

## A case of cerebellar infarction presenting as thunderclap headache

Yoon-Sik Jo · Jin-Yong Choi · Sang-Don Han ·  
Yong-Duk Kim · Sang-Jun Na

Received: 10 May 2011 / Accepted: 15 June 2011 / Published online: 1 July 2011  
© Springer-Verlag 2011

**Abstract** Thunderclap headache (TCH) refers to a sudden-onset, severe headache that features in subarachnoid hemorrhage, unruptured intracranial aneurysm, cerebral venous thrombosis, pituitary apoplexy, cervical artery dissection, and hypertensive reversible posterior leukoencephalopathy. TCH is a rare manifestation in cerebral or cerebellar infarctions. Herein, we report on a 60-year-old woman with a thunderclap headache as the first symptom of cerebellar infarction, in the absence of abnormal findings in the brain computed tomography (CT), CT angiography, and lumbar puncture. An urgent brain MRI showed an acute infarction of the right cerebellar hemisphere. The next day, the patient presented with right side ataxia. In emergency cases presenting with thunderclap headache, one should consider an expanded evaluation and/or close observation, with frequent neurological examinations, even though the findings are normal on the initial neurological examination, cerebrospinal fluid analysis, and brain CT.

**Keywords** Headache disorders · Brain infarction · Ataxia · Magnetic resonance imaging

### Introduction

Headaches are a relatively common phenomenon in cerebrovascular disease. About 25% of stroke patients experience headaches and half of such headaches develop before the onset of other stroke manifestations [1]. One such headache is the thunderclap headache (TCH), a sudden, severe headache with a maximum intensity at onset [2]. A TCH might be the first sign of a serious intracranial vascular disorder. TCH can also be a manifestation of primary headache, and it is now also recognized as an idiopathic headache. Headache is not uncommon in ischemic stroke. However, cerebral and cerebellar infarctions rarely present with TCHs. A formula for the diagnostic assessment of TCH should be established. The management of this type of headache is controversial. The established diagnostic assessment of TCH includes a computed tomography (CT) of the head and cerebrospinal fluid (CSF) examination in order to exclude a subarachnoid hemorrhage. In patients whose CT scans and CSF analyses are normal, further testing is rarely considered to be indicated by most clinicians. Herein, we report a case in which TCH was the first symptom of cerebellar infarction diagnosed by diffusion-weighted magnetic resonance imaging.

### Case report

A 60-year-old woman visited the emergency room with a sudden-onset headache. The patient described headache as maximal at, or within 1 min of the onset. The patient said it

---

Y.-S. Jo · J.-Y. Choi · S.-D. Han  
Department of Neurology, School of Medicine,  
Konkuk University, Chungju Hospital, Chungju,  
Republic of Korea

S.-D. Han  
Department of Neurology, College of Medicine,  
Chungbuk National University, Cheongju,  
Republic of Korea

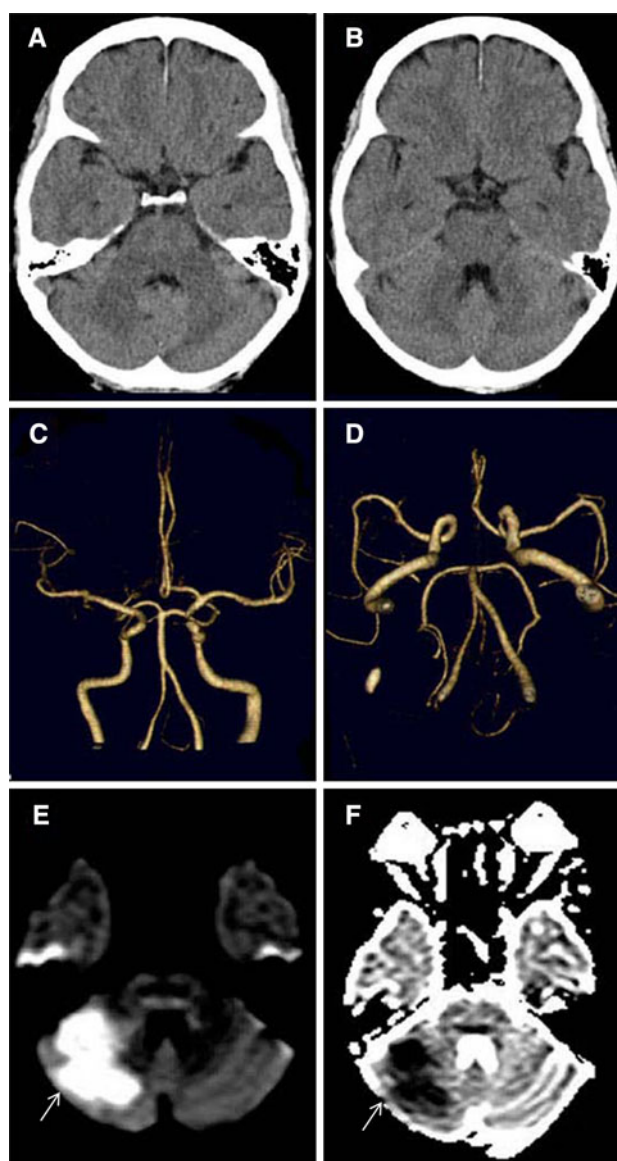
Y.-D. Kim · S.-J. Na (✉)  
Department of Neurology, College of Medicine,  
Konyang University Hospital, 685 Gasuwon-dong,  
Seo-gu, Daejeon 302-718, Republic of Korea  
e-mail: nukedoc@hanmail.net

rapidly generalized from its origin, in the right occipital region, to the patient's entire head, and it occurred while the patient was resting. The patient described the pain as the worst headache the patient had ever experienced. The patient's associated symptoms included nausea, without any phonophobia, and photophobia. The patient's medical history revealed hypertension and diabetes mellitus, both of which were well controlled.

On the physical examination, the patient seemed irritated and distressed, with a temperature of 36.6°C, a regular pulse of 98 beats per minute, a respiratory rate of 17 breaths per minute, and oxygen saturation of 99%. Blood pressure monitoring revealed hypertension with its peak at 190/90 mmHg. A cardiopulmonary examination revealed no jugular venous distension or peripheral edema, patient's breath sounds were equal bilaterally and heart sounds were tachycardic without a murmur. A neurological examination disclosed no focal, long-tract, or lateralizing neurological findings, and there were no specific signs of raised intracranial pressure, such as papilledema, visual obscuration, or diplopia. Patient's isocoric pupils measured 3 mm. Initial laboratory tests, including blood count, electrolytes, C-reactive protein, and D-dimer, were normal. An electrocardiogram showed an atrial fibrillation with tachycardia. A two-dimensional transthoracic echocardiography (TTE) was performed to detect a possible cardiac embolic source as vegetation lodged in the mitral valve.

Upon patient's admission, a brain CT did not reveal any abnormalities (Fig. 1a, b). A lumbar puncture had an opening pressure of 150 mm H<sub>2</sub>O (reference range 70–180 mm H<sub>2</sub>O), with clear, colorless cerebrospinal fluid (CSF). An analysis revealed no erythrocytes or leukocytes, with protein at 44 mg/dL and glucose at 83 mg/dL. A brain computed tomography (CT) angiography showed no aneurysmal malformations (Fig. 1c, d). An urgent, diffusion-weighted MRI revealed an infarction in the right cerebellar hemisphere, and an apparent diffusion coefficient (ADC) map showed low ADC values corresponding to the lesion (Fig. 1e, f, arrows). The headache intensity gradually subsided, and the headache resolved completely within 24 h.

At the second day of admission, the patient presented with gait difficulty. Neurological examination showed that the patient's cranial nerves were intact. Noted symptoms were right-side dysmetria with right-side limb ataxia, and mild, hypotonic right hemiparesis. Sensory examination findings were normal. The findings of additional laboratory tests such as, blood counts, basic chemistries, prothrombin time, partial thrombin time, homocysteine, erythrocyte sedimentation rate, anti-nuclear antibodies, anticardiolipin antibodies, and cardiac enzymes, were normal. The patient received anticoagulant treatment. The patient was then discharged. At the patient's follow-up 2 months later, the



**Fig. 1** Neuroradiological studies of the patient. The brain CT shows no abnormalities (a, b). Brain CT angiography shows no aneurysmal malformations (c, d). A diffusion-weighted image shows a high signal-intensity lesion on the right cerebellum (e, arrow). An ADC map shows low ADC values in the corresponding location (f, arrow)

patient reported no headache. Though the ataxia on the right side gradually improved, slight cerebellar signs persisted.

## Discussion

Day and Raskin initially used the term “thunderclap headache” in referring to the pain associated with an unruptured intracranial aneurysm [2]. The diagnostic criteria of TCH are the following: severe head pain, sudden-onset reaching maximum intensity in less than 1 min (generally 30 s), a duration between 1 h and 10 days, no regular recurrence

over subsequent weeks or months (headache may recur within the first week after onset), no evidence of another disorder (in particular, normal CSF and normal brain imaging are required) [3]. A number of other disorders present with a TCH include subarachnoid hemorrhage, cerebral venous sinus thrombosis, pituitary apoplexy, spontaneous intracranial hypotension, hypertensive encephalopathy, and retroclival hematoma [4]. On the other hand, TCH is a rare manifestation of a cerebral or cerebellar infarction. To our knowledge, TCH caused by cerebellar infarction was reported by Gossrau et al. [5] but our patient differs from the previous case report in the point that this case developed delayed neurologic symptoms after headache onset.

The mechanisms of headache in cerebral or cerebellar infarction are multifactorial. Trigeminovascular system's activation in the intracranial vessels seems to be essential for the production of headache in ischemic stroke [6]. Headaches are more frequent in vertebrobasilar strokes than in carotid territory strokes and are still more frequent in posterior cerebral artery and cerebellar infarctions, but infrequent in subcortical infarctions [2]. A large clinical series study has stressed particularly the frequent association between headaches at onset with cerebellar infarction [7]. The posterior circulation is more densely innervated by the trigeminal system. This may explain the frequency with which headaches occur in posterior circulation infarctions [6]. However, the severity of the headache is not related to the size of the ischemic lesion. The pathophysiology of primary and secondary TCHs is unclear. In our view, it is possible that a secondary TCH may be caused by direct activation of intracranial pain-sensitive structures, especially the cerebral blood vessels. The proximal portions of the intracranial arteries are innervated by neuropeptide Y and noradrenaline-containing sympathetic afferents, which modulate vascular tone [8]. An increased response to endogenous circulating catecholamines or abnormal antidromic discharge of sympathetic afferents (autonomic dysreflexia) could cause acute vasoconstriction or alterations in vascular tone that result in the production of headache [9]. In addition, a pattern of neurogenic inflammation is created during ischemic stroke with release of vasoactive neuropeptides from sensory afferents, inflammatory cytokines, nitrogen oxide from damaged tissue, and bradykinins [10]. This response results in increased nociceptive input into the nervous system and leads to headache. Vasoactive and spasmogenic substances, such as endothelin-1 and 3, norepinephrine, prostaglandin E2, thromboxane A2, leukotriene C4, and neuropeptide Y, may play important roles in the mechanisms of primary and secondary TCHs, including reversible cerebral vasoconstriction [4, 6, 11–13].

In our case, clinical features, radiologic and cardiologic investigations are consistent with an embolic cerebellar infarction presenting with TCH. Based on our case and the previous reports, we suggest that, when a patient presents with a TCH, physicians should consider the possibility of various vascular disorders, particularly cerebral or cerebellar infarction. Recent studies have shown that diffusion-weighted MRI (DWI) can detect and localize acute ischemic stroke with remarkable sensitivity [14]. Shortly after the onset of ischemic stroke, DWI shows infarction as a homogeneous lesion of high-signal intensity resulting from diffusion changes caused by cytotoxic edema [14]. In this case, we report our experience using DWI to evaluate thunderclap headache patients with negative brain CT and lumbar puncture. Such patients require close observation, with frequent neurologic examination and brain diffusion-weighted MR imaging.

## References

1. Dodick DW (2002) Thunderclap headache. *Headache* 42: 309–315
2. Ferro JM, Melo TP, Oliveira V, Salgado AV, Crespo M, Canhao P, Pinto AN (1995) A multivariate study of headache associated with ischemic stroke. *Headache* 35:315–319
3. Casucci G, d'Onofrio F, Torelli P (2004) Rare primary headaches: clinical insights. *Neurol Sci* 25:S77–S83
4. Edvardsson BA, Persson S (2009) Cerebral infarct presenting with thunderclap headache. *J Headache Pain* 10:207–209
5. Gossrau G, Dannenberg C, Reichmann H, Sabatowski R (2008) Thunderclap headache caused by cerebellar infarction. *Schmerz* 22:82–86
6. Valenca MM, Andrade-Valenca LP, Bordini CA, Speciali JG (2008) Thunderclap headache attributed to reversible cerebral vasoconstriction: view and review. *J Headache Pain* 9:277–288
7. Tentschert S, Wimmer R, Greisenegger S, Lang W, Lalouschek W (2005) Headache at stroke onset in 2,196 patients with ischemic stroke: a transient ischemic attack. *Stroke* 32:e1–e3
8. Edvinsson L, Gulbenkian S, Barroso CP, Cunha E, Sa M, Polak JM, Mortensen A, Jorgensen L, Jansen-Olesen I (1998) Innervation of the human middle meningeal artery: immunohistochemistry, ultrastructure, and role of endothelium for vasomotility. *Peptides* 19:1213–1225
9. Schwedt TJ, Matharu MS, Dodick DW (2006) Thunderclap headache. *Lancet Neurol* 5:621–631
10. Dray A (1995) Inflammatory mediators of pain. *Br J Anaesth* 75:125–131
11. Zimmermann M, Seifert V (1998) Endothelin and subarachnoid hemorrhage: an overview. *Neurosurgery* 43:863–876
12. Mulvany MJ, Aalkjaer C (1990) Structure and function of small arteries. *Physiol Rev* 70:921–961
13. Kallela M, Färkkilä M, Saijonmaa O, Fyhrquist F (1998) Endothelin in migraine patients. *Cephalalgia* 18:329–332
14. Chung SP, Ha YR, Kim SW, Yoo IS (2003) Diffusion-weighted MRI of intracerebral hemorrhage clinically undifferentiated from ischemic stroke. *Am J Emerg Med* 21:236–240