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Postconcussion syndrome (PCS) in the emergency department: predicting and pre-empting persistent symptoms following a mild traumatic brain injury

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ABSTRACT

Head injuries across all age groups represent an extremely common emergency department (ED) presentation. The main focus of initial assessment and management rightly concentrates on the need to exclude significant pathology, that may or may not require neurosurgical intervention. Relatively little focus, however, is given to the potential for development of post-concussion syndrome (PCS), a constellation of symptoms of varying severity, which may bear little correlation to the nature or magnitude of the precipitating insult. This review aims to clarify the aetiology and terminology surrounding PCS and to examine the mechanisms for diagnosing and treating.

INTRODUCTION

Postconcussion syndrome (PCS) is a much maligned complex of symptoms that includes headache, dizziness, nausea and cognitive impairment occurring commonly following a mild traumatic brain injury (mTBI).[1] While the majority of patients experiencing mTBI will have a rapid and complete resolution of any adverse sequelae within days, if not weeks, a significant proportion will suffer a protracted course of symptoms which may result in considerable disability.[2] The symptoms can be quite profound and are associated with significant morbidity from the physical, emotional, occupational and social sequelae of the disorder.[3] Even though its existence is widely acknowledged by the so-called experts,[1 3] the vague nature of its symptoms, its unpredictability, a perceived association with mental health disorders and the pursuit of litigation and difficulties with treatment lead to persistent controversy and debate about its existence. With head injury representing an extremely common emergency department (ED) presentation across all age groups, the potential for large numbers of patients to suffer protracted symptoms in the form of PCS is high, yet ED management strategies still focus on excluding significant structural sequelae, with little or no attempt made to either predict those at risk of PCS or present potential management strategies.

METHODOLOGY

A comprehensive search strategy was developed to include both the peer-reviewed and non-peer-reviewed literature. The databases searched included Medline (1950 to end of November 2007), EMBASE (1974 to end of November 2007) and the Cumulative Index for Nursing and Allied Health Literature (1986 to end of November 2007). The search terms '*mild head injury*', '*minor head injury*' and '*mild traumatic brain injury (mTBI)*' were used in conjunction with '*post-concussion syndrome (PCS)*', '*predictors*', '*concussion*', '*tools*', '*neurocognitive testing*', '*follow-up*' and '*management*'.

The bibliographies of relevant papers were examined and cross-referenced along with the 'related article' feature on PubMed. Key publications such as the *Emergency Medicine Journal* and the *British Journal of Psychiatry* were also searched using the same terms. The review was focussed by excluding papers relating to significant structural brain injury or moderate/severe head injury and the complex management of such.

DEFINITION AND TERMINOLOGY

Much of the confusion relating to head injuries and their sequelae may relate to the variety of different terms used to define different grades of injuries in various different contexts, many of which are used interchangeably. TBI, mTBI, minor head injury and concussion have all been used in similar settings and no single definition is universally accepted.

Similarly, there is no universally accepted definition of PCS, yet the two most often cited diagnostic criteria are those of the diagnostic and statistical manual of mental disorders (DSM-IV) and international statistical classification of diseases (ICD-10).[4 5] The former has a far tighter criteria for definition, requiring objective cognitive impairment, disturbance in social or occupational functioning and persistence for at least 3 months. It is little surprising, therefore, that the rates of PCS after TBI vary considerably depending on the criteria used with DSM-IV criteria being met by 11% and 64% by the ICD criteria in one study.[5] Some of the diagnostic criteria for these classifications are shown in table 1.

mTBI

The term 'traumatic brain injury' was introduced in the USA following the Traumatic Brain Injury Act of 1966, referring to a brain injury resulting from direct or indirect head trauma. Perhaps counterintuitively, mTBI has a fairly concise definition, beyond what one might expect of just a bang on the head. The defining features of mTBI are a 'traumatically induced physiological disruption of brain function',[3] with at least one of the following features:

- any period of loss of consciousness
- any antegrade or retrograde amnesia

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Table 1 Diagnostic criteria for PCS for the ICD-10 and DSM-IV classifications

Symptom	ICD-10	DSM-IV	PTSD
Headache	✓	✓	
Dizziness	✓	✓	
Fatigue	✓	✓	
Irritability	✓	✓	✓
Sleep problems	✓	✓	✓
Concentration problems	✓	–	✓
Memory deficit	✓	–	
Problems tolerating stress/emotion/alcohol	✓	–	✓
Alteration in affect/anxiety/depression	–	✓	✓
Personality alteration	–	✓	✓
Apathy	–	✓	

Diagnostic criteria vary according to which classification is used. The final column shows similarities with the diagnostic criteria for PTSD.
DSM, diagnostic and statistical manual; ICD, international statistical classification of diseases; PCS, postconcussion syndrome; PTSD, post-traumatic stress disorder.

- any alteration of mental state at the time of the incident (feeling dazed, disorientated, confused)
- focal neurological deficits.

Technically, the definition of mTBI does not preclude structural brain abnormalities; however, the clinical sequelae must be transient. Post-traumatic amnesia must not be greater than 24 h, loss of consciousness greater than 30 min or glasgow coma score (GCS) diminished for greater than 30 min.[6] Most recently, TBI has been defined by a working group as '*an alteration in brain function or other evidence of brain pathology, caused by an external force*',[7] with concise definitions of the component parts provided.

Concussion

'Concussion' is derived from the latin '*concutera*', meaning to shake violently. The International Conference in Sport[7] has defined this as a 'complex pathophysiological process affecting the brain due to either direct or indirect injury', clearly recognising that significant blows to the torso with forces transmitted to the head are as likely to result in concussion as are direct injuries. The defining feature is a 'brief' neurological deficit that typically resolves spontaneously.[8] The course, however, may be protracted and PCS may result. Concussion probably represents a subgroup of mTBI. Loss of consciousness is *not* a requirement for the development of concussive symptoms and indeed 75% of concussed sportspersons have no history of loss of consciousness. Characteristically, there will be no abnormality on standard neuroimaging, if performed, emphasising its functional nature, although new, currently experimental modalities such as diffusion tensor imaging may prove useful in the future for detecting the early subtle changes associated with concussion.[9]

Postconcussion syndrome

PCS refers to a complex constellation of symptoms that follow a concussive injury. By amalgamating these criteria, the syndrome can be defined as the presence of three or more of the symptoms in table 1, occurring in the weeks and months following a head injury.[10] Such symptoms may persist up to 6 months following the head injury, though in some cases may continue indefinitely.[11] The differences in symptoms required for diagnosis (table 1) highlight the uncertainty clinicians have regarding this condition and also how differently PCS can manifest itself. Moreover, as many of the symptoms below are subjective and are common to, or exacerbated by, other disorders, there is a significant risk of misdiagnosis.

EPIDEMIOLOGY

Head injury represents one of the commonest presentations to UK EDs,[12] with TBI representing the biggest cause of death and disability in children and working age adults. Across the country there is an annual incidence of over 300/100 000 population,[3 4] representing in excess of 1 million ED attendances per year in the UK. The majority of these fall in the category of 'mild' although some studies suggest that as many as 80% of such patients will experience some symptoms of PCS.[5] A recent Canadian study revealed as many as 63% of patients experiencing postconcussive symptoms at 1 month after minor head injury according to the Rivermead Post Concussion Symptom Questionnaire.[12] Symptoms may be relatively trivial, self-limiting and relieved with conservative measures or severe, persistent, resistant to treatment and associated with significant morbidity. The severity of the symptoms may bear no relation to the nature or severity of the precipitating insult, meaning that relatively minor injuries may be associated with inexplicably incapacitating symptoms. This means that a large number of patients are at risk from the often severe and persistent symptoms that are common to PCS. Of these, approximately 40 per 100 000 (15%) will go on to have symptoms lasting longer than 3 months (the so-called persistent PCS).[2] Head injuries most commonly result from simple falls, sporting injuries, assaults and road accidents[13] and are most commonly seen among male subjects (although PCS predominates in female subjects), with peaks in the very young (below 5), adolescents and young adults. Though UK data are lacking, evidence from the USA suggests that head injury (and subsequent sequelae) costs $48 billion per annum.[14] If translated across to this country, PCS and TBI represent a significant financial burden on the NHS.

AETIOLOGY: PSYCHOLOGICAL OR PHYSIOLOGICAL?

The aetiology of PCS would seem to be multi-factorial and with a combination of both physical and psychogenic factors (physiogenesis vs psychogenesis) being responsible, perhaps in the presence of as yet undescribed predisposing factors.[15] Preinjury stress has been hypothesised as a factor implicated in the long-term maintenance of symptoms.[16] Conclusive evidence for any single cause is lacking. While some studies have shown that psychological factors may be present early, others using specialised imaging modalities such as single-photon emission CT or MRI have shown the presence of persistent organic brain injury in some cases up to 1 year.[17 18]

The theory that PCS results from diffuse axonal injury with micro-trauma to axons, triggering a cascade of reactions at a cellular level, has received much attention. Postmortem studies of patients with TBI have demonstrated pathological changes undetectable with conventional neuroimaging studies, although it is not clear how this relates to PCS.[19] Further to this, the extent of the changes seen does not consistently relate to the extent of the symptoms bringing their clinical relevance into question.

Brain injuries to boxers sustaining repeated head trauma with concussive or subconcussive blows have long been recognised, with its effects being known as dementia pugilistica. More recently, a variant has been described in American footballers, known as chronic traumatic encephalopathy.[20] With its aetiology thought to be similar, resulting from repeated significant blows transmitted to the brain despite seemingly robust protective head gear, its existence has been given significant credence by

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postmortem studies of several high profile American footballers that showed significant and characteristic degenerative change and the accumulation of a micro-tubule associated protein known as 'tau'.[21]

A psychological origin would seem compelling however, and this is supported by the considerable overlap between PCS and anxiety/depression.[22] Up to 46% of PCS sufferers have premorbid depression and levels of daily stress have been shown to be correlated to PCS symptoms in both mildly brain injured subjects and controls.[23] The development of PCS may be due to a combination of factors such as adjustment to the effects of injury, preexisting vulnerabilities and brain dysfunction.[24] There is a widely held view in some circles that some patients with supposed PCS may be embellishing their symptoms for some form of personal gain. In one review of 30 000 court cases, over a third of subjects either feigned or exaggerated their symptoms in those cases which involved mild head trauma.[25] Whether such symptom severity and duration correlate with the stresses of litigation claims remains unclear.

IS PCS AN ACUTE STRESS RESPONSE?

Post-traumatic stress disorder (PTSD) is increasingly recognised as a critical factor in the development of PCS, occurring more commonly in patients after mTBI compared with non-TBI controls (11.8% vs 7.5%)[3 26] but also seemingly contributing to PCS symptomatology, with rates of PCS three times higher for individuals with existing PTSD. It was previously held that TBI and PTSD were incompatible, the former insult perhaps being protective, masking the memories of a 'traumatic' event. However, the consensus view seems to be that this is not the case, with factors such as 'memory islands' or 'confabulated memories' providing a focus for the development for some form of stress response to the injury.[3] Some groups believe that TBI may not in fact be required for the development of PCS and that PTSD may be a key factor in its development, making it more of an acute stress disorder.[3] That many of the symptoms associated with PCS occur in the absence of mTBI has undoubtedly complicated the diagnostic process. Up to 80% of 'healthy, uninjured' people have reported three or more postconcussive symptoms in some studies.[18] In recent years many studies have focused on military personnel returning from combat in an attempt to delineate the relative roles of TBI, PCS and PTSD. Improvements in protective equipment have led to the survival of many personnel with injuries that previously may have been fatal, with almost a quarter having injuries to the head and neck. A recent study of military personnel returning from Iraq and Afghanistan showed that a history of mTBI predicted a range of health problems with 40% of those with loss of consciousness fulfilling the criteria for PTSD.[27] The implications of this are complex as the effects were significantly decreased after PTSD and depression were considered, suggesting that the traumatic event may be the major precipitant of the sequelae rather than the TBI. What is evident is that the interaction is extremely complex and that the precise relationship remains unclear and may remain so because of the inherent difficulties of teasing out physical and psychological precipitants in a scenario where both have a part to play.

From an Emergency Medicine perspective, the fact that PCS is a genuine entity with significant morbidity that requires early recognition and prompt intervention is more important, perhaps, than its aetiology. While our initial attention correctly focuses on the exclusion of significant pathology that may perhaps require prompt neurosurgical intervention, to ignore the potential for the development of a condition with such associated morbidity is akin to ignoring the potential for adverse outcome in a patient with a normal ankle radiograph after injury, but in whom significant ligamentous disruption may cause major limitation of normal function.

PREDICTING PCS: WHO IS AT RISK?

Regardless of the exact mechanism of PCS, it remains a common sequela of a common ED presentation. The problems for emergency physicians who acknowledge its existence are twofold. First, to be able to try and predict which of the many patients who present with head injury are at increased risk of developing PCS, and second, to consider what interventions, if any, are appropriate for patients identified as being at risk. The national institute for health and clinical excellence (NICE) head injury guidelines[28] acknowledge the importance of forewarning patients of the possibility of longer term sequelae and of the existence of appropriate support services. This, however, would usually entail follow-up by a general practitioner who may or may not feel appropriately skilled to manage PCS symptoms, with neuropsychological support services notoriously difficult to access. There is little guidance available on which patients, if any, with mTBI should be followed-up routinely or indeed whether there are any reliable acute-phase predictors which would deem a head-injury patient to be at 'high-risk' of developing PCS in the future. Early recognition or better pre-empting symptoms of PCS permits more rapid intervention and as will be discussed this may facilitate a more favourable outcome.

For many years, much attention has been paid to a whole variety of methods and tools intended to achieve just this, to predict which patients are likely to suffer from PCS symptoms. Strategies have included consideration of premorbid features (pre-existing mental health problems, female sex, mechanism of injury),[22 23 29 30] features at the time of first presentation (reports of significant amnesia, either antegrade or retrograde, dizziness, severe headache, nausea, noise sensitivity)[31] or the use of specialist neuropsychological tests.[32] Different studies have advocated the use of the SAC (standardised assessment of concussion),[33] speed of word retrieval,[34] HADS (hospital anxiety and depression scale),[35] the IES (impact of event scale),[35] the BESS (balance error scoring system)[11] and the PASAT (paced auditory serial addition task)[36] among numerous other acronyms. Some of the risk factors are summarised in table 2. The reason that most emergency physicians will not be familiar with these tests is that there is no compelling evidence to advocate

Table 2 Risk factors for persistent symptoms and/or poorer overall outcomes (adapted from US Department of Defence mTBI guidelines 2009)

Preinjury	Peri-injury	Postinjury
Age (older) Gender (female) Low socio-economic state Less education/lower levels of intelligence Pre-existing neurological conditions Preoccurrence or co-occurrence of mental health disorders (depression, anxiety, traumatic stress or substance use)	Lack of support system Acute symptom presentation (eg, cognitive impairment, headaches, dizziness or nausea in the ED) Context of injury (stress, combat-related, traumatic)	Compensation Litigation (malingering, delayed resolution) Co-occurrence of psychiatric disorders Co-occurrence of chronic pain conditions Lack of support system

ED, emergency department; mTBI, mild traumatic brain injury.

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the use of any single test or feature and that they are time consuming to perform, when there is already insufficient time to carry out our existing tasks.

Sheedy *et al*[31] reported promising results using a combination of a visual analogue scale to assess injury related pain and a simple measure of immediate and delayed recall of word lists as having reasonable sensitivity (80%) and specificity (76%) for predicting PCS. This they reported as being seen in 25.6% of cases compared with 2.2% in controls ($p>0.001$). Work has also been done looking at the use of serum biochemical markers for predicting those at risk of PCS following head injury, notably protein s100,[29 37 38] but perhaps predictably none have proved as yet to be reliable indicators. Local guidelines at the Royal Devon and Exeter hospital mandate follow-up to a head injury clinic for patients in the ED with CT negative head injury, those with prolonged antegrade or retrograde amnesia or those with specific clinician concern. This is a clinic run by a psychologist and an ED consultant using both paper and computer based neuropsychological tests for further assessment and a combination of behaviour modification and positive reassurance therapies.

STANDARDISED ASSESSMENT OF CONCUSSION

The SAC was developed in the sporting arena to assess the severity of cognitive, physical and neurological impairment immediately following a head injury (see online supplementary appendix 1).[39] It is simple, relatively quick to administer and numerous trials have shown its efficacy in identifying impairment after head-injury when compared with premorbid baseline or age-matched controls.[40] It relies on objective markers (neurological and neurocognitive measures) rather than subjective symptoms (headache, dizziness and nausea) whose presence or absence can be fabricated depending on the expectation/desired outcome of the patient (eg, wanting to return home early). A recent trial has also demonstrated the positive predictive value of acute neurocognitive impairment immediately following mTBI in patients who had persistent PCS at 3 months.[41]

BALANCE ERROR SCORING SYSTEM

A second tool, again devised for sporting head injuries, is the BESS (see online supplementary appendix 2). This method works by evaluating postural stability after a head injury to evaluate dizziness and fine motor control.[42] Studies have shown that the BESS score is easy to use and is able to identify poor balance, which generally lasts, if present, up to 72 h after head injury.[43] Whether or not scoring low on the BESS correlates to prolonged PCS at 3 months is unknown to date, but certainly acute-phase dizziness has been shown to make patients at a high-risk of developing PCS and persistent PCS.[44]

In the professional sporting arena, unsurprisingly, progress has been made. Historical guidance has mandated a 3-week stand down period for athletes after sustaining a 'concussion' in order to minimise the negative effects of repeated concussions and also that of the so-called 'second impact syndrome'.[45] The pressures of modern day sports have dictated that systems such as *Cogsport*[46] and *Impact*[47] have been developed that use a computer based series of neurophyschological tests to permit early testing and a more rapid return to play. While such systems are useful for determining which athletes may return to training and playing, they are as yet not helpful in the early diagnosis of PCS, although this may well be a part of the future.

MANAGEMENT

Perhaps one of the reasons that PCS may not be considered an ED problem or given higher regard is the perception that treatments are ineffective, unavailable and that the responsibility should fall with other agencies (usually primary care). As with most conditions that we deal with on a regular basis, primary prevention for PCS is far more effective than secondary intervention of an established or establishing condition.[48] This should start as early as possible and this will invariably be in the ED.[49]

The mainstays of early intervention are education and empowerment in conjunction with appropriate symptomatic relief.[3 6] A number of controlled studies have examined the role of education and reassurance in ameliorating the effects of PCS. Beneficial interventions have included provision of an information booklet detailing the natural history of PCS, possible symptoms and avoidance strategies and arranging a single follow-up session to give further reassurance and consolidate education already provided.[50 51] Studies have shown that early intervention with a single follow-up session of telephone counselling, focusing on symptom management, is beneficial in both adults and children.[52] Education about the effects of concussion shortens overall symptom duration and severity. Forewarning patients of the symptoms that they may expect following a head injury and reassuring them both of their normality, in terms of the absence of significant structural injury, and their prognosis (symptoms *will* improve) is extremely powerful. Removing negative thoughts relating to outcome at an early stage has been shown to be beneficial.[53–56]

Patients should be encouraged to observe good 'sleep hygiene'.[57] Education may be required about the use of stimulants such as caffeine late in the day, or the use of personal electronic equipment such as laptops or computer games, all of which can interfere with normal sleep patterns. A short course of benzodiazepines may be warranted in those where insomnia is present and exacerbating symptoms of PCS.

No compelling case exists for the use of any specific agent in the management of patients with PCS, though various drugs have been trialled, including Sertraline, Propanol, Donepezil (a central anitcholinesterase inhibitor) and Dihydroergotamine. Use of simple analgesia for symptomatic relief, such as Paracetamol or NSAIDS where tolerated, would seem to be beneficial.[23 58 59] The ProTECT study[60] demonstrated that in a well conducted, randomised, placebo controlled trial of 100 patients with moderate to severe brain injury, progesterone was well tolerated, with a trend towards better neurological outcomes and reduced overall mortality. A phase III trial is currently recruiting and while this focuses on gross outcomes in terms of overall function, there is some thought that there may be an impact on more subtle cognitive and neurological dysfunction.

Anderson *et al*[19] outlined the concept of patients using their symptoms as a 'temperature gauge', with symptoms increasing with physical and cognitive exertion and settling with rest. Anecdotally, patients seem comfortable with the analogy of the brain being akin to an injured muscle following injury, helping them to recognise that a short period of early enforced rest is often beneficial. Premature return to exertion, be it physical or cognitive, may lead to a resurgence of acute symptoms, with adverse psychological consequences.

RECOMMENDATIONS AND CONCLUSIONS

PCS is a controversial condition, particularly in its protracted form. The absence of objective neurological findings, the variance in duration, severity and presentation as well as the poor

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understanding of the underlying aetiology have all spawned debate. Despite such speculation, however, the fact remains that the condition can be severe and debilitating, regardless of whether it has an organic or psychogenic derivation.

No individual tool has both the accuracy to reliably predict those at risk and the simplicity to enable its use by busy emergency physicians in the acute setting.

Pre-empting the development of PCS is the key to its management, recognising that all patients who present with head injury are at some risk, with frequency not being proportional to the extent of injury. Head injury advice leaflets that highlight symptoms that patients may experience with some simple mechanisms and strategies for their management may be all that is required for the majority of patients.

There are a number of risk factors for protracted PCS, and these should be taken into account in the ED so that such patients can be referred for early intervention. It may be impractical to review every patient with mTBI, but those at 'high-risk' should be prioritised for optimum management. If potential sufferers were picked up earlier, this would translate to decreased patient morbidity and potential less days off work, less burden on primary care services and decreased benefits claims.

However, risk factors for PCS are often subjective and vague, and more studies are needed to validate objective predictors of PCS (ie, neuropsychiatric testing). PCS definitions, stratification of patients (ie, ED guidelines) and PCS management all need to be standardised so that patients could potentially be prevented from suffering from this often debilitating condition.

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