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CASE REPORT
OLGU SUNUMU

Kafa Travmasından Sonra Gelişen Bilateral Abdusens ve Sağ Fasiyal Sinir Paralizisi

Bilateral Abducens Nerve And Right Facial Nerve Palsy Occuring After Head Trauma

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ÖZET

Nervus abdusens lezyonu yaralanmaya eğilimi nadir olup genelde aniden gelişen kafa travmasından sonra meydana gelmektedir. 11.01.2014 tarihinde iş kazasına bağlı yüksekten düşme hikayesi olan 43 yaşında bir hasta sunduk. Kraniyal tomografisinde bilateral epidural hematom saptanmıştı. Epidural hematom cerrahi olarak boşaltıldı. Cerrahiden sonraki göz muayenesinde bilateral lateral bakış kısıtlılığı dışında görme kaybı saptanmadı. Diplopi nedeniyle hasta yürüyemeyince hastaya tek gözünü kapatması tavsiye edildi. Sağ yüzünde santral fasiyal sinir ve bilateral abdusens sinir paralizisi bulguları vardı. Kafa travmasından sonra birden fazla kraniyal sinir hasarı olabilir. Bu yüzden tüm kraniyal sinir muayeneleri ayrıntılı biçimde yapılmalıdır.

Anahtar Kelimeler: kraniyal sinir, abdusens paralizi, fasiyal paralizi

ABSTRACT

Lesions of the nervus abducens, the 6th cranial nerve tend to be rare, usually occur suddenly following head injuries. A 43-year-old male patient presented with a history of fall from a height due to an occupational accident on the date of 11.01.2014. Cranial tomography demonstrated bilateral epidural hematoma.

The epidural hematoma was drained during the operation. After the surgery, eye examination showed no vision loss, except limited bilateral lateral gaze.

When the patient was unable to walk due to diplopia, he was advised to close one eye. On the right side, there were findings suggesting central facial paralysis. There may be multiple cranial nerve damage following head injury.

Therefore, all cranial nerves should be thoroughly examined.

Keywords: cranial nerve, abducens palsy, fascial paralysis

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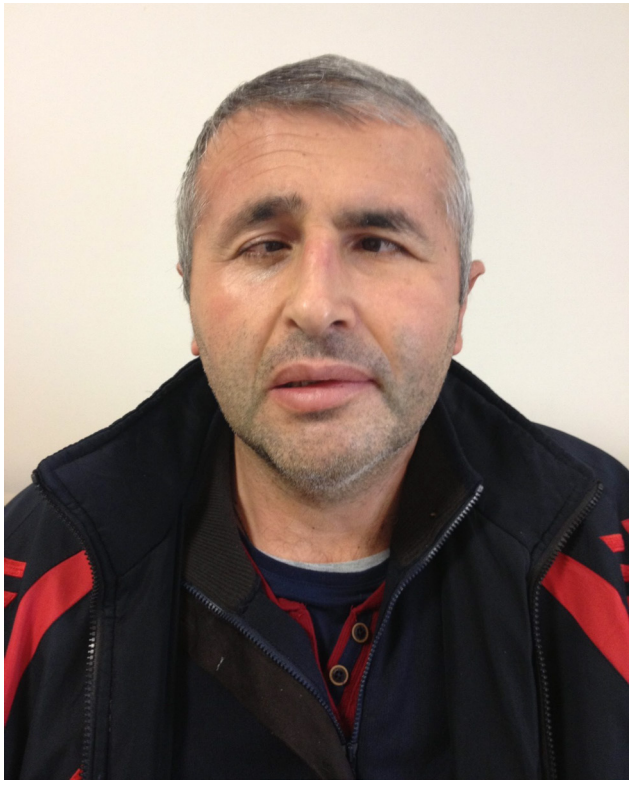
INTRODUCTION

The nervus abducens, sixth cranial nerve, rarely exposes to injury and lesion of this nerve usually occurs suddenly following head trauma. Vascular pathologies and idiopathic causes are likely to develop after the age of 50. Unilateral abducens nerve palsy forming after head injury occurs at a rate of 1 to 2.7% and bilateral lesions of the 6th cranial nerve are commonly rare, usually accompanying intracranial or cervical spine injuries (1). Diplopia (double vision) is the most significant symptom in many patients. It should certainly come to mind in the case of such patients with limited lateral gaze or double vision. In this article, we present a case with a bilateral lesion of the abducens and right facial nerve injury developed following severe head injury. Involvement of the 6th and 7th cranial nerves following head injury is seen extremely rarely.

CASE REPORT

A 43-year-old male patient presented with a history of fall from a height due to an occupational accident on the date of 11.01.2014. The patient who suffered from the loss of consciousness was admitted to the emergency department in an intubated state. Physical examination revealed rib fracture and pelvic and acetabular and the initial computed tomography (CT) scan showed a minimal edema, fracture in the left parieto-occipital bone, bitemporal fractures, cerebellar edema, and hemorrhage in the paranasal sinuses. Repeated CT at 6 hours demonstrated bilateral epidural hematoma. The patient underwent an emergency operation. The epidural hematoma was drained during the operation and a drainage tube was placed after duraplasty and cranioplasty was performed. The left hip CT showed fractures in the left acetabulum pubic segment and a fissure in the anterior cortex of the left iliac bone near the sacroiliac joint. The patient was not considered an eligible candidate for surgery for pelvic fractures based on the judgement of the orthopedic surgeon. Bilateral chest tubes were inserted upon the detection of the rib fractures, a massive right-sided pneumothorax, bilateral atelectasis and pleural effusion in the CT scan in the department of thoracic surgery, and later removed after scheduled follow-ups. Hemoglobin levels continued to fall, while biliary sludge was observed in the abdominal ultrasonography along with thickening consistent with hemorrhage in the left abdominal anterior wall, heterogeneous dense appearance consistent with retroperitoneal bleeding in the left retroperitoneum, fracture in the anterior column of the left acetabulum and vertebral endplate height loss at L5 in the high-contrast lower abdominal CT scan. An emergency laparoscopy was performed by a general surgeon and a large retroperitoneal hematoma was observed, however, no surgical intervention was made. Hemoglobin levels returned to normal limits through blood transfusions. The patient was extubated four days after the admission to the intensive care unit. On Day 11, the patient was transferred to the neurosurgery unit. As liver enzymes and acute phase reactants increased, the patient was closely monitored. Antibiotherapy was also initiated. Bilateral abducens nerve palsy and right central facial paralysis were noticed (Figure 1).



(Figure 1)

The patient was consulted by the neurology department. In the repeated thoracic CT scan, bilateral pleural effusion exceeding 1 cm on the right side and a partially displaced fracture in the left 6th, 7th and 8th posterior ribs were detected. The patient was discharged in a stable condition. Eye examination showed no vision loss, except limited bilateral lateral gaze. When the patient was unable to walk due to diplopia, he was advised to close one eye. On the right side, there were findings suggesting central facial paralysis. Based on our examination, we observed no findings indicating other cranial nerve lesions. His upper and lower limb neurological examination results together with his cerebellar examination findings were normal. The range of motion of the hip was full, however, it was painful. The patient was scheduled for follow-up visits with exercise program recommendations.

DISCUSSION

The mechanism of cranial nerve injuries occurring after the head trauma without fractures is still controversial. After severe injuries, a nerve can be damaged at the dural entry point, the petrous apex, and around the lateral wall of the internal carotid artery. As bilateral nerve damage tends to be seen in injuries to the lateral region of the head, it occurs in transverse basilar skull fractures, in particular (2). Brain stem lesions, the subarachnoid space, the petroclival region, and the cavernous sinus or areas within the eye, where the nerve travels, may trigger abducens nerve palsy. The abducens nucleus is localized in the pontine tegmentum, seated beneath the ventral of the 4th ventricle and the 7th cranial nerve axons (3). Due to the proximity of the nuclei of both nerves, the joint occurrence of abducens and facial nerve lesions after brain stem injuries injuries may suggest nuclear lesions (4). In addition, central lesions can be displayed with CT or magnetic resonance imaging (MRI) studies. Lesions of the 5th, 6th, 7th and 8th cranial nerves may simultaneously develop following petrous bone fractures (5). Therefore, all cranial nerves, pyramidal system involvement, petrous bone, and the orbita should be carefully examined.

Loss of function of the abducens nucleus and fascicles usually stems from intraaxial tumors in the brain stem, demyelinating diseases, or inadequate vascular nourishment related to ischemic syndromes. Posterosuperior displacement of the brain from the skull base secondary to the injury may lead to avulsion of the nerve in any area. Injury to the branches of the meningodorsal artery supplying blood to the abducens nerve may also result in acute or progressive paralysis along with mild to severe cases of ischemia. The postmortem studies of patients who died due to head injury showed that the abducens nerve was damaged in the area of anastomosis with the sympathetic plexus and at the dural entry porus in the petroclival region (6). It was also reported that nerves in these regions were compressed in cases of severe cervical and cranial injuries (7,8).

Furthermore, bilateral nerve damage is usually caused either by the bilateral nerve being individually affected due to trauma or indirectly by an increase in intracranial pressure (9). In a study including 11 patients with bilateral 6th cranial nerve damage secondary to injury, fractures of the cervical vertebra were detected in four and skull fractures in three patients. Five patients remained comatose. As a result, severe injuries may lead to bilateral 6th nerve damage (2). Our case had a skull fracture, but no cervical vertebral fractures. The presence of a bilateral temporal fracture suggests that there may be direct nerve damage.

In the differential diagnosis, peripheral nerve injuries occurring jointly with basilar skull fractures, clival fractures, in particular, brain stem lesions, and lateral rectus muscle injury or entrapment should be considered (9). Intracranial pressure and compression along the course of the nerve may be also risk factors. Additionally, the nucleus of the abducens nerve may become involved in the case of congenital diseases such as the Moebius syndrome and the Duane retraction syndrome (10). Medial orbital wall injuries leading to any damage in the medial rectus muscle may also mimic 6th nerve palsy. The most critical prognostic factor in 6th cranial nerve injury is a partial loss of function of the lateral rectus muscle. Recovery may be more rapid in

the absence of skull fractures following injury without intracranial pathology and transection.

In conclusion, there may be multiple cranial nerves damage following head injury. The nervus abducens nerve lesion along with other cranial nerves, though rare, may occur as in our case. Therefore, patients should be thoroughly examined after the head injury involving all cranial nerves.

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