Revista Chilena de Neurocirugía 40: 2014

Lesiones de múltiples nervios craneales luego de traumatismo craneal cerrado: Reporte de caso y Revisión del tema

Multiple Cranial Nerve Injuries Following Closed Head Trauma: Case Report and Review

Magalhães MJS[1], Mendes CAA[2], Farnese JM[3], Hoffmann EJ[4]

1 Medical Assistant Professor, Faculdades Unidas do Norte de Minas (FUNORTE) Medical School. Department of Neurosurgery, Aroldo Tourinho Hospital, Montes Claros-MG, Brazil.
2 Medical Assistant Professor, Universidade Estadual de Montes Claros (UNIMONTES) Medical School. Department of Neurosurgery, Aroldo Tourinho Hospital, Montes Claros-MG, Brazil.
3 Medical Student, FUNORTE Medical School, Montes Claros-MG, Brazil.
4 Medical Assistant Professor, FUNORTE Medical School, Montes Claros-MG, Brazil.

Rev. Chil. Neurocirugía 40: 37-41, 2014

Resumen

La lesión de los nervios craneales es un acompañamiento común de un trauma en la cabeza. Lesiones de los nervios craneales asociados con la lesión cerrada de la cabeza ha sido encontrado para ser asociado con una mayor gravedad de la lesión. Los objetivos de este estudio son documentar la incidencia de lesiones de los nervios craneales en lesiones en la cabeza, que se correlaciona con la incidencia de los hallazgos radiológicos, para evaluar el tiempo de recuperación con respecto a los signos y síntomas en la presentación inicial. Se presenta un caso de un varón de 51 años de edad, con lesiones nerviosas del segundo, sexto, séptimo, octavo, noveno y décimo después de una lesión grave en la cabeza. Lo admitieron a la víctima con un historial de conducir una motocicleta utilizando un casco y con una caída a alta velocidad. Su Resonancia Magnética (RM) presenta contusión en el tronco cerebral y su tomografía computarizada (TC) simple mostró pequeña hemorragia a la derecha del tronco cerebral y el paciente fue tratado de forma conservadora. Por otra parte, si se detectan múltiples lesiones de los nervios craneales hay una necesidad de evaluar más a fondo la lesión del tronco cerebral por RM con el fin de evaluar mejor el tronco cerebral. La mayoría de las lesiones de los nervios craneales pueden recibir tratamento conservador, aunque algunos autores indican intervención quirúrgica temprana para el tratamiento de la parálisis facial con fractura a través del canal facial.

Palabras clave: Traumatismos Craneocerebrales, Nervios Craneales, Parálisis.

Abstract

Injury to the cranial nerves is a common accompaniment of head trauma. Cranial nerve injuries associated with closed head trauma has been found to be associated with injuries of a higher severity. The incidence of cranial nerve injury in head trauma varies in the literature, ranging from 5 to 23 percent. The objectives of this study are: to document the incidence of cranial nerve injuries in head trauma; to correlate the incidence with radiological findings and to assess recovery time according with signs and symptoms at initial presentation. We report a case of a 51-year-old male having second, sixth, seventh, eighth, ninth and tenth nerve injuries after severe head trauma. He was admitted after an accident with a history of riding a motorcycle wearing a helmet and falling at high speed. Study by Magnetic Resonance Imaging (MRI) of this case presented stem contusion and a plain Computerized Tomography (CT) showed small hemorrhage on the right of the brain stem. The patient was managed conservatively. Multiple cranial nerve palsies after head injury may not carry a bad prognosis as previously thought, and may be reversible. Moreover, if multiple cranial nerve injuries are detected, a better evaluation of the stem brain is needed by MRI in order to evaluate possible lesions in

this site. The majority of the cranial nerve injuries are treated conservatively, although some authors indicate early surgical intervention to treat facial palsy with fracture through the facial canal.

Key words: Craniocerebral Trauma, Cranial Nerves, Paralysis.

Introduction

Injuries to the third, fourth, sixth and seventh cranial nerves are common after head trauma and can be missed during the initial assessment[1,2]. The incidence of cranial nerve injury in head trauma ranges from 5 to 23 percent[2,3]. Cranial nerve lesions associated with closed head trauma has been found to be associated with a higher severity of injury[4]. The kind of presentation of the trauma is an important etiological factor for multiple cranial nerve palsies[5]. We report a case of a 51-year-old male having right injury of second, sixth, seventh, eighth, ninth, tenth nerves after a severe head trauma. The patient was managed conservatively.

Case report

A 51-year-old male patient was admitted after an accident with a history of driving a motorcycle wearing a helmet and falling at high speed. The patient immediately lost consciousness. Right ear bleed was present. There was no history of seizures, vomiting or cerebrospinal fluid (CSF) leak. The patient was not intubated, and his Glasgow Coma Score (GCS) was 13. There was no abdomen or long bone injury. CT evaluation showed small hemorrhage on right of the brain stem (Figure 1). MRI of this patient showed contusion on right cerebellopontine angle. His vitals, hematological and biochemical parameters were all stable. On a more detailed neurological examination we found right lower motor type facial palsy (Figure 2), without ptosis. Pupillary light reflex was present, both direct and consensual. There also could be found palsies of 6th, 9th and 10th right nerves (Figure 3 and 4). The patient also presented hoarseness, and hearing loss and abnormal vision could be found on the right side. Other cranial nerves had no alterations. Motor and sensory examinations were normal. After five days of hospitalization under a conservative management, the patient was released.

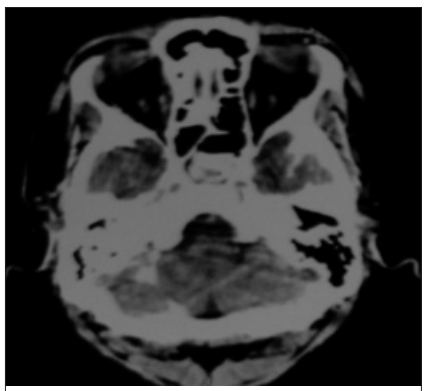

Figure 1. CT image showing hemorrhage on right of the brain stem.

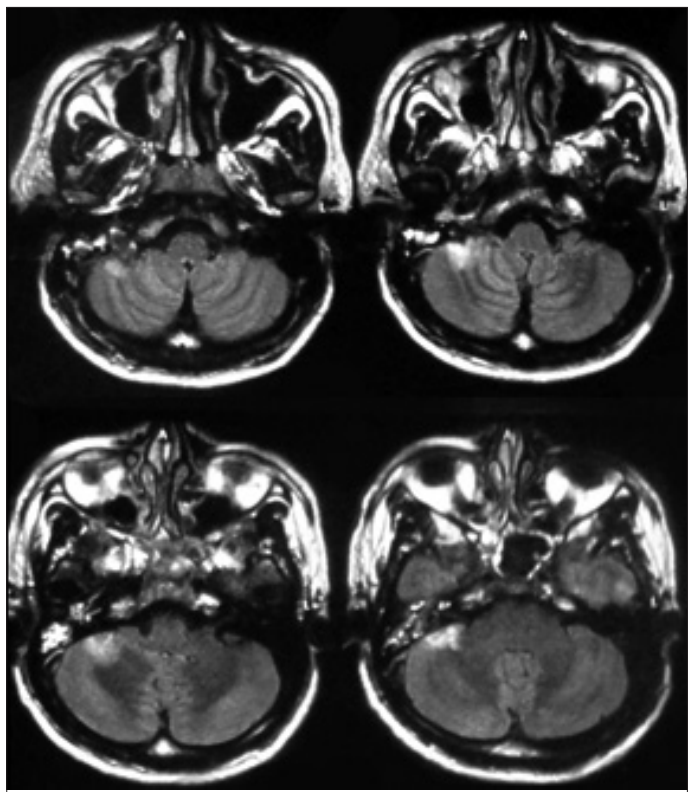

Figure 2. MRI showing hemorrhage and contusion on right cerebellopontine angle.

Discussion

In an Indian study involving 794 consecutive cases of head injury, 100 of these were found to have cranial nerve injuries. Of these 100 patients, 50 had mild, 26 had moderate and 24 had severe injury. There was a preponderance of male patients (87 males; 13 females). Sixty seven patients (67%) had single cranial nerve injury[6].

Olfactory Nerve Injury is rare. The same study only 0.88% had post-traumatic olfactory dysfunction. This injury was associated with cribiform plate fracture and CSF rhinorrhea. During the trauma, either shearing of olfactory filaments (as they pass through the cribiform plate) or contusion to the olfactory bulb may oc-

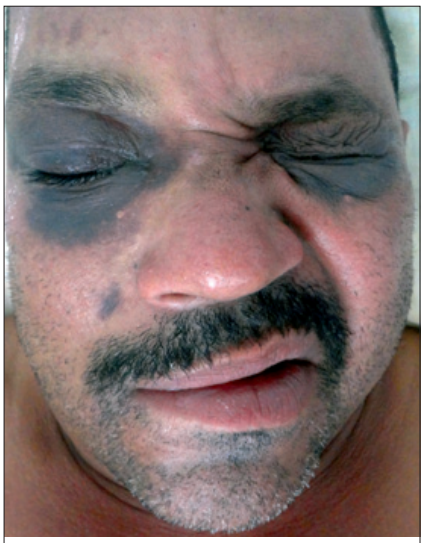

Figure 3. Picture showing palsy of right Seventh cranial nerve.

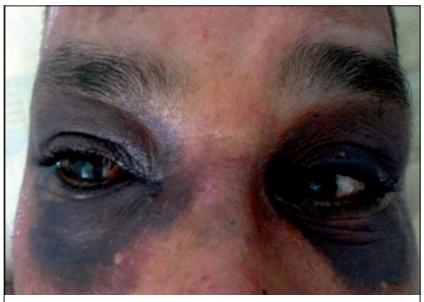

Figure 4. Picture showing palsy of right sixth cranial nerve.

cur. Moreover, contusion or a shearing injury to the cerebral cortex (frontal and temporal lobes) may occur[12,13,14]. Most of them have shown no improvement over a follow up of 10 to 18 months[6]. In a review involving 268 patients who had suffered head trauma, anosmia occurred in about 66.8% of the cases, and only rarely olfactory function became normal again[12].

Optic-traumatic nerve injury happens in about 2.78% of head injury[6]. It is commonly associated with mild head injury compared to moderate and severe head injury[6]. This kind of nerve injury is associated in almost all cases with orbital fractures[6].

Oculomotor nerve injury developed after a head injury is present in about 2,9% of cases[6]. Severity of injury was diverse[6]. Significant improvements in extra-ocular muscle paresis in patients with non-penetrating trauma suggest neuropraxia type of injury to some degree[6]. The symptoms more frequently found were ptosis and mixed horizontal and vertical binocular diplopia. Near normal medial gaze recovery was more commonly seen within the first two months[6] . Pathologic reports have demonstrated injury to cranial nerve 3 at its exit from the brainstem, from the superior orbital fissure, and at the tentorial shelf after herniation secondary to traumatic brain edema[9,10]. Upward and downward gaze recovery was incomplete and delayed (more than 3 months)[6].

Trochlear Nerve injury associated with head injury is rare and it´s find in about 2.14% of patients[6]. Mild head trauma associated with this nerve injury is more common compared with moderate or severe head trauma.

Post-mortem examinations of patients in motor vehicle accidents have demonstrated injury to cranial nerve 4 at its exit from the brainstem in the dorsal midbrain[9]. Spontaneous complete recovery of trochlear nerve was seen in almost half of the patients with severe head injury and are more prone to partial and delayed recovery (more than one year)[6].

The commonest injuries to the trigeminal nerve are in its peripheral branches (supraorbital or infraorbital nerves). The infraorbital nerve is frequently damaged by maxillary fractures. The area of numbness usually diminishes without any special treatment[11].

Abducens Nerve injury was present in 3,02% of the cases in the Indian study[6]. Skull base fracture involving the clivus was the most common finding. Mild and moderate head injury was more common associated to this nerve injury[6]. There are two hypotheses to the mechanism of injury to this nerve: the first is petrous bone injury transmitted to the nerve at this region; and the second one is neck hyperextension causing stretch of this nerve[7]. This second mechanism would be secondary to upward and posterior displacement of the brainstem causing stretch injury to the sixth cranial nerve as it passes through Dorello's canal under the rigid petrosphenoidal ligament[8].

Post-traumatic facial nerve palsy is relatively common and is associated to temporal bone fracture[17]. It can be classified as either an immediate or a delayed onset[18]. The most common type of injury is the one that show up at an immediate onset, which usually results from direct laceration or contusion of the nerve at the site of a temporal bone fracture[17]. Delayed onset palsy is commonly caused by edema, external compression by hematoma fluid, arterial or venous thrombosis and is not usually noticed until four or five days after head injury[17]. High resolution CT scanning of the petrous temporal bones is indicated in all cases of immediate onset facial nerve palsy so that the relation between a fracture and the facial canal can be determined[17]. Some authors indicate immediate onset facial palsy with fracture through the facial canal as an indication for early surgical intervention[19,20]. Others report a slow recovery of function after traumatic facial nerve palsy, with the degree of palsy having a greater influence on recovery of function than the time of onset[17]. In a study involving 25 patients with post-traumatic facial nerve palsy, complete recovery had occurred at the four month point in 62% of patients who had a partial lesion, but in none of those who had complete paralysis[18]. So the prognosis for these patients is highly variable and depends on the extension of the initial involvement. Unilateral traumatic cases resolve spontaneously in 72% of cases, against only 12% of bilateral cases after a 6 month follow-up period[22].

Post-traumatic hearing loss is a well-known occurrence[14]. All head injuries, with or without skull base fracture, may cause hearing loss which, in both instances, can be conductive as well as sensorineural[15]. The site of the hearing impairment can be peripheral or central and the finding of a hearing loss after skull trauma may be associated to Benign Paroxysmal Positional Vertigo. The middle ear or cochlea appear to be the most frequently involved areas. In these cases, the hearing loss is most commonly due to a temporal bone fracture with involvement of the otic capsule or disruption of the ossicula.

A syndrome consisting of unilateral lesions of the last four cranial nerves was first described by Collet and Sicard and is now called the Collet-Sicard syndrome[23,24]. This syndrome has been described in association with Jefferson's fracture, idiopathic cranial polyneuropathy, multiple myeloma, internal carotid artery dissection and coiling, Lyme disease[23], and also with skull base tumors of primary or metastatic (prostate, lung, breast, and renal tumors) origins[25-27]. One of the rare causes of the Collet-Sicard syndrome is the occipital condyle

fractures[28]. Although they are among the most rare traumatic lesions of the skull base and usually receive little attention, occipital condyle fractures constitute of an anatomoclinical entity because of its special features[28]. Occipital condyles are in close relationship with the hypoglossal canal and the jugular foramen, which includes the cranial nerves IX, X, and XI[28]. Occipital condyles also have a vital anatomical relationship to the brain stem and vascular structures[28].

Injuries to the hypoglossal nerve (cranial nerve XII) are rare. The majority of reported cases result from malignancy; traumatic causes are less common[29]. Penetrating trauma, iatrogenic causes, and occipital-cervical junction fractures from motor vehicle crashes account for the majority of traumatic cases[30]. The prognosis for isolated unilateral hypoglossal palsies is good, most of them resolving by about 6 months[30]. When palsy lasted longer than 6 months, near-normal clinical function was reported within 12 months[30].

Unilateral traumatic hypoglossal nerve palsy has been shown to have a better prognosis than bilateral palsies, probably because a unilateral lesion is usually caused by a nerve root injury, whereas bilateral lesions suggest an intrinsic spinal cord lesion[29].

Conclusions

Multiple cranial nerve palsies after head injury may not carry a bad prognosis as previously thought and may be reversible, hence the need to actively treat such patients. Moreover if multiple cranial nerve injuries are detected there is a need to evaluate the brain stem by MRI in order to detect possible injuries at this site. The majority of the cranial nerve injuries are treated in a conservative way, although some authors indicate early surgical intervention to treat facial palsy with fracture through the facial canal.

Recibido: 15 de junio de 2013
Aceptado: 14 de agosto de 2013

References

1. Rucker CW. Paralysis of the third, fourth and sixth cranial nerves. Am J Ophthalmol. 1958; 46: 787-794.
2. Jin H, Wang S, Hou L, et al. Clinical treatment of traumatic brain injury complicated by cranial nerve injury. Injury. 2010; 41: 918-923.
3. Keane JR, Baloh RW. Post-traumatic cranial neuropathies. In: Evans RW, ed. The Neurology of Trauma. Philadelphia: Saunders; 1992: 849-868.
4. Dhaliwal A, West AL, Trobe JD, Musch DC. Third, fourth, and sixth cranial nerve palsies following closed head injury. J Neuroophthalmol. 2006; 26: 4-10.
5. Keane JR. Multiple cranial nerve palsies: analysis of 979 cases. Arch Neurol. 2005; 62: 1714-1717.
6. Purav Patel, S Kalyanaraman, J Reginald, P Natarajan, K Ganapathy KR Suresh Bapu, A Vincent Thamburaj, B Chendhilnathan, M Balamurugan. Post-traumatic Cranial Nerve Injury. Indian Journal of Neurotrauma 2005; 2(1): 27-32.
7. Lavin PJ, Troost BT. Traumatic fourth nerve palsy: Clinicoanatomic correlations with computed tomographic scan. Arch Neurol 1984; 41: 679-680.
8. Lindenberg R, Freytag E. Brainstem lesions characteristic of traumatic hyperextension of the head. Arch Path 1970; 90: 509-515.
9. Heinze J. Cranial nerve avulsion and other neural injuries in road accidents. Med J Aust 1969; 2: 1246-1249.
10. Keefe WP, Rucker CW, Kernohan JW. Pathogenesis of paralysis of the third cranial nerve. Arch Ophthalmol 1960; 63: 585-592.
11. Lewin W. Injuries to cranial nerves and visual pathways. In, Lewin W, Bailliere, Tindall and Cassell (eds). The management of Head injuries 1996:137-146.
12. Doty RL, Yousem DM, Pham LT, Kreshak AA, Geckle R, Lee WW. Olfactory dysfunction in patients with head trauma. Arch Neurol 1997; 54: 1131-1145.
13. Zusho H. Posttraumatic Anosmia. Arch Otolaryngol 1982; 108: 90-92.
14. Costanzo RM, Becker DP. Smell and taste disorders in head injury and neurosurgery patients. In: Meiselman HL, Rivlin RS, editors. Clinical Measurements of Taste and Smell. New York: Macmillan; 1986. p. 565-578.
15. Fitzgerald DC. Head trauma: hearing loss and dizziness. J Trauma 1996; 40: 488-496.
16. Bergemalm P-O, Borg E. Long-term objective and subjective audiologic consequences of closed head injury. Acta Otolaryngol 2001; 121: 724-734.
17. Li J, Goldberg G, Munin MC, et al. Post-traumatic bilateral facial palsy: a case report and literature review. Brain Injury 2004; 18: 315-320.
18. Diamond C, Frew I. The facial nerve. Oxford: Oxford University Press,1979: 162-256.
19. Darrouzet V, Bonfils-Dindart C, Bebear JP. Management of post-traumatic facial paralysis. A decision based on a series of 85 cases. Neurochirurgie 1998; 44: 235-246.
20. Darrouzet V, Duclos JY, Liguoro D, et al. Management of facial paralysis resulting from temporal bone fractures: our experience in 115 cases. Otolaryngol Head Neck Surg 2001; 125: 77-84.
21. Adegbite AB, Khan MI, Tan L. Predicting recovery of facial nerve function following injury from a basilar skull fracture. J Neurosurg 1991; 75: 759-762.
22. Mutyala S, Holmes JM, Hodge DO, Younge BR. Spontaneous recovery rate in traumatic sixth-nerve palsy. Am J Ophthalmol 1996; 122: 898-899.
23. Collet FJ. Sur un nouveau syndrome paralytique pharyngolarynge par blessure de guerre (Hemiplegie glosso-laryngo-scapulopharyngee) Lyon Med. 1915; 124: 121-129.
24. Sicard JA. Syndrome du carrefour condylo-dechire posterieur (type pur de paralysie laryngee associee) Marseille Med. 1917; 53: 383.
25. Comacchio F, D'Eredita R, Poletto E, Poletti A, Marchiori C. Hemangiopericytoma of the skull base and Collet-Sicard syndrome: a case report. Ear Nose Throat J. 1995; 74: 845-847.
26. Mohanty SK, Barrios M, Fishbone H, Khatib R. Irreversible injury of cranial nerves 9 trough 12 (Collet-Sicard syndrome). Case report. J Neurosurg. 1973; 38: 86-88.

27. Anderson PA, Montesano PX. Morphology and treatment of occipital condyle fractures. Spine. 1988; 13: 731-736.
28. Sharma BS, Mahajan RK, Bhatia S, Khosla VK. Collet-Sicard syndrome after closed head injury. Clin Neurol Neurosurg. 1994; 96: 197-198.
29. Castling B., Hicks K. Traumatic isolated unilateral hypoglossal nerve palsy-case report and review of the literature. Br J Oral Maxillofac Surg. 1995; 33(3): 171-173.
30. Hong JT, Lee SW, Son BC, Sung JH, Kim IS, Park CK. Hypoglossal nerve palsy after posterior screw placement on the C-1 lateral mass: case report. J Neurosurg Spine. 2006; 5(1): 83-85.

Correspondencia a:
Dr. Marcelo José da Silva de Magalhães
Neurosurgeon.
Mail to: Marcelo JS Magalhães. 375 Capelinha Street, Antônio Pimenta. Zip Code 39402-315.
Montes Claros, Minas Gerais. Brazil.
E-mail: marcelo7779@yahoo.com.br