



Symptomatic cerebral vasospasm manifested 18 days after aneurysmal subarachnoid haemorrhage

Takakazu Kawamata, Nobuhiko Aoki, Tatsuo Sakai & Koji Arai

To cite this article: Takakazu Kawamata, Nobuhiko Aoki, Tatsuo Sakai & Koji Arai (1993) Symptomatic cerebral vasospasm manifested 18 days after aneurysmal subarachnoid haemorrhage, *Neurological Research*, 15:3, 209-211, DOI: [10.1080/01616412.1993.11740137](https://doi.org/10.1080/01616412.1993.11740137)

To link to this article: <https://doi.org/10.1080/01616412.1993.11740137>



Published online: 20 Jul 2016.



Submit your article to this journal [↗](#)



Article views: 1



Citing articles: 3 [View citing articles](#) [↗](#)

Case Report

Symptomatic cerebral vasospasm manifested 18 days after aneurysmal subarachnoid haemorrhage

Takakazu Kawamata, Nobuhiko Aoki, Tatsuo Sakai and Koji Arai

Department of Neurosurgery, Tokyo Metropolitan Fuchu Hospital, Tokyo, Japan

A patient who suffered rapid deterioration due to cerebral vasospasm 18 days after aneurysmal subarachnoid haemorrhage is reported. Unusually delayed onset of ischaemic neurological deficits and its preventative management is discussed. [Neurol Res 1993; 15: 209–211]

Keywords: Subarachnoid haemorrhage; cerebral vasospasm; cerebral aneurysm; delayed ischaemic neurological deficit

INTRODUCTION

Symptomatic cerebral vasospasm is known to occur commonly in the period between 4 and 14 days after the onset of aneurysmal subarachnoid haemorrhage (SAH)^{1,2}. To our knowledge, only a few investigators have documented the occurrence of delayed ischaemic neurological deficit (DIND) that developed after 15 days of aneurysmal SAH^{3,4}. However, serial cerebral blood flow (CBF) measurements revealed prolonged reduction of CBF during the 3rd week after SAH^{5,6}. In the practical setting, unreported cases of DIND with the onset later than 15 days after the ictus, though rarely, seem to be experienced at most neurosurgical institutions.

Recently the authors encountered a patient whose clipping and postoperative course was uneventful until 18 days after SAH, when unexpectedly she was complicated by severe DIND with an unsatisfactory response to aggressive treatment. Few publications have specifically addressed as to how long prophylactic management for vasospasm be continued. It seems worthwhile to discuss the value of preventative management for vasospasm during the 3rd week after aneurysmal SAH.

CASE REPORT

A 57-year-old housewife with a history of mild hypertension was brought to the emergency room at our institution for the sudden onset of severe headache on October 24, 1990.

Examination

Upon arrival the patient was alert and did not show any neurological abnormalities, including neck stiffness.

Correspondence and reprint requests to: Dr Takakazu Kawamata, Department of Neurosurgery, Neurological Institute, Tokyo Women's Medical College, 8-1 Kawada-Cho, Shinjuku-Ku Tokyo 162, Japan. Accepted for publication November 1992.

Computed tomography (CT) scans revealed thin layers of SAH, predominantly in the ambient cistern and the sylvian fissure on the right side (*Figure 1*). Cerebral angiography delineated two aneurysms at the junction of the internal carotid and the posterior communicating artery, and at the bifurcation of the middle cerebral artery on the right side (*Figure 2*).

Operation

With a preoperative condition of Hunt and Kosnik grade 2⁵, the patient underwent clipping of both

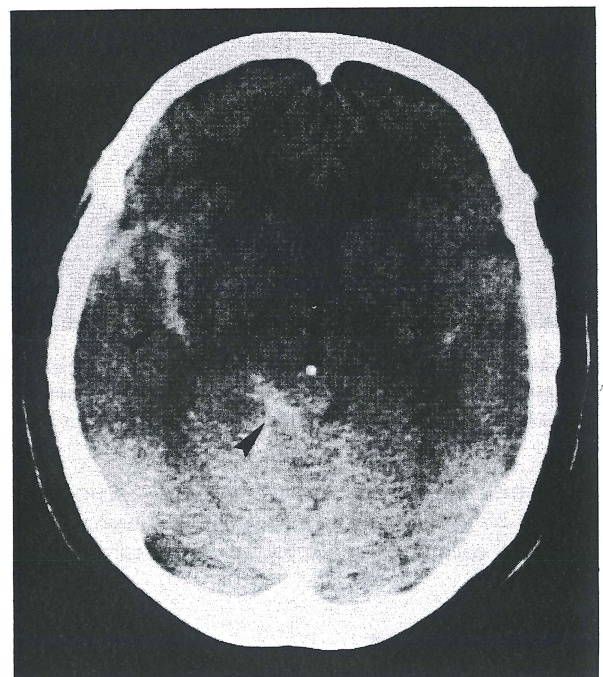


Figure 1: A CT scan on admission shows thin layers of SAH, mainly in the ambient cistern and the sylvian fissure on the right side (arrows)

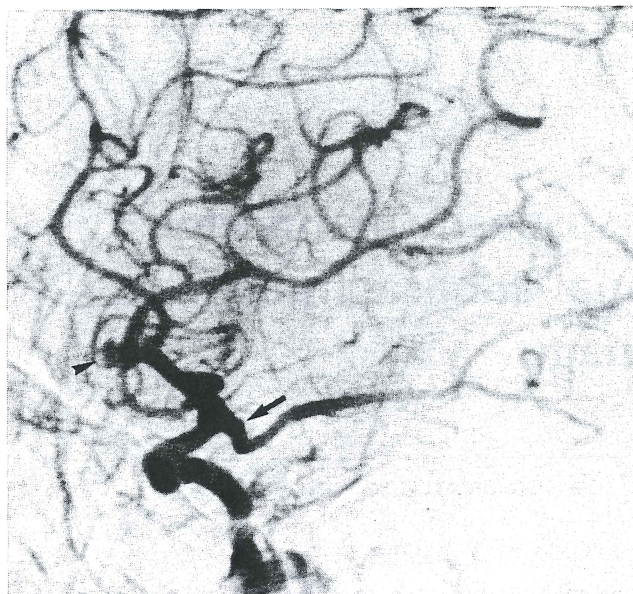


Figure 2: Right carotid angiography on admission discloses two cerebral aneurysms at the junction of the internal carotid artery and the posterior communicating artery (arrow), and at the bifurcation of the middle cerebral artery on the right side (arrowhead)

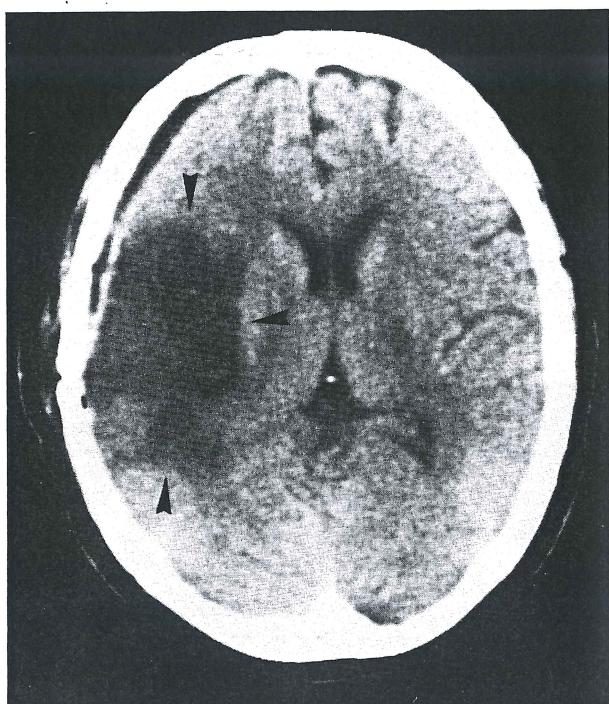


Figure 3: A CT scan 20 days after SAH (i.e., 2 days after the onset of symptomatic vasospasm) reveals large low-density lesions in the territory of the right middle cerebral artery (arrowheads)

aneurysms through a right pterional approach on October 25. At surgery, rupture of the internal carotid aneurysm was confirmed to be responsible for the current SAH.

Post-operative course

The patient awoke from general anaesthesia without neurological deficits. Her post-operative course was uneventful with the management including hypervolaemic haemodilution therapy. Post-operative CT

scans taken 14 days after SAH showed a small low-density lesion in the left thalamus. No other ischaemic lesions were delineated. The patient was free of neurologic deficit until early morning of November 10 (18 days), when she was noted to have mild hemiparesis on the left side. In addition, she was not fully alert. Immediately after detection of these symptoms, hypervolaemic hypertension treatment with low molecular dextran and urokinase was initiated without sufficient response. Neurological evaluation the following day showed complete hemiplegia and severe dysarthria. Serial CT scans exhibited a small low-density area in the right prefrontal region on November 10 (18 days), and then diffuse low-density lesions in the territory of the middle cerebral artery on November 12 (20 days) (Figure 3).

Post-operative cerebral angiography performed on November 14 (22 days) disclosed high-grade narrowing (70–80% reduction in caliber) of the superior and inferior trunk of the M2 portion of the right middle cerebral artery (Figure 4). Although the patient showed resolution of dysarthria, dense hemiparesis remained despite a 3-month course of rehabilitation.

DISCUSSION

Cerebral vasospasm secondary to ruptured aneurysms is recognized to have its earliest onset of 4 days after SAH, and to reduce in severity within 2 weeks^{2,7}. Coinciding with the narrowing of the vessels, neurological symptoms such as DIND commonly occur within 14 days after SAH^{1,2}. In the practical setting, however, though extremely limited, the occurrence of DIND with its onset 14 days or later after SAH is documented^{3,4}. This uncommon phenomenon may deserve to be discussed as to how long prophylactic management for vasospasm should be continued.

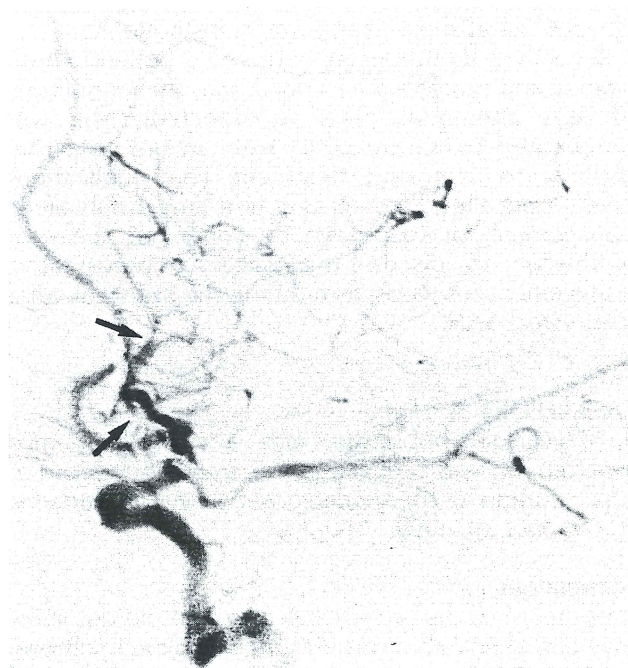


Figure 4: Post-operative carotid angiography on the right side 22 days after SAH demonstrates severe narrowing (70–80% reduction in caliber) at the proximal part of the M2 portion of the right middle cerebral artery (arrows)

Although there is a recent report describing symptomatic arterial luminal narrowing presenting months after SAH⁸, the mechanism of this pathology differs from vasospasm exclusively caused by SAH *per se*⁹.

According to Abiko and his colleagues³, very delayed onset of vasospasm tends to develop in patients with a large volume of SAH on the initial CT scans. The outcome of their five patients included 'good' in 2, 'poor' in 2, and 'died' in one, suggesting that symptomatic vasospasm manifesting 15 days or later is not necessarily benign in prognosis. The patient reported here presented with a relatively mild degree of SAH (Fisher group 2), which resolved early in the post-operative course. Furthermore, aggressive treatment for the prevention of vasospasm was carried out for 14 days after SAH. Despite these measures, relentless progression of DIND began 18 days after SAH. Severe narrowing of the middle cerebral artery was confirmed on post-operative angiography. Retrospectively, mild dehydration and low blood pressure seen 15 days after SAH may have contributed to the occurrence of catastrophe in this patient.

Meyer *et al.* studied change in CBF after SAH⁶. They concluded that CBF fell progressively during the first 2 weeks and remained abnormally low throughout 3 weeks after SAH. Also, Knuckey and his colleagues revealed that even in good clinical grade patients, with low CBF on admission, flows may not return to normal for 3 to 4 weeks after SAH¹⁰. Thus, serial CBF measurements indicate that reduction of CBF lasting more than 2 weeks after SAH is not exceptional.

From a prophylactic standpoint, Öhman *et al.* stressed the effect of their protocol including oral administration of nimodipine for up to 21 days after SAH¹¹. In the most recent publication, Origitano *et al.* demonstrated the persistent elevation of CBF during 21 days after SAH in patients who were treated with hypertensive hypervolaemic haemodilution therapy for 10 to 14 days after surgery¹². From these observations, the possibility of occurrence of symptomatic vasospasm is thought to be present for more than 2 weeks after SAH. This may explain the mechanism of very delayed ischaemic neurological deficits manifesting following the discontinuation of aggressive prophylactic management.

To date, reports of patients showing symptomatic vasospasm 15 days or later after SAH are very few^{3,13}, however, unpublished experience derived from personal communications suggests that this phenomenon is not as rare as it might seem. In an effort to minimize this complication, there is merit maintaining sufficient hydration and blood pressure, and continuing the administration of vasodilating drugs during the 3rd

week after SAH, based on the findings of cerebral angiography and serial transcranial Doppler evaluation. Once patients become symptomatic, emergency treatment with aggressive therapeutic modalities is mandatory.

In conclusion, cerebral vasospasm may become symptomatic during the 3rd week after SAH. This is supported by the results of serial CBF measurements, indicating reduced CBF lasting more than 2 weeks after SAH. It is important to continue supportive treatment, at least including sufficient hydration and avoidance of low blood pressure, during the 3rd week after SAH.

REFERENCES

- 1 Fisher CM, Roberson GH, Ojemann RC. Cerebral vasospasm with ruptured saccular aneurysm – The clinical manifestations. *Neurosurgery* 1977; **1**: 245–248
- 2 Saito I, Ueda Y, Sano K. Significance of vasospasm in the treatment of ruptured intracranial aneurysms. *J Neurosurg* 1977; **47**: 412–429
- 3 Abiko H, Mizoi K, Suzuki J, Yoshimoto T. Evaluation of patients manifesting very delayed cerebral vasospasm. In Ota T, ed. *Proceedings of the 3rd Cerebral Vasospasm Symposium in Kyoto*, Tokyo: Chugai Igakusha, 1988; pp. 117–122
- 4 Taneda M, Wakayama A, Ozaki K, Kataoka K, Hayakawa T, Mogami H. Biphasic occurrence of delayed ischemia after early aneurysm surgery. Case report. *J Neurosurg* 1983; **58**: 440–442
- 5 Hunt WE, Kosnik EJ. Timing and perioperative care in intracranial aneurysm surgery. *Clin Neurosurg* 1974; **21**: 79–89
- 6 Meyer CHA, Lowe D, Meyer M, Richardson PL, Neil-Dwyer G. Progressive change in cerebral blood flow during the first three weeks after subarachnoid hemorrhage. *Neurosurgery* 1983; **12**: 58–76
- 7 Weir B, Grace M, Hansen J, Rothberg C. Time course of vasospasm in man. *J Neurosurg* 1978; **48**: 173–178
- 8 Kondziolka D, Bernstein M, Spiegel SM, ter Brugge K. Symptomatic arterial luminal narrowing presenting months after subarachnoid hemorrhage and aneurysm clipping. *J Neurosurg* 1988; **69**: 494–499
- 9 Shiokawa Y, Aoki N, Saito I. Delayed ischemic deficit after SAH and aneurysm clipping. Letter. *J Neurosurg* 1989; **70**: 970
- 10 Knuckey NW, Fox RA, Surveyor I, Stokes BAR. Early cerebral blood flow and computerized tomography in predicting ischemia after cerebral aneurysm rupture. *J Neurosurg* 1985; **62**: 850–855
- 11 Öhman J, Servo A, Heiskanen O. Long-term effects of nimodipine on cerebral infarcts and outcome after aneurysmal subarachnoid hemorrhage and surgery. *J Neurosurg* 1991; **74**: 8–13
- 12 Origitano TC, Wascher TM, Reichman OH, Anderson DE. Sustained increased cerebral blood flow with prophylactic hypertensive hypervolemic hemodilution ('Triple-H' therapy) after subarachnoid hemorrhage. *Neurosurgery* 1990; **27**: 729–740
- 13 Ohno K, Masaoka H, Suzuki R, Monma S, Matsushima Y. Symptomatic cerebral vasospasm of unusually late onset after aneurysm rupture. *Acta Neurochir (Wien)* 1991; **108**: 163–166