

Managing post-traumatic headache:

guidance for primary care

INTRODUCTION

Traumatic brain injury (TBI) is an acute pathological event related to penetration, accelerational, decelerational, or rotational forces. In primary care whiplash is very common and management is poor.¹

Estimates of post-TBI headache range from 30–90% but many people will not consult.² Other symptoms include physical, behavioural, emotional, and cognitive difficulties. Headache is the most important as it is common, often complicated by medication overuse headache (MOH), and can respond to treatment. Ninety-five per cent will reflect a recognised primary headache, most commonly migraine. Ninety per cent present within a week, resolving by 3–6 months, but headache can be problematic up to 5 years or longer.³

Previous history of a primary headache, female gender, and the presence of psychiatric disorders are risk factors for headache development. The area is complicated by:

- *Lack of agreement on terminology.* In primary care the terms head injury, concussion, and traumatic brain injury are often used interchangeably; similarly post-concussion syndrome and chronic traumatic encephalopathy are often conflated, although the neuropathology between these entities differ significantly.
- *Limited understanding of underlying mechanisms.* Early dysfunction results from axonal damage with an inflammatory reaction providing defensive responses and return to normality. However, this can persist with a complex cascade of pathophysiological changes that lead to impaired neurotransmission.
- *No prognostic indicators.* Coma and post-traumatic amnesia are best indicators of early structural damage but paradoxically do not necessarily equate to post-traumatic pain syndromes. Later problems occur more commonly with

mild and moderate TBI.

- *Limited evidence base.* Epidemiological studies demonstrate a wide range of estimates and there is no good evidence base to direct treatment. A systematic review found one uncontrolled study showing amitriptyline helpful.⁴
- *There is a significant population prevalence of similar symptoms in the absence of trauma.* Symptoms of TBI occur commonly with a wide range of other problems and a coexisting post-traumatic stress disorder may give similar symptoms.
- *Emotional and psychological responses may play a role,* particularly when litigation is a factor, although the contribution of litigation to symptoms is unclear.

MAKING THE DIAGNOSIS

TBI is taken as an alteration in brain function in close temporal relationship to an external force to head or neck, and is arbitrarily classified as injury with immediate, early, or late effects. Box 1 shows the features of this classification, although invariably there will be a degree of overlap in all these characteristics.

GUIDANCE FOR PRIMARY CARE

The aim of management is to exclude a causal secondary pathology, exclude MOH, and treat to closest phenotype. Secondary headache can be caused by:

- *Dissecting carotid artery.* Dissection of carotid or vertebral artery can be found in 1% of all traumas that are imaged with angiography. The majority occur within 72 hours and 2% of patients can experience pain for months. The pain is ipsilateral to the injury and can be associated with Horner's syndrome because of damage of the sympathetic plexus around the artery.⁵
- *Intracranial haematoma.* Fifteen per cent of patients with TBI demonstrate

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Box 1. Time of onset of symptoms after traumatic brain injury and pathophysiological insights

Traumatic brain injury	Time to onset	Symptom duration	Predominant clinical feature	Pathophysiology
Immediate symptoms (concussion)	Seconds to minutes	Hours to days	Alteration in consciousness	Direct axonal damage
Early symptoms (post-traumatic syndrome)	Hours to days	Weeks to years	Headache as part of post-traumatic syndrome	Inappropriate inflammatory cascade
Late symptoms (chronic traumatic encephalopathy)	Years	Years	Cognitive dysfunction	Neuronal loss, protein deposition, inappropriate inflammatory response

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intracranial abnormalities. However, findings that alter management may be much lower.

- Intracranial hypotension due to dural tear.** This occurs particularly after injuries to the neck. Headache is relieved by lying flat.
- Hypopituitarism.** Hypopituitarism is largely unrecognised and may be as high as 27% in TBI cases because of damage to hypophyseal vessels or direct trauma to the gland. Undiagnosed pituitary adenoma can be a factor. Screening for pituitary dysfunction has been suggested at 3 to 6 months post-injury, when admission time is greater than 48 hours or when symptomatic.⁶ Relevant symptoms include fatigue, low mood, poor motivation, reduced appetite, sexual dysfunction, and oligomenorrhoea/amenorrhoea.

GUIDANCE FOR PRIMARY CARE PRACTITIONERS

At presentation

- Take a headache history including relief lying flat. Coexisting connective tissue disorders may be at higher risk of dissection and dural tear.
- A basic clinical examination includes skull and neck, funduscopy, and blood pressure as a minimum.
- If symptoms suggestive of hypopituitarism or if patient admission >48 hours, check pituitary function.
- Consider imaging if symptoms are deteriorating or if focal neurological signs are present. Discuss the possibility of incidental abnormalities or non-specific findings secondary to trauma, the significance of which may not be known. Angiography will be needed if dissection

is suspected. Have a low threshold for patients with connective tissue disorders.

- Offer a simple explanatory model to the patient, for example, '*Post-traumatic headache often occurs after inflammation to repair brain damage that is initially beneficial but, for reasons that are unclear, develops into a complex chain of unhelpful biochemical reactions.*' Reassure that symptoms will settle in most cases by 6 months. Explore for other symptoms associated with TBI.
- Exclude MOH. This can occur if analgesics or non-steroidal anti-inflammatory drugs (NSAIDs) are consumed on >15 days per month over a 3-month period.
- Offer simple analgesics or NSAIDs, bearing in mind the potential for MOH. Avoid opioids. If further pharmacological treatment is indicated, treat to the nearest phenotype, including triptans if the phenotype is migrainous, and use appropriate preventive treatments when considering other symptoms (for example, mood, sleep issues, cognitive slowing).
- Emphasise the importance of sleep hygiene and reduction or elimination of alcohol and caffeine intake.
- Review at 3 and 6 months if symptoms are present as a minimum requirement. Monitor for depression, MOH, and new symptoms or signs.

At 3 months

- Carry out imaging if symptoms are deteriorating or there are clinical signs.
- Explore other symptoms, particularly sleep and cognitive issues. Professional consensus guidelines are available for their management.⁷

At 6 months

- Undertake pituitary function if not previously done. Carry out imaging if symptoms are not improving. There may be coincidental pathology causing headache. Consider referral to a headache specialist or community-based neuro-rehabilitation as appropriate.

CONCLUSION

Post-traumatic headache is common but poorly managed. Understanding underlying mechanisms may offer future therapeutic possibilities.

A structured approach combined with clear explanation to patients of what is happening may reduce the morbidity associated with this problem.