CONCUSSION IN CHILDREN:
DIFFERENT FROM ADULTS

Diagnosis and collaborative care for pediatric concussion in primary care optometry.

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With heavy emphasis on concussion in the media recently—whether in news, sports, or movies—it is not surprising that concussion is on everyone’s mind. A surge of research has come to the surface in recent years looking at the impact of concussion on cognition, behavior, and the visual system. This research shows how much we do not know about concussion and highlights the role that optometrists and ophthalmologists can play in the multidisciplinary management of concussion sequelae in the future.

Although there has been a lot of research on concussion in adults, there have been limited studies in pediatric and adolescent populations. Among the questions remaining to be answered are how concussions are different in the pediatric population and how we need to change the ways that we practice to accommodate this younger population.

There is yet to be a universally accepted definition of what a concussion is, and the criteria for what classifies a diagnosis of concussion is constantly evolving. At present, a concussion is considered to be a type of mild traumatic brain injury (TBI) in which an external biomechanical force to the head, face, or neck causes a complex pathophysiologic injury to the brain. Unlike previous definitions, the current understanding of concussion acknowledges that the patient may or may not have lost consciousness and that standard structural neuroimaging studies such as computed tomography CT scans and low-field magnetic resonance imaging (MRI) are normal, suggesting that concussion causes a functional disturbance rather than a structural injury to the brain.

However, as technology improves with specialized neuroimaging techniques such as functional MRI (fMRI) and high-field MRI with diffusion tensor imaging, emerging research is suggesting that microstructural and axonal injury are evident after concussion, and the definition of concussion may change again.

EPIDEMIOLOGY

According to the US Centers for Disease Control and Prevention (CDC), approximately 173,285 children and adolescents less than 19 years old were treated for nonfatal recreation- and sport-related concussions or TBIs in emergency rooms annually from 2001 to 2009, with an increase from 153,375 in 2001 to 248,418 in 2009.

Although these numbers are staggering and have led to the villainization of contact sports in the media, what is most surprising is that sports and recreation are not the leading causes of TBI. From 2006 to 2010, the leading causes of TBI among children aged 0 to 14 years old were falls (approximately 55%), followed by unintentional blunt trauma and then motor vehicle accidents. Even more alarming, these numbers are reportedly underestimations of the true incidence of concussion and TBI in the population, as these numbers do not include individuals who did not receive medical care, received medical care in an outpatient or office-based clinic, or received care in a federal facility. Not only this, but, with the ever-changing definition of concussion, some individuals who may not have been classified as having a concussion previously would be diagnosed with one based on newer criteria, and vice versa. Furthermore, many adolescents, coaches, and parents may not recognize the symptoms of concussion or may choose to ignore the symptoms to continue playing in a game, leading to further underestimation of concussion’s incidence and prevalence. All of these numbers indicate that concussion is a public health problem and will continue to become an increasingly larger one.

PATHOPHYSIOLOGY

There are numerous models based on animal studies evaluating the pathophysiology of concussion. In general, these models postulate about what primary mechanical injuries and secondary injuries occur based on cellular, inflammatory, neurochemical, cerebral blood flow, and metabolic responses. The primary brain injury causes biomechanical changes to tissue, ranging from focal injury to diffuse axonal injury.

The initial external force of a concussion causes a release of excitatory neurotransmitters, which leads to changes in neuronal cell wall permeability, changing the concentrations of sodium and potassium and thus pH of the cell, leading to
cellular damage. As damaged cells die, they release cytokines that upregulate an inflammatory response, causing secondary injury. It is hypothesized that it is the secondary inflammatory cascade that can lead to a delay of symptoms for 6 to 24 hours after the primary injury.12

Additionally, changes in cerebral blood flow and glucose metabolism after a concussion can lead to further exacerbation of cellular edema and ischemic injury.11,13,14 The reversibility of neuronal injury from concussion is still under investigation, as controversial postmortem studies suggest that there may be chronic traumatic damage to the brain that has been previously undetected, known as chronic traumatic encephalopathy.15

CONCUSSION IN THE PEDIATRIC POPULATION

A concussion is considered a mild TBI because the direct force and trauma to the brain is mild; however, from the patient’s and parent’s perspective, the symptoms are anything but mild. An acute concussion is diagnosed based upon clinical symptoms and physical signs. The symptomatic presentation of a concussion depends upon the part of the brain that is injured and the individual’s complex pathophysiologic response to the injury itself. Thus, postconcussion symptoms vary on a case-by-case basis, making the diagnosis of concussion difficult and dependent upon the physician’s clinical experience.12

Although in most cases a concussion causes a rapid onset of symptoms, some patients may note a delay in their symptom onset from minutes to hours or even days after injury.12,16 Clinical symptoms can vary from physical symptoms such as headache, nausea, and light sensitivity, to cognitive difficulties with attention or emotional issues such as depression and anxiety (Table 1).2,16,17

Studies in the pediatric population have shown that the majority of children present acutely with physical symptoms such as headache, dizziness, and fatigue, and that some of the emotional and cognitive symptoms may present with delayed onset and last longer than the physical symptoms.16

Although in adults the majority of concussion signs and symptoms resolve within 7 to 10 days after injury, children and adolescents tend to have longer recovery times.5,12,16 The mechanism for longer recovery time in children is unknown, but it is considered to be due to differences in response to excitotoxic injury by a developing adolescent brain.18

POSTCONCUSSION SYNDROME

When the symptoms of a concussion persist beyond 3 months after injury, the patient is considered to have post-concussion syndrome (PCS). The pathophysiologic mechanism that causes some patients to develop PCS and others not to is unknown. Studies have shown that the symptoms are unrelated to the severity of the TBI, and ongoing research is exploring what treatment is best to resolve PCS.19

SECOND IMPACT SYNDROME

The diagnosis of concussion in an adolescent, as well as the ability to ascertain when the adolescent has fully recovered from the brain injury, is critically important. There have been a few clinical cases reported of adolescent athletes suffering from a second concussive injury prior to resolution of an initial concussive injury, leading to diffuse cerebral edema, bleeding, increased intracranial pressure, and ultimately death.20–22 This has been termed second impact syndrome and has interestingly caused mortality only in athletes younger than 20 years. It is hypothesized that it is differences in the developmental brain that make it more susceptible to this syndrome, which has a morbidity rate of 100% and mortality rate of 50%.22

RETURN-TO-PLAY AND RETURN-TO-LEARN

Once a concussion is diagnosed, management revolves around minimizing further secondary injury and determining resolution. Given the absence of a single test to diagnose concussion, management of concussion can be just as difficult as its diagnosis, especially when it is based on the subjective symptoms of the patient.

The primary treatment for acute concussion is physical and cognitive rest.12 Due to increased expression of sodium channels on neuronal axons from diffuse axonal injury, the brain is more vulnerable to additional stress and injury,23 so tasks that increase metabolic demands and cerebral blood flow such as prolonged concentration on school work, video games, and physical activity including nonimpact activities such as running and weightlifting, should be avoided.2,12,17

However, how much rest is needed for optimal recovery is unknown.2 Given the high cognitive demands on children in school work, the physician must balance the need for cognitive rest with the impact of cognitive rest and school absence on academic performance, especially in cases of PCS.24

ROLE OF EYE CARE PROVIDERS

Given the complex nature of vision and visual processing and the vast expanse of neurologic networks that these activities require, it is not surprising that vision can be affected after head injury. Most postconcussion checklists used by physicians assess nonspecific visual symptoms or are limited to blurred vision, double vision, and light sensitivity.17

Over the past few years, however, clinical research has shown that visual signs and symptoms after concussion or TBI can be more subtle than these aforementioned symptoms. They can include reading difficulties, eyestrain or eye fatigue, eye focusing problems, eye tracking problems, vision-derived nausea, visual inattention, and difficulties interacting with visually complex situations.15–17 Many of these symptoms correlate with difficulties with accommodation, convergence, and oculomotor tracking.

Not only have these problems been noted with high prevalence in 30% to 42% in adult civilian and military
populations, but they have also been shown to have a high prevalence in the adolescent population. In a study of 100 postconcussion adolescents aged 11 to 17 years, 51% had accommodative disorders, 49% had convergence insufficiency, 29% had saccadic dysfunction, and 46% had more than one of these vision-related diagnoses. Additionally, the presence of vestibulo-ocular dysfunction in acute concuss ... have recommended a return to cognitive activities, prescription of glasses and/or vision therapy may help alleviate symptoms and aid the patient in his or her return to academics.

CONCLUSIONS

The aforementioned guidelines are subjective, based on clinical experience and the literature. The role of vision care in concussion is still being defined by ongoing research. Studies using fMRI have suggested that oculomotor assessment has the potential to provide objective diagnosis of concussion and could be used as a tool to assess for resolution and management of concussion. The timeline of resolution of visual signs and symptoms is yet to be established, as some visual signs and symptoms self-resolve, and some require active vestibulo-ocular vision therapy for complete resolution. Given the need for rest in the acute recovery period, further clinical trials are needed to assess the usefulness and timeliness of vision therapy after concussion.

It is important to recognize that concussion is different in the pediatric and adolescent population than in adults physically, cognitively, and emotionally. Timelines for recovery are different, and underestimation of concussion resolution can lead to catastrophic events in the younger population. Additionally, the demands on adolescents for academic performance add another stressor to the importance of symptomatic recovery. As experts in eye care, we can help.
primary care physicians, pediatricians, and sports-medicine physicians collaboratively manage concussion, especially as vision’s role in concussion becomes more well-defined.