# Ischemic Infarction Evoked by Reversible Cerebral Vasoconstriction Syndrome Due to Unruptured Intracranial Aneurysms

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**Abstract**

**Objective:** To identify the underlying secondary causes of reversible cerebral vasoconstriction syndrome (RCVS).

**Methods:** We present the case of a 52-year-old woman who developed thunderclap headache and subsequent watershed infarction resulting from reversible vasospasm triggered by unruptured intracranial aneurysms (UIAs).

**Results:** Spontaneous cerebral vessel spasm in the absence of subarachnoid blood implicates that multiple factors other than blood components may induce reversible vasospasm.

**Conclusion:** It is increasingly evident that UIAs can be direct triggers for RCVS. This case contributes to the body of evidence and implies that the incidence of UIA-induced vasospasm may be greater than currently appreciated.

**Keywords:** Reversible cerebral vasoconstriction syndrome; unruptured intracranial aneurysm; watershed infarction

Reversible cerebral vasoconstriction syndrome (RCVS) is a complex neurovascular disorder characterized by severe thunderclap headaches and reversible cerebral vasoconstriction. Its clinical presentation can mimic that of an aneurysmal subarachnoid hemorrhage (SAH), which is complicated by cerebral vasospasm in approximately one-third of patients. Notably, vasospasm occurs sporadically after surgical clipping of unruptured intracranial aneurysms (UIAs)[1], and the development of subsequent delayed ischemic stroke is a scarcely documented complication[2,3]. In addition, RCVS directly attributable to UIAs has been rarely reported.

## Case report

A 52-year-old woman was transferred to our hospital for further management of weakness of the left leg following thunderclap onset of headache. The headache peaked in intensity within 2 minutes and then gradually subsided. The limb weakness partially improved after 2 hours, leaving a residual weakness that persisted at rest. The patient had an unremarkable past medical history and denied any substance abuse or medication use. She had no risk factors for cerebrovascular disease and denied any history of headache. On admission physical examination, neurological deficits were presented as a left facial droop with some loss of the nasolabial fold and slightly left lower extremity weakness. The National Institutes of Health Stroke Scale score was 2, indicating minor neurological deficits. SAH was highly suspected but excluded based on a head computed

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tomography (CT) scan (Figure 1) and subsequent lumbar puncture.

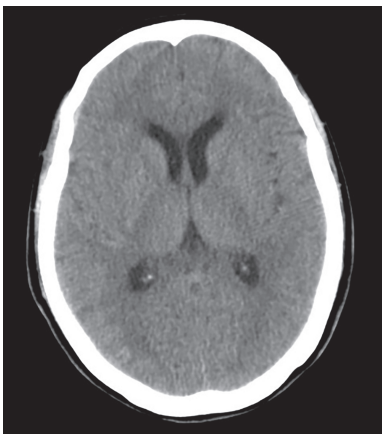

**Figure 1:** The head CT scan showing no evidence of subarachnoid hemorrhage.

Cerebral magnetic resonance imaging (MRI) evealed an internal watershed infarction in the right hemisphere, localized to the border zone between the superficial and deep branches of the middle cerebral artery (MCA) (Figure 2(a) and 2(b)). Corresponding magnetic resonance angiography (MRA) demonstrated severe segmental narrowing of the right M1 segment of MCA, consistent with the underlying hemodynamic abnormalities of the infarction. Somewhat unexpectedly, MRA also demonstrated two intracranial aneurysms located at the supraclinoid portion and terminal segment of the right internal carotid artery (ICA), respectively (Figure 2). Cerebral CT perfusion demonstrated increased cerebral blood volume (CBV), prolonged mean transit time (MTT) and time to peak (TTP) while constant cerebral blood flow (CBF) in the right MCA territory, suggesting a stenosis in the MCA (Figure 3).

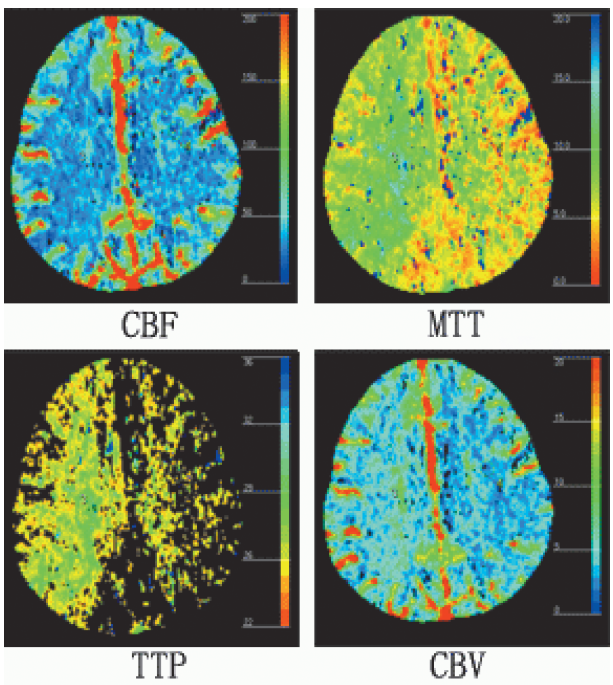


**Figure 3:** Right middle cerebral artery stenosis-related brain CT perfusion alteration.

Subsequent cerebral angiography (digital subtraction angiography, DSA) confirmed multiple aneurysms along the right ICA while normal lumen diameter of MCA, indicating the narrowing was reversible vasoconstriction

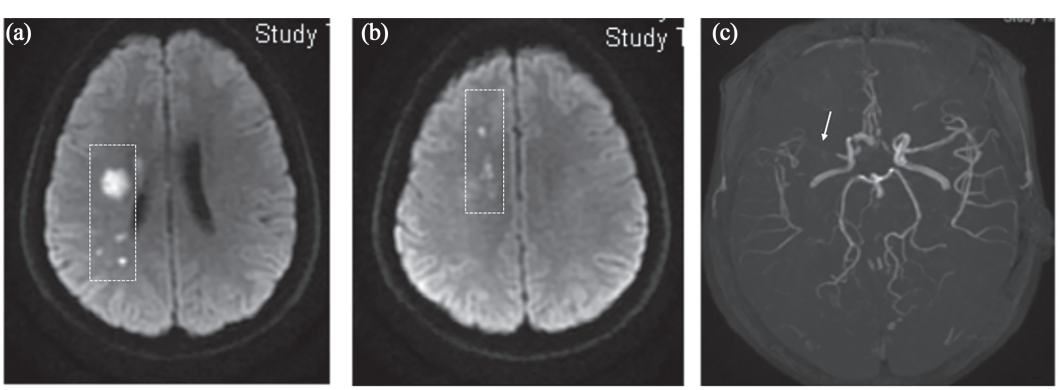


**Figure 2:** Abnormalities detected on cerebral MRI scanning ((a) and (b) Diffusion-weighted MR imaging showed acute infarcts (rectangular box) in the right internal watershed territories; (c) 3D-TOF MRA demonstrated severe stenosis (arrow) in the M1 segment of the right middle cerebral artery).

(Figure 4). Therefore, the acute infarction was attributed to spontaneous cerebral vasospasm, rather than to mass effect from an enlarging aneurysm or an existing atherosclerotic plaque in the MCA.

## Discussion

Recurrent thunderclap headaches, seizures, strokes, and non-aneurysmal subarachnoid hemorrhage can all reveal RCVS. RCVS was overlapped with primary thunderclap headache, posterior reversible encephalopathy syndrome, Takotsubo cardiomyopathy, transient global amnesia, and other conditions[4]. The main imaging finding is segmental constriction of intracranial arteries, which can be associated with SAH and/or ischemic lesions[5].

Rare cases of RCVS have been linked to UIAs, as in a notable report by Dodick et al.[6] describing two patients with the triad of thunderclap headache, reversible cerebral vasoconstriction, and an unruptured aneurysm. Another case report documented a female patient who developed vasospasm in the context of an unruptured posterior communicating artery aneurysm, followed by complete resolution of the vasospasm[7]. In previously reported cases, vasospasm typically presented with severe headache rather than cerebral infarction. In contrast, we report a distinctive case of ischemic stroke resulting from reversible vasospasm associated with multiple unruptured aneurysms along the ICA.

The pathophysiology underlying RCVS remains largely unknown. The occurrence of vasospasm in the absence of subarachnoid blood suggests that factors other than blood components can trigger this phenomenon. Given its distribution, the vasospasm likely originated from the predisposing factors intrinsic to the aneurysm. Pathoanatomical alterations of the aneurysm wall may elicit vasoconstriction in the neighbouring vessels adjacent to the parent artery. Such a change might be either spontaneous, such as abrupt enlargement of the aneurysm, or iatrogenic, as seen with surgical clipping or carotid artery revascularization[8,9], which is postulated to damage the adjacent endothelium and impair its ability to produce prostacyclin, a vasorelaxant substance[7].

The thrombo-embolic events derived from the aneurismal cavity were another plausible explanation involved the provoking of vasoconstriction and cerebral infarction. The setting of vasospasm in return elevated shear stress, which might lead to platelet activation and microthrombus formation accompanied by impaired washout of emboli in hypoperfusion states[8].

We have noticed that aneurysms of this patient are close to the circle of Willis. It is now established that the circle of Willis receives rich sensory perivascular innervation from the trigeminal ganglion and sensitizes the "trigeminovascular system" (TGVS), which could be responsible for the spasm of vessels without aneurysm rupture[10].

This report reminds of us that UIAs can be direct triggers for RCVS. This case contributes to the body of

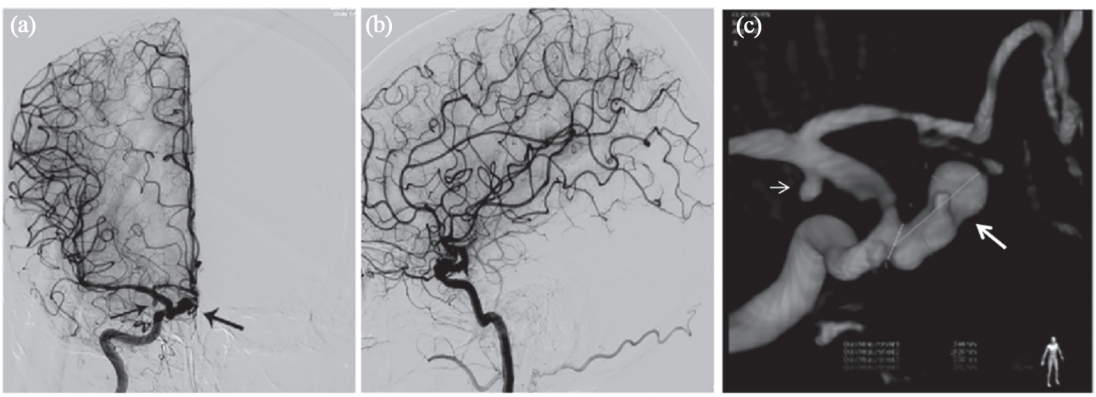


**Figure 4:** Right ICA angiogram ((a) Anteroposterior view; (b) Lateral view; (c) Magnified view of the aneurysms. DSA demonstrated normal lumen diameter of the M1 segment of the right middle cerebral artery. Multiple aneurysms were detected at the supraclinoid portion (large arrow) and terminal segment (small arrow) of the right ICA).

evidence and implies that the incidence of UIA-induced vasospasm may be greater than currently appreciated.

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## Authors' contributions

Dan Su contributed to collection and assembly of the data; Kefu Cai wrote the manuscript and performed the critical revision of article.

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## Ethics approval and consent to participate

The Ethics Commission of the Affiliated Hospital of Nantong University approved the data collection (2025-L270). Informed consent was obtained from the patient.

## Disclosure of artificial intelligence (AI) use

All authors declare that no AI was used in the writing and publication of this article.

## Competing interests

The authors declare that they have no competing interests.

## Consent for publication

All the authors consent to the publication of identifiable details, which can include figures and data details within the text to be published by *Translational Neurology and Neurosurgery*.

## Data availability statement

The raw data supporting the conclusions of this case report will be made available by the authors, without undue reservation.

## References

1. Paolini S, Kanaan Y, Wagenbach A, et al. Cerebral vasospasm in patients with unruptured intracranial aneurysms. *Acta Neurochir (Wien)*. 2005;147(11):1181-1188; discussion 1188.
2. Kim JG, Kang CH, Sheen JJ, et al. A case of severe delayed vasospasm after clipping surgery for an unruptured intracranial aneurysm. *Neurointervention*. 2024;19(2):123-128.
3. Matsubara M, Takizawa Y, Saito Y, et al. Symptomatic cerebral vasospasm after clipping of an unruptured intracranial aneurysm: a case report and literature review. *Cureus*. 2025;17(8):e90339.
4. Singhal AB. Reversible cerebral vasoconstriction syndrome: a review of pathogenesis, clinical presentation, and treatment. *Int J Stroke*. 2023;18(10):1151-1160.
5. Perillo T, Paolella C, Perrotta G, et al. Reversible cerebral vasoconstriction syndrome: review of neuroimaging findings. *Radiol Med*. 2022;127(9):981-990.
6. Dodick DW. Thunderclap headache. *J Neurol Neurosurg Psychiatry*. 2002;72(1):6-11.
7. Friedman P, Gass HH, Magidson M. Vasospasm with an unruptured and unoperated aneurysm. *Surg Neurol*. 1983;19(1):21-25.
8. Nickele C, Muro K, Getch CC, et al. Severe reversible cerebral vasoconstriction syndrome mimicking aneurysmal rupture and vasospasm. *Neurocrit Care*. 2007;7(1):81-85.
9. Constant Dit Beaufils P, Lecluse A, Guillon B, et al. Reversible cerebral vasoconstriction syndrome following carotid artery revascularization: about three case reports and review of literature. *J Med Vasc*. 2024;49(5-6):195-202.
10. Edvinsson L, Juul R, Jansen I. Perivascular neuropeptides (npy, vip, cgrp and sp) in human brain vessels after subarachnoid haemorrhage. *Acta Neurol Scand*. 1994;90(5):324-330.