Unruptured aneurysm producing thunderclap headache treated with endovascular coil embolization

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Introduction

Thunderclap headache (TCH) is a severe and explosive headache with peak intensity at onset, coming on full force in less than a minute, lasting at least 5 minutes, and often appearing without any trigger. It is described as sudden as a ‘clap of a thunder’. The term “thunderclap headache” was first coined in 1986 by Day and Raskin to describe headache that was the presenting feature of an unruptured cerebral aneurysm.[1,2] Today, TCH describes the abrupt onset headache seen with many other emergency conditions like subarachnoid haemorrhage, cerebral venous sinus thrombosis, cervical artery dissection, acute hypertensive crisis, spontaneous intracranial hypotension, ischaemic stroke, retroclival haematoma, pituitary apoplexy, third ventricle colloid cyst, and intracranial infections.[3]
Primary TCH is diagnosed when no underlying cause is discovered.\[4\]

Subarachnoid haemorrhage (SAH) is one of the most common causes of secondary TCH. Aneurysmal SAH accounts for the majority of cases although other causes should also be considered which include perimesencephalic haemorrhage, arteriovenous malformations, and dural arteriovenous fistula as well as reversible cerebral vasoconstriction syndrome.\[5\]

TCH related to unruptured cerebral aneurysm is not a very common entity. Different retrospective series showed that 9–25% of the patients presenting with TCH were found to have cerebral aneurysms of which most of them were together with subarachnoid haemorrhage (SAH).\[1,4,6\]

Although sudden and severe headache is the most common presentation of an acutely ruptured cerebral aneurysm, it is rarely due to an unruptured cerebral aneurysm in the absence of SAH. This may indicate a high risk of future rupture or growing of the aneurysms.\[1,4,6\] Also some retrospective surveys of patients with subarachnoid haemorrhage suggest that minor episodes with sudden headache (warning leaks) may precede rupture of an aneurysm, and that early recognition and treatment might lead to improved outcome.\[7\]

In different studies the mechanisms underlying TCH due to unruptured aneurysms were diffuse, multifocal and segmental cerebral vasospasm in the absence of haemorrhage, morphologic changes like stretching, expansion, dissection,\[8\] local thrombosis in the wall\[9\] and limited leakage of blood which implies that partial rupture has occurred in to the subarachnoid space.\[6,10\]

**Case Report**

A previously healthy 32-year-old woman developed a sudden, severe headache since one week. She described it as being the worst headache of her life. The attacks were reaching the maximum intensity in a minute and each attack was lasting for 3–4 hours. Associated symptoms included neck pain, nausea and vomiting. The headache attacks repeated during the day. The pain included both sides of the head and neck. She had no history of headache before. Visual Analogue Scale (VAS) was 10. She had been to emergency three times during this period with severe headache but discharged after limited relief. There was no apparent response to parenteral or oral NSAIDs. The cranial tomography was also performed which was reported normal. She could not sleep at nights because of this headache. She realized drop in her left eyelid in her last emergency service application. The patient was alert, oriented and appropriate. There was no nuchal rigidity. Neurologic examination revealed complete oculomotor nerve palsy on the left side (Fig. 1). Computed tomography was interpreted as normal. Brain MRI together intracranial cerebral MRI angiography revealed posterior communicating artery broad neck saccular aneurysm with two associated daughter aneurysms. Balloon assisted endovascular coil embolization of the aneurysm was performed under DSA (Fig. 2a, b). No evidence of diffuse segmental intracerebral arterial vasoconstriction was observed during DSA scan. After endovascular intervention, TCH was resolved sponta-
neously. She was discharged on the third day of the endovascular intervention with oral steroids and 100 mg aspirin. Complete oculomotor nerve palsy recovered with mild ptosis in three months (Fig. 3). At 1 year follow up evaluation she had no subjective symptoms.

Discussion

TCH headache can be a signal of a very serious underlying etiology.[2]

Most of the life threatening secondary TCH conditions may present with a normal brain CT and a normal lomber puncture as in an unruptured intracranial aneurysm.[11]

Any thunderclap headache triggered by activity, including sexual activity or straining to have a bowel movement, coming on after an injury, accompanied by fever or any focal neurological deficits like weakness, loss of vision or sensation, or accompanied by confusion or changes in speech or thinking are particularly suspicious of being life threatening and needs immediate evaluation.[2]

In this presented case, ictal headache associating with acute oculomotor nerve palsy necessitated further diagnostic studies although the brain CT was reported normal. In return, MR angiography showed the posterior communicating artery aneurysm which was associated with a higher risk of subsequent rupture in this case. We cannot perform vessel wall imaging in the 1.5 Tesla MRI which could be an early predictor but ruptured or unruptured intracranial aneurysm should be also considered even if focal signs are absent. In this case oculomotor nerve palsy was not present in the first TCH attacks. It developed on the fifth day of the initial presentation.

In the study of Schwedt et al. 11% of the patients with aneurysmal subarachnoid haemorrhage, reported a distinctive and unusual severe warning or sentinel headache in the days or weeks preceding such a haemorrhage.[4] Also in another study of Gilard et al. thunderclap headache were present in 17.5% in the studied cohort of ruptured intracranial aneurysm in the 3 previous months, but none in the control cohort. This was proposed to be a sign of aneurysm instability.[12]

In a case report, a patient who had severe headaches in the weeks before rupture which seemed to be caused by an unruptured cerebral aneurysm had accompanying diffuse cerebral vasospasm.[10] A similar case was also reported to have symptoms as a result of acute aneurysm expansion with no associated neurological deficit who was treated with craniotomy and clip obliteration of the aneurysm.[6]

In a study of patients who had chronic headaches before the diagnosis of unruptured intracranial aneurysm, 89.8% reported that their headaches recovered after the treatment of intracranial aneurysm either by surgical clipping or coil embolization.[13] This finding is in line with our case who reported marked relief of the TCH attacks after the coil embolization.

This is a case of symptomatic unruptured cerebral aneurysm producing thunderclap headache which resolved after endovascular coil embolisation. Although TCH due to unruptured aneurysm is an uncommon type of headache, it is a neurological emergency which mandates a swift evaluations for early and accurate diagnosis as it may associate with significant neurologic morbidity and mortality. Endovascular intervention avoided a potentially catastrophic subarachnoid haemorrhage in this patient.

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Informed consent was obtained from the patient for the pictures used in this case report.

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References