Mild Head Injury in Young Athletes
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Abstract
Mild head injury is of growing interest because of its underdiagnosed prevalence and underestimated clinical importance. Half of the patients in emergency departments report sport-related head injuries or concussions. Knowledge of symptoms and appropriate management can be improved and is of practical interest for healthcare providers as well as coaches, parents and athletes in order to improve patient care and prevent future injuries. An acute concussion presents with a combination of physical, cognitive, and emotional symptoms, which are usually self-limited and resolve within a week. However, significant sequelae from even mild impacts, such as headaches, prolonged cognitive impairment, or even death, are known. A second impact before full recovery from the first may have deleterious consequences and should be avoided by observing strict rules for ‘return to play.’ Recent research suggests that repetitive minor hits may cause delayed brain damage (dementia pugilistica or the ‘punch-drunk syndrome’). A link to neurodegenerative diseases such as dementia, Alzheimer’s disease (AD) and parkinsonism (tauopathies) is described by amyloidβ plaques in those brains. A genetic predisposition (apolipoprotein) is discussed.

Introduction
Mild head injuries in general are a serious healthcare problem contributing to 0.5% of all visits to emergency departments and resulting in 10% of all pediatric admissions. In adolescents mild head injuries are often related to sport. Recent studies have shown that 20–50% of the minor head injuries treated in an emergency department are sport-related. Public knowledge about mild head injuries is astonishingly poor: only less than 10% of the population believe that a head injury could cause a brain injury, more than 25% think it is harmless and never causes long-term problems and up to 40% believe a second blow to the head may help someone remember things they forgot after a first injury. To prevent athletes from incurring subsequent injuries, initial symptoms (Table 1) should be recognized and athletes as well as trainers encouraged to report symptoms.

The lack of a consensus on the definition and terminology of mild head injury, traumatic brain injury (TBI) and concussion contributes to the misunderstanding and underestimation of this disease entity. For this reason, mild head injuries or concussions are often misjudged as minor and not recognized by the children who sustain them, their parents, coaches, or by physicians. Often only concussions with loss of consciousness are regarded as significant. To prevent athletes from incurring subsequent injuries, initial symptoms (Table 1) should be recognized and athletes as well as trainers encouraged to report symptoms.

Table 1: One or more symptoms may suggest a concussion. This may or may not include loss of consciousness (LOC).
logical and functional rather than the structural aspects of the injury and is defined as a complex process affecting the brain, induced by traumatic biochemical forces, or as a brain injury with transient, traumatic disruption of brain activity. A concussion is described as a direct blow to the head, face, or neck, which results in short-lived impairment of the neurological function of the brain.

Pathophysiology

Pathological studies have demonstrated that head injuries—predominantly inflicted by diffuse rotational and, to a lesser extent, linear forces—destroy the axonal and cytoskeletal structures, which causes axonal swelling and bulb formation. Failed axonal transport in the injured axons causes an accumulation of several proteins such as amyloid precursor protein (APP), presenilin-1 (PS-1), and β-APP cleaving enzyme (BACE). Condensation of APP, PS-1 and BACE causes amyloid β (Aβ) formation and accumulation. The damaged neuronal cells then release the accumulated Aβ. The enzyme neprilysin (NEP), that clears Aβ, is also increased and released by the damaged axons. This may mitigate the negative effect of increased Aβ deposition in the brain parenchyma, which sometimes persists for years and results in neurodegeneration and brain atrophy seen many years after brain injury.

Additionally, the destruction of the cytoskeletal structure increases the amount of tau proteins in the brain. These features, Aβ plaques and abnormal tau accumulation, share many characteristics with Alzheimer’s (AD) and Parkinson’s disease (parkinsonism). The deposition of tau proteins in the CSF is also an ominous sign in severe head injury patients.

Recently, another proteinopathy was described in former athletes that is associated with the loss of the first motor neuron and corticospinal tract degeneration: amyotrophic lateral sclerosis (ALS) has been repeatedly observed and confirms reports that Italian soccer players with previous repetitive head injuries have a higher risk for ALS.

Symptoms

Symptoms of mild head injury or concussion are physical, cognitive, emotional, or related to balance, coordination and sleep disturbance (Table 1). Symptoms may or may not involve loss of consciousness as well as subtle neuropsychological, cognitive symptoms and balance disturbances may last up to 30–45 days. Brain electrical activity is abnormal at the time of injury and still at day 8, despite the fact that symptoms had already disappeared by that time. Standard structural neuroimaging is normal.

The subsequent course is unpredictable. In general, children need a longer period for resolution of symptoms than do adults, and most children will recover unremarkably. Risk factors for a slower recovery are loss of consciousness > 1 min, prior injuries, attention deficit hyperactivity syndrome (ADHS), psychiatric disease, depression, and anxiety. In some patients recovery is complicated by the post-concussion syndrome, a mix of physical, emotional, and behavioral problems similar to depression, anxiety and attention deficit disorder. The post-concussion syndrome is seen in more than half of concussion patients after one month, in more than 10% after three months, and still in 2.3% of patients (e.g. headache) after one year. Symptoms are more likely to persist in children older than 6 years, in those with preexisting injuries, ADHS, or psychiatric diseases. Recent studies report that after a mild head injury, 75% of the young patients will show endocrinological dysfunction after six months and almost 30% will suffer from thyroid hypofunction, growth hormone deficiency or premature puberty after one year.

Investigations

On the field, athletes should be managed according to standard emergency principles (ABC rule), giving particular attention to the cervical spine. On the sideline, a neuropsychological assessment must follow either briefly using the Maddocks questions or in more detail with the Sport Concussion Assessment (SCAT2) or other tests. The Maddocks questions pertain only to orientation (time, place, person) and are not a substitute for a proper and more detailed examination in the locker room or the coach’s office. The SCAT2 is a standardized tool that includes symptom evaluation, calculation of the Glasgow Coma Scale score, sideline assessment (Maddocks questions) and importantly—a cognitive, balance and coordination assessment. However, the SCAT2 has never been evaluated in a prospective study. Consequently, no normative data or cut-offs are available for it. Nevertheless, it is a useful, practicable and standardized tool for estimating the risk for athletes following a head injury. Other neuropsychological tests are web-based (imPACT®, HeadMinder®, CogState®) and developed specifically for athletes’ injuries. For all these tests a pre-injury test should ideally be obtained at the beginning of the season to assist in interpreting any post-injury results.

Return to Play Rule

The main message is that any player with a diagnosed concussion should never be allowed to return to play on the same day, no matter how brief the symptoms are or how well the player looks. They should never be left alone in the hours subsequent to sustaining a hit to the head in order to not miss any deterioration, and they are not allowed to drive a vehicle until medically cleared. The return to play protocol is a six step process (Table 3), where each step takes 24 hours. If any symptoms recur, the athlete is put back to the previous level until he/she is finally symptom-free. This protocol is to be applied in all athletes, regardless of whether the concussion is simple or complex, or the athletes are professionals or amateurs. The concept is based on physical, mental and cognitive rest. This means...
no physical (work, sport) or cognitive (school, noise, television, internet or text messaging, bright light, sleep shortage) activities should be undertaken. Light aerobic (stationary biking, jogging to sprinting) is followed by sport-specific exercise, then non-contact, contact training and practice. Any medication prescribed to reduce symptoms must be discontinued before training starts. There is no cure for a concussion, but treatment can help. For prolonged symptoms antidepressants or amantadine (NMDA receptor antagonist) may be an option.

An exception from the rule «no return to play the same day» can be made if a player was consistently free of symptoms for 20 minutes following the concussion, had a negative test and the team physician has seen the «punch-drunk syndrome» or «dementia pugilistica» described. It is characterized by dysarthria, pyramidal and cognitive signs as well as other neuropsychiatric symptoms like long-term cognitive impairment, anxiety or depression. This disorder, currently defined as chronic traumatic encephalopathy (CTE), is a rare event, given the high number of concussions occurring each year. Case studies demonstrate that a significant percentage (15–20%) of former and retired athletes in any contact sport will develop symptoms of CTE, with a higher prevalence in those with three or more concussions in their history and those with an apolipoprotein E4 (ApoE4) polymorphism. These plaques are similar to those found in the early stages of AD; they occur rapidly within a few hours and appear not only in the white, but also in the grey matter of the brain parenchyma.

These recommendations are reflected in various laws enacted in recent years in the United States. In 2007 Texas passed the Will’s Bill, which requires high school coaches, athletics trainers and even players to undergo safety training such as in cardiopulmonary resuscitation. The Zackery Lystad Law (May 2009) urged the development of guidelines and educational materials for athletes, parents and coaches. All athletes suspected of having a concussion must be removed from play and may not return before they have been cleared by a licensed healthcare professional. In Switzerland guidelines for athletes, coaches and parents are published by the Swiss Ice Hockey League, but no obligatory mandatory law has passed the legislature.

**Repetitive Mild Head Injury – Chronic Traumatic Encephalopathy, CTE**

Recently, concerns about repetitive minor traumatic head injuries came to public attention in the media. National Geographic (February 2011 issue) reported the history of a 21-year-old football defensive back, who took 537 (7) hits to the head in games and practice during one season. Several (>20) had an impact equal to that sustained when hitting a windshield in a car crash at approx. 20 mph. After two hits he showed symptoms of a concussion without loss of consciousness.

Repeated concussions have a long-lasting effect on cognitive and motor function, even decades after the last injury. Already in the 1920ies, in boxers (prize fighters) with recurrent head injuries a syndrome called the punch-drunk syndrome or ed-ementia pugilistica was described. It is characterized by dysarthria, pyramidal and cognitive signs as well as other neuropsychiatric symptoms like long-term cognitive impairment, anxiety or depression. This disorder, currently defined as chronic traumatic encephalopathy (CTE), is a rare event, given the high number of concussions occurring each year. Case studies demonstrate that a significant percentage (15–20%) of former and retired athletes in any contact sport will develop symptoms of CTE, with a higher prevalence in those with three or more concussions in their history and those with an apolipoprotein E4 (ApoE4) polymorphism.

Recent research suggests that there is a link between TBI and the development of neurodegenerative diseases like AD, Parkinsonism, or dementia. The more severe and the more often a TBI occurs, the greater the risk for and the earlier the onset of AD is. Striking arguments for this hypothesis are found in autopsies of former athletes, who sustained multiple minor head injuries. Aβ plaques are found even in children who died following a TBI. These plaques are similar to those found in the early stages of AD; they occur rapidly within a few hours and appear not only in the white, but also in the grey matter of the brain parenchyma. It is unclear and the subject of discussion whether all athletes with recurrent injuries have the same risk for developing CTE, or only those with a genetic predisposition. Susceptibility depends on (epi)genetic factors, such as age, sex and ApoE4 and other unknown genetic factors. In the non-sporting population, ApoE4 polymorphism is a risk factor for AD and is associated with a significantly poorer outcome for all degrees of head injury, especially in children aged less than 15 years. ApoE4 is also associated with earlier onset of Aβ deposition and a decreased volume of the entorhinal cortex and hippocampus as compared to those without ApoE4. Consequently, an ApoE4 polymorphism is a highly suspicious risk factor for the development of CTE in athletes with repetitive mild head injuries.

**Second-Impact Syndrome (SIS)**

Sustaining a second head trauma before full recovery from the previous one may bring on the second-impact syndrome. The proposed pathophysiological pathway is not known exactly. It is a rare condition seen only in young athletes (boxers, football, baseball but also soccer players) under the age of 21 years. It is often fatal (1–2 deaths per year in the US) and may occur within up to two weeks after taking the first hit, and can even be triggered by the mildest degree of second head injury. For this reason, all recent guidelines strongly advocate adherence to the return to play rule as outlined above.

**Prevention**

In order to reduce and minimize the risk for mild head injury in athletes, an attempt must be made to improve several aspects: firstly, injuries should be prevented by changing the athletes’ attitude: respect the...
bodies and heads of the players on your and your opponent’s team. Wear a intact and fixed helmet and a mouth guard and play heads-up. Secondly, laws must be passed to ensure mandatory facilities (require emergency and safety training for sports officials in high schools, emergency-trained physicians on the field) and procedures (return to play rule). Thirdly, trainers must be educated to recognize symptoms and adhere to safety guidelines, and the competence of our profession as medical doctors must also be improved. 

**References**


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