Minor head injury

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Abstract

Head injury is one of the most important public health issues. Minor head injury accounts for 75% or more of all brain injuries. Perhaps 50% of patients with minor head injury will develop post-concussion syndrome.

In spite of the frequent occurrence of minor head injury, the post-concussion syndrome has received little attention from the medical community. Nevertheless, the syndrome is a well defined clinical entity with anatomopathological, electrophysiological and image properties.

Key words: minor head injury, post-concussion syndrome.

Introduction

Mild head injury (MHI) is one of the most common neurological conditions; only migraine has higher prevalence and incidence.[1]

Around 50% of the patients with MHI develop post-concussion syndrome.[2]

The high prevalence of this pathology has not been discussed thoroughly for several reasons: on one hand, the treatment of head injury in the acute phase is the responsibility of different specialists; on the other, patients often recover spontaneously. Also contributing to this fact is that symptomatic patients are attributed with the desire to obtain monetary compensation.

However, MHI is a clinically defined entity, like post-concussion syndrome. In other wise, there are risk factors and clinical syndromes that are well known; it is known that their treatment may prevent chronic disability, the prognosis of these conditions is predictable and, essentially, anatomopathological, electrophysiological and imaging anomalies exist in this group of patients.[1,2,3]

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Head injury (HI) is one of the most significant problems related to Public Health. MHI accounts for approximately 75% of all head injuries.[4] In an industrialized country, the causes of head injury are as follows: car accidents 45%; falls 30%; occupational accidents 10% and assaults 5%.[5] Falls in the elderly and car accidents in young people are among the most common causes. Males are more frequently affected, in a 2:1 ratio. Approximately 50% of all patients with HI are aged between 15 and 34 years.[4,5]

Definition

MHI is defined according to the following characteristics:[3]

1 - Existence of a brief period of unconsciousness (lasting from seconds to minutes); in most cases there is no loss of consciousness, but rather, a brief period of confusion.

2 - When the patient is treated immediately or soon after the HI event, the Glasgow score 13 to 15. Probably only patients with a score of 15 will actually have MHI. Scores of 13 and 14 correspond to confusion or disorientation, and are associated with a longer period of amnesia.

3 - Post-traumatic amnesia lasts, by definition, less than 24 hours, usually from minutes to hours.

4 - Patients have no focal neurological signs, although they may be pale, diaphoretic and ataxic, and have nausea right after HI.

5 - By definition, neuroradiological tests show no changes; however, this is not always the case.

Pathophysiology and anatomopathological changes

The primary change is a diffuse axonal injury (DAI), caused by deceleration forces. These forces affect

mainly structures that are oriented longitudinally in the skull, such as the axons and small-caliber vessels.

Axonal injury causes failure of axonal transport, leading to edema and, sometimes, lysis of the axon with Wallerian degeneration.[6,7] The role in the release of excitatory neurotransmitters by the synapses of the damaged axons as a cause of cellular loss is still unclear.[2,3]

Vascular injury can disrupt small veins and cause petechial hemorrhages or focal edema.[3]

The distribution of injury appears to occur preferentially in the parasagittal deep white matter, spreading from the cortex to the brainstem.[8]

There is experimental evidence for non-human primates in which the magnitude of the diffuse axonal injury is proportional to the intensity of deceleration force of the HI. The stronger the force, the larger will be the DAI. For this reason, car accidents and falls cause more severe HI, while occupational accidents and assaults cause milder HI.[8]

Small intracerebral petechial hemorrhage was observed in cases of clinically mild HI. These injuries appear not to affect the prognosis significantly,[9] unlike the focal cortical contusions rarely observed in these conditions.[3]

Clinical findings

1 - Headaches

Headaches occur in 30% to 90% of patients who are asymptomatic after a MHI.[2] Paradoxically, headaches are more frequent in these patients than in patients with more severe HI.

There are several types of headaches, but tension headache and occipital neuralgia make up 85% of post-traumatic headaches. Many patients have more than one type of headache. Headaches can occur episodically or daily.

Tension headache – It has varied different distribution (generalized, occipital, bifrontal, cap-like or headband) and is described as a constant or intermittent pressure of variable duration. This type of headache, which is sometimes associated with cervical injury, can be perpetuated by a cervical pathology, such as myofascial injury or intervertebral disc injury.[2,10]

Occipital neuralgia - occurs frequently and may be caused by direct injury of the large occipital nerve or, most commonly, by contraction of the trapezius or paravertebral muscle. Patients report burning pain in the occipital region, which may spread to the temporoparietal, frontal or retro-orbital regions and lasts hours to days, or paroxysmal shock-like pain occurring several times a day.

It may be bilateral, and is sometimes triggered in trigger points of other cervical muscles, such as the digastric or sternocleidomastoid muscle.[2]

Migraine - Migraine can occur with or without aura after a MHI and, sometimes, a MHI may accelerate a migraine episode.[11]

In the first case, the interval between MHI and the first migraine episode can take hours to weeks. In the second case, the episodes are mostly migraine with aura and an explanation appears to exist for the presence of transitory neurological injuries (hemiparesis, somnolence and vomiting, visual changes and signs of brainstem injury) which occur, sometimes, without headaches, in teenagers and young adults after a MHI.[12]

Both conditions described above occur more frequently in patients with a family history of migraine.

Cluster headache – May occur, though rarely, several weeks after the MHI. It appears to be more resistant to treatment than the idiopathic form.[2]

Supraorbital neuralgia – May result from an injury to the supraorbital branch of the first division of the trigeminal nerve. Patients have hypoesthesia in the region and shock-like pain. The same occurs with injury to the infraorbital nerve.[13]

Dysautonomic headache - It has the same characteristics as the previous injury. Patients report acute pain in that region, which may be followed, weeks or months later, by severe unilateral temporo-frontal headache, increased unilateral facial sweating, dilation of the ipsilateral pupil, photophobia, blurred vision and nausea. It may occur several times a day or from month to month[14]. It appears to be caused by stimulus of the sympathetic autonomic nervous system.

Dysaesthesia - Dysaesthesia with scalp lacerations occur frequently. However, even when there are no lacerations, pain (shock-like and burning pain) may occur in the region of the injury. It may last weeks or months, but it rarely lasts more than one year.

2 - Dizziness and changes in hearing

These are frequent and occur in 50% of the patients with MHI one week after the injury and continue after two years, in 10% of cases.[15] Involuntary nystagmus or postural tremor was observed in 30% of the patients

with dizziness and MHI.

Even if no temporal bone fractures are identified, labyrinthine concussion may occur with vertigo. Sometimes, perilymphatic fistula may occur, leading to the development of acute vertigo or acute hearing loss. Sensorineural hearing loss may occur due to temporal bone injury, as well as hemotympanum conductive hearing loss. Bilateral sensorineural hearing loss may also occur, with no fracture, and its cause is not known.[16]

3 – Changes in vision

Blurred vision occurs in 14% of the patients after a MHI.[2] Convergence insufficiency is the cause in most cases and, although the exact location where it is produced is unknown, occipital lobe or mid-brain injuries might cause it.[17] Diplopia caused by traumatic injury of the cranial nerves III, IV and VI can be caused my mild HI.[18] Also the contusions of the optic nerve may lead to impaired visual acuity.[2] HI is the most frequent cause of anosmia. It occurs in 5% after MHI due to damage to the olfactory filaments.

4 - Psychological complaints

These are frequently reported and include personality changes, irritability, anxiety and depression.

Three months after MHI, 50% to 80% of patients have these symptoms. These are persistent symptoms, with a prevalence of 15% after three years.[2] Changes in sleep habits occur in approximately 15% of the cases, and difficulty falling asleep and waking up many times during the night are also common.[19]

5 - Cognitive changes

Four weeks after MHI, 20% of the patients complain of loss of memory and 20%, of difficulty concentrating.[2,3]

6 - Rare sequelae

Seizures - Seizures may be a sequela arising from any type of HI. The risk of seizures five years after a MHI is 0.8%, which is similar to the incidence in the general population. Nevertheless, even the rare occurrence of seizures soon after MHI doesn't rule out a link between seizures and MHI.

Transient global amnesia - May occur within two to 24 hours after a MHI. Some patients experience associated headache. Most patients have a family history of transient global amnesia.[21]

Movement disorders - Tremor can be of any type and involve the hands, tongue and trunk. It may occur immediately after, or up to four weeks after the injury. Imaging tests for all the patients were normal. Primidone does not appear to be effective, but this is not the case for clorazepam and beta-blockers.[22] Goltz demonstrated that HI or stress caused by car accidents would possibly temporarily increase dysfunction in Parkinson's disease.[23,24]

Complications in neurosurgery - The incidence of neurosurgical complications after MHI was estimated at approximately 2%. These complications include epidural haematomas, subarachnoid hemorrhage and intra-parenchymatous hematomas.[25]

Haematomas rarely develop at a later stage, even if an early imaging test is performed and gives a normal result.[26] Any late development may clinically simulate a post-concussion syndrome.

Additional tests

Skull X-ray - The incidence of intracranial complications is the same for patients with MHI with or without visible fracture on a skull x-ray. Therefore, skull x-ray appears to have no benefits in the evaluation of MHI.[27]

Computerized Axial Tomography (CAT scan) in cranio-cerebral injuries - The neurosurgical complications of MHI are rare and yet the use of a CAT scan of the brain in the evaluation of this condition is low. The following are recommended:[28]

• If in the initial stage after HI, the Glasgow score is lower than 15, and changes to mental state or focal neurological deficits are observed, a CAT scan of the brain should be performed. Even if this scan does not reveal any neurosurgical changes, the patient should be monitored, since there is a risk of neurological worsening.

• If in the initial stage after HI, a Glasgow score of 15, normal mental state and no focal neurological deficits are observed, a CAT scan of the brain is not necessary and the patient can be discharged, provided they are monitored by a family member (even if skull x-ray reveals fracture, or consciousness is momentarily lost or cranial nerve injury exist).

Nuclear magnetic resonance (NMR) imaging of the brain - In comparative studies, MRI of the brain is far more sensitive than a CAT scan of the brain.

In the cases of MHI, cortical injuries are usually observed, with hypersignal in T2 that subsequently

disappear. These cortical injuries are interpreted as regional edema, with no areas of contusion or infarction.[29] No clinical or prognostic correlation has been found for this type of injury, therefore, this test is not indicated in cases of MHI.

Electroencephalogram (EEG) - A few anomalies have been observed (Diffuse delta and theta activity and decreased alpha amplitude and frequency) in the EEG of patients with MHI. These changes seem to disappear spontaneously, and no clinical correlation was found. Therefore, this test is not indicated for the evaluation of MHI.[2]

Encephalic Trunk Auditory Evoked Potentials (AEP) - Changes have been observed in AEP (interval I-V) after MHI. These anomalies, if detected early, appear to predict a greater risk of persistence of symptoms; however, these data are not definitive.[30]

For that reason, AEPs are not indicated for the routine evaluation of MHI.

Nevertheless, specific additional tests are indicated for patients reporting auditory, visual or cognitive conditions, to clarify the cause of these complaints.

Treatment

Treatment is individual to each patient, and depends on the specific complaints of each one (*Table I*). Reassuring patients that most of the effects disappear three months after the MHI is essential and is of great practical benefit.

Prognosis

In the last few years, several prognostic studies have been conducted. However, comparing these studies is difficult because they used very different data (including different definitions of MHI, different additional tests, and diverse follow-up periods).

The prognosis appears to depend on several factors:[2,3]

Loss of consciousness - The likelihood of developing of post-concussion syndrome is the same for a patient who has not lost consciousness as for a patient who has lost consciousness for a period of less than one hour.

Post-traumatic amnesia - The duration of post-traumatic amnesia was related, in most studies, to the existence of post-concussion syndrome.

Additional tests - No relation whatsoever was found between the changes in the additional tests and post-concussion syndrome, except for the presence of cortical contusions, which are associated with a poorer prognosis.

Individual characteristics - Age over 40 years, females and lower socio-economical level appear to be predominant factors in the persistence of the symptoms.

Also, the existence of previous HI, even if mild, consumption of alcohol or other drugs, and personality characteristics are risk factors for the presence and persistence of symptoms.

The association of other injuries (orthopedic, soft tissue, etc.) not only contributes to the existence of a post-concussion syndrome, but can also be a source of depression and anxiety.

TABLE I

Treatment of post-concussion syndrome

1 – Tension headache
Non-steroidal anti-inflammatory drugs
Antidepressants
Muscle relaxants
T E N S
Biofeedback
2 - Migraine
Prophylactic treatments
Beta-blockers
Antidepressants
Calcium channel blockers
Sodium valproate
Acute phase
Non-steroidal anti-inflammatory drugs (NSAID)
Ergotamine
Dihydroergotamine
Sumatriptan
3 - Occipital neuralgia
Large occipital nerve block
A I N E
Muscle relaxants
Carbamazepine
T E N S
Surgery (rarely)
4 - Psychological support
5 - Cognitive rehabilitation (?)

In general, the recovery rate is very high three months after MHI, corresponding to 50% to 70% of the patients. Symptoms may reoccur in some patients, in periods of greater anxiety. One year after the MHI, approximately 90% of the patients are recovered. The minority of patients who still complain of disorders report aggravation of symptoms and sometimes the persistence of only one symptom (headache, neck pain, or dizziness being the most frequently reported ones).[1,2,3]

Conclusion

Mild head injury is a very common condition that accounts for approximately 75% of all head injuries.

Of all patients with MHI, approximately 50% will have post-concussion syndrome: the most frequently reported complaints are headaches, dizziness, fatigue, irritability, insomnia, and loss of memory and concentration. The characteristics of this condition are well defined through anatomopathological, neuroradiological and neurophysiological tests.

In most patients, the manifestations of post-concussion syndrome disappear three months after the MHI. ■

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