Concussion and Post-traumatic Headache

Concussion

From his days spent playing high school football, this author recalls that "getting your bell rung" was practically a rite of passage. As you stood there dazed on the sideline following a blow to the head, the coach would exhort you to "shake it off", "get back in the game" and "take your hits". To do otherwise was to risk his scorn and perhaps some teasing from your teammates.

Things clearly have changed. Rarely does an eighth grade lacrosse game go by when a player does not lie motionless on the turf after even a minor collision, undergo an immediate "concussion assessment" by someone presumably trained in the evaluation of head injury and then arise, leaving the field to thunderous applause from all the parent spectators watching anxiously from the stands. A discussion involving "concussions" invariably follows.

So what *is* a concussion, anyway? The definition of a concussion is clinical in nature; while acute brain CT or MRI scans occasionally may demonstrate areas of brain contusion (bruising), much more often the scans are normal, and neither a scan nor another other test reliably can confirm the diagnosis. Concussion is a diagnosis made by listening to and observing the patient, and any testing in the acute or subacute period is performed to exclude other, potentially serious complications arising from the trauma (e.g., fracture/dislocation of the neck).

Concussions result from a sudden rotational acceleration of the head, skull and the brain contained within. Neither a direct blow to the head nor any loss of consciousness is required for a concussion to be said to have occurred. The "whiplash" injury to the neck that most commonly results from a motor vehicle accident can cause concussion and post-concussion syndrome (see below) even in the absence of any direct impact to the head, and the minority of adults and only about 5% of children or adolescents with concussion experience loss of consciousness.

The signs and symptoms of a concussion typically begin soon after the injury, and in descending order of frequency they include headache (>90%), dizziness/unsteadiness, confusion and difficulty concentrating, sensitivity to light, nausea and amnesia (<25%).

So why all the fuss? What really brought concussion to the public's attention was the discovery of late-developing dementia in NFL football players with a history of multiple concussions, with the clinical and pathologic identification of "*chronic traumatic encephalopathy*" in these individuals and others who had sustained multiple head injuries (e.g., boxers, amateur and professional soccer players). Dramatically fueling interest in the serious consequences of concussion were scattered reports of "*second impact syndrome*", typically occurring in young males who had a second concussion shortly after the first and suffered clinically serious and even fatal acute swelling of the brain.

The vast majority of children, adolescents and adults who suffer a concussion will experience no serious acute or long-term consequences. On rare occasions a concussion which is accompanied by brain contusion will produce delayed bleeding within the bruised areas of brain, resulting in blood clot formation within the brain and associated neurologic deterioration which occurs within the several days following the head injury. Infrequently, the acute concussive head trauma may lacerate a large vein or artery lying inside the skull and on the surface of the brain, causing the gradual development of a blood clot that compresses the brain from the outside and producing a progressive decline in consciousness; depending on the vessel lacerated, neurologic worsening may occur within hours to days of the trauma. Concern for these "*delayed hematomas*", whether occurring inside the contused brain or on the surface of the brain, is the primary reason family members are advised to confirm at regular intervals that the concussed individual can be easily awakened from sleep.

Post-concussion syndrome

A far more common complication of concussion is post-concussion syndrome. The associated symptoms occur from

injury to the brain, head or neck, typically develop shortly following the inciting trauma and most commonly include headache, "dizziness" (ranging from mild disequilibrium to incapacitating vertigo) psychological disturbance (e.g., irritability, anxiety), fatigue, impaired concentration and memory, disrupted sleep, sensitivity to noise and a variety of visual symptoms which most commonly involve difficulty focusing and sensitivity to motion in the fields of vision.

There is not a clear correlation with the degree of initial head and/or neck injury and the either the likelihood of developing post-concussion syndrome or its duration. Successful management and suppression of post-concussion syndrome symptoms can prove difficult to achieve, but the measures most commonly employed involve pharmacologic treatment for headache (see below), psychological counseling and vestibular rehabilitation (intended to assist with the symptoms of disequilibrium/vertigo and the associated visual sensitivity to motion). At times, formal neuropsychological testing can prove quite helpful in determining the presence, type and degree of any cognitive impairment present as well as determining whether a patient's cognitive symptoms may in fact be psychological in origin.

Post-traumatic Headache

So why does a magazine devoted to migraine include an article on concussion, post-concussion syndrome and post-traumatic headache?

A couple of reasons. For one, there is a fair amount of evidence to suggest that individuals with a pre-existing history of migraine are more likely to develop chronic post-traumatic headache (see below) than are individuals who experience an equivalent amount of head injury but have no established history of migraine. For another, and one that is far more relevant to providers who practice headache medicine, a number of patients presenting to specialized headache clinics do so for management of chronic post-traumatic headache.

The International Classification of Headache Disorders (ICHD), a widely accepted guide for headache diagnosis, is not especially helpful in describing diagnostic criteria for post-traumatic headache or providing suggestions for management of that disorder. About all the ICHD states is that the headache should begin within 7 days of the inciting head or neck trauma, a criterion it freely admits is arbitrary. The ICHD also states that if the headache resolves within 3 months it should be termed acute post-traumatic headache, whereas persistence beyond 3 months changes its designation to chronic post-traumatic headache.

The chronic variant is the type most commonly seen in headache subspecialty clinics, and it may or may not be accompanied by the other clinical manifestations of post-concussive syndrome. The descriptions patients provide of their chronic post-traumatic headache disorder typically falls into 1 of 2 "clinical phenotypes": a headache disorder that resembles chronic tension-type headache versus a headache disorder that resembles chronic migraine. In both instances the chronic post-traumatic headache patient typically reports daily and even constant low intensity head pain, but with the clinical phenotype of chronic migraine that low intensity baseline headache is punctuated by spikes in intensity and associated symptomatology that resemble migraine.

Does this matter? It does if the clinical phenotype predicts response to therapy. While our arsenal of evidence-based therapies for primary/non-traumatic chronic tension-type headache is thin at best, we have on hand a number of therapies that have been demonstrated to be effective in suppressing primary/non-traumatic chronic migraine. Will those therapies be effective in chronic post-traumatic headache with the phenotype of chronic migraine?

> **...symptoms of a concussion typically begin soon after the injury...**

Put simply, there is no clear answer as yet. Preliminary data presented at the American Headache Society's annual scientific meeting in June indicated that serial onabotulinumtoxinA injection therapy, may be significantly more effective in suppressing chronic post-traumatic headache with a chronic migraine clinical phenotype then a chronic tension-type headache phenotype, and in another paper presented at that meeting both onabotulinumtoxinA and erenumab (Aimovig) were more or less equally effective in reducing headache burden significantly in approximately 50% of patients with chronic post-traumatic headache.

Again, immediately striking to any provider who sees a high volume of patients with chronic posttraumatic headache and the clinical phenotype of chronic migraine is the similarity between their headache characteristics and those of patients with primary/non-acquired chronic migraine. If the pathophysiology generating headache in both types of patients is also similar, then therapy is effective for both acute and preventive therapy that is effective in primary chronic migraine theoretically should be effective in acquired/post-traumatic chronic headache as well. At least one large scale, prospective and well-designed clinical trial is under way to help address this issue in regards to preventive therapy with erenumab.

A last word about treatment. Especially for patients with post-traumatic headache who have sustained a flexion/extension ("whiplash") neck injury, occipital nerve blocks may be particularly helpful in suppressing headache and neck pain regardless of the headache's other clinical features.

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