Isolated Abducens Nerve Paresis Associated With Incomplete Horner’s Syndrome Caused by Petrous Apex Fracture
—Case Report and Anatomical Study—

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Abstract

A 17-year-old male presented with a wound on the right temporal region, oozing hemorrhagic necrotic brain tissue and cerebrospinal fluid, following a fall. Computed tomography showed temporoparietal and petrous apex fractures on the right. Neurological examination revealed abducens nerve paresis, ptosis, and myosis on the right side. The patient was treated surgically for the removal of the free bony fragments at the fracture site and to close the dural tear. The abducens nerve paresis, ptosis, and myosis persisted at the 3rd monthly postoperative follow-up examination. The anatomy of the abducens nerve at the petroclival region was studied in four cadaveric heads. Two silicone-injected heads were used for microsurgical dissections and two for histological sections. The abducens nerve has three different angulations in the petroclival region, located at the dural entrance porus, the petrous apex, and the lateral wall of the cavernous segment of the internal carotid artery. The abducens nerve had fine anastomoses with the trigeminal nerve and the periarterial sympathetic plexus. There were fibrous connections extending inside the venous space of the petroclival area. The abducens nerve seems to be vulnerable to damage in the petroclival region, either directly by trauma to its dural porus and petrous apex or indirectly by stretching of the nerve through the nervous and/or fibrous connections. Concurrent functional loss of the abducens nerve and the periarterial sympathetic plexus clinically manifested as incomplete Horner’s syndrome in our patient.

Key words: abducens nerve, incomplete Horner’s syndrome, petrous apex fracture, sympathetic plexus

Introduction

The incidence of traumatic unilateral abducens nerve palsy is 1% to 2.7% in patients with head trauma. Both severe head traumas and severe cervical traumas can lead to abducens nerve palsy. The low incidence of abducens nerve palsy among patients with severe cervical and head trauma may be attributed to the low survival rate after the traumatic episode. Stretching of the abducens nerve at the petrous apex and dural entrance porus may be the mechanism of injury.

However, abducens nerve palsy has never been associated with incomplete Horner’s syndrome caused by petrous apex fracture. We report a case of this condition and a cadaveric anatomy study of the abducens nerve in the petroclival region to investigate the mechanism of injury leading to isolated abducens nerve paresis and incomplete Horner’s syndrome.

Case Report

A 17-year-old male was admitted to the emergency unit of Firat University Hospital, Department of Neurosurgery, following a fall from a height of 7 meters. He had a scalp hematoma of 10-cm diameter in the right temporoparietal region. There was an 8-cm vertical wound 1 cm behind the auricle. There was no lateralization finding, but a slight amount of hemorrhagic necrotic brain and cerebrospinal fluid oozed out of the wound. He also had otorrhagia...
through the right external ear meatus. The Glasgow Coma Scale score was 12 points. The patient complained of diplopia. Neurological examination revealed lateral gaze restriction, myosis, and ptosis on the right side (Fig. 1). Computed tomography revealed diastatic fracture of the posterior part of the right temporal bone, a fracture in the right temporal region, a fracture in the lateral wall of the right side of the sphenoid sinus, and a bony fragment at the right petrous apex (Fig. 2).

Surgery was performed 7 hours after the trauma under emergency conditions. The upper edge of the wound was extended to the right frontal region of the scalp to form a temporoparietal scalp flap. A dural tear extended from the temporal region to the border of the transverse sinus on the right side under the fracture. There was a small tear on the upper side of the sinus. The dural tear was repaired with a periosteal graft, and the sinus bleeding was stopped by the periosteal graft and a tissue sealant. Free bony fragments were also removed during the operation.

The patient awakened with preoperative findings of diplopia on right lateral gaze, and myosis and ptosis on the right side on the postoperative first day. The Glasgow Coma Scale score increased to 15 points. There was no sweating abnormality on either side of the face. However, his complaints persisted at the 3rd monthly follow-up examination.

**Anatomical Study**

I. Materials and methods

Four adult fresh cadaveric heads, 65 to 80 years of age, were used. The coagulated blood remnants in the vessels were removed with isotonic NaCl in two heads, then both the common carotid and vertebral arteries, and internal jugular veins were perfused with colored silicone; red for arteries and blue for veins. The skull vaults and the brains were removed and the heads were preserved in 10% formalin solution. Both petroclival areas were dissected under an OPMI MD surgical microscope (Carl Zeiss Co. Ltd., Berlin, Germany). Contax 167 M camera (Kyocera Co. Ltd., Tokyo) was used for photographic documentation. The petroclival dura mater was dissected and partially removed to expose the structures in this space, protecting the part around the abducens nerve dural entrance porus.

Both the common carotid and vertebral arteries, and internal jugular veins of two fresh cadaver heads were perfused with 10% formalin in 0.1 M phosphate buffered solution (pH 7.4) for histological examination. The heads were kept in the same fixative solution for 10 days. The calvarium and the brains were removed. The dura mater around the petroclival region covering the cranial base was carefully lifted away including all the membranous, nervous, and ligamentous structures. Right and left sides were separately embedded in paraffin after processing in a graded ethanol series from 70% to 100%, and serial sections of 5-μm thickness were made. The sections were stained with hematoxylin-eosin and Masson-trichrome stains and observed under the light microscope.

II. Results

The abducens nerve follows three different routes in its course through the petroclival region. The ab-
ducens nerve runs laterally from its exit point in the brainstem until piercing the dura mater. At first, its direction changes medially at the dural entrance porus, forming the first angulation; then laterally at the petrous apex, forming the second angulation. The third angulation occurs at the tangential passing of the lateral wall of the cavernous segment of the internal carotid artery (ICA). The round shape of the abducens nerve flattens vertically at the third angulation point where the abducens nerve anastomoses with the periarterial sympathetic plexus surrounding the ICA (Fig. 3). The abducens nerve is covered by arachnoid membrane and dura mater between the dural entrance porus and the third angulation point (Fig. 4). The dural sleeve of the abducens nerve becomes thinner, and transforms into a semitransparent membrane around this area. This transformation point is located where the lateral wall of the cavernous segment of the ICA tangentially approaches the medial wall of Meckel’s cave just before the third angulation. Fine anastomoses between the abducens and trigeminal nerves were observed at this location (Fig. 5). There were fibrous trabeculations extending between the layers of dura mater which were attached to the membranous coverages of the trigeminal and abducens nerves, the periarterial sympathetic plexus, and the external layer of the ICA (Fig. 5).

Discussion

The abducens nerve may be damaged directly by bony fragments caused by the skull base fracture. However, the mechanism of abducens nerve paresis in severe head or cervical trauma without fracture is controversial. The petrosphenoidal ligament acts as a contusion point of the abducens nerve in upward and posterior displacement of the brain in cervical spine fracture cases. However, this mechanism...
has been rejected because the abducens nerve is fixed downward below the petrosphenoidal ligament by the dura mater and the apex of the petrous pyramid. The petrosphenoidal ligament constitutes a bridge between the petrous apex and the dura mater. According to our dissection findings, a second layer of the petrosclival dura mater apparently fixes the abducens nerve between the petrosphenoidal ligament and the petrous apex. Therefore, we suggest that direct damage of the abducens nerve by the petrosphenoidal ligament is not the mechanism of injury as previously stated.

Cerebrospinal fluid suspends the brain and the spinal cord in a watery medium and cushions the central nervous system from external forces applied to the skull or vertebral column. The brain weighs only 50 g when floating in the cerebrospinal fluid. However, a transfer of high velocity to the brain by the sudden acceleration of the head in severe trauma causes the rupture of vessels and nerve fibers. Considering the movement of the brain in the skull, both abducens nerves may be stretched downward by a linear acceleration in the mid-sagittal plane during an impact. Then, the nerve is compressed against the petrous apex, resulting in injury. Two points may be the site of abducens nerve damage in cases of traumatic bilateral abducens nerve palsy, the dural entrance porus and the petrous apex. Vectorial analysis of the flexion and extension forces in cervical injuries demonstrated that there was a consistent vertical component in both types of injuries. This finding indicates an upward movement of the brain in cervical trauma leading to functional loss of the nerve at the dural entrance porus of the abducens nerve. In our opinion, the rigid structure formed by the borders of the dural entrance porus is significant, especially in cervical trauma, because of the upward movement of the brain. On the other hand, since the abducens nerve crosses the petrous apex, acute downward displacement of the brain may compress and injure the nerve at this location.

The right temporoparietal fracture indicated the site of the impact in our patient. The brain definitely moved upwards and right and then downwards in the skull. The dural entrance porus of the abducens nerve acted as a fulcrum in the upward movement, and the nerve stretched at the petrous apex in the downward movement. We believe that the isolated abducens nerve paresis was caused by injury at the two locations mentioned above. The presence of petrous apex fracture specifically suggests direct damage of the nerve. Incomplete Horner’s syndrome indicates definite damage of the periarterial sympathetic plexus at the third angulation point.

Based on our anatomical dissections, damage of the abducens nerve is quite possible due to the anastomosis of the abducens nerve with the periarterial sympathetic plexus at the third angulation point.

Abducens nerve has three angulations in the petroclival region, at the dural entrance porus, petrous apex, and the lateral wall of the cavernous segment of the ICA. The dural sleeve of the abducens nerve, which protects the nerve from venous blood, becomes thinner at the end of the petrous apex and the abducens nerve anastomoses with the periarterial sympathetic plexus on the lateral wall of the cavernous segment of the ICA, where the last angulation is located. The sympathetic connections to the abducens nerve were studied in detail, finding that the connecting fibers must process some of the functions absent in Horner’s syndrome. Abducens nerve paresis and Horner’s syndrome can be caused by intracavernous aneurysm and petrous apex tumors, but the sweating function, which is another component of Horner’s syndrome, was not mentioned. However, involvement of the periarterial sympathetic plexus in the dissection of ICA present as incomplete Horner’s syndrome, which is characterized with ptosis and myosis but not anhydrosis, due to sparing of the external carotid plexus, which innervates the facial sweat glands. Similarly, the absence of anhydrosis in our case indicates incomplete Horner’s syndrome.

The results of our anatomical study indicate that the abducens nerve may be tethered by the periarterial sympathetic nerve fibers during the strong sudden backward stretching of the nerve. The anastomoses between the abducens nerve and the trigeminal nerve were weaker than those with the periarterial sympathetic plexus. Therefore, their effect during entrapment may be slight. On the other hand, the use of long electrodes through the foramen ovale was considered responsible for the abducens nerve palsy complication during treatment for trigeminal neuralgia. In our opinion, the abducens nerve palsy complication during the rhizolysis procedure provides more evidence for the connections between the abducens and trigeminal nerves and implies the tethering of the abducens nerve by these structures.

Two types of connective tissue occur inside the cavernous sinus; adipose tissue and a network of collagenous sheaths. The fibrous trabeculations extending between the two dural leaves limit the venous space of the petroclival region as well as suspend the arteries. The petrosphenoidal ligament is the largest of the fibrous trabeculations at the petroclival region and forms a ligament between the

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petrous apex and the dorsum sellae including the posterior clinoid process supporting the posterior petroclinoid fold. These trabeculations fix the dural sleeve between the dural entrance porus and the petrous apex, and thin membranous coverage of the abducens nerve at the third angulation point. Therefore, the fibrous trabeculations form another tethering structure of the abducens nerve.

In our case, the direct injury was the main mechanism of abducens nerve paresis, inflicted by the bony fragment at the petrous apex. However, the association of incomplete Horner’s syndrome indicates damage to the periarterial sympathetic nerve fibers through the nervous and/or fibrous connections between these two structures. Based on our anatomical dissections and histological findings, we suggest that the fixation of the abducens nerve by the periarterial sympathetic plexus and fibrous trabeculations may form another mechanism of nerve damage.

In conclusion, the three angulation points of the abducens nerve in the petroclival region are critical locations where the nerve is highly vulnerable to damage. Association of incomplete Horner’s syndrome with abducens nerve paresis suggests the involvement of the periarterial sympathetic plexus surrounding the ICA. Although the origin of abducens nerve paresis seems to be a direct bony injury in our patient, these indirect mechanisms may also be involved.

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References


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