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Thunderclap Headache: A Review of Different Aetiologies

Rubesh Gooriah and Fayyaz Ahmed*

Department of Neurology, Hull Royal Infirmary, Hull, UK

***Corresponding author:** Fayyaz Ahmed, Department of Neurology, Hull Royal Infirmary, Hull, UK, Tel: 00447973144051; E-mail: fayyaz.ahmed@hey.nhs.uk

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Abstract

Thunderclap headache refers to a severe headache of abrupt onset. It is a neurological emergency with potentially catastrophic consequences and should therefore be recognized and investigated promptly. Several conditions may cause thunderclap headache. Subarachnoid haemorrhage remains the most common condition that needs to be ruled out in the first instance. When no cause is identified, the term primary thunderclap headache is used but this is a diagnosis of exclusion and should only be used after other conditions have been systematically ruled out with clinical evaluation and different imaging modalities. This review will discuss the clinically serious conditions to present as thunderclap headache that should be carefully considered.

Keywords: Thunderclap; Sentinel headache; Subarachnoid haemorrhage; Lumbar puncture; Xanthochromia

Abbreviations:

CAD: Carotid Artery Dissection; Cead: Cervical Artery Dissection; CVST: Cerebral Venous Sinus Thrombosis; PRES: Posterior Reversible Encephalopathy Syndrome; RCVS: Reversible Cerebral Vasoconstriction Syndrome; SAH: Subarachnoid Haemorrhge; SIH: Spontaneous Intracranial Hypotension.

Introduction

First used by Day and Raskin [1], the term thunderclap headache (TCH) refers to a headache that is of sudden-onset (a clap of thunder), severe and usually peaks very rapidly [2]. It is a headache that reaches a severity of 7 or more on an 11 point scale (0 no pain; 1-3 mild pain; 4-6 moderate pain; 7-9 severe pain; 10 worst pain ever) within 1 minute [3] and lasting for more than 5 minutes [4]. It is frequently described as the 'worst ever' headache by patients, although many of those with such a description of their headaches do not have a thunderclap onset. TCH has been reported to have an incidence of around 43 per 100,000 adults per year [5]. Subarachnoid haemorrhage (SAH) is the most commonly identified disorder and together with other conditions such as cervical artery dissection and cerebral venous sinus thrombosis, can all have serious consequences. For this reason, such a presentation should always be looked into diligently. We are now also increasingly aware of a number of other conditions that can present with a TCH [6]. This review will focus on the clinically important disorders that can present with a TCH.

Vascular Causes

Subarachnoid haemorrhage and sentinel headache

SAH accounts for around 3% of all strokes [7]. The incidence of SAH is approximately 9 per 100 000 population-years but this varies widely across geographical areas [8]. Between 12-25% of patients who present to the emergency department with the 'worst ever' headache turn out to have SAH [9,10]. Mechanisms behind the occurrence of the headache have been hypothesised to include stretching of the vessel, irritation of the meninges and increased intracranial pressure [2]. In a prospective study involving 102 patients with acute severe headache of which 42 had aneurysmal SAH and 23 had non-aneurysmal perimesencephalic SAH (NAPSAH), a severe headache peaking within a minute was found in 74% of the patients with aneurysmal SAH and 51% in the NAPSAH group [11]. Transient loss of consciousness was reported in 26% of patients with SAH compared to 4% with NAPSAH [11]. Other symptoms that usually accompany SAH include visual disturbance, seizures, delirium, neck stiffness, photophobia, nausea, vomiting and focal stroke [12]. Patients presenting with these signs and symptoms are diagnosed without much difficulty. However, in another prospective study by Linn et al. [9], 70% of patients with SAH presented with a headache alone. There has been a decrease in case-fatality rates and an improvement in outcome as a result of new diagnostic techniques and treatment options made available over the last three decades [13]. However, mortality remains high following aneurysmal SAH [14].

Patients with SAH frequently describe a similar severe headache days or weeks preceding the bleed, the so-called sentinel headache (SH) [15]. Its true incidence is unknown and is reported to be preceding 10-43% of cases of SAH, although the results are potentially affected by recall bias [16]. The sentinel headache has a sudden onset similar to SAH, is poorly localized, and usually subsides after a couple of days. It can sometimes continue for two weeks or until a major SAH occurs [17]. Gillingham first used the term 'warning leak' suggesting that the headache was caused by a small bleed from an aneurysm [18]. However, not all cases of SH is due to a minor haemorrhage as stretching and dissection of the vessel wall are also thought to be causative [19]. The importance of recognizing a SH is that early detection and treatment of the aneurysm can prevent a second potentially catastrophic bleed. Unfortunately, more often than not, SH is a retrospective diagnosis. In a prospective study involving 422 patients with aneurysmal SAH, SH occurred in 75% of patients within 2 weeks of the SAH [20].

Sign or Symptom	Conditions
Photophobia	SAH, infection
Neck stiffness	Infection, SAH
Neck pain	CeAD
Visual disturbance	PRES
Horner's syndrome	CAD
Papilloedema	Intracranial hypertension
Fever	Infection and sometimes haemorrhage (SAH, CVST, haemorrhagic stroke, pituitary apoplexy)
Hypertension	PRES, RCVS,
Third nerve palsy	Aneurysmal SAH
Postural headache worse on sitting up or standing	SIH

Figure 1: Signs and symptoms that provide clues to a possible cause of TCH.

Reversible cerebral vasoconstriction syndrome

Reversible cerebral vasoconstriction syndrome (RCVS) is a clinico-radiological entity presenting with recurrent TCH attacks, with or without concomitant neurological signs and symptoms, and in the context of reversible vasoconstriction of cerebral arteries [21,22]. It is a collective term used to encompass several vasoconstrictive syndromes with different nomenclatures depending on their cause and presentation [23]. The term RCVS was introduced by Calabrese et al. [24] in 2007 and is now favoured instead of the eponymous 'Call-Fleming syndrome'. The pathophysiology is unknown and the condition could be related to a transient dysfunction of cerebral arterial tone [25]. RCVS affects females predominantly, and they account for around 70% of those affected [26]. The mean age is around 42 years [27,28]. The incidence remains uncertain and it has been reported that 45.2% of patients with sudden headache and 45.8% of those with a TCH have RCVS [21]. Recurrent TCH is the hallmark of RCVS and occur in more than 95% of patients [22] and can be the only symptom in around 75% of patients [27]. Triggers are reported in 79% of patients, and include sexual intercourse, defecation, physical exertion, urination, coughing, sneezing and bathing [27]. Associated risk factors include the puerperium, together with the pregnancy complications of eclampsia and pre-eclampsia, vasoactive medications, including selective serotonin reuptake inhibitors, alpha-sympathomimetic decongestants, and acute migraine medications [22]. Cocaine and cannabis use have also been implicated [29,30]. In a retrospective analysis of 139 cases of RCVS, 55% of patients demonstrated no abnormality on initial head computed tomography (CT) or magnetic resonance imaging (MRI) [28]. Follow-up imaging showed brain lesions in 81% and these included ischaemic stroke, convexity SAH, lobar intracerebral haemorrhage and brain oedema [28]. The diagnosis of RCVS relies on the demonstration of diffuse reversible cerebral vasoconstriction. The gold standard test is catheter angiography. CT and MR angiography have a sensitivity of around 70% [27]. The prognosis is usually good with 90% having favourable clinical outcomes, but 10% have permanent neurological disability [28].

Posterior reversible encephalopathy syndrome

Posterior reversible encephalopathy syndrome (PRES) is a clinico-radiological entity characterised by headaches, confusion, seizures and visual disturbance, in association with oedema affecting predominantly the posterior cerebral white matter. It is associated with hypertension, eclampsia, renal failure, immunosuppressants, cancer chemotherapy, allogeneic bone marrow transplantation, sepsis and some autoimmune diseases [31-35]. The pathophysiology is not fully understood and the current and most popular hypothesis suggests that hypertension leads to failure of autoregulation with subsequent vasogenic cerebral oedema [36]. PRES also often overlaps with RCVS [28,37]. PRES is associated with a sudden increase in blood pressure in most cases [38]. Headache occurs in 20% of hypertensive emergencies but this is usually throbbing and non-distinct [38]. However, PRES can present with a TCH and this is supported by 3 case reports in the setting of hypertensive crisis [38-40]. Imaging, especially MRI, plays an essential role in diagnosing this condition. Diffusion-weighted imaging shows an increase in the diffusion coefficient consistent with vasogenic oedema [41]. The parietal or occipital lobes are affected in 98% of cases [41]. Full recovery, both clinically and radiologically, is usually expected although the brainstem and deep white matter, if involved, are less likely to achieve complete resolution [42].

Cerebral venous sinus thrombosis

Cerebral venous sinus thrombosis causes less than 1% of all strokes [43]. The presentation of CVST is highly variable. It is more common in women and this is due to pregnancy, the puerperium and the use of oral contraceptive pills [44]. The predisposing factors for CVST are similar to those for venous thrombosis in other parts of the body except for a few local causes (e.g. head trauma, sinus infections, and brain tumours) [43]. Headache is the most common symptom. It occurred in around 89% of patients in a prospective observational study involving 624 cases of CVST [45] but it can also be the only symptom throughout the course of the disease [46]. It is more frequently diffuse than localised. Other common signs and symptoms include seizure, paresis, aphasia, mental status disorders, diplopia and papilloedema [45]. The onset of headache is usually subacute over several days [46]. However, a thunderclap onset can be seen in CVST. In a study of 59 patients with CVST who presented with isolated headache, 4 (7%) had a thunderclap headache [46], and this was found to be as high as 17.5% in another study [47]. The diagnosis of CVST is based on neuroimaging. CT venography may show a filling defect while MR venogram shows a lack of flow signal. The cord sign, representing a hyperdense thrombosed cortical vein, and the dense triangle sign (or empty delta sign), suggesting a thrombus in the posterior superior sagittal sinus, can be seen on unenhanced CT scans [43]. Other signs on neuroimaging that indirectly suggest CVST include venous oedema or infarction, subarachnoid or parenchymal haemorrhages [48]. Anticoagulation is the first-line management.

Cervical artery dissection

Carotid and vertebral artery dissections (CAD, VAD) are both rare. CAD has an annual incidence of around 2.5 to 3 per 100,000 [49,50].

VAD is around twice less frequent. Yet, cervical artery dissection is a major cause of stroke in younger patients [51,52]. Risk factors include trauma, chiropractic manipulation, hypertension, smoking, diabetes mellitus, migraine and connective tissue disorders [53,54]. In a study of the headache characteristics of 161 consecutive symptomatic patients with spontaneous cervical artery dissections, headache was reported in 68% of patients with internal CAD as opposed to 33% in those with VAD [55]. It was the presenting symptom in 58.5% of internal CAD [56]. Arnold et al [57] identified 20 of 245 (8%) patients with spontaneous CAD who presented with pain as the only symptom. 20% of them had a thunderclap headache at onset [57]. Thunderclap headache has also been found to be more common in those with VAD than those with CAD [58]. The diagnosis relies on vascular imaging. MRA has the highest sensitivity [59]. Anticoagulation is recommended for extracranial cervical artery dissections although this is not supported by robust evidence [60].

Ischaemic stroke

Headache is a frequently reported symptom of both ischaemic and haemorrhagic strokes. In a large study involving 2196 patients, headache was present in 27% of patients at stroke onset [61]. Vestergaard et al. [62] reported similar findings. Stroke involving the posterior cerebral circulation is more often associated with headaches than in the carotid distribution. Headaches with lacunar infarction are less common [62]. The character of the headache has been described as 'pressing' in 73%, 'throbbing' in 18% and 'stabbing' in 9% of patients with ischaemic strokes. In patients with a history of a primary headache disorder, the headache developing in association with stroke may resemble their usual headache [62]. Although ischaemic stroke is not considered to be a textbook cause for TCH, there are a few case reports of TCH as a presenting feature of ischaemic stroke, occurring with both cerebral and cerebellar infarcts [63-66]. The mechanism of headache in ischaemic stroke is not clearly understood but is probably through activation of the trigeminovascular system [67]. CT and lumbar puncture, which are usually carried out to investigate TCH, may be inadequate in this situation, suggesting the need for MRI in these cases.

Non-vascular Causes

Spontaneous intracranial hypotension

Initially described by Schaltenbrand [68], spontaneous intracranial hypotension (SIH) is an uncommon condition which can cause a new daily persistent headache. It is caused by spontaneous spinal cerebrospinal fluid (CSF) leaks resulting in intracranial hypotension. A history of minor trauma sometimes precedes the onset of SIH but in most cases the exact cause remains unknown, and it is suggested that an underlying structural defect in the spinal meninges might exist [69]. Orthostatic headache is the most common manifestation of SIH, occurring within seconds or minutes after assuming the upright position and relieved by recumbency. A specific definition is however not provided in the 3rd edition of the International Classification of Headache Disorders (ICHD3 beta). The headache due to SIH may be throbbing or non-throbbing, and is localised to the occipital region in the majority of cases [70]. However, SIH can also present with a thunderclap headache [71,72]. In other cases, it can take the form of exertional headaches, non-positional headaches, intermittent headaches and paradoxical orthostatic headaches [73-75]. MRI has radically changed the way SIH is diagnosed. MRI with and without gadolinium is recommended in suspected cases. Intracranial abnormalities include pachymeningeal enhancement, sagging of the brain, subdural fluid collections, decrease in the size of the ventricles, and pituitary enlargement [76]. MRI may also show pachymeningeal enhancement of the spine, engorgement of epidural venous plexus and extradural collections [76]. However, MRI of the spine rarely identifies the level of the leak as the collections tend to extend multiple levels [77]. Conservative management options involve bed rest and fluid rehydration. Epidural blood patching is the next step. The definitive management is with fibrin glue injection or surgical repair [77].

Pituitary apoplexy

Pituitary apoplexy is a rare potentially life-threatening condition caused by pituitary infarction, haemorrhage, and/or necrosis which presents with headache, nausea and vomiting, fever, meningism, visual disturbances and impaired mental status in the context of a pre-existing pituitary mass [78,79]. Several factors increase the risk of apoplexy and these include size of the tumour, cavernous sinus invasion, coughing and sneezing, trauma, pregnancy and the use of exogenous oestrogen therapy [78]. In a retrospective analysis of 45 patients who presented with pituitary apoplexy, headache was the most common symptom affecting 96% of cases, all of which reported a sudden-onset severe headache [80]. There are also case reports of TCH as the presenting feature of pituitary apoplexy but with normal physical examination findings, CT scan and CSF results [81,82]. CT scan may reveal a haemorrhage but is often inadequate with a yield of only 21% and MRI, which detects around 88% of cases, should therefore be performed [83]. Death is the most feared complication, presumably from adrenal insufficiency. Therefore the initial management revolves around corticosteroids and fluids replacement and correction of electrolyte abnormalities. Most cases require prompt surgical intervention [79].

Primary thunderclap headache

Primary thunderclap headache refers to those cases of thunderclap headaches whereby no underlying aetiology has been identified. It is therefore a diagnosis of exclusion. It was previously known as 'benign thunderclap headache'. Diagnostic criteria for primary thunderclap headache includes a severe head pain of abrupt onset, reaching maximum intensity within 1 minute and at least lasting 5 minutes, and for which no intracranial pathology has been identified [4]. However, the evidence for its existence is poor and some advocate the use of the term 'thunderclap headache of undefined origin' [3]. Nevertheless this condition is self-limiting and the prognosis is therefore good.

Other Conditions

Over a hundred conditions are known to cause TCH [6]. These include third ventricle colloid cyst [84], pneumocephalus [85], aqueductal stenosis [86], ruptured arachnoid cyst [87], ruptutred spinal dermal tumour [88], subdural haematoma [89] and cerebellar tonsillar herniation [90].

Approach to thunderclap headache

TCH is a neurological emergency and this cannot be overemphasized. Initial investigation should be with a plain CT scan of the head followed by a lumbar puncture if the former is negative. If both are unrevealing, the setting, symptoms and physical examination findings will usually provide the necessary clues to the diagnosis (e.g. a

post-partum patient with TCH, high blood pressure and visual disturbances probably has PRES) and will also influence the choice of additional investigations. However, the dilemma of whether to investigate further or not arises in cases where TCH occurs with normal CT scan and CSF analysis but in the absence of any further symptoms or examination findings. Savitz et al suggest that supplementary investigations are unnecessary and lack cost-effectiveness given that follow-up studies of at least 1 year did not reveal any patient with SAH or sudden death [91]. It is also likely that the aneurysms identified in such cases are incidental and therefore the risks of invasively investigating these aneurysms with digital subtraction angiography (DSA) outweigh the potential benefits [91]. Moussouttas et al. [92] argue that the accurate diagnosis of several of the conditions that cause thunderclap headache relies on further imaging techniques such as CTA, MRA, CTV and MRV. Indeed, as discussed earlier, there are cases of CVST and CAD presenting with isolated TCH. However, these are very rare. We acknowledge that there are authors who advocate systematic imaging with CTA/MRA if initial CT scan and lumbar puncture are normal [3], especially if the history is typical. However, we do not support the systematic investigation of TCH beyond CT scanning and CSF analysis unless the history or examination supports a specific diagnosis other than SAH. We, nonetheless, cannot stress enough the importance of a thorough history-taking and physical examination in the setting of TCH. It is important to remember that CSF xanthocromia is 100% sensitive up to 2 weeks post-ictus and is therefore still useful to look for in those who present late [93], while the sensitivity of CT imaging decreases to 30% after 2 weeks and almost nil after 3 weeks [94]. In those who present beyond the 3 weeks window, lumbar puncture, CT and MRI are all unhelpful and CT angiography is increasingly used to detect an aneurysm [95].

Conclusion

Thunderclap headache, a sudden-onset severe headache, is a presentation with potentially life-threatening causes. It therefore requires a meticulous assessment and prompt investigation with a CT scan of the brain and, if normal, with lumbar puncture to rule out subarachnoid haemorrhage. The list of causes of TCH is ever-expanding. In those with no significant history and examination finding, and no identifiable cause following a CT scan and CSF analysis, we do not recommend further investigations as the prognosis is benign. However, careful history-taking and examination is required to ensure an important cause for TCH is not missed.

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