Mild traumatic brain injury

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Traumatic brain injury (TBI) is either a temporary disturbance of the brain function or a permanent damage to the structure of the brain, in both cases caused by an external force. The most common cause for TBI in the developed countries is a ground level fall, and a remarkable number of patients are under the influence of alcohol. Possible intracranial damages which require immediate (surgical) treatment are detected or ruled out by computer tomography (CT) scan. Furthermore, it is essential during the first contact with the patient to recognize and record the clinical symptoms and signs as well as the mechanism of the injury. For example, the time of the possible amnesia cannot be determined a week later and the recognition of a possible unconsciousness requires a witness.

Most of the TBIs are mild and a good recovery can be expected in weeks or months. The recovery is more predictable if the information about the injury and symptoms, as well as the good prognosis, are available (1).

The term concussion refers to the mildest form of TBI, mTBI, when there is the immediate, but transient symptoms of a TBI, and thus the symptoms reflect a functional disturbance rather than a structural injury seen on the standard structural neuroimaging studies.

Sports related concussion

Sports related concussion (SRC) may be caused either by a direct blow to the head or elsewhere on the body with an impulsive force transmitted to the head. As in any concussion, it typically results in a rapid onset of short-lived impairment of neurological function that resolves spontaneously. Similarly to "normal" concussion, the prognosis is very good. However, there are some important and special issues related to SRC that need attention. These include the possible harmful effect of repetitive concussions during an athlete’s career, the possible need for rest vs. training during the recovery, and the timing of the return to play. Finally, as after any concussion, there is a question of how best to help the minority of patients who have prolonged symptoms.

Increased risk for dementia?

In 2012 it was reported that former NFL players (n=3439, playing at least five seasons between 1959 – 1988) had an increased risk for neurodegenerative disease (2). By comparing standardized mortality ratios (SMR) between the ex-players and normal population, the researchers concluded that there "should have" been four deaths because of neurodegenerative disease among the ex-players, but there were as many as ten deaths (+6). This suggests that the risk indeed for these players clearly increased. On the other hand, there "should have" been 630 deaths among the ex-players, but there were only 334 deaths, thus "saving" 290 lives (630 – 334 – 6) by playing as professional as compared to the normal population, even taking into account the extra six players died because of neurodegenerative disease. Since the season played started already in 1959, years before the invention of CT scanner, it is likely that not all injuries at the time were concussions, but rather contusions known to have a bigger risk for neurodegenerative disease later in life (3). On the other hand, mTBI does not seem to increase the risk for dementia or cognitive impairment later in life (4).

Chronic traumatic encephalopathy

Especially in the USA the possibility of former collision sports athletes for developing a chronic, progressive neurodegenerative disease, often termed as
chronic traumatic encephalopathy (CTE), has raised tremendous interest. CTE, which some researchers especially before 2015 believed to be a progressive neurodegenerative disease which can be triggered even by one mTBI, is a rare neuropathological, post-mortem diagnosis with no established or validated criteria. Whereas some researchers still believe in CTE as a specific diagnosis, it is now established that the neuropathology of CTE is present in people with other neurodegenerative diseases. Also, there are very few cases of CTE diagnosed despite the huge amount of athletes involved in contact sports. The symptoms claimed to be caused by CTE, such as depression, are common in normal population. Further, there is only minimal scientific evidence to support the progressive nature of the diagnosis (5). Therefore, CTE is probably not the major worry after an SRC. In fact, it may even have negative iatrogenic effects to “diagnose” or warn about a progressive neurodegenerative disease in someone with a psychiatric illness due to entirely other factors.

According to some studies the patients who present at the ER after a minor head trauma have a clearly diminished life expectancy compared to the normal population (6). This, at the first sight, might support a theory of a kind of a progressive disease triggered by a minor head trauma. However, the excess mortality is thought to be caused by the same reason that caused the accident in the first place, alcohol. Obviously, alcohol does not play a common role in SRs, but is not totally non-existent among retired athletes.

Need for “brainrest”?

Clinicians especially in the USA have recommend strict physical and cognitive rest after a concussion. Rest has been described as “the cornerstone of treatment” after a concussion. The theory behind this is that the concussed brain is in a vulnerable state placing it at an increased risk of delayed recovery should it sustain more trauma. Whereas it seems clear that in contact sports the risk for new injury is increased if a player continues playing in an altered state of consciousness, the scientific basis for the need to restore the brains metabolic homeostasis by total rest is not as clear. In fact, the idea of the need for “brainrest” has never been widely accepted in Finland. The need for “brainrest” has lately been widely challenged (7). An active recovery and training after concussion has recently been encouraged, and the benefit of sub-symptom threshold aerobic training program as compared to the treatment as usual has been demonstrated among pediatric athletes despite of temporary (<24h) symptom exacerbations (8).

After SRC – to play or not to play?

The latest consensus statement about concussion in sports was published in 2017 (9). In brief, it recommends that when a player shows any symptoms or signs of an SRC, he should be removed from the field and assessed by a healthcare provider. After the first-aid, an assessment of the concussion should be made using the SCAT5 (or similar) sideline assessment tool which typically takes about 10 minutes. A player with a diagnosed SRC should not be allowed to return to play on the day of the injury. The consensus statement supports the idea that after an SRC the player probably should take it easy and rest according to his feelings for a day or two, but after that he should gradually return to his normal activities (9). It advisable to stay below the cognitive and physical symptom-threshold-level, but the aim is to gradually return to the normal sport. Typically, this takes a week.

Prolonged symptoms – why and what to do?

Roughly, the “normal” recovery time for an athlete after an SRC is by two weeks for an adult and by four weeks for a child. The “prolonged recovery” reflects a longer than expected time period, but is not necessarily linked to the ongoing physiological injury (1, 9). In fact, there is a growing body of evidence showing that the prolonged recovery after a concussion correlates much better with pre-injury personal factors such as resilience, mood or behavioral issues, and post-traumatic stress reaction, rather than the time of amnesia or unconsciousness, or even with a minor finding in a CT such as a small contusion (1, 9). In many studies the strongest predictor of a slower recovery from an SRC has been the severity of the person’s initial symptoms in the first day or a few days after the injury.

Headache is the most common symptom of a brain injury. For that, as for any specific symptom,
a symptomatic treatment is advised. Sedative or “brain injury specific” drugs are not recommended. Some prolonged symptoms may be caused by autonomic nervous malfunction, and physical therapy might help to improve it. This is similar to dizziness caused by benign paroxysmal positional vertigo that may react well to therapeutic exercise. Physical therapy may also help patients with cervical spine or vestibular dysfunction. A collaborative approach including cognitive behavioral therapy is recommended to deal with any persistent mood or behavioral issues. In general, the few patients with prolonged symptoms should be managed in a multidisciplinary collaborative setting by healthcare providers with experience in SRC.

References


