



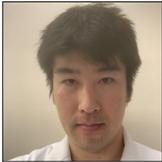
Case Report

Intracerebral steal phenomenon induced focal reversible vasogenic edema and decrease in cerebral blood flow after carotid endarterectomy

Yu Shimizu, Katsuhiko Tsuchiya, Norihiro Fujisawa

Department of Neurosurgery, Kanazawa Medical Center, 1-1 Shimoishibikimachi, Kanazawa, Ishikawa, Japan.

E-mail: *Yu Shimizu - bleuler3a@yahoo.co.jp; Katsuhiko Tsuchiya - bleuler5a@gmail.com; Norihiro Fujisawa - bleuler4a@yahoo.co.jp



***Corresponding author:**

Yu Shimizu,
Department of Neurosurgery,
Kanazawa Medical Center, 1-1
Shimoishibikimachi, Kanazawa,
Ishikawa, Japan.

bleuler3a@yahoo.co.jp

Received : 05 October 2019

Accepted : 05 June 2020

Published : 20 June 2020

DOI

10.25259/SNI_508_2019

Quick Response Code:



ABSTRACT

Background: Intracerebral steal phenomenon (ISP) is a rare complication following surgical treatment of carotid stenosis. However, the factors responsible remain unknown. We described the rare case of the ISP who had vasogenic edema and cerebral blood flow (CBF) decline and presented with hemiparesis after carotid endarterectomy (CEA).

Case Description: A 72-year-old male with stenosis of the bilateral carotid artery (NASCET right 90% and left 70%) presented with cerebral hypoperfusion manifesting as right hemiparesis, after left CEA. Fluid-attenuated inversion recovery images showed edema of the motor area around an old infarction and a decrease in CBF. This lesion was an area of vasogenic edema caused by ISP and focal cerebral hypoperfusion. CBF of the contralateral cerebral hemispheres had increased. The treatment with an intravenous infusion of a free radical scavenger and glycerol improved the patient's symptoms and brain edema. Magnetic resonance imaging showed a gradual decline in the brain edema, which completely disappeared 2 weeks after CEA. He was discharged with no neurological deficit.

Conclusion: In this report, we described the case of a patient with ISP who had vasogenic edema induced by CBF decline and presented with hemiparesis following CEA. This is the first report of progressing focal vasogenic edema caused by ISP after endarterectomy.

Keywords: Cerebral blood flow, Endarterectomy, Intracerebral steal phenomenon, Single-photon emission computed tomography, Vasogenic edema

INTRODUCTION

Intracerebral steal phenomenon (ISP) is a rare complication after the treatment of carotid artery stenosis. ISP after the stent is reported, but the ISP after the carotid endarterectomy (CEA) has not been reported yet.^[8] Cerebral hypoperfusion induced by ISP can cause local vasogenic edema in the acute stages after surgery. Revascularization of cervical carotid stenosis can change ipsilateral cerebral blood flow (CBF). A postoperative increase in CBF on the operated side is common. In patients with bilateral stenotic lesions, CBF on the side contralateral to revascularization can increase.^[2] This phenomenon is unique and is due to increased blood flow through the anterior communicating artery (Acom). ISP is characterized by headaches, seizures, focal neurological deficits, and cognitive impairment, which appears

This is an open-access article distributed under the terms of the Creative Commons Attribution-Non Commercial-Share Alike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

©2020 Published by Scientific Scholar on behalf of Surgical Neurology International

within a few hours to 1-month postsurgery. ISP of CBF is an important factor in the initiation of decreased perfusion, which may result in brain edema. Here, we report a case involving ISP in a patient who presented with reversible brain edema and neurological symptoms after endarterectomy.

CASE PRESENTATION

A 72-year-old male initially presented with the right-sided hemiparesis and underwent magnetic resonance (MR) imaging. MR imaging revealed acute infarction of the left frontal lobe [Figure 1] and bilateral internal carotid artery (ICA) stenosis (Rt: NASCET 90%, peak systolic velocity 425 cm/s and Lt: NASCET 70%, peak systolic velocity 212 cm/s). After 2 months of rehabilitation, his neurological status improved to a modified Rankin

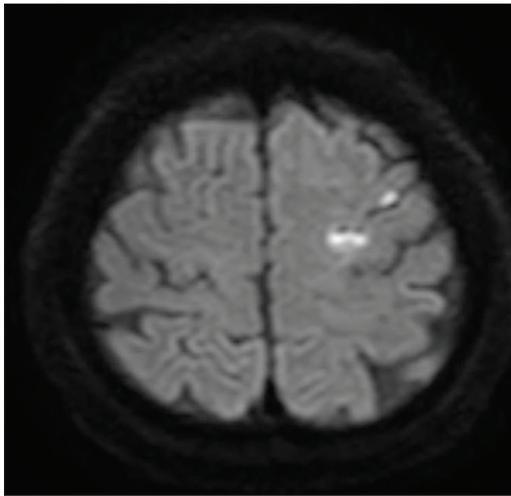


Figure 1: Diffusion-weighted magnetic resonance imaging revealing hyperintensity in the left motor area.

scale score of 0, and single-photon emission computed tomography (SPECT) revealed a decrease in CBF in the right cerebral hemisphere [Figure 2a]. Endarterectomy was successfully performed 3 months after the onset of minor stroke. We performed CEA on the left side because it was symptomatic. The patient was prescribed a dose of an antiplatelet agent (aspirin, 100 mg) daily for 7 days before the operation. We used the INVOS-4100 cerebral oximeter at several stages during CEA to measure the effect of carotid clamping and shunting on oxygen saturation (rSO_2). Contralateral rSO_2 readings were lower than ipsilateral rSO_2 readings during the preoperative period. However, the rSO_2 value increased by 2% (from 50 to 51) and 38% (from 47 to 65) on the ipsilateral and contralateral sides, respectively, the day after the CEA. The blood pressure did not have a change through the perioperative period. Neurological deterioration was observed after surgery, with the appearance of right-sided hemiparesis and dysarthria. Significant changes were not evident from the emergency CT scan. The apparent diffusion coefficient of this lesion was slightly high, but the DWI signal did not show a significant increase, suggesting the formation of local vasogenic edema. SPECT showed a decrease in CBF around the infarction [Figure 2b]. MR angiography showed a patent left ICA, whereas SPECT indicated a contralateral increase in CBF [Figure 2b]. Administration of a free-radical scavenger, edaravone, an antiepileptic agent, and glycerol, gradually relieved the patient's symptoms during the next 7 days. The CBF improved, and laterality disappeared 2 weeks after surgery [Figure 2c and d]. The blood flow of both sides equaled 2 weeks later [Figure 3]. Subsequent MR imaging with fluid-attenuated inversion recovery (FLAIR) images revealed a local high-intensity lesion around a previous infarction in comparison with before operation [Figure 4a and b]. FLAIR MR imaging

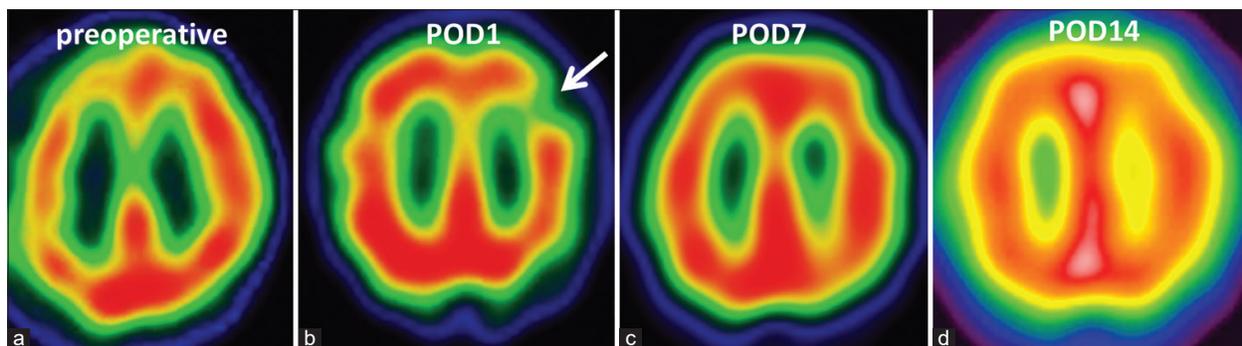


Figure 2: (a) Single-photon emission computed tomography (SPECT) revealing a significant decrease in cerebral blood flow (CBF) in the bilateral hemispheres, predominantly on the right side (CBF of middle cerebral artery area, R: 47 ml/100 g/min and L: 50 ml/100 g/min). (b) SPECT showing focal hypoperfusion (white arrow) around an old infarction (CBF of middle cerebral artery area, R: 65 ml/100 g/min and L: 51 ml/100 g/min). (c) SPECT demonstrating gradually normalized CBF in the lesion (CBF of middle cerebral artery area, R: 60 ml/100 g/min and L: 56 ml/100 g/min). (d) SPECT is demonstrating completely normalized CBF of the lesion (CBF of middle cerebral artery area, R: 59 ml/100 g/min, and L: 60 ml/100 g/min).

2 weeks after initial surgery confirmed the complete disappearance of the high-intensity lesion [Figure 4c]. The patient recuperated and did not exhibit any cerebrovascular events during the 2-year postoperative period.

DISCUSSION

Following CEA, the previously reduced CBF due to stenosis or occlusion on the contralateral side increased.^[2,7] The presence of the Acom was related to this phenomenon.^[2]

It was reported that communicating arteries caused contralateral CBF increase.^[5] Collateral blood flow plays an important role in sustaining adequate tissue perfusion during flow-limiting carotid artery disease.^[6] Our patient had a well-developed Acom. Leptomeningeal anastomoses on the cerebral surface can cause this CBF increase. A postoperative increase in perfusion pressure on the ipsilateral side may increase collateral circulation to the contralateral CBF deficient hemisphere through the leptomeningeal

anastomoses, resulting in increased CBF in the contralateral hemisphere.

Vasogenic edema occurs due to local cerebral hypoperfusion and subsequent dis-autoregulation/blood-brain barrier (BBB) disruption after revascularization.^[1] Therefore, the association of focal cerebral hypoperfusion with local vasogenic edema, which was transiently responsible for the focal neurological deterioration in our case, is unique.

Sato *et al.* reported that cortical FLAIR high-intensity signs can be caused by postoperative hemodynamic conditions including hypoperfusion.^[9] Vasogenic edema can be caused by a variety of hemodynamic conditions after CEA, and an accurate diagnosis of the perioperative condition is necessary when symptomatic vasogenic edema is evident after surgery. Although the exact mechanism underlying this rare association is unknown, the intrinsic vulnerability of intracranial vascular wall structures and the increased vascular permeability after infarction may partly explain this unique pathophysiological condition.^[3] The excessive hemodynamic stress on the fragile vessel after infarction may cause increased vascular permeability in the pial artery and anatomical fragility.^[10] BBB breakdown is an important initiating factor in neurological deficits and vasogenic edema following carotid procedures. Reactive oxygen species produced by reperfusion in the chronic ischemic brain may affect vascular permeability by direct endothelial cell damage through lipid peroxidation and by activating MMP-9.^[4]

To counteract these deleterious cascades, we used a free-radical scavenger in addition to glycerin in the present case. These postoperative treatments, which were intended to prevent symptomatic cerebral hypoperfusion, may play a role in avoiding further progression of the vasogenic edema. Our patient recovered completely 2 weeks after surgery.

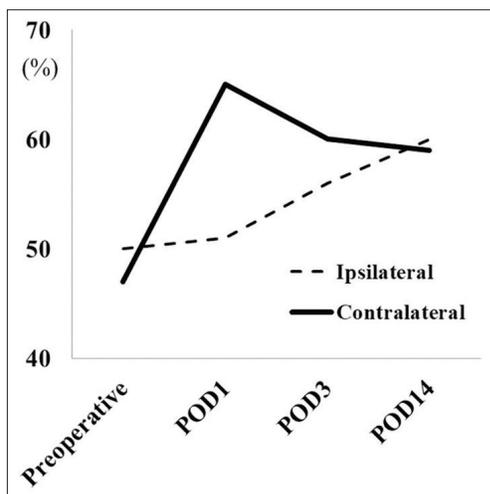


Figure 3: Time course of changes in cerebrovascular oxygen saturation and blood pressure in our patient.

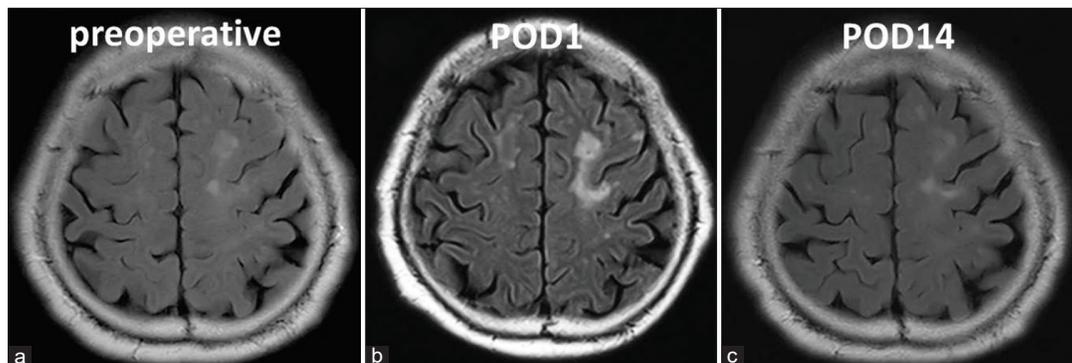


Figure 4: (a) Fluid-attenuated inversion recovery images 1 week before surgery with inconspicuous edema of the left frontal lobe. (b) Deterioration of edema the next day after surgery. (c) Almost complete disappearance of edema 2 weeks after surgery.

CONCLUSION

ISP caused focal vasogenic edema and neurologic deficits in our patient, who had a prior infarction. MR imaging showed focal vasogenic edema around the old infarction, and SPECT revealed a decrease in focal CBF. Focal vasogenic edema was subsequently found to have occurred around the fragile vessels of the old infarction. After treating the edema and ischemia, the neurologic deficits were alleviated.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. AY Lau, Wong EH, Wong A, Mok VC, Leung TW, Wong KS. Significance of good collateral compensation in symptomatic intracranial atherosclerosis *Cerebrovasc Dis* 2012;33:517-24.
2. Baracchini C, Meneghetti G, Manara R, Ermani M, Ballotta E. Cerebral hemodynamics after contralateral carotid endarterectomy in patients with symptomatic and asymptomatic carotid occlusion: A 10-year follow-up. *J Cereb Blood Flow Metab* 2006;26:899-905.
3. Hamano E, Kataoka H, Morita N, Maruyama D, Satow T, Iihara K. Clinical implications of the cortical hyper intensity belt sign in fluid-attenuated inversion recovery images after bypass surgery for Moyamoya disease. *J Neurosurg* 2017;126:1-7.
4. Hashimoto N, Tominaga T, Miyamoto S, Nagata I, Houkin K, Suzuki N, *et al.* Guidelines for diagnosis and treatment of Moyamoya disease (spontaneous occlusion of the circle of Willis). *Neurol Med Chir (Tokyo)* 2012;52:245-66.
5. Kataoka T, Hyogo T, Hayase K, Nakamura H. Cerebral blood flow change before and after carotid angioplasty and stenting (CAS) in cases with contralateral carotid artery occlusion. *Interv Neuroradiol* 2006;12:201-4.
6. Lagrèze HL, Levine RL, Pedula KL, Nickles RJ, Sunderland JS, Rowe BR. Contralateral flow reduction in unilateral stroke: Evidence for transhemispheric diaschisis. *Stroke* 1987;18:882-6.
7. Oka F, Ishihara H, Kato S, Higashi M, Suzuki M. Cerebral hemodynamic benefits after contralateral carotid artery stenting in patients with internal carotid artery occlusion. *AJNR Am J Neuroradiol* 2013;34:616-21.
8. Sadato A, Maeda S, Hayakawa M, Adachi K, Toyama H, Nakahara I, *et al.* Carotid stenting for unilateral stenosis can increase contralateral hemispheric cerebral blood flow. *J Neurointerv Surg* 2018;10:351-4.
9. Sato Y, Ito K, Ogasawara K, Sasaki M, Kudo K, Murakami T. Postoperative increase in cerebral white matter fractional anisotropy on diffusion tensor magnetic resonance imaging is associated with cognitive improvement after uncomplicated carotid endarterectomy: Tract-based spatial statistics analysis. *Neurosurgery* 2013;73:592-9.
10. Takagi Y, Kikuta K, Nozaki K, Hashimoto N. Histological features of middle cerebral arteries from patients treated for Moyamoya disease. *Neurol Med Chir (Tokyo)* 2007;47:1-4.

How to cite this article: Shimizu Y, Tsuchiya K, Fujisawa N. Intracerebral steal phenomenon induced focal reversible vasogenic edema and decrease in cerebral blood flow after carotid endarterectomy. *Surg Neurol Int* 2020;11:161.