ISSN 2689-8268 | Volume 8

American Journal of Surgery and Clinical Case Reports

Editorial Open Access

Concussion, Persistent Concussion Symptoms (PCS) and 'Pseudo-PCS': A Neurosurgeon's Medicolegal Perspective

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Received: 12 Aug 2024
Accepted: 25 Sep 2024
Published: 30 Sep 2024
J Short Name: AJSCCR

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Keywords:

Biopsychosocial; Concussion; Diffuse axonal injury; Medicolegal; Persistent concussion; Traumatic brain injury

Citation:

Vini G. Khurana. Concussion, Persistent Concussion Symptoms (PCS) and 'Pseudo-PCS': A Neurosurgeon's Medicolegal Perspective.
Am J Surg Clin Case Rep. 2024; 8(2): 1-4

1. Definition and Aetiology

Concussion is essentially a transiently altered level of consciousness of sudden-onset. There is no requirement for an actual loss of consciousness (LOC) to meet the definition of concussion [1,9]. The word itself is derived from Latin *concutere* ('to shake violently'). In the media, and in lay discussions, concussion is often trivialized as a 'head knock' or 'ding'. Clinically, concussion has been equated to 'mild traumatic brain injury' (TBI), typically associated with a presenting Glasgow coma score (GCS) of 13-15, and post-traumatic amnesia (PTA) of less than a day, and either no LOC or LOC less than 30 minutes [3]. It can be associated with amnesia, antegrade and/or retrograde [11]. Most concussive events are without gross radiology. In the Australian Football League (AFL), it is estimated that there is an average of 6-10 concussions per team per season. In the United States National Football League (NFL), it is estimated that there is an average of 1.6 evaluations for concussion per game, a number that is rising. There is currently a 2:1 male:female preponderance. In terms of aetiology, concussion typically results from a direct impact to the head [9]. However, if another part of the body is struck with sufficient impulsive force, that force can also be transmitted (indirectly) to the head, resulting in the same. In adults, falls, motor vehicle accidents (MVA) and professional contact sports feature causally in concussion. In children and adolescents, contact sports and bicycle accidents feature more often. It is currently accepted that rotational or angular (as opposed to linear acceleration-deceleration) forces result in neuronal stretch and shearing [9]. This appears to be associated with changes in rotational velocity of the head from the physical impact, direct or indirect.

2. Pathophysiology and Key Imaging Features

Axonal stretch and shear with microhaemorrhages are frequently associated with concussion at a brain tissue level. However, at an intracellular level, there is a neurometabolic cascade of events underpinning the pathophysiology of this condition [6,9]. Massive ion fluxes occur, with potassium ion net efflux and extracellular accumulation, accompanied by calcium ion net influx and intracellular accumulation. There is initially substantial neuronal depolarization and release of excitatory neurotransmitter molecules. However, with ion pumps in cell membranes being in overdrive, and glucose catabolism initially generating more adenosine triphosphate (ATP), the ion fluxes eventually lead to mitochondrial dysfunction, lactate accumulation, and impairment of ATP production [6,9]. Calpain, a calcium-dependent enzyme, is activated leading to apoptosis/cell death. Axons swell and become disrupted, with local neurotransmission failure. The development of chronic traumatic encephalopathy (CTE; from recurrent concussions in the same individual) and its pathophysiology are discussed elsewhere [9]. The key imaging features of concussion, if present and detected, are punctate microhaemorrhages and foci of demyelination, collectively referred to as diffuse axonal injury (DAI) [9]. The most common DAI locations, from perhaps least to most severe concussions, respectively, are the grey-white junction, corpus callosum and brainstem. It should be noted that computerized tomography (CT) and magnetic resonance imaging (MRI) studies are frequently normal in concussion [9]. Of the various MRI sequences, the susceptibility weighted imaging (SWI) sequence, previously referred to as gradient echo (GRE) sequence or blood oxygenation level dependent (BOLD) imaging,

remains a current benchmark for DAI detection. With TBI greater than a mild degree, other structural abnormalities can be identified on CT and MRI, including contusions, extra axial collections of blood, traumatic subarachnoid haemorrhage, traumatic oedema, brain shift and even herniation (the latter being imminently life-threatening radiological hallmarks). However, again concussion (mild TBI) does not usually involve these more severe imaging associations [9].

3. Symptoms and Signs of Concussion

The most common symptoms are headache and dizziness, while other symptoms include nausea, vomiting, vertigo, imbalance and clouded thinking [9,11]. There may be impairment in vision and/or hearing, tinnitus, photophobia, phonophobia, concentration difficulty, memory impairment, fatigue and a feeling of sadness. Signs of concussion could be the transient LOC itself (if LOC occurred), drowsiness, disorientation/delirium [9,11]. There may be an appearance of being dazed, with slowed verbal and reaction time responses. Amnesia, either antegrade or retrograde, [11] may be found. There may also be impaired balance, coordination, and/or gait. A seizure may occur, as may general behavioral irritability and emotional lability. Signs of cranial and scalp injury or other body trauma can be evident.

4. Persistent Concussion Symptoms (PCS)

The current terminology supported by the international statistical classification of diseases and related health problems (ICD) version 11 (ICD-11) of the World Health Organization (WHO), refers to "mild neurocognitive disorder" [5]. This was previously known as post-concussion syndrome [9,11]. With its original acronym, PCS. Post-concussion syndrome is now obsolete as a term, as mentioned in, for example, the position statement of multiple sports-related medical and paramedical societies of Australia and New Zealand [1], which refers to the new term replacing post-concussion syndrome to be persistent concussion symptoms (same acronym, PCS). The supersedence over previous terminology has been well explained by Clark et al. [5]. Based on their concept of PCS being "an interface disorder of neurology, psychiatry and psychology". That is, reframing the aetiological basis of persistent symptomatology of mild TBI according to a "bio-psycho-socio-ecological" model, superseding what they referred to as the previous "one-dimensional concept" of PCS in older ICD versions, such as ICD-9 [5,9].

4.1. Features of PCS

While most concussion events are clinically mild and self-limiting between a few days and a few weeks, a limited subset (approximately 20%) of concussed individuals will develop PCS [1]. The symptoms of concussion mentioned above may continue for some months, and in a smaller subset (probably less than 5%) for numerous months. A term used as an alternative to persistent concussion symptoms can be "mild neurocognitive disorder" from ICD-11, which usually commences less than one month between the index injury and determination of an ongoing cluster of aforementioned concussion symptoms [5]. From the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5) of the American Psychiatric Association, the equivalent term is "neurocognitive disorders due to TBI" [5]. Resolution of the symptom and sign cluster is typically less than 3 months. It is the author's interpretation that concussion will be self-limiting in most affected individuals, by less than one month, with around 20% progressing to persistent concussion symptoms for up to 3 months, and less than 5% with PCS of numerous months' duration [1,5,9]. However, in the small subset of individuals with prolonged PCS (i.e., numerous months), there should be a plausible explanatory mechanism and severity of the index trauma, consistent symptoms and signs being reported/observed, and substantiating adjunct testing, including, for example, auditory, visual, balance tests, and corroborating neuropsychological assessment data [9].

4.2. Features of Pseudo-PCS

In 2011, Dr Jonathan Silver, Professor of Psychiatry at New York University School of Medicine, published an article reviewing, in essence, non-organic symptom persistence after concussion [13]. He wrote "although most individuals who suffer a mild traumatic brain injury have complete recovery, a number experience persistent symptoms that appear inconsistent with the severity of the injury…ascribed to malingering, exaggeration or poor effort on cognitive testing" [13]. This theme is also well recognised in the compensation-litigation arena, as detailed and referenced elsewhere [2,8,14]. As well explained by Clark et al. [5]. Such symptomatic persistence may be reflective of perceived adversarial employment and insurance interpersonal interactions, the financial implications, and the overall litigation process, if commenced. Aspects of dissatisfaction [4], perceived injustice [10], catastrophizing [10], and non-organic signs [14], described in the spinal literature, are probably applicable to a small PCS subset of patients, but in this particular context 'pseudo-PCS'. It is the author's view that in the pseudo-PCS subset, there will be an unusual persistence of symptoms over many months, and a clinicoradiological mismatch (i.e., examination, mechanism and radiological testing do not support such persistence) [7,8]. Adjunct ophthalmological, balance/vestibular, and hearing testing results, as well as formal neuropsychological evaluation may reveal further discrepancies. It could also be expected that such a presentation may be in the context of the 'compensation-litigation umbrella', and there may be co-diagnoses of anxiety, depression, post-traumatic stress disorder (PTSD) and/or various psychosocial economic stressors [5,12,13], all subject to evaluation by appropriate clinical psychology and psychiatry experts. It is important that, for example, neurosurgeons seeing patients or assessees in a clinical or medicolegal context, respectively, refer or recommend referral of individuals for further evaluation by

neuropsychologists, occupational physician, neurologists, and/or psychiatrists.

5. Brief Case Reports

The following three independent medicolegal examination (IME) cases assessed by the author illustrate salient aspects of concussion and PCS, including 'pseudo-PCS' mentioned above.

5.1. Case 1

A young man working at a mining site reported domestic and work-related stress. While washing a work vehicle with a coworker on a hot day, he experienced probable vasovagal syncope. He struck his head on a cement gutter, lost consciousness for a few minutes, and suffered antegrade amnesia from the time of cleaning and some days into his hospitalisation. He was diagnosed clinicoradiologically with moderate TBI, and his treatment included decompressive craniectomy and intracranial pressure monitoring. He did not develop a classic PCS symptom cluster, but rather a very good recovery by several months following the incident. He reported headache and some cognitive impairment (mild psychomotor slowing), and had some mild upper motor neuron lesion signs. He appeared to be well motivated towards return to suitable, gainful employment when cleared by his neurosurgeon. He was recommended by the author following neurosurgical IME for formal neuropsychology testing and review by an occupational physician, neurologist, and psychiatrist for prognostication, including return to work recommendations.

5.2. Case 2

A female in her 60s, still working in a desk-job in a family business, was assaulted by a neighbor with a mallet. The assault was captured on closed-circuit television. There was no LOC, but there was brief antegrade PTA. A local soft tissue injury to the scalp, from the mallet, was noted, of length less than 2cm, with some edema and skin laceration, but no underlying calvarial fracture. MRI and CT imaging were normal but for the aforementioned focal scalp injury. Neurological examination was normal, but for unilateral upper trigeminal neuropraxia. However, substantial disability and complete work incapacity were self-reported many months post-injury. Other symptoms included (paradoxical) ipsilateral body weakness, in addition to persistent lack of concentration, loss of problem solving ability, panic attacks, and reduced socialization. There was also self-reported "speaking gibberish" and self-declared unemployability. A co-diagnosis by a psychiatrist of PTSD was made from the injury. The author diagnosed what is referred to above as pseudo-PCS.

5.3. Case 3

A middle-aged female working in an administrative job was involved in a MVA 4 years prior to an IME with the author. She ceased working a few weeks after the MVA and never returned to work. During the IME, she was noted to be stuttering, with word-finding difficulties and apparent confusion. She recurrently mentioned her antegrade amnesia. The mechanism of the injury was a T-bone accident, with no LOC, no emergency services attending, and no evidence of external injury. The assessee had emerged from her own vehicle and actually assisted the other (allegedly offending) driver, who had no significant injury either. The assessee presented with a relatively bizarre cluster of longstanding symptoms stated to be "stuttering…memory issues…confused…no longer driving…on and off headaches…dizzy…off balance…can't tell difference between left and right". She was found to be neurologically normal, but for excessive vocalization, collapsing gait, and visible distress. While she was diagnosed earlier with a meningioma, it had been uneventfully resected 2 years prior to the IME, with some mild local gliosis of the anterior medial temporal lobe near where it had arisen. A neuropsychologist opined that she was exhibiting psychological "decompensation" which he found to be associated with "unusual…atypical…enigmatic" cognitive data, and recommended psychiatry input. The author diagnosed initial *bona fide* concussion and PCS, but by numerous months later, 'pseudo-PCS' superseding the aforementioned. The tumor was regarded by the author as a confounding variable, and so too the presence of the compensation-litigation context.

6. Conclusion

Concussion is a clinical state involving sudden and transient altered LOC, but not necessarily actual LOC. The mechanism usually involves rotational or angular forces, directly or indirectly transmitted to the head/brain. The structural hallmark appears to be neuronal shearing with focal microhemorrhages. Imaging is usually normal, but MRI SWI sequence can pick up a DAI-like pattern of injury. Persistence of concussion symptoms and signs, particularly beyond 3 or 4 weeks can morph into a PCS, which in a small minority can persist for numerous months, but needs to be clinically evaluated to verify an organic versus non-organic basis if such latent persistence occurs.

7. Box 1. Take-Home Message on Concussion and PCS:

- Concussion is a sudden, transient altered level of consciousness
- Does not require actual loss of consciousness
- Involves rotational or angular forces (direct or indirect transmission) to head/brain
- Structural hallmark is neuronal shearing with microhemorrhages
- Imaging usually normal but MRI SWI sequence can detect DAI pattern in mild TBI, including concussion
- Usually resolves within days to a few weeks, but can persist as PCS
- Careful multimodal, multidisciplinary approach to determine if PCS persisting for many months is organic (small minority) versus non-organic.

8. Acknowledgements

The subject of this manuscript was delivered by Dr Khurana participating as an invited speaker at the combined Australian Orthopaedic Association (AOA) and Australian Medico-Legal College (AMLC) meeting in Hamilton Island, Queensland on Friday, 13 September 2024. Dr Khurana would like to thank experts at AMLC, AOA and MEDirect for their insightful discussions and support. The author is grateful to Ms Cathy Zhou (Practice Manager, CNS Medicolegal and CNS Neurosurgery) for her invaluable assistance in the preparation of this manuscript. The author has no known conflict of interest to declare.

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