Post-concussion syndrome in sport

Síndrome postconmoción en el deporte

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Introduction

Post-concussion syndrome is a common consequence of a traumatic brain injury, and it is defined as a set of symptoms including headaches, dizziness, neuro-psychiatric symptoms and cognitive deterioration[1,2]. Post-concussion syndrome is most frequently described as the scenario of a mild trauma, although it can also occur after a moderate or severe TBI. Similar symptoms are also described after whiplash injuries. The underlying physiopathology is not defined. Test results can be abnormal, or not; when they are present, test anomalies do not consistently follow a defined pattern.

A mild traumatic brain injury comes after a non-penetrating head trauma, and it is usually defined as mild due to a Glasgow Coma Scale (GCS) score of 13 to 15, 30 minutes after the trauma[3]. Concussion is an alteration in the mental state caused by a trauma that might imply loss of consciousness.

Epidemiology

Between 30 and 80 percent of patients with mild to moderate head injuries experience some post-concussion syndrome symptoms. This apparently wide incidence range demonstrates variability among the studied patient population and the criteria used to diagnose post-concussion syndrome, either using individual symptoms or defined clinical criteria. Two clinical criteria are commonly used: the International Disease Classification, 10th edition (ICD-10) and the Diagnostic and Statistics Manual of Mental Disorders, 4th edition (DSM-IV)[4]. They give very different results, even within the same patient population.

Several studies have attempted to associate head injury severity with post-concussion syndrome among patients with a mild traumatic brain injury (TBI) using a variety of measurements including the Glasgow Coma Scale (GCS), how long consciousness was lost or post-traumatic amnesia lasted, and the presence or extension of abnormalities seen in the CT or MRI scan. In general, the severity of the injury does not correlate clearly with the risk of post-concussion syndrome. However, at least one study suggests that prior history of concussion, particularly if recent or multiple, is a risk factor for prolonged symptoms post-concussion[5-9].

Studies of patient cohorts with mild and moderate TBI have consistently found that being female and older are risk factors for PCS. While the nature of the head injury has not been systematically studied as a risk factor, some studies suggest that patients with sports-related concussion have a better natural history than any with mild TBI resulting from a car accident, a fall or an attack. This can reflect different severity for the physical and/or psychosocial impact, and/or a different inclination for PCS. This can also contribute to gender differences, as the relative weighting of accident vs sports injuries as a cause for the TBI may be greater in women than in men.

Physiopathology

There are various theories for the pathogenesis of PCS. Some maintain that the disorder is structural and biochemical and that it is the direct result of the head injury; others state it has a psychogenic origin. It is possible, even probable, that both contribute, and can have a different impact on different symptoms at various points in the syndrome.

Neuro-biological factors

A series of structural and biochemical changes have been documented in animal models of head injuries and in neuropathological studies on humans. One study compared regional brain volumes in magnetic resonance images (MRI)[10-11] in 19 patients one year after a mild

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traumatic brain injury (TBI) with 22 paired control subjects. The patients had measurable global atrophy compared to the controls. Certain areas of regional volume loss (such as the cingulate cortex) were correlated with lower neurocognitive measurements, clinical scores for anxiety and post-concussive symptoms.

Physiological and functional neuroimaging (computerised tomography due to emission of a single photon [SPECT], tomography due to emission of positrons [PET][12-13] and functional magnetic resonance) also documents more extensive areas of abnormality than observed in the computerised tomography (CT scan), supporting the idea that a structural or physiological head injury plays a role in causing PCS. However, many of these neuroimaging findings are not specific to head injuries and can also be observed in patients with migraines or depression. Furthermore, the studies do not consistently show a relationship between the extent of the abnormalities observed in these studies and the degree of deterioration or the severity of the symptoms experienced by the patient. One exception is a study that correlated acute findings in the CT perfusion examination at the time of the TBI with disability at six months. The role of these factors in producing the clinical PCS symptomatology remains unclear.

Psychogenic factors

The psychogenic contribution to PCS is suggested by several empirical and clinical observations. The PCS symptom complex (headaches, dizziness and sleeping issues) is similar to the somatization that is observed in psychiatric disorders such as depression, anxiety and post-traumatic stress disorder (PTSD). Furthermore, anxiety and depression can cause subjective and objective cognitive deficits similar to those observed in PCS, that improve with antidepressant treatment.

Several studies suggest that both psychiatric inclination (low capacity for confrontation, limited social support and negative perceptions) such as psychiatric comorbidity (depression, anxiety and panic, acute stress and PTSD) are more prevalent in patients with PCS compared to the general population controls and/or to patients with head injuries who do not develop persistent PCS.

However, studies on the interaction of depression, anxiety and cognitive performance in other populations with mild TBI are limited. Some researchers do not find a substantial correlation between the level of the depressive symptoms and the cognitive deficits in patients with mild TBI, while others have found a correlation in the response to antidepressant treatment in a subset of patients.

The association between psychiatric disease and PCS has not been established. Limitations in the methodology, including cross-discipline design and the selection bias for patients and control groups make it impossible to draw solid conclusions. Furthermore, this association may have several explanations. Patients with prior psychiatric diseases may be more likely to suffer head injuries as a result of more prevalent alcoholism, motor or physical impediments derived from their illness or medicines, and for other reasons. Alternately, patients with psychiatric diseases may be more likely to develop PCS after a head injury. Finally, the head injury may cause or precipitate the psychiatric disease in susceptible individuals.

Other factors

The lowest rates or even absence of post-concussion brain symptomatology, occasionally reported in some countries and among children, suggest a prominent role of sociocultural factors in the PCS pathogenesis.

The idea that pending compensation claims contribute to the presence and duration of PCS symptomatology go back to the original reports at the end of the 19th century. The studies show a relationship between persistent PCS and potential financial compensation. However, association does not clearly imply causality. Some patients with pending lawsuits improve with or without treatment and PCS occurs in the absence of lawsuits. On the other hand, the fact that patients do not recover after the claims have been resolved does not necessarily invalidate this theory, as a financial agreement can, in fact, reinforce the disease.

Clinical characteristics

The most common complaints in PCS are headaches, dizziness, fatigue, irritability, anxiety, insomnia, loss of concentration and memory, and sensitivity to noise. The relative preponderance of these symptoms varies from one study to another depending on the clinical environment, the time passed since the injury and other variables. For example, among 118 patients who volunteered for a mild traumatic brain injury (TBI) treatment study, one month after the injury, there were reports of headaches in 78%, dizziness in 59%, fatigue in 91%, irritability in 62%, anxiety in 63%, sleep disorders in 70%, memory lapses in 73% and noise sensitivity in 46%. Among the patients sent to a headache clinic, approximately half had cognitive complaints and a quarter had psychological complaints; 17 percent had an isolated headache complaint.

Headaches

Headaches are estimated to occur in 25 to 78 percent of persons after a mild TBI. Paradoxically, the prevalence, duration and severity of the headaches is greater among people with mild head injuries compared to any with more severe trauma[15-16]. A significant number of patients have pre-existing headaches, but studies contradict themselves regarding whether this is a risk factor for post-traumatic headaches.

According to criteria from the International Headache Society (IHS), the headache should start within seven days of the injury. The seven-day

start is arbitrary, particularly because the aetiology of the post-traumatic migraine is not understood. Three months seems a more reasonable latency for the start than seven days.

Most post-traumatic headaches can be classified by the IHS type in a similar way to non-traumatic headaches. Migraines and tension headaches predominate. In most series, tension headaches are the most frequent (75 to 77 percent). Many patients (27 to 75 percent) have more than one type of headache.

Tension headaches can occur daily, either as a constant pain or intermittently for a variable duration. Distribution can be generalised, nuchal-occipital, bifrontal, bitemporal, like a head band or a cap, and they are characteristically described as pressure, oppressive or deaf pain. Excessive use of analgesics complicated 42 percent of post-traumatic headaches in a series.

The migraine headache is typically lateralized, pulsing or stabbing, with associated photophobia and nausea. This occurs with and without a visual aura[17].

Recurring migraine attacks, with and without an aura, can be the result of a mild head injury. The impact can also cause acute migraine episodes, often in adolescents with a family history of migraines. Originally known as a "footballer's migraine" to describe young men who played football and had multiple migraine attacks with aura, triggered only by impacts, similar attacks can be triggered by a mild head injury in any sport.

In the same way, there are many post-traumatic cranial pains, such as post-traumatic temporomandibular pain where patients might complain of pain in their jaw or hemicrania or ipsilateral frontotemporal pain or post-traumatic neural pain. Headache syndromes attributed to the trauma in the case reports include cluster headaches: continuous hemicrania; short unilateral neuralgiform headache attacks with conjunctival injection, tears, sweats and nasal discharge (SUNCT), short unilateral headaches with cranial autonomic symptoms; and paroxysmal hemicrania.

Dizziness

Approximately half the patients report dizziness after a light head injury. While some patients with PCS have unspecific dizziness (vertigo), others report real vertigo that can be due to a benign paroxysmal positional vertigo or a labyrinthine concussion. Several studies suggest that dizziness complaints at the time of the injury, and afterwards, identify patients who run the risk of a lengthy recovery.

Sleep disorders

Sleep disorders are also reported, generally insomnia, in approximately one third of patients in the acute phase after a mild injury and in approximately half of patients in the chronic phase.

The most common manifestations of sleep disorders and wakefulness after a TBI are excessive daytime drowsiness, an increased need to sleep and insomnia. Less commonly, patients experience variations in their circadian rhythms; abnormal movements or behaviour during sleep, such as talking in their sleep, grinding their teeth and representation of dreams; and sleep-disordered breathing.

Psychological and cognitive symptoms

Over 50 percent of patients report personality changes, irritability, anxiety and depression after a mild TBI. They might find that they are intolerant to noise, emotional excitation and large crowds, and more susceptible to the effects of alcohol. Patients also report memory and concentration impairment[18-20]; this can be corroborated by the objective deficits of the neuropsychological tests. In a typical case, these are more prominent immediately after the injury and they are resolved in the following weeks and months.

A significant number of patients (15 to 20 percent) will develop symptoms that meet the criteria of a psychiatric disease. These include acute stress and post-traumatic stress disorders (PTSD) plus anxiety, panic disorder and depression.

Diagnostic tests

Wise use of tests should be individualised for each patient. Patients with persistent complaints of visual or vertigo symptoms should be referred to an ophthalmologist or an ear, nose and throat specialist. A psychiatric assessment must be considered for patients with prominent psychiatric symptoms.

Neuropsychological tests: these tests are not useful in most patients with symptoms following a concussion. However, when they are performed by an expert and experienced psychologist, the neuropsychological evaluation can be useful to assess selected patients with prominent cognitive or psychological complaints, ensuring their mild nature and their limited reach. The monitoring studies for non-selected patients after a mild traumatic brain injury (TBI) demonstrate small measurable deficits in the neuropsychological tests. Cognitive domains that seem particularly vulnerable to the effects of the head injury include attention, working memory, processing speed and reaction time. Deficits are generally light; severe intelligence and memory deficits are not associated with a mild TBI. Abnormalities are most prominent in the first week after the TBI and disappear over time. After three months, mild TBI patients have a similar performance as a group to the control subjects. In one study, approximately 15 percent of patients have persistent cognitive deficits.

Neuroimaging

Many patients assessed for a mild TBI will have had a CT scan or an MRI scan, as part of their acute assessment. Approximately 10 percent of CT scans in mild TBI are abnormal, demonstrating a mild subarachnoid haemorrhage, subdural haemorrhage or contusions. The MRI scan is

more sensitive than the CT scan, demonstrating abnormalities in approximately 30 percent of patients with normal CT scans.

Patients with PCS who have not had an MRI scan and have incapacitating complaints should have an MRI on their brain to exclude a more serious pathology that would identify either a worse prognosis or an alternative cause for their symptoms.

Other advanced neuroimaging techniques, including functional magnetic resonance, magnetic resonance spectroscope and diffusion tensing image (DTI), are being investigated in TBI patient assessment. In one study, the patients with evidence of a traumatic axon injury in DTI were more likely to demonstrate objective evidence of cognitive impairment compared to patients with normal studies. A meta-analysis concluded that although DTI is sensitive to a wide range of group differences in the diffusion metrics, DTI currently lacks the specificity required for significant clinical application in individuals.

Treatment

PCS treatment is individualised according to the patient's particular complaints. Peace and quiet is often the main treatment, as most patients will improve in three months. In the absence of specific treatments to prevent or treat PCS, most doctors take a symptomatic approach[20].

Cognitive or physical rest

Cognitive or physical rest after a concussion has shown no convincing evidence of improvement in terms of faster recovery or in the long-term clinical results. Patients must avoid activities that might lead to a second concussion while they still have symptoms from the initial event. We do not formally recommend any other type of rest period. Patients must limit activities that worsen their symptoms in the first few days after the injury and then gradually return to their previous level of activity according to what they can tolerate.

Headache treatment

Information on the treatment of headache syndromes, specifically in the post-traumatic environment, is limited to series of cases:

- Amitriptyline has been widely used for post-traumatic tension headaches, and for unspecific symptoms such as irritability, dizziness, depression, fatigue and insomnia[21].
- Occipital neuralgia frequently responds to blocking the major occipital nerve with local anaesthetic, and it can also be combined with an injectable corticosteroid.
- Propranolol or Amitriptyline alone or in combination produce a response rate of 70 percent in 21 of a series of 30 patients properly treated with post-traumatic migraines.
- Excessive use of analgesics was a common contributing factor to the post-traumatic headache in 19 to 42 percent of patients. These patients respond to withdrawing analgesics as favourably as patients whose headaches were not post-traumatic.
- Patients with post-traumatic paroxysmal hemicrania and hemicrania continua have responded to treatment with indomethacin.

Donepezil has had positive results in preliminary studies featuring patients with more severe TBI, but it has not been extensively studied in PCS. Six patients with chronic symptoms after a mild head injury reported a subjective cognitive improvement in an open study on Donepezil[22].

In the absence of specific controlled studies for PCS, this data suggests that post-traumatic headaches probably respond to treatments used for migraines and tension headaches in other environments. Doctors caring for these patients state that the recovery delay from post-traumatic headaches might be due to an inappropriately aggressive treatment, excessive use of analgesics or co-morbidity.

Treatment of sleep disorders and wakefulness: there are behavioural and pharmacological treatments for most sleep disorders and wakefulness in patients with traumatic brain injuries (TBI). Treatment varies according to the dominant symptom or the specific sleep disorder, and the relevant comorbidities. Beyond improving symptoms, potential benefits of the successful treatment for sleep disorders and wakefulness in the population with TBI include improving functional results and quality of life.

Psychological and cognitive complaints

Current evidence does not provide information to treat these complaints that are specific to the post-trauma environment.

Use of cognitive rehabilitation for cognitive difficulties after a mild head injury is controversial. Although there was good support for using a systematic review in military/veteran populations, studies are lacking on other populations. Given that cognitive rehabilitation can be expensive, prospective studies are required to demonstrate their efficacy before being able to recommend generalised application. When psychological symptoms are particularly prominent, support psychotherapy and use of antidepressants and tranquillisers can be useful. Once again, there is only limited data to support a specific treatment approach for the PCS environment. In one study, 15 patients with mild TBI who also meet the criteria for major depression were treated with Sertraline for eight weeks, achieving substantial remission of depressive symptoms, and an improvement to cognitive measurements. An open study among 20 patients with depression after a TBI demonstrated symptomatic improvements with treatment using Citalopram and Carbamazepine. Small random trials have found that cognitive-behavioural therapy improved anxiety and/or depression symptoms in patients who had had a mild TBI[23-24].

Random and control simulated hyperbaric oxygen studies on treating persistent PCS have not systematically demonstrated a benefit in the symptoms or in the cognitive tests.

Education

One of the most important roles for the doctor is patient education. Many patients find peace of mind knowing that their symptoms form part of a well-described syndrome.

Early education and support can also affect the PCS progress.

This was illustrated in a monitoring study on 73 patients with a mild TBI. Those who report a belief at the time of the injury that they were likely to have lasting negative effects had a greater chance of lasting symptoms after three months than anyone who did not back this belief.

Most of the studies, although not all, suggest that early intervention with information and peace and quiet can benefit patients with mild TBI by reducing the PCS severity.

Prognosis

Natural history

The symptoms and disability attributed to PCS are greater in the first 7 to 10 days for most patients. After one month, symptoms improve and in many cases are resolved. A greater symptom load in the initial presentation seems to be associated with a greater risk that symptoms will last for over one month. The vast majority of patients have recovered to a large extent after three months[25-26].

A minority (10 to 15%) have symptoms that last for one year or more. Due to biased information, it is possible that this number is inflated, and the general prevalence is much lower[27-28].

Persistent post-concussion syndrome

Patients with incapacitating symptoms lasting for several months or one year can become more disabled than immediately after the injury. Although the whole complex of symptoms persists in most cases, emotional symptoms seem to be particularly prominent. In general, the studies could not define the risk factors for this sub-set; it has not been systematically demonstrated that the premorbid psychosocial factors or psychiatric disease define which patients run the risk of a lengthy issue.

An exhaustive review of the studies that examine the recovery prognosis after a mild traumatic brain injury (TBI) made the following points:

- Medical-legal problems are a consistent, strong risk factor for persistent symptoms and disability after a mild TBI.
- Repeated concussions can lead to more severe, longer cognitive deficits, but the cross-discipline design of the studies excludes causal inference.
- Gender is an inconsistent risk factor for persistent symptoms.
- Patients with a score of 13 on the Glasgow Coma Scale (GCS) have higher disability rates than those with a GCS of 15, although this can be attributed to other injuries. Patients with complicated TBI (intracranial hematoma or depressed skull fracture) can also be at risk of more persistent symptoms.

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