.³ It includes the oculosympathetic plexus around the internal carotid artery and the first two divisions of the oculomotor, trochlear, abducens, and trigeminal nerves.⁴ These anatomical features are the causes of various disorders of the cranial nerves, alone or in combination, and postganglionic Horner syndrome.³

Horner's syndrome is caused by a lesion in the sympathetic pathway supplying the head and neck, including the oculosympathetic fibers. The causes can be central, preganglionic, or postganglionic. Traumatic Horner syndrome is less common than idiopathic causes, tumors, and postoperative causes. Cervical traumas are among the most important causes of preganglionic Horner syndrome. A study reported that the rate of traumatic Horner syndrome was 2.5% among all patients, and non-iatrogenic traumas are less than iatrogenic traumas.⁵

6th cranial nerve is rarely injured, and a lesion of this nerve usually occurs suddenly following severe head trauma. Its nucleus is located in the pons, ven-



FIGURE 2: A) Esotropia in the right eye, anisocoria, and ptosis on the left eyelid; B) Lateral gaze limitation in the right eye.



FIGURE 3: 3rd-month post-trauma control, spontaneous recovery abducens nerve palsy.

tral to the floor of the 4th ventricle. The nucleus of the 6th cranial nerve is in close relationship with the 7th cranial nerve fascicle at the level of the pons. Therefore, a lesion of the nucleus does not cause isolated 6th nerve palsy. After leaving the brainstem, it passes under the petrosphenoidal ligaments enters the cavernous sinus, and is located next to the carotid artery. It enters the orbit from the medial 1/3 of the superior orbital fissure. The 7th cranial nerve in the brainstem is adjacent to the 3rd, 4th, and 5th cranial nerves within the cavernous nerve. Lesions in these areas cause combined cranial nerve paralysis rather than isolated abducens paralysis.⁶ Brainstem lesions, subarachnoid space, petroclival region, and cavernous sinus or nerve course areas may trigger abducens nerve palsy.⁷ Approximately 50% of traumatic 6th nerve palsies resolve within 6 months. This process can be followed by closing one's eyes and using a prism. Botulinum injections may also be used to treat medial rectus muscle weakness. If there is no improvement during the follow-up period, it is evaluated for surgery.

Facial nerve damage occurs in 7-10 percent of temporal bone fractures, most commonly in road traf-

fic accidents.⁸ If not treated and rehabilitated promptly and effectively, these injuries can result in permanent facial deformity. Longitudinal fractures of the temporal bone are the most common (70-80%), followed by transverse and mixed fractures.⁹

6th nerve palsy associated with ipsilateral Horner's syndrome is localized in the posterior cavernous sinus, also known as Parkinson's syndrome.¹ However, in our case, there was an abducens paresis contralateral to Horner syndrome, not ipsilateral.

We think that in our case, linear temporal bone fracture is the main mechanism of abducens and facial nerve palsy. Most of the traumatic Horner syndrome cases in the literature were related to neck and chest injuries.¹⁰ In our presented case; head, neck, and chest trauma is considered to be the cause of traumatic Horner syndrome.

Conservative management including corticotherapy, eye protection, and passive facial physiotherapy was used in our case. At the 3rd month follow-up after the trauma, the abduction restriction had disappeared and the signs of facial paralysis had alleviated. Ji et al. presented a case of abducens and

facial nerve palsy after a temporal bone fracture. Facial nerve palsy required surgical decompression, and abducens nerve palsy occurred and healed after 2 months without the need for any treatment, as in our case.¹¹

As a result, it may cause Horner syndrome and traumatic nerve paralysis due to its proximity to temporal bone fracture. It is thought that steroid treatment given in the early period contributes to the recovery process.

Source of Finance

During this study, no financial or spiritual support was received neither from any pharmaceutical company that has a direct connection with the research subject, nor from a company that pro-

vides or produces medical instruments and materials which may negatively affect the evaluation process of this study.

Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

Idea/Concept: Mehmet Atila Argın; **Design:** Pınar Eröz; **Control/Supervision:** Mehmet Atila Argın; **Data Collection and/or Processing:** Pınar Eröz; **Analysis and/or Interpretation:** Pınar Eröz; **Literature Review:** Pınar Eröz; **Writing the Article:** Pınar Eröz; **Critical Review:** Mehmet Atila Argın; **References and Fundings:** Mehmet Atila Argın; **Materials:** Mehmet Atila Argın.

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