Punch Drunk: Repetitive Concussions in an Adolescent Student-Athlete

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Case

Cody is a 16-year-old male with no previous psychiatric history who presented 7 months after sustaining a concussion with a loss of consciousness lasting 6 min. On his initial presentation, Cody complained of low mood, initial insomnia, poor appetite, difficulty with concentration, and repetitive ego-dystonic intrusive thoughts of suicide. Cody said he began suffering from some of these symptoms 2 weeks after sustaining his concussion and denied any premorbid psychiatric symptoms.

Cody had no recollection of the injury, but his mother reported he sustained his concussion while playing goalie for his elite level hockey team. She reported that another player struck Cody across the forehead with a hockey stick while skating at full-speed directly toward him. The impact of the blow knocked Cody's protective mask off, and he immediately fell to the ground in a prone position, striking his head a second time.

Upon regaining consciousness, Cody reported numbness and being unable to move the left side of his body. By the time he got back to the locker room, he began regaining feeling and control of the left side of his body. He was cleared by the team trainer and went home with an outpatient neurology evaluation scheduled. That night, he had nausea, and the following day, he woke up with a headache.

By the time Cody was seen the following day, his symptoms had subsided. He was advised to begin physical therapy as he continued to have left-sided weakness. Over the next several weeks, Cody regained his strength but began to suffer from intrusive thoughts about suicide. These thoughts increased in intensity, frequency, and duration and were subsequently accompanied by depressed mood, initial insomnia, and poor appetite. Six months after the initial injury, he experienced command auditory hallucinations to kill himself and asked his parents to schedule an appointment with a psychiatrist.

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Clinical Pearl 1
Athletes are ill-equipped to recognize the sequelae of concussions, and they often have strong incentive to actively ignore these symptoms in order to continue to play. As such, it is unsurprising that Cody did not attribute his mood changes, difficulty with concentration, and sleep disturbance to the head injury. It is incumbent on the clinician to elucidate the mechanism of the injury and the ensuing symptom development in order to accurately arrive at the diagnosis and shape the treatment plan.

Developmental History Cody was the result of a full-term uncomplicated pregnancy. His mother was 31 years old when he was conceived. She received prenatal care and had an unremarkable perinatal course. There were no in utero exposures to alcohol, tobacco, illicit drugs, or medications. Cody was born full-term via normal spontaneous vaginal delivery. His birth weight was 7 lbs 9 oz. He met all of his developmental milestones within expected ranges. His elementary school teachers described him as a friendly child who loved to participate in school. His parents said that he was always a gifted athlete, excelling at various sports, planning to play collegiate hockey ever since his father took him to his first game. Cody's father was also a highly successful athlete, recruited at the college level.

Social History Cody lives with his parents and his younger sister. He earns A's and B's and has a close-knit group of friends. He spends his free time playing sports, hanging out with friends, and playing video games.

Family History of Psychiatric Illness There is no family history of medical or psychiatric illness, including dementia or a primary mood or psychotic disorder.

Disease Course Cody and his family were amenable to the use of medication, but Cody was reluctant to engage in individual therapy. He stated that his schedule didn't allow for him to make a weekly commitment, nor did he have anything that he needed to talk about. Cody started citalopram 10 mg daily which was titrated over 4 weeks to 40 mg daily. His school was contacted to coordinate accommodations that would allow him to engage in psychotherapy.

Over the course of 8 weeks, Cody reported an improvement in his mood symptoms and resolution of the hallucinations. He was sleeping very well and was able to complete his final exams for the school year. Over the summer, Cody remained psychiatrically stable, enjoyed family trips, and also visited college campuses. By winter, he had returned to playing hockey at an elite level and was being actively recruited to play at highly competitive college programs.

Cody remained compliant with his treatment and was stable until late spring. At that time, he reported the return of hearing a voice telling him to kill himself. He described this as intrusive and ego-dystonic. His grades declined, and he reported feeling irritable, aggressive, and depressed. He expressed concern as he also began having headaches, short-term memory loss, and difficulty focusing on the hockey puck. He disclosed that during the last hockey season, he suffered five additional concussions without loss of consciousness. On several of these occasions, he had nausea and vomiting, ear ringing, visual changes, and headaches lasting for several days. Additionally, he had significant amnesia for events on those particular days. He didn't tell anyone about his symptoms because the starting goalie position was highly competitive, and he didn't want to jeopardize his position as a starter. He also did not want to negatively impact his college recruitment process.

A family meeting was held with Cody, and, after obtaining a second opinion from a neurologist at a local academic tertiary care center, Cody made the decision to no longer participate in organized sports.

Diagnostic Impression

Given the seemingly abrupt onset of symptoms, lack of premorbid dysfunction, and lack of family history, it seems likely that the head injury that Cody sustained may have contributed to or even have caused his current symptoms. A recent study by Wallace et al. [1] demonstrates that high school athletes are ill-equipped to recognize the sequelae of concussions; so the fact that Cody did not attribute his mood changes, difficulty with concentration, and sleep difficulties to the head injury is unsurprising. It is incumbent on the clinician to elucidate the mechanism of the injury and the ensuing symptom development in order to accurately arrive at the diagnosis and shape the treatment plan.

Background of Concussion

Dementia pugilistica, also known as punch drunk syndrome, was first described in the 1920s after boxers were observed to have memory lapses, changes in speech, unsteady gait, and tremors. The first article, titled "Punch Drunk" by Dr. Harrison Martland, appeared in JAMA in 1928 [2]. Dr. Martland postulated that symptoms manifested years after repeated brain injuries occurred "…in the deeper portions of the cerebrum." It is now speculated by some that dementia pugilistica is a variant of chronic traumatic encephalopathy (CTE), a recently discovered neuropathological condition seen in individuals with history of repeated head injuries. CTE has gained national attention along with the increasing need to understand the sequelae of concussions experienced by players in the National Football League in the USA [3–5].

Concussion is defined as "any transient neurologic dysfunction resulting from a biomechanical force" [6]. Concussion is distinguished from other forms of

traumatic brain injury (TBI) by the fact that there are no structural injuries to the brain visible on neuroradiological imaging, and symptoms tend to completely resolve over time. However, there is increasing evidence that repeated concussions can cause long-term dysfunction, with children and adolescents being particularly vulnerable.

Between 2001 and 2005, there were over 200,000 emergency room visits for concussions, and over 65% of them were for children between 5 and 18 years old [7]. From 2001 to 2009, the number of TBI-related emergency room visits increased by over 60% [8]. Bakhos et al. [9] showed that approximately 40% of emergency room visits for sports-related concussion are younger children (8–13 years old). Younger children appear more susceptible to the diffuse injury, seen in concussions, which may lead to long-term effects on learning and development. Younger children are also more likely to return to play less than 24 h after sustaining concussion [10]. This same group has increased risk for the catastrophic outcome of second-impact syndrome, herniation of the brainstem from a seemingly minor injury that is sustained after a concussion.

With the emphasis on concussions largely coming from investigations into the biomechanics of football, the incidence and treatment of concussion in girls have received markedly less attention. However, girls are more likely than boys to sustain concussions and typically have more severe symptoms. Girls have more sleep disturbances after one concussion, and sleep disturbance is a harbinger for headaches and mood changes [11]. The need for concussion education and surveillance for young female athletes warrants additional resources as girls have poorer outcomes [12].

Relevant Neuroanatomy and Pathophysiology

Given that the symptoms of concussion typically resolve completely, it is reasonable to assume that symptoms of concussion are caused by neuronal dysfunction and not cell death [6]. The biomechanical force of concussion leads to disruption of the cell membranes which allows for an efflux of potassium and an influx of calcium. The membrane alteration described is evidenced by the increase in choline [13]. In an effort to restore membrane potential, voltage-gated ion channels are activated resulting in an increased need for adenosine triphosphate (ATP). This demand coupled with the disruption in cerebral blood flow leads to glycolysis. With increased glycolysis, more lactic acid is produced. The metabolism of lactate is inhibited by the disruption of oxidative metabolism caused by the sequestration of calcium in the mitochondria. The resulting lactic acidosis causes neuronal dysfunction by altered membrane permeability and cerebral edema.

After the initial transient period of glucose hypermetabolism, hypometabolism can persist for weeks [14]. The neuropsychiatric sequelae seen after concussion may result from the sustained decreased metabolism of glucose in the brain. Further, axonal stretching and the resulting microtubule breakdown may impair axonal transport and cause axotomy.

As indicated above, no consistent structural changes result from a single incidence of concussion. Additionally, a causal relationship has not yet been definitively established; however, the potential for developing CTE in an individual with repetitive head trauma must be considered [15]. Unlike concussion, CTE is associated with the following gross anatomical features including cortical thinning [16], frontal and temporal atrophy [17], smaller hippocampal volumes [18], enlarged ventricles [17], and cavum septum pellucidum (CSP) which may occur. Koerte and colleagues [19] showed that retired American professional football players experiencing psychiatric symptoms had a higher rate of CSP and greater ratio of CSP to septum, which is associated with memory and language deficits. Additionally, the histological lesion that is pathognomonic for CTE is an accumulation of p-tau in cortical sulci in an irregular pattern [20]. The presence of TDP-43-positive inclusions and neurofibrillary tangles is also common in the neuropathology of CTE. Importantly, CTE is a histopathological diagnosis, meaning it can only be diagnosed after viewing a sample of brain tissue under the microscope. As such, it is considered to be a postmortem diagnosis and cannot yet be definitively identified in individuals based on symptoms or biomarkers.

Treatment Strategies

The opportunity to treat anyone who has sustained a concussion rests on that individual's ability and willingness to report his or her symptoms. There is robust evidence that sports-related concussions are underreported [21]. In a study done with high school athletes in Michigan, only 21% reported all of their concussions, and over 55% did not report any concussions [1]. Athletes may think that the injury doesn't warrant medical attention, fear losing playing time, not want to be seen as weak by teammates, or want to avoid disapproval from coaches and trainers [22]. Given the increased risk for children and adolescents to suffer from second-impact syndrome, the implications for an athlete continuing to play after sustaining a concussion are grave.

Though the Centers for Disease Control and Prevention (CDC) recommends all athletes receive concussion education prior to playing any sport, Wallace et al. [1] demonstrated that even high school athletes who receive the CDC "Heads Up" educational documents are unaware of the signs and symptoms of concussion, with many believing that loss of consciousness is a required element of the injury. Further, the type of school environment (i.e., urban vs. suburban, access to athletic trainers, etc.) influences the likelihood that an athlete will report a concussion.

Though there is considerable heterogeneity in pre- and post-concussion assessments, they are often used to elucidate the presence of an injury, its severity, and the need for treatment. These assessments include some or all of the following: clinical evaluation, symptom scales, and computerized neurocognitive testing.

The main treatment for concussion is "brain rest," where the athlete curtails physical and cognitive activities (e.g., no school, no homework, no screens, no

physical activity, no social visits, etc.). Lovell et al. [23] showed that hyperactivation of the brain on fMRI was predictive of longer recovery times. While there are no guidelines on the parameters of brain rest, the resumption of activities depends on the resolution of symptoms [24]. Should any symptoms reemerge, the brain rest protocol should be restarted. Clinicians treating adolescents who have suffered a concussion should consider their readiness to resume driving, as reaction time and concentration are commonly affected [25].

While most symptoms of a concussion resolve over the course of a few days, some patients will experience post-concussion syndrome, also known as persistent concussion symptoms. This occurs anywhere from 6% to 59% of the time and is marked by recurrent headaches, neuropsychiatric changes, and behavioral changes. There is no consensus on the parameters of post-concussion syndrome, such as duration and minimum number of symptoms [26].

All 50 states have Return-to-Play laws that mandate the institution of a protocol for assessment of head injuries and an athlete's ability to resume sports after a concussion. In 2010, the National Collegiate Athletic Association (NCAA) adopted a policy to reduce the incidence of concussions by requiring education, surveillance, and a process for medical clearance. The National Federation of State High School Associations also published educational materials for coaches, trainers, parents, and students. Despite these mandates, compliance remains variable, and enforcement is lacking.

Clinical Pearl 2

In a study of nearly 500 student-athletes, 1/3 reported a previously undiagnosed concussion. Athletes reporting previously undiagnosed concussions had a higher mean Post Concussion Symptoms Scale (PCSS) score and were more likely to have lost consciousness with their current injury than athletes without previously undiagnosed concussions [27].

Lessons Learned About Neuropsychiatry

Until recently, concussions were not regarded as causing significant morbidity and even less so as having the potential for mortality. Cody's experience demonstrates that even one concussion can result in a severe constellation of symptoms. Tragic events have illuminated that repeated concussions have cumulative effects that are destructive. Case reports show that high school athletes can have histopathology consistent with CTE [28, 29].

Because CTE can only be definitely diagnosed postmortem, a valid calculation of incidence and prevalence is challenging. Although traditionally thought of as a diagnosis relevant to middle-age football players, there is limited evidence for its incidence in adolescents and young adults. In a recent study on the postmortem brains of four adolescent athletes, ages 17–18, two of whom committed suicide, changes consistent with a diagnosis of CTE were found [29]. This raises the troubling possibility

that severe neuropathological repercussions of repeated TBI may occur in athletes in adolescence and are not only present later in life. Given that by 10 years of age, 16% of children will have sustained a head injury [26], the inclusion of adolescents in research revolving around repeated head trauma and CTE is warranted.

Educational Tools

1. CDC Heads Up educational materials (https://www.cdc.gov/headsup/youthsports/training/index.html)
2. CDC Heads Up Concussion and Helmet Safety App
3. Consensus Statement on Concussion in Sport – the 5th International Conference on Concussion in Sport held in Berlin, October 2016
4. NCAA Sport Science Institute concussion educational resources (http://www.ncaa.org/sport-science-institute/concussion-educational-resources)
5. National Federation of State High Schools concussion course (https://nfhslearn.com/courses/61129/concussion-in-sports)
6. National Operating Committee on Standards for Athletic Equipment (NOCSAE) (http://nocsae.org/)

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