Post-ischemic hyperperfusion after clipping of a ruptured internal carotid-posterior communicating artery aneurysm under suction decompression

Yoshiteru Tada a,⁎, Junichiro Satomi a, Yasuhisa Kanematsu a, Kazuyuki Kuwayama a, Nobuhisa Matsushita a, Takashi Abe b, Masafumi Harada b, Shinji Nagahiro a

a Department of Neurosurgery, School of Medicine, The University of Tokushima, Tokushima City, Japan
b Department of Radiology, School of Medicine, The University of Tokushima, Tokushima City, Japan

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A B S T R A C T

A 64-year-old woman presenting with subarachnoid hemorrhage (World Federation of Neurosurgeons grade IV) from the rupture of a right large internal carotid-posterior communicating artery aneurysm suffered neuronal damage associated with post-ischemic hyperperfusion after neck clipping of the aneurysm under suction decompression. She did not completely recover consciousness after the operation. Diffusion-weighted imaging (DWI) performed on the first postoperative day showed subtle cortical hyperintensity in the parietal lobe. Arterial spin-labeling (ASL) and 123I-iodoamphetamine (123I-IMP) single photon emission computed tomography (SPECT) demonstrated hyperperfusion in the right tempo-parietal lobes. Delayed 123I-iomazenil (123I-IMZ) SPECT images showed a reduced IMZ uptake in the right tempo-parietal lobe corresponding to the hyperperfusion area on ASL images. DWI repeated on postoperative day 3 revealed progression of a hyperintensity lesion in the right parietal lobe. Blood pressure control and the use of a free radical scavenger relieved her symptoms. One month later, the area of reduced IMZ uptake was further expanded. Our findings suggest that post-ischemic hyperperfusion after suction decompression may result in neuronal damage demonstrated on 123I-IMZ SPECT images.

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Case report

A 64-year-old woman with a history of uncontrolled hypertension lost consciousness and was admitted to our hospital. Her neurological status was World Federation of Neurosurgeons grade 4. There was no paresis of her extremities. Computed tomography (CT) revealed thick basal subarachnoid hemorrhage (SAH). Initial diffusion-weighted imaging (DWI) showed no cerebral infarction. Angiograms demonstrated a right large IC-PC aneurysm and patency of the anterior and posterior communicating artery. As the aneurysmal neck involved the origin of the posterior communicating artery we performed surgery. We inserted a balloon catheter into the distal cervical segment of the right ICA. The balloon was inflated and a temporary clip was placed on the ICA distal to the aneurysm. After the aspiration of blood through the catheter, the collapsed aneurysm was obliterated with two clips. The duration of the temporary occlusion was 14 minutes. Postoperatively, she did not completely regain consciousness. DWI performed on the first postoperative day showed subtle cortical hyperintensity in the parietal lobe. We report a patient with a ruptured internal-posterior communicating artery (IC-PC) aneurysm who underwent neck clipping under suction decompression. This case was complicated by post-ischemic hyperperfusion on ASL and 123I-IMP SPECT images. We posit that her hyperperfusion state led to progressive neuronal damage.

Introduction

The treatment of large and giant internal carotid artery (ICA) aneurysms may require suction decompression for the safe and effective occlusion of the aneurysmal neck. No patients with cerebral hyperperfusion after suction decompression have been reported [1]. Cerebral hyperperfusion after vascular construction surgery has been documented to result in intracranial hemorrhage and neuronal damage [2–4]. While 123I-iodoamphetamine (123I-IMP) single photon emission computed tomography (SPECT) remains the gold standard for the accurate diagnosis of postoperative hyperperfusion, arterial spin-labeling (ASL) has been used to evaluate cerebral perfusion in stroke patients [5]. It is a magnetic resonance imaging (MRI) technique that helps to identify and quantify hypoperfusion areas. 123I-iomazenil (123I-IMZ), a central benzodiazepine receptor ligand, is an ideal marker of neuronal integrity [6]. Early and delayed 123I-IMZ SPECT images reveal cerebral perfusion and neuronal viability, respectively [6].

⁎ Corresponding author at: Department of Neurosurgery, Institute of Health Biosciences, The University of Tokushima Graduate School, 3-18-15, Kuranoto-cho, Tokushima, Tokushima 770-8503, Japan. Tel.: +81 88 633 7149; fax: +81 88 632 9464.
E-mail address: yoshiteru.tada@hotmail.co.jp (Y. Tada).

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first postoperative day (POD) demonstrated multiple high-intensity lesions in the right caudate head and the splenium of the corpus callosum suggesting primary brain injury. The signal change in the right temporo-parietal lobes was subtle (Fig. 1A). ASL showed a hyperperfusion area in the right temporo-parietal regions (Fig. 1B). On POD 2 we noted decreased IMZ binding in the right temporo-parietal lobe on delayed 123I-IMZ SPECT images; the area coincided with the hyperperfusion area on ASL images (Fig. 2A). DWI on POD 3 revealed hyperintensity in the right temporo-parietal lobes (Fig. 1C). ASL findings were similar to those obtained on POD 1 (Fig. 1D). Angiograms confirmed the absence of vasospasm and total obliteration of the aneurysm.

Based on a diagnosis of post-ischemic hyperperfusion we continued to control her blood pressure at normotension and administered glycerol and edaravone, a free radical scavenger. 123IMP-SPECT performed on POD 6 confirmed hyperperfusion in the same area (Fig. 2B). Her symptoms started to resolve on POD 7. 123I-IMZ SPECT images obtained one month after the operation demonstrated decreased IMZ uptake in the right hemisphere (Fig. 2C). T1-weighted images acquired 2 months after the operation revealed cortical linear hyperintensities in the affected area, a typical radiological characteristic of cortical laminar necrosis. DWI and ASL performed 5 months after the operation revealed a hypointensity- and a hypoperfusion area in the right temporo-parietal lobe (Fig. 1E and F). Five months post-onset she manifested no paresis of her extremities, no unilateral spatial neglect, and no apraxia. Her mini-mental state examination score was 28 out of 30. Her modified Rankin scale was 1.

**Discussion**

We suggest that post-ischemic hyperperfusion after neck clipping of a ruptured aneurysm using suction decompression may result in neuronal damage. We document that ASL studies, which can be performed repeatedly, are useful for evaluating hyperperfusion and that 123I-IMZ SPECT can detect neuronal damage due to hyperperfusion.

Cerebral hyperperfusion is defined as a significant increase in the cerebral blood flow (CBF) on the side of arterial stenosis or occlusion after reconstruction surgery. In the presence of cerebral hyperperfusion, the blood supply exceeds the metabolic demands of brain tissue. Hyperperfusion after the clipping or coiling of ruptured and unruptured aneurysms has been reported [7,8]. Putative causative factors include...
chronic hypertension, decreased preoperative CBF, SAH, flow changes after giant aneurysm clipping, and prolonged temporary occlusion. Ours is the first reported case of post-ischemic hyperperfusion after suction decompression.

Suction decompression is required to facilitate clipping of large and giant ICA aneurysms. It effectively produces shrinking and softening of the aneurysm and facilitates the full dissection of the ICA, of branches, and of the aneurysm, and allows aneurysmal clipping. According to Fulkerson et al. [1] the complication rates in patients with ophthalmic artery aneurysms treated with and without endovascular suction decompression were similar. However, placement of the catheter in the ICA and balloon occlusion of the vessel risk ischemic complications. We do not know whether suction decompression per se added to the inherent risks posed by temporary occlusion and led to post-ischemic hyperperfusion. Continuous suction is necessary for the aneurysms to remain collapsed because of the persistent collateral arterial blood supply. As vacuum pressure exerted by the suction force applied within the cerebral vasculature may exacerbate cerebral ischemia we suggest that the proper suction force must be predetermined.

In most patients with cerebral hyperperfusion without intracranial hemorrhage, neurological deficits are reversible. Although it rarely leads to neuronal damage [2,3], lihara et al. [3] documented that hyperperfusion after extracranial-intracranial bypass surgery for a large unruptured cerebral aneurysm resulted in progressive neuronal death. In our patient the hyperperfused area on ASL images acquired on POD 1 was larger than the DWI lesions and the signal change on DWI was subtle in the temporo-parietal lobes. This suggests that post-ischemic hyperperfusion contributed at least partly to her neuronal damage.

Long-standing hypertension, poor collateral blood flow, high-grade carotid artery stenosis, and an impaired cerebral hemodynamic reserve are risk factors related to cerebral hyperperfusion [9]. A disruption in autoregulation has been observed in patients with SAH. Although we did not evaluate the cerebral perfusion reserve in our patient, we posit that, in addition to her undergoing suction decompression, impaired cerebral-vascular autoregulation related to uncontrolled hypertension and SAH may have played a role in the manifestation of hyperperfusion [10]. After control of her blood pressure at normotension and the administration of a free radical scavenger, her symptoms resolved. Although we cannot confirm the effects of the free radical scavenger on her post-ischemic hyperperfusion, it is possible that it reduced the amount of reactive oxygen species produced during post-ischemic hyperperfusion.

For the accurate diagnosis of cerebral hyperperfusion 123I-IMP SPECT remains important, but ASL as an additional diagnostic tool offers several benefits [5]. It is noninvasive and can be performed repeatedly with other sequences such as DWI. We confirmed hyperperfusion not only by 123I-IMP SPECT but also by ASL. Although signal changes on DWI obtained on POD 1 were subtle, the areas of decreased IMZ uptake in the acute phase on 123I-IMP SPECT images corresponded to the areas of decreased IMZ uptake and hypoperfusion on ASL images obtained in the chronic phase. This suggests that 123I-IMP SPECT is useful for predicting eventual neuronal damage. Although cortical atrophy was observed on MRI obtained 5 months postoperatively, the neurological status was apparently recovered in our patient. As shown on T1-weighted images obtained 2 months postoperatively, post-ischemic hyperperfusion induced cortical laminar necrosis rather than frank infarction in the right temporo-parietal lobe.

### Conclusion

We caution that suction decompression for ruptured aneurysms may result in post-ischemic hyperperfusion and neuronal damage. Patients with neurological deterioration after suction decompression of ruptured aneurysms should be evaluated immediately by MRI and SPECT studies.

### References


