J Neurosurg 113:547–555, 2010

Cranial nerve injury after minor head trauma

Clinical article

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Object. There are no specific studies about cranial nerve (CN) injury following mild head trauma (Glasgow Coma Scale Score 14–15) in the literature. The aim of this analysis was to document the incidence of CN injury after mild head trauma and to correlate the initial CT findings with the final outcome 1 year after injury.

Methods. The authors studied 49 consecutive patients affected by minor head trauma and CN lesions between January 2000 and January 2006. Detailed clinical and neurological examinations as well as CT studies using brain and bone windows were performed in all patients. Based on the CT findings the authors distinguished 3 types of traumatic injury: no lesion, skull base fracture, and other CT abnormalities. Patients were followed up for 1 year after head injury. The authors distinguished 3 grades of clinical recovery from CN palsy: no recovery, partial recovery, and complete recovery.

Results. Posttraumatic single nerve palsy was observed in 38 patients (77.6%), and multiple nerve injuries were observed in 11 (22.4%). Cranial nerves were affected in 62 cases. The most affected CN was the olfactory nerve (CN I), followed by the facial nerve (CN VII) and the oculomotor nerves (CNs III, IV, and VI). When more than 1 CN was involved, the most frequent association was between CNs VII and VIII. One year after head trauma, a CN deficit was present in 26 (81.2%) of the 32 cases with a skull base fracture, 12 (60%) of 20 cases with other CT abnormalities, and 3 (30%) of 10 cases without CT abnormalities.

Conclusions. Trivial head trauma that causes a minor head injury (Glasgow Coma Scale Score 14–15) can result in CN palsies with a similar distribution to moderate or severe head injuries. The CNs associated with the highest incidence of palsy in this study were the olfactory, facial, and oculomotor nerves. The trigeminal and lower CNs were rarely damaged. Oculomotor nerve injury can have a good prognosis, with a greater chance of recovery if no lesion is demonstrated on the initial CT scan. *(DOI: 10.3171/2010.6.JNS091620)*

Key Words • **head injury** • **cranial nerve** • **minor head trauma**

Numerous authors in the literature have analyzed CN palsy after severe head injury; however, there are no specific studies about CN injury following mild head trauma (initial GCS score of 14–15 after the trauma).[2,16,17,28] The aim in this study was to document the incidence of CN injury after mild head trauma and to correlate the initial CT findings with the final outcome 1 year after the trauma. The obtained information could then be used to offer a prognosis assessment of CN injury following mild head trauma.

Methods

We studied 49 consecutive patients affected by minor head trauma and CN lesions between January 2000 and January 2006. We considered those patients who had a GCS score of 14–15[12] as suffering from minor head trauma. Detailed clinical and neurological examinations were performed in all patients. Computed tomography scanning using brain and bone windows was performed when at least 1 of the following risk factors was present—posttraumatic amnesia, loss of consciousness, posttraumatic seizure, headache, vomiting, focal neurological deficit, skull fracture, coagulopathy, anticoagulant therapy—and in patients older than 60 years of age. Every patient with a neurological deficit, CT scan abnormalities, or persistent cerebral commotion was admitted to our neurosurgical unit for clinical monitoring and to receive specific therapy. Anterior fossa, petrous bone, and posterior fossa CT scans; audiograms; visual evoked potentials monitoring; and other investigations were conducted in specific circumstances. Visual evoked potentials monitoring was undertaken when a visual pathway disorder

Abbreviations used in this paper: CN = cranial nerve; GCS = Glasgow Coma Scale; SAH = subarachnoid hemorrhage.

was suspected, and brainstem auditory evoked responses allowed us to investigate the auditory pathway. From the CT scans, 3 types of traumatic injury were differentiated: no lesion, that is, no traumatic lesion was identified; skull base fracture, that is, occipital, temporal and petrous, ethmoid, sphenoid, maxillary, or orbital roof fractures were identified; or other CT abnormality, that is, contusion, hematoma, or SAH. Three grades of clinical recovery from CN palsy were distinguished: no recovery (no evidence of clinical improvement in the nerve function), partial recovery (some objective clinical recovery), or complete recovery (at the end of the follow-up period, the patient was asymptomatic). Patients were followed up for 1 year after head injury. Four check-ups were conducted by the same senior staff neurosurgeon (A.G.C.) during this period: in the 1st, 3rd, 6th, and 12th month after trauma. Patients with mild head injury and nonadmission criteria were followed up by the general practitioner and readmitted if any neurological deficit was observed.

The linear association Mantel-Haenszel chi-square test was used to determine whether the ordinal variable CT scan abnormality (skull base fracture, other abnormality, or no abnormality) was associated with outcome after 1 year (deficit vs no deficit). The Fisher exact test was used in cases in which the independent variable CT scan abnormality fit 2 categories. Group comparisons of demographic data were conducted using the nonparametric Kruskal-Wallis test or the linear association Mantel-Haenszel chi-square test. All tests were 2-tailed. Values are expressed as means and ranges. A significance level of 5% ($p < 0.05$) was accepted in all cases. The SPSS software, version 15.0 (SPSS, Inc.), was used for statistical analysis.

Results

Of the 19,800 patients admitted to our hospital with head trauma between January 2000 and January 2006, 16,440 had mild head injury. Forty-nine patients had CN injury, an incidence of 0.3%. There was a preponderance of male patients: 14 were female and 35 were male. The median age was 52 years. Posttraumatic single nerve palsy was observed in 38 (77.6%) patients and multiple nerve injury in 11 (22.4%). Cranial nerves were affected in 62 cases. The most affected CN was the olfactory nerve (CN I), followed by the facial nerve (CN VII; Table 1). When more than 1 CN was involved, the most frequent association occurred between CNs VII and VIII (Table 2).

One year after head trauma, a CN deficit was present in 26 (81.2%) of the 32 cases with a skull base fracture, 12 (60%) of 20 cases with other CT abnormalities, and 3 (30%) of 10 cases without CT abnormalities (Table 3 and Fig. 1). The difference between these proportions was statistically significant ($p = 0.002$). The proportion of male patients was significantly higher in the group with skull base fractures compared with the group with other CT abnormalities and without CT abnormalities ($p = 0.013$). There was no significant association between the sex of patients and the prognosis of the CN deficit ($p > 0.05$; Table 4).

Olfactory Nerve

Among 49 patients with a combined total of 62 CN injuries, we found 13 olfactory nerve injuries (21%). Only 3 patients had multiple CN injuries involving the olfactory nerve (CNs I and II, and I and VIII). There were 8 male and 5 female patients with olfactory nerve injuries, with a median age of 56 years. The initial GCS score after the head trauma was 15 in 9 patients and 14 in 4 patients. Anosmia was the most common symptom, occurring in 12 of the 13 patients. All 12 of these patients also presented with taste affection. Rhinorrhea was seen in only 2 cases, with 1 requiring surgical treatment to repair the CSF leakage. Other clinical findings were observed when more than 1 CN was involved. Computed tomography scans showed bone fractures in 6 patients: frontal sinus, temporal and petrous, ethmoidal (Fig. 2), sphenoid, maxillary, orbital roof, and LeFort I and II fractures, which were seen in 2 of the 6 patients. In most of these patients, the fracture was not the only injury. Five of the 6 patients also had hemorrhagic lesions. The most frequent intracranial lesion was a frontal base contusion, which was observed in 7 of the 13 patients with olfactory nerve injuries. Four patients had bilateral frontal base contusions, and only 1 of these (25%) showed clinical improvement after long-term follow-up. Three patients had unilateral frontal base contusions, and 2 of these (66.6%) showed clinical improvement. Other associated intracranial lesions were pneumoencephalus in 2 patients: epidural hematoma in 1 patient and traumatic SAH with temporal contusion in 1 patient. A nondiagnostic CT scan was obtained in 3 pa-

TABLE 1: Cranial nerve injury distribution

CN	No. of Nerves Injured
olfactory	13
optic	5
oculomotor	7
trochlear	7
trigeminal	1
abducens	8
facial	11
acoustic	7
glossopharyngeal	1
vagus	1
spinal accessory	1
hypoglossal	0
total	62

TABLE 2: Multiple CN injury associations

Nerve Group Affected	No. of Patients
VII & VIII	3
I & II	2
III & IV	2
III, VI, & VII	1
I & VIII	1
VI & VII	1
IX, X, & XI	1

TABLE 3: Correlation between CT scan findings and clinical recovery

Clinical Recovery	CT Findings: No Lesion	Other CT Findings	Skull Base Fracture	Total
no recovery	1	6	10	17
partial recovery	2	6	16	24
complete recovery	7	8	6	21
total	10	20	32	62

tients. Five patients showed partial clinical improvement during the follow-up period, with the remaining 8 patients suffering the same deficits. None of the 13 patients had fully recovered after 1 year. No statistical association was found between the kind of lesion demonstrated on CT and olfactory nerve recovery.

Optic Nerve

Five patients (8.1%) were affected by injury to the optic nerve. In 3 cases, the optic nerve was the only CN affected; in the other 2 cases, the olfactory nerve was injured as well. All patients were male. The median age was 52 years. The initial GCS score after trauma was 15 in 4 patients and 14 in 1 patient. In all patients, visual acuity was affected, and 2 patients were completely blind in the injured eye. In 1 case, a burst eye required surgical extraction of the ocular globe. In another case, an associated epidural hematoma required surgical treatment. In all cases, CT scans revealed bone fractures: temporal bone fracture, orbital fracture, sinus occupation, and a LeFort I fracture. Contusions and an epidural hematoma were associated in 3 of the 5 cases. In 1 case, an orbital and temporoparietal fracture caused a pneumocompressive neuropathy of the optic nerve. In 4 of the 5 patients, a skull base fracture was associated with optic nerve lesions. Visual acuity improved in 3 patients during the follow-up period, 1 of whom recovered normal

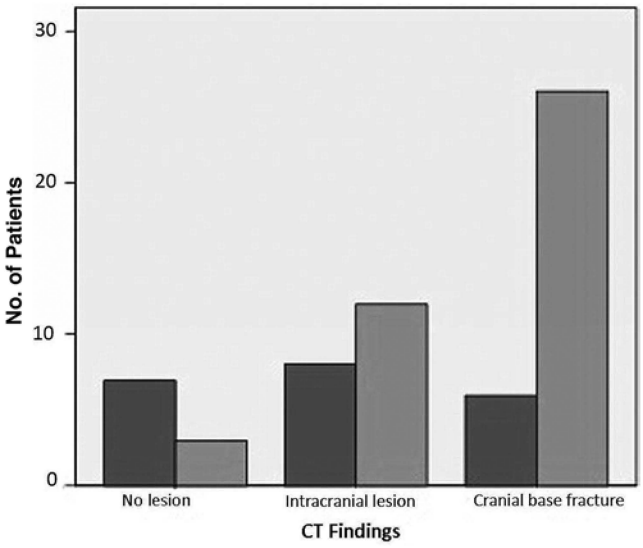

Fig. 1. Bar chart relating CT findings with the final outcome after 1 year of follow-up. *Dark gray bars,* no deficit; *light gray bars,* deficit.

TABLE 4: Statistical correlation between CT findings and clinical recovery

Factor	No Lesion	Other CT Abnormalities	Skull Fracture	p Value
no. of patients	10	17	22	
sex (M/F)	5:5	11:6	19:3	0.013*
mean age (yrs)	45	45	42	>0.05
multiple nerves affected	0/10	4/17	7/22	>0.05
CN I	3/13	4/13	6/13	
complete recovery	0/3	0/4	0/6	>0.05
partial recovery	2/3	0/4	3/6	
no recovery	1/3	4/4	3/6	
deficit	3/3	4/4	6/6	
CN II	0/5	1/5	4/5	>0.05
complete recovery	0/0	0/1	1/4	
partial recovery	0/0	1/1	1/4	
no recovery	0/0	0/1	2/4	
deficit	0/0	1/1	3/4	
CN III	0/7	4/7	3/7	<0.029*
complete recovery	0/0	4/4	0/3	
partial recovery	0/0	0/4	3/3	
no recovery	0/0	0/4	0/3	
deficit	0/0	0/4	3/3	
CN IV	3/7	2/7	2/7	<0.029*
complete recovery	3/3	0/2	0/2	
partial recovery	0/3	1/2	2/2	
no recovery	0/3	1/2	0/2	
deficit	0/3	2/2	2/2	
CN V	0/1	1/1	0/1	>0.05
complete recovery	0/0	1/1	0/0	
partial recovery	0/0	0/1	0/0	
no recovery	0/0	0/1	0/0	
deficit	0/0	0/1	0/0	
CN VI	4/8	3/8	1/8	>0.05
complete recovery	4/4	2/3	0/1	
partial recovery	0/4	1/3	1/1	
no recovery	0/4	0/3	0/1	
deficit	0/4	1/3	1/1	
CN VII	0/11	4/11	7/11	>0.05
complete recovery	0/0	1/4	2/7	
partial recovery	0/0	2/4	4/7	
no recovery	0/0	1/4	1/7	
deficit	0/0	3/4	5/7	
CN VIII	0/7	1/7	6/7	>0.05
complete recovery	0/0	0/1	0/6	
partial recovery	0/0	0/1	1/6	
no recovery	0/0	1/1	3/6	
deficit	0/0	1/1	6/6	
CN IX	0/1	0/1	1/1	>0.05
complete recovery	0/0	0/0	1/1	

(continued)

TABLE 4: Statistical correlation between CT findings and clinical recovery *(continued)*

Factor	No Lesion	Other CT Abnormalities	Skull Fracture	p Value
CN IX	0/1	0/1	1/1	>0.05
partial recovery	0/0	0/0	0/1	
no recovery	0/0	0/0	0/1	
deficit	0/0	0/0	0/1	
CN X	0/1	0/1	1/1	>0.05
complete recovery	0/0	0/0	1/1	
partial recovery	0/0	0/0	0/1	
no recovery	0/0	0/0	0/1	
deficit	0/0	0/0	0/1	
CN XI	0/1	0/1	1/1	>0.05
complete recovery	0/0	0/0	1/1	
partial recovery	0/0	0/0	0/1	
no recovery	0/0	0/0	0/1	
deficit	0/0	0/0	0/1	
CN XII	none	none	none	

* Statistically significant.

visual acuity by the end of the follow-up. The 2 patients who required surgery (epidural hematoma and burst eye) suffered a complete visual loss (Fig. 3). No statistical association was found between the kind of CT-demonstrated lesion and CN recovery.

Oculomotor Nerve

Seven patients (11.3%) had posttraumatic oculomotor nerve injury. Three of these patients had multiple CN palsies; the most common associated nerve injury was to the trochlear nerve. Other associated nerve injuries affected CNs VI and VII (Table 2). There were 4 male and 3 female patients. The median age was 32 years. The initial GCS score after trauma was 15 in 3 patients and 14 in 4 patients. Oculomotor nerve palsy produced a binocular diplopia in all patients. In 2 cases with multiple nerve palsy (associated CN III and IV palsy), there was a mixed horizontal and vertical binocular diplopia. Every case showed an intracranial bleeding lesion when the CT scan was obtained: 1 case of epidural hematoma, 2 cases of bifrontal contusion, 1 cerebellar contusion, 1 occipital contusion, and 2 cases of perimesencephalic SAH (Fig. 4). The patient with the associated epidural hematoma required a craniotomy and clot extraction. None of the 3 patients with an oculomotor nerve deficit and skull base fracture completely recovered from the deficit. However, all patients with an oculomotor nerve deficit and a CT abnormality other than a skull base fracture completely recovered from the deficit. The difference between these 2 proportions was statistically significant ($p = 0.029$).

Trochlear Nerve

Seven patients had posttraumatic trochlear nerve injury, for an incidence of 11.3%, including 2 patients with multiple nerve injuries involving CNs III and IV. Five patients were male and 2 were female. The median age was 32 years. On the initial trauma, 6 patients had a GCS Score 15 and 1 patient had a GCS Score 14. All patients presented with binocular diplopia. In 3 cases, the CT scan showed no traumatic lesions. Hemorrhagic lesions were identified in 4 patients: 2 patients had parenchymal contusions in both frontal lobes (1 of these also had a cerebellar contusion), another patient showed an acute subdural hematoma (Fig. 4) that did not need surgical treatment, and 1 patient had subarachnoid bleeding. Complete spontaneous recovery of the trochlear nerve was observed in 3 (42.8%) of 7 cases; all patients improved except 1 who showed no clinical evolution. None of the 4 patients with a trochlear nerve deficit and skull base fracture or other CT abnormality complete-

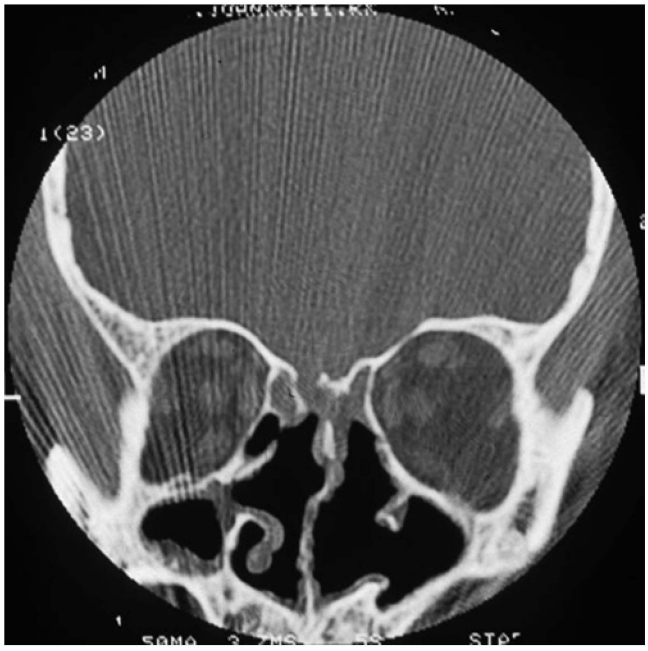

Fig. 2. Coronal CT scan, bone window setting, demonstrating a fracture through the cribriform plate in a 58-year-old man with associated bifrontal contusions. This kind of fracture causes disruption of the olfactory rootlets and subsequent impairment of smell. This patient showed partial clinical improvement by the end of the follow-up period.

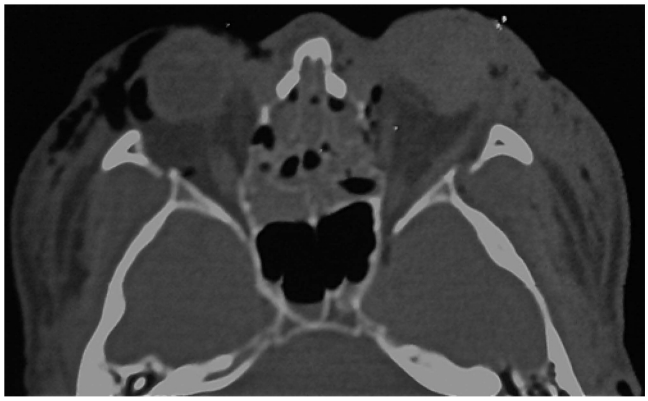

Fig. 3. Axial CT scan showing multiple fractures and bone fragments affecting both orbits in a 57-year-old man with associated LeFort I fracture, frontal contusions, and a burst eye that required surgical extraction of the ocular globe.

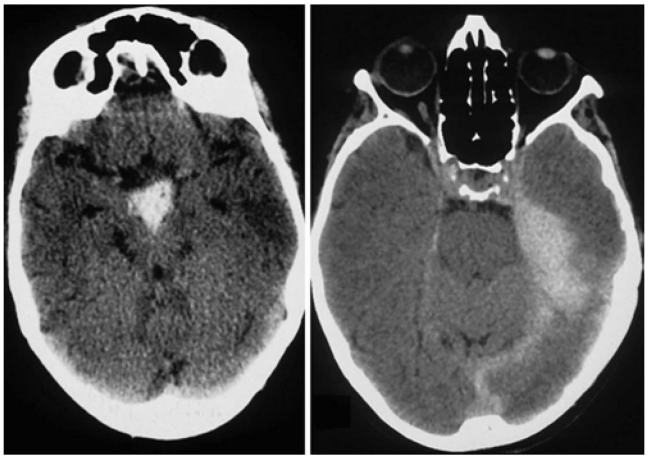

Fig. 4. Left: Axial CT scan showing an anterior perimesencephalic SAH in a 21-year-old woman with CN III palsy that became completely asymptomatic. **Right:** Axial CT scan demonstrating a tentorial subdural hematoma affecting the CN IV in a 53-year-old man who partially recovered from a vertical diplopia in the 3rd month of follow-up.

ly recovered from the deficit. However, all patients with a trochlear nerve deficit and no CT abnormality completely recovered from their deficit. The difference between these 2 proportions was statistically significant ($p = 0.029$).

Trigeminal Nerve

Only 1 patient (1.4%) was affected by a trigeminal nerve lesion: a 71-year-old woman who had posttraumatic neuralgia associated with a subdural and tentorial hematoma (GCS Score 15). This patient required medical management and showed complete remission of symptoms after 6 months.

Abducens Nerve

There were 8 patients (12.9%) with abducens nerve injury. Three had a bilateral nerve injury. Multiple CN palsies developed in 2 patients; 1 of these cases became a complete facial nerve palsy (House-Brackmann Grade VI), the other case involved CNs III, VI, and VII. Five patients were male and 3 were female. The median age was 52.5 years. Four patients had a GCS Score 14, the other 4 a GCS score of 15. Diplopia occurred in all patients and was associated with facial palsy in the 2 cases with more than 1 affected nerve. Four patients had nondiagnostic CT scans; the scans obtained in the other 4 patients demonstrated frontal and cerebellar contusions. A longitudinal temporal bone fracture and cavernous sinus and sphenoid bone fractures were revealed in 1 patient. All patients had improved nerve function 1 year after the trauma. Six of the 8 patients completely recovered nerve function, and 2 patients had a partial recovery. No statistical association was found between the kind of lesion demonstrated on CT and CN recovery.

Facial Nerve

Eleven patients (17.7%) had a facial nerve injury after head trauma. Five patients had more than 1 affected CN, and CN VIII was the most commonly affected nerve (3 patients). Other associated injured nerves included CNs III and VI. Ten patients were male and 1 was female. The median age was 28 years. Seven patients had GCS scores of 14; 4 patients had GCS scores of 15. Facial nerve injury was graded according to the House-Brackmann scale.[9] Hearing loss occurred in 5 cases with associated acoustic nerve injury. Blood in the external auditory canal was observed in 7 patients, whereas a CSF fistula was present in only 2 cases. A temporal bone fracture was evidenced in 7 patients. Other CT findings included occupation of the mastoid cells (4 cases), lobe contusions (4 cases), and epidural hematomas (2 cases). If damage to the facial nerve was recorded, a course of oral steroids was given. We saw that facial palsy improved in 6 of the 11 patients by the end of the observation period, and 3 patients showed normal facial nerve function (Table 5). No statistical association was found between the kind of lesion demonstrated on CT and CN recovery.

Vestibulocochlear Nerve

Seven patients (11.3%) were affected by acoustic nerve injury; all patients were male, and the median age was 44 years. Three patients had a single vestibulocochlear nerve lesion, whereas 4 patients showed multiple CN involvement (3 patients had CN VII and VIII lesions, and 1 patient had associated CN I and VIII lesions). Six patients had a GCS score of 15, and 1 patient had a GCS score of 14. On clinical examination, hearing loss was found in all patients and vertigo in 1 patient. Orthoscopic examination of the external auditory canal revealed otorrhagia due to tympanic perforation in 3 patients and hemotympanum in 1 patient. Computed tomography scans showed temporal or occipital bone fractures (6 patients; Fig. 5), occupation of the mastoid process (3 patients), and frontal and temporal lobe contusions (1 patient).

All patients suffered sensory-neural hearing loss. Four patients showed no improvement, whereas the other 3 partially recovered hearing loss. No statistical association was found between the kind of lesion demonstrated on CT and CN recovery.

Lower Cranial Nerves: Glossopharyngeal, Vagus, Spinal Accessory, and Hypoglossal

One patient presented with a lesion of the lower CNs

TABLE 5: Facial nerve injury: clinical recovery after 1 year

Case No.	House-Brackmann Score	
	Initial	Final
1	V	IV
2	V	II
3	IV	IV
4	IV	II
5	V	V
6	II	I
7	III	I
8	IV	II
9	III	I
10	VI	IV
11	VI	III

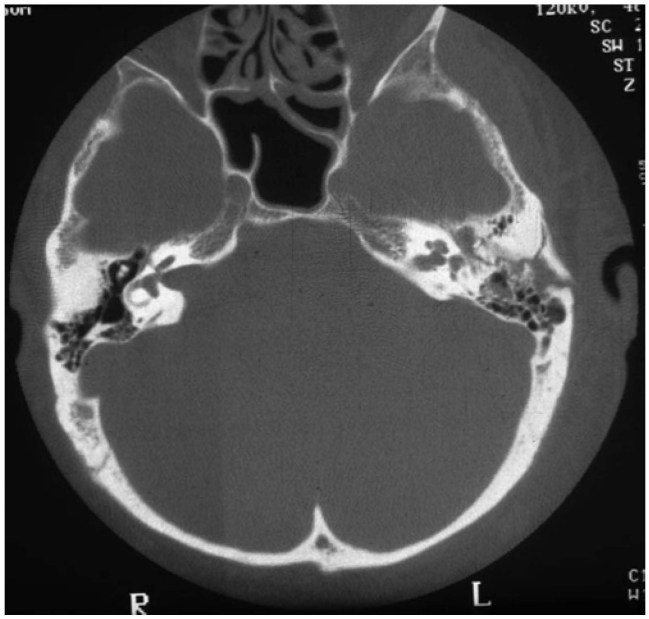

Fig. 5. Temporal bone CT scan showing a longitudinal left temporal bone fracture in a 60-year-old man with facial and vestibulocochlear nerve palsy. Otorrhagia and impairment of audition were associated with the facial palsy, which had not improved by the end of the follow-up period.

(1.4%): a 17-year-old boy with a GCS score of 15 on admission together with associated CN IX, X, and XI injuries that caused dysphagia, loss of gag reflex, and saliva retention in the piriform sinus. A fracture passing through the jugular foramen was observed on CT, along with small bifrontal contusions. Dysphagia was persistent until the 6th month when improvements began; it finally resolved after 1 year.

Discussion

Cranial nerve injury following severe head trauma has been extensively described in the literature, but far less attention has been given to CN injury after minor head trauma. In 2005, Patel et al.[28] reported on a series of 100 patients with CN injury following head injury: 50 patients had CN injury following mild head trauma, 67 patients had a single CN lesion, and 32 patients had multiple nerve lesions. Follow-up was performed from 6 months to 2 years in the patients. The single nerve most affected was CN VII, and the most frequent associations were with CNs II, III, IV, and VI; VI and VII; and VII and VIII. Other series documented in the literature do not have enough data to be compared with the results of our study.[16]

We studied 49 patients with CN injury following mild head trauma to document its incidence, correlate CN lesions with CT scan findings, and evaluate the clinical outcome of every CN injured. Sixty-two CNs were affected in our analysis. Posttraumatic single nerve palsy was seen in 38 patients (77.6%), and multiple nerve injuries were seen in 11 (22.4%). These results are similar to those obtained by Patel et al.,[28] where single nerve palsy was seen in 67% of patients and multiple nerve injuries were observed in 32%. Only 50% of the patients had mild head injury. The most affected CN in our study was the olfactory nerve, and the second most affected CN was the facial nerve. In the Patel series, CN VII was most often affected. Chung et al.[3] suggested that CNs I, VII, and VIII are injured the most often and that the lower CNs are affected the least frequently. The results of both our study and Patel and colleagues' are consistent with this information. In our study, when more than 1 CN was involved, the most frequent association occurred between CNs VII and VIII.

It is worth noting that the CN injury following minor head trauma in this series may have been underestimated, as some patients with mild deficits and a rapid recovery may not have reported their deficit, and consequently the trauma was not diagnosed.

Overall analysis of our results revealed a better prognosis 1 year after the head trauma in patients without CT-demonstrated abnormalities and the worst prognosis in patients with skull base fracture. Patients with other abnormalities, excluding skull base fracture, had an intermediate chance of recovery. Therefore, the data suggest that a skull base fracture is the radiological finding most frequently associated with sequelae. In addition to this initial analysis, it is important to analyze each CN separately.

The olfactory nerve is the most frequently injured CN in most published articles.[10,19,31] In accordance with the literature, we observed that in mild head trauma the olfactory nerve is also the most frequently damaged CN. Damage to the olfactory nerve results in anosmia, which may be due to injury to the nerve filaments at the cribriform plate, to the olfactory bulb, or to the olfactory tracts. Olfaction can also be impaired by edema, hematoma, ischemia, or injury to orbitofrontal and temporal lobes in closed-head injuries.[5] In our series, anosmia was present in 12 of 13 patients with olfactory nerve injury, making it the most important symptom in accordance with the literature.[3,17,19,20,31] Only the patients who had olfactory or taste symptoms after trauma were included in this series. As previously explained, this strategy may have caused an underestimation in the incidence of olfactory damage following minor head trauma. Two of our patients had CSF rhinorrhea, which usually occurs due to a fracture in the cribriform plate or frontal sinus associated with a dural tear. In such cases, there is an increased risk of posttraumatic meningitis,[2,3,21] although that complication was not seen among our series. Five patients (38.5%) with an olfactory deficit showed clinical improvements during the follow-up period. Chung et al.[3] reported that 40% of their patients with posttraumatic anosmia recovered from loss of smell, and Keane et al.[17] found that recovery occurred in more than one-third of their cases. Early recovery is directly related to the trauma, such as local hematoma, concussion, or edema, whereas delayed recovery indicates olfactory axon regeneration.[2,3,11] Trivial blows, as in the case of mild head injury, can also cause permanent loss of smell. In our series, CT scans showed bone fractures in 6 patients. In 5 of the 6 patients, hemorrhagic intracranial lesions were also present. Among these patients, only

2 showed clear clinical improvements; the rest retained the same symptoms. Severe disruption of lamina cribrosa and fibrotic tissue can mean that the olfactory neuron axons are unable to reconnect with the olfactory bulb.[2,3,11] Another mechanism of injury can be the direct impact of the frontal lobes against the anterior skull base fossa, as demonstrated by the presence of multiple frontal base contusions in most of these patients. Seven patients had basal frontal lobe contusions that were bilateral in 4 cases and unilateral in 3. There was a tendency for anosmia to improve less in the bilateral group (25%) than in the unilateral group (66.6%), which could be explained by the bilateral olfactory nerve lesion in the bilateral group. Olfactory nerve injury following mild head trauma can appear by the same mechanism as frontal base contusions, from the direct impact of the olfactory nerve against the anterior cranial base, causing hematoma, concussion, or edema in both olfactory nerve fibers and increasing the chance of a permanent lesion.

The optic nerve, the largest CN, is divided into 4 segments: intraocular, intraorbital, intracanalicular, and intracranial. The intracanalicular portion of the optic nerve is the most susceptible to injury in closed head trauma because of its dural covering, which becomes contiguous with the periosteum of the optic canal and the orbit. Optic nerve injury is described in the literature as a rare injury.[4,8,23,25,28] In our study, there were 5 patients affected by CN II injury. Optic nerve injury can result in blindness or a degree of visual loss;[14,17,18] in fact, visual acuity was affected in all 5 patients with an optic nerve lesion, and 2 patients were completely blind in the injured eye. One case had a burst eye requiring surgical extraction of the ocular globe; in this particular case the lesion affected the eye globe and the optic nerve. Immediate visual loss is usually produced by direct optic nerve trauma; delayed visual deterioration indicates secondary factors like ischemia, edema, or compression that make this situation potentially reversible.[2,17,24] Fractures of the optic canal, orbit, or skull base can damage the optic tracts.[5,14] In all 5 patients, bone fracture was observed on the CT scan. Mahapatra and Tandon[24] showed that spontaneous visual recovery occurred in 51%–57% of cases following conservative management. In our series, 3 patients (60%) demonstrated improved visual acuity during the follow-up period; however, the 2 cases that required surgery (epidural hematoma and burst eye) suffered complete visual loss.

If we consider the ocular motor nerves (CNs III, IV, and VI) as a whole, they are the most often affected CNs following head injury. In our series, 22 patients were affected by ocular motor palsies. The complex anatomy of the oculomotor nerve makes clinical findings highly variable—not only because of the various brainstem nuclei that are involved, but also given the nerve tract and the separation of its branches upon entering the orbit. Traumatic isolated CN III palsy is not common.[13,26,27,29,32,34] In our series, 3 of the 7 patients had multiple CN palsy. In these cases, the most commonly associated CN was the trochlear nerve. Keane[16] reported that in 285 of 979 cases with the simultaneous involvement of 2 or more different CNs, the most common combination was CNs III and VI. Oculomotor nerve paresis generally causes mydriasis, impairment of eye adduction, upward and downward gaze palsy, and eyelid drop in the affected eye. In our study, all patients with CN III lesions presented with binocular diplopia. In the 2 cases that involved CN III and IV palsy, there was a mixed horizontal and vertical binocular diplopia. Muthu el al.[27] reported that when isolated CN III palsy occurs, the closed head injury must be more severe than mild and that minor head trauma may precipitate oculomotor nerve palsy in patients with an occult intracranial mass lesion because of the mechanical stress.[6,37] Our 7 cases showed bleeding complications when CT scanning was performed, which could explain nerve impairment. This clinical feature could happen with any abnormality, however, and is perhaps caused by damage to the oculomotor nerve by the ipsilateral posterior petroclinoid ligament, as suggested by Kaido et al.[13] The muscle paresis in all our patients improved, and 4 patients (57.14%) became completely asymptomatic. In our patients, palsy due to a compressive lesion was likely to be completely resolved on removal of the mechanical damage, although aberrant function regeneration has been described when the fascicles are injured.[3,36]

According to the literature, trauma is the most common cause of CN IV palsy.[29] The trochlear nerve is the thinnest and longest ocular motor nerve and the only CN to decussate its fibers and exit the brainstem on its dorsal surface, and thus CN IV malfunction can occur after apparently trivial head trauma.[3] Patients report seeing double images that are vertically or obliquely oriented to each other. In 3 of the 7 cases in our study, the CT scan showed no traumatic lesions. These 3 patients completely recovered nerve function by the end of the 3rd month. In contrast, none of the 4 patients with a trochlear nerve deficit and a skull base fracture or other CT abnormalities completely recovered the deficit. Sydnor et al.[33] reported that spontaneous recovery occurs in 65% of patients with unilateral trochlear palsy. This figure is consistent with our series in which the spontaneous recovery rate was 57.14%.

The abducens nerve is frequently damaged in head injuries; in fact, it is the most frequently injured oculomotor nerve.[2,3,30] Its long intracranial route up to the clivus and over the petrous ridge, along with its strong dural attachments in the cavernous sinus, explain its vulnerability to stretching and displacement by trauma. Eight patients presented with diplopia due to CN VII palsy, making it the most affected ocular nerve in our series as well. All patients with a normal CT scan eventually became asymptomatic. Three of 5 patients, who had an excellent evolution, were asymptomatic after 6 months of follow-up, and the other 2 were asymptomatic by the end of the first year. These data are consistent with reports from Bhatoe [2] and Chung et al.,[3] who suggested an observation period of 6–12 months to allow sufficient time for maximal spontaneous recovery. However, Katzen et al.[14] said that most cases of abducens nerve injury spontaneously recover after 4 weeks. Ocular muscle surgery is offered in cases of lateral rectus palsy that persists beyond the observation period.[2]

Damage to the trigeminal nerve most often occurs to its peripheral branches during severe maxillofacial and

skull base injuries. Sensation along the cutaneous distribution of the involved nerve is affected. Only 1 patient with an interhemispheric and tentorial hematoma had posttraumatic neuralgia that required treatment with carbamazepine; a complete recovery was made by the 6th month of follow-up. In this case, we considered that the tentorial part of the subdural hematoma observed on the CT scan was the cause of the nerve affliction. When a trigeminal nerve lesion is associated with a skull base fracture, the outcome is usually less fortunate: incomplete transaction or scarring on the branches of the trigeminal nerve may result in intractable facial pain and neuroma formation, in such cases medical management is not enough to treat the neuralgia.[2,14]

The facial nerve has a potentially precarious route through the temporal bone, leaving it liable to injury when the base of the skull is fractured at this level. The auditory nerve also courses with the facial nerve in the petrous portion of the temporal bone, and as a result conductive hearing loss frequently occurs. Posttraumatic facial nerve palsy is relatively common, especially when the temporal bone is fractured.[22] This kind of fracture is the most common cause of facial nerve injuries.[1,7] Temporal bone fracture was seen in 7 of the 11 patients with facial nerve palsy in our study. Of these patients, 4 had a clinical improvement and 2 recovered from the deficit. Three patients that did not have a skull base fracture (epidural hematoma, bifrontal contusions, and occupation of mastoid cells) had improved facial nerve function by the end of the follow-up period. In total, 9 patients had improved by the end of the follow-up period. It seems that CN VII improvement after injury is relatively common, whereas complete recovery seems to be achievable but infrequent. No statistical association was found between the kind of CT lesion demonstrated and CN recovery. Facial nerve palsy can be categorized as either immediate or delayed onset.[3] Immediate onset results from transaction or other forms of severe neural trauma and carries the worst prognosis. Delayed onset palsy occurs due to external compression by edema, hematoma, or swelling of the nerve, and in these cases prognosis is more favorable.[2] Turner[35] found a satisfactory return of facial function in 82% of cases of delayed facial palsy, whereas only 53% showed a good recovery after immediate paralysis. In our series, only 1 patient had delayed-onset palsy, 2 days after the blow. In this case, the patient became completely asymptomatic in the 3rd month of the follow-up period. Recognizing a delayed onset facial nerve injury is important in relation to the final outcome.

The vestibulocochlear nerve was the fourth most injured CN in our series. Trauma to the CN VIII varies from concussion of the cochlea and semicircular ducts to fractures of the petrous bone. Damage to the CN VIII is common after transverse fractures of the temporal bone from frontal or occipital impact. Six of the 7 patients in our series had a skull base fracture. Typical symptoms after CN VIII injury are deafness and labyrinthine damage (vertigo, nausea, and dizziness). All our patients presented some degree of hearing loss, as evaluated by audiometry and brainstem evoked potentials. Conductive deafness usually shows signs of recovery; sensorineural deafness, however, has a worse prognosis, especially if the initial hearing loss was complete.[2] This finding is consistent with our own results: 7 patients had audition impairment at the end of the follow-up period (all of whom had neurosensorial hearing loss). Labyrinthine concussion with vertigo occurs frequently, even with mild head injury and the absence of a skull fracture; this condition is often partially or completely reversible.[17] Our patients also presented labyrinthine symptoms. Keane and Baloh[17] reported posttraumatic positional vertigo as the most common symptom of head injury, with spontaneous remission within 3 months and complete remission for almost everyone within 2 years of head injury.

Traumatic injury to the glossopharyngeal, vagus, and accessory nerves is infrequent and usually follows a fracture through the jugular foramen.[2,3,14,17] A fracture passing through this structure was observed on the CT scan obtained in our patient. Dysphagia was treated by feeding the patient via a nasogastric tube. Once swallowing was reestablished at the end of 6 months, oral feeding was resumed.

None of the patients in our series were affected by hypoglossal nerve palsy. Isolated unilateral hypoglossal nerve palsy after fracture of the occipital condyle is rare and usually occurs after a major trauma.[15]

Conclusions

Trivial head trauma that causes minor head injury (GCS Scores 14–15) can cause CN palsies with a distribution similar to that for moderate or severe head injuries. The CNs that presented the highest incidence of palsy in our study were the olfactory, facial, and oculomotor nerves. In contrast, the trigeminal and lower CNs are rarely damaged. Oculomotor nerve injury can have a good prognosis, with more chances of recovery if no lesion is demontsrated on the initial CT scan.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Coello. Acquisition of data: Canals. Drafting the article: Coello. Critically revising the article: Martín. Reviewed final version of the manuscript and approved it for submission: all authors. Statistical analysis: Gonzalez.

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Manuscript submitted November 23, 2009.
Accepted June 8, 2010.
Please include this information when citing this paper: published online July 16, 2010; DOI: 10.3171/2010.6.JNS091620.

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