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Surgical Neurology International
Editor-in-Chief: Nancy E. Epstein, MD, Professor of Clinical Neurosurgery, School of Medicine, State U. of NY at Stony Brook.
SNI: Neurovascular

Editor
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Case Report

A case of temporary occlusion of donor artery after secondary generalized seizure in a patient with superficial temporal artery-middle cerebral artery bypass

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Received: 21 July 2023
Accepted: 26 August 2023
Published: 15 September 2023

DOI
10.25259/SNI_612_2023

Quick Response Code:

ABSTRACT

Background: To prevent stroke recurrence, a superficial temporal artery-middle cerebral artery (STA–MCA) bypass for atherosclerotic cerebrovascular occlusive disease is performed. Post stroke epilepsy is known as serious sequelae of stroke. Herein, we present a case of a 60-year-old man who underwent STA–MCA bypass for the prevention of stroke recurrence; however, the donor artery was deemed to be temporally occluded secondary to generalized seizure.

Case Description: A 60-year-old man was referred to our hospital with a diagnosis of the left cervical internal carotid artery occlusion presenting with mild aphasia and right hemiparesis. He underwent STA–MCA bypass to prevent the recurrence of stroke 1 month after the onset of symptoms. On postoperative day 7, patency of the donor artery was confirmed on magnetic resonance imaging (MRI), and no complications were noted. However, on postoperative day 14, he presented with a secondary generalized seizure. MRI was immediately performed and the donor artery was not patent with no new lesions. Several hours thereafter, the blood flow of the donor artery was confirmed using pulse Doppler; however, during mouth opening, the flow of the donor artery decreased. Computed tomography-angiography confirmed donor artery patency. An encephalogram was conducted and revealed a focal epilepsy which was compatible with stroke on MRI.

Conclusion: Post stroke epilepsy caused an unintended and forced mouth opening which led to a temporary occlusion of the donor artery after STA–MCA bypass. Thus, this complication should be recognized, and seizures should be prevented through the administration of prophylactic anti-seizure medication based on risk stratification assessment of post stroke epilepsy.

Keywords: Open mouth, Post stroke epilepsy, Secondary generalized seizure, Superficial temporal artery-middle cerebral artery bypass

INTRODUCTION

In Japan, superficial temporal artery-middle cerebral artery (STA–MCA) bypass for atherosclerotic cerebrovascular occlusive disease is currently performed to prevent stroke recurrence. Specifically, patients with severe hemodynamic impairment who demonstrate cerebral blood flow <80% at rest and cerebral vascular reserve <10% are considered to be at high risk of stroke recurrence.[7-9] In these patients, according to the 2021 Japan Stroke Guidelines, extracranial–intracranial bypass is recommended.[11] Post stroke epilepsy is a serious complication of stroke which can occur in approximately 5–8% of patients and negatively affects functional outcomes.[5,6] Here, we present a

patient who underwent STA-MCA bypass for the prevention of stroke recurrence, in which the donor artery was temporally occluded due to a secondary generalized seizure as a manifestation of post stroke epilepsy.

CASE PRESENTATION

A 60-year-old man who is a smoker with a medical history of hypertension, hyperlipidemia, and diabetic mellitus was transferred to our hospital with a diagnosis of acute ischemic stroke secondary to a left cervical internal carotid artery occlusion. One of his pertinent medical histories was a left-sided depressed fracture in which he underwent reduction surgery during childhood. Three days before admission, he complained of mild aphasia, dysarthria, and right hemiparesis. Magnetic resonance imaging (MRI) and computed tomography (CT) angiography showed occlusion of the proximal left cervical carotid artery and ipsilateral ischemic stroke [Figures 1a-f]. Dual antiplatelet therapy (aspirin 100 mg and clopidogrel 75 mg) was administered, and rehabilitation was started. Three weeks after the onset of symptoms, he had a modified Rankin Scale of 1, indicating recovery. Cerebral angiography was performed to examine the cerebral blood flow in the left hemisphere [Figures 2a-c]. Thereafter, hemodynamic impairment was assessed using ^{123}I-iodoamphetamine single-photon emission CT (^{123}I-IMP SPECT), and the results showed a reduced cerebral blood flow at rest and cerebral vascular reserve in response to acetazolamide in the left hemisphere [Figures 2d-f]. At 1 month after the onset, he underwent STA–MCA bypass to prevent stroke recurrence, and prophylactic anti-seizure medication was administered for 3 days. On the first postoperative day, clopidogrel 75 mg per day was taken to prevent ischemic complications. A day and 7 days after the surgery, magnetic resonance angiography (MRA) showed good patency of the donor artery [Figures 3a and b], and the patient had an absence of new lesions or neurological deficits [Figures 3c and d]. Rest ^{123}I-IMP SPECT showed no region of hyperperfusion 9 days after the operation [Figure 3e]. However, 2 weeks thereafter, he suddenly presented an adversive seizure with a groan, neck rotation to the right and tonic elevation of the right arm lasting for <5 min which occurred at night time. He received intravenous administration of diazepam (5 mg). MRI was immediately performed and showed no new lesions. Furthermore, MRA revealed a diminished signal intensity of the donor artery [Figures 3f-h]. Several hours after the onset of a seizure, blood flow of the donor artery was confirmed using a pulse Doppler at the bedside; however, the blood flow of the donor artery decreased during mouth opening. Therefore, ultrasonography

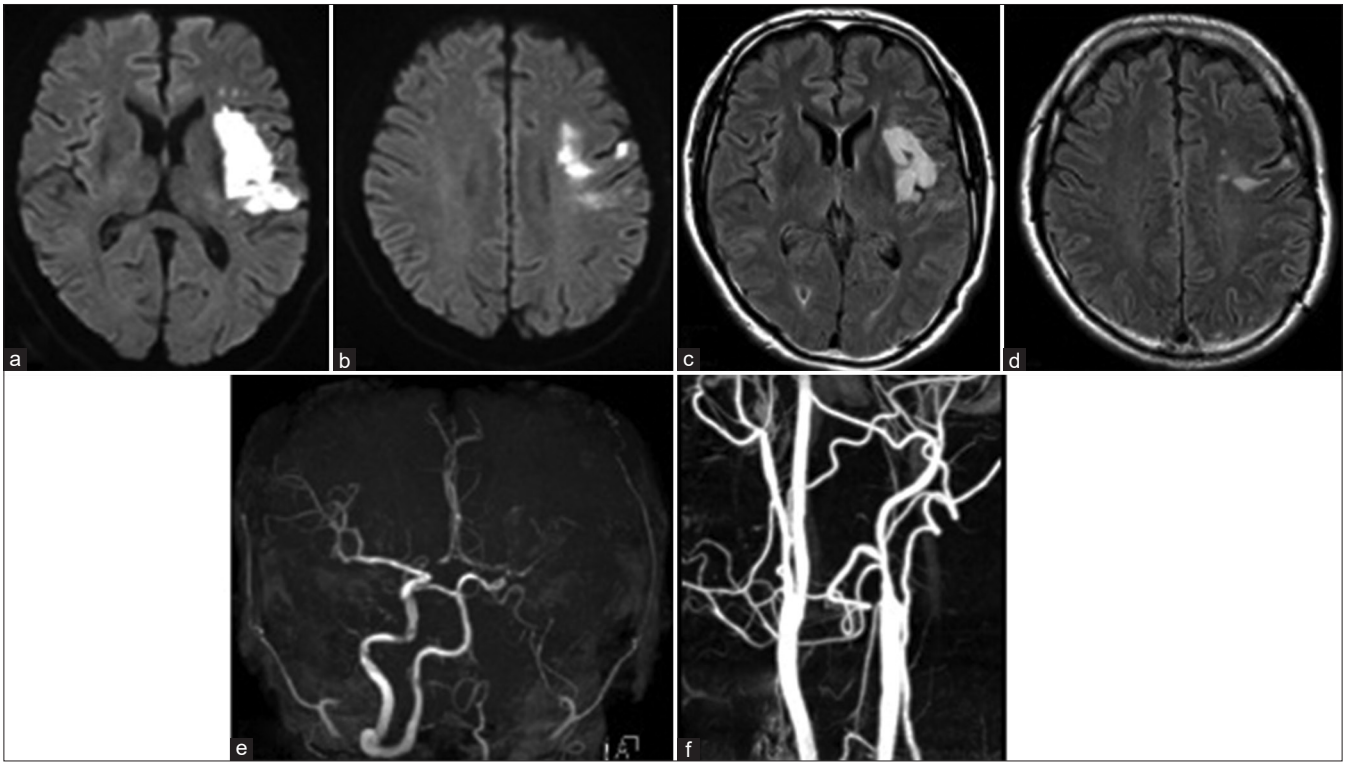

Figure 1: (a and b) Magnetic resonance imaging (MRI) diffusion-weighted imaging (DWI) demonstrating an infarct involving the left basal ganglia, insula, and opercular region. (c and d) MRI fluid-attenuated inversion recovery showing an infarct compatible with that of DWI. (e and f) Magnetic resonance angiography reveals no flow signal intensity in the left neck internal carotid artery (ICA) and contrast computed tomography demonstrates occlusion of the left neck ICA.

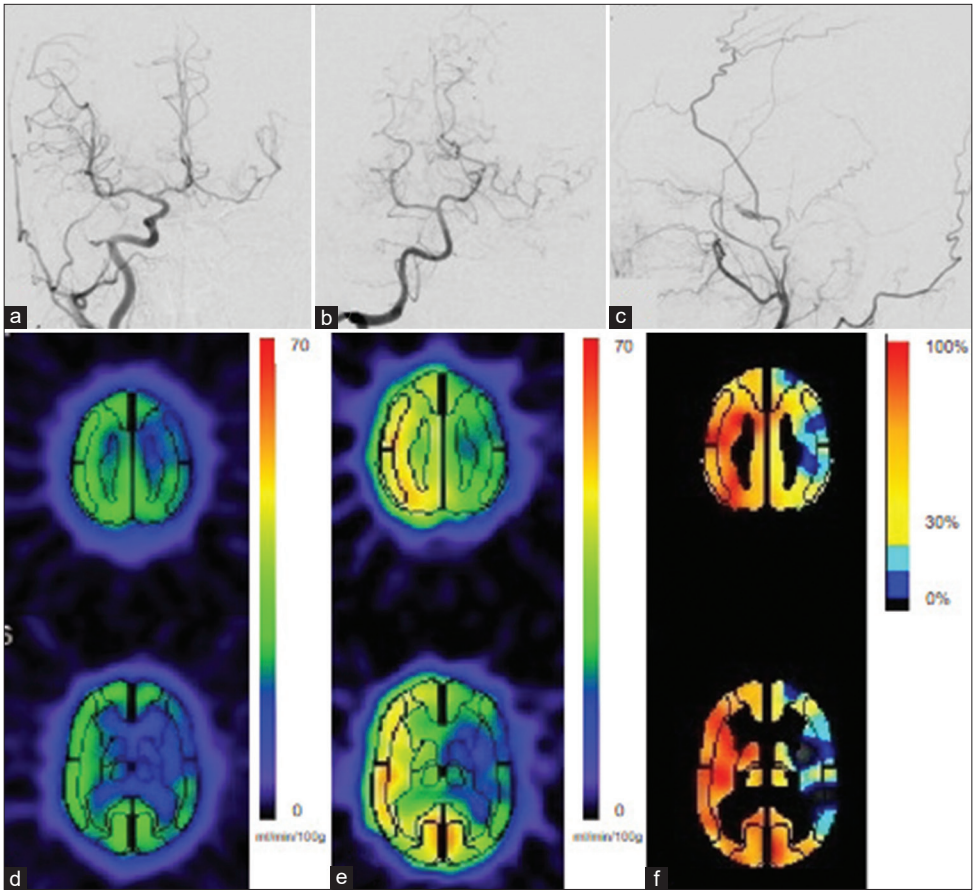

Figure 2: (a and b) Cerebral angiography demonstrating collateral flow to the left hemisphere through the anterior communicating artery and leptomeningeal anastomoses from the posterior cerebral artery. (c) Left external carotid artery angiography showing opacification of the internal carotid artery and ophthalmic artery through the middle meningeal artery. (d-f) ^{123}I-iodoamphetamine single–photon emission computed tomography demonstrating cerebral blood flow reduction at rest and cerebral vascular reserve in response to acetazolamide in the left hemisphere.

was performed to evaluate the donor artery during the opening and closing of the mouth. Opening of the mouth caused a 32% reduction in the peak systolic velocity than that of mouth closing, which showed good donor artery patency on CT angiography [Figures 4a-c]. The end-diastolic velocity (ED) ratio of STA, calculated from the ED during closing of the mouth divided by the ED of mouth opening, was 3.2. An encephalogram was conducted and revealed a focal epilepsy which was compatible with stroke on MRI and semiology of a seizure.

DISCUSSION

STA–MCA bypass is currently performed in Japan for atherosclerotic cerebrovascular disease, moyamoya disease, and complex aneurysm surgery, among others. Although some complications have been described following STA–MCA bypass, this procedure is considered relatively safe.[2] Some of the complications include donor artery occlusion, subgaleal hematoma, scalp necrosis, and postoperative intracranial hemorrhage.[12] Donor artery occlusion is a rare event except during the perioperative period. Furthermore, temporary occlusion of the donor artery is extremely rare, although complications of temporary occlusion of the donor artery have been reported in the literature.[1,10] The authors assessed and analyzed the blood flow of the donor artery during the closing and opening mouth in the chronic stage after STA–MCA bypass. It has been reported that blood flow of the donor artery is decreased during mouth opening for some patients. It has been proposed that one rationale for the temporary occlusion is attributed to the big bite ischemic phenomenon, where compression of the donor artery occurs at the site of the bone flap or there is kinking of the donor artery coursing through the muscle layer, which is deemed to be mechanisms for this phenomenon.[1,10] In our case, we believe that the unintended and forced opening of the mouth due to the secondary generalized seizure from post stroke epilepsy led to the temporary occlusion of the donor artery:

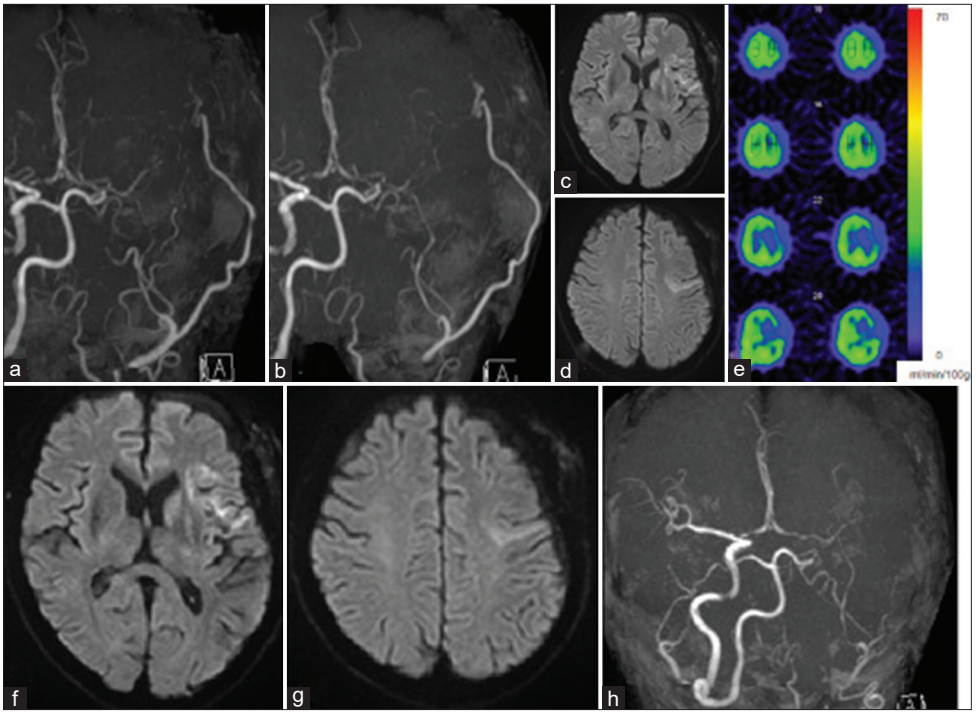

Figure 3: (a and b) Intracranial magnetic resonance angiography (MRA) showing patency of the donor artery following a day and 7 days after superficial temporal artery-middle cerebral artery bypass, respectively. (c and d) Magnetic resonance imaging (MRI) diffusion-weighted imaging (DWI) revealed no new infarction on postoperative day 7. (e) ^{123}I-iodoamphetamine single-photon emission computed tomography showed no apparent region of hyperperfusion on postoperative day 9. (f-h) Immediate MRI after secondary generalized seizure demonstrated no new infarction on DWI and MRA revealing diminishing signal intensity of the donor artery.

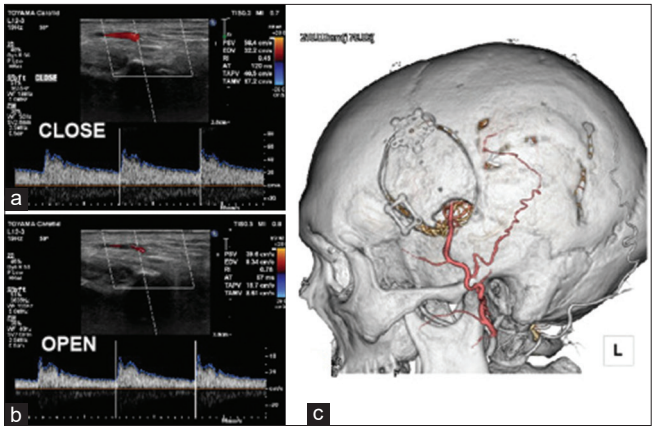

Figure 4: (a and b) Evaluation of the blood flow of the donor artery using ultrasonography which revealed a prominent decrease in blood flow during mouth opening. (c) Computed tomography angiography confirms good patency of the donor artery after the event.

(1) During the postoperative care, the patient was instructed not to compress his surgical site to avoid donor artery occlusion, and no complications were observed until 2 weeks after the surgery when seizure the occurred. (2) A groaning sound was noted during the secondary generalized seizure, which suggests an unintended and forced opening of the mouth due to the seizure.[3] (3) Hypoperfusion of the brain after a seizure could be considered as another explanation for the temporary occlusion of the donor artery[4] and might induce decreased blood flow; however, this could not explain the change of the blood flow during the opening and closing of the mouth. (4) Blood flow results of the donor artery by pulse Doppler and ultrasonography had significantly changed, that is, the blood flow decreased during the opening of the mouth. Besides the decreased systolic velocity of the STA, an ED ratio >3 indicates occlusion of the distal STA axis. Ultrasonic assessment of the ED ratio, obtained from the ED of the nonaffected side of the common carotid artery divided by the ED of the affected side, was used to predict the site of occlusion in case of an acute cardioembolic stroke.[13] ED is known to reflect peripheral resistance.[13] Therefore, we assumed that the distal STA which perfuses the MCA territory occluded during mouth opening. In our case, the lower part of the bone flap was resected to avoid donor artery compression, and a slight redundancy of the donor artery was observed between the zygoma and margin of the craniotomy [Figure 4c]; kinking of the donor artery was considered as the cause of the decrease in the blood flow during opening

of the mouth. Post stroke epilepsy is now recognized as a preventable complication of stroke, and anti-seizure medications should be prescribed in accordance with the published risk stratification score (the SeLECT score).[6] In our case, although the SeLECT score was 5, considering the delicate state after surgery, the risk for post stroke epilepsy is high. It has been reported that permanent neurologic deficits from big bite ischemic phenomenon are rare,[10] and this complication is preventable. This rare complication of STA–MCA bypass should be recognized.

CONCLUSION

Secondary generalized seizure following stroke causes unintended and forced mouth opening, which leads to the temporary occlusion of a donor artery after STA–MCA bypass. The serious complication of STA–MCA bypass should be recognized, and seizures should be prevented through the prophylactic administration of anti-seizure medication based on risk stratification assessment of post stroke epilepsy.

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

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The author(s) confirms that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

REFERENCES

1. Abe H, Katsuta T, Miki K, Higashi T, Inoue T. Temporary steno-occlusive change in the donor artery during mouth opening (big bite ischemic phenomenon) after superficial temporal artery to middle cerebral artery bypass in adult patients with moyamoya disease and atherosclerosis. Acta Neurochir Suppl 2016;123:123-8.
2. Chida K, Ogasawara K. Systematic review of complication for proper informed consent (11) STA-MCA bypass surgery. No Shinkei Geka 2013;41:1111-8.
3. DeToledo JC, Ramsay RE. Patterns of involvement of facial muscles during epileptic and nonepileptic events: Review of 654 events. Neurology 1996;47:621-5.
4. Farrell JS, Gaxiola-Valdez I, Wolff MD, David LS, Dika HI, Geeraert BL, *et al.* Postictal behavioural impairments are due to a severe prolonged hypoperfusion/hypoxia event that is COX-2 dependent. Elife 2016;5:e19352.
5. Ferreira-Atuesta C, Döhler N, Erdélyi-Canavese B, Felbecker A, Siebel P, Scherrer N, *et al.* Seizures after ischemic stroke: A matched multicenter study. Ann Neurol 2021;90:808-20.
6. Galovic M, Döhler N, Erdélyi-Canavese B, Felbecker A, Siebel P, Conrad J, *et al.* Prediction of late seizures after ischaemic stroke with a novel prognostic model (the SeLECT score): A multivariable prediction model development and validation study. Lancet Neurol 2018;17:143-52.
7. Hishikawa T, Date I. Evidence of efficacy of superficial temporal artery-middle cerebral artery bypass in Japan. No Shinkei Geka 2022;50:745-51.
8. Horie N, Okazaki T. Indications for extracranial-intracranial bypass surgery. No Shinkei Geka 2022;50:727-34.
9. Kataoka H, Miyamoto S, Ogasawara K, Iihara K, Takahashi JC, Nakagawara J, *et al.* Results of a prospective cohort study on symptomatic cerebrovascular occlusive disease showing mild hemodynamic compromise [Japanese extracranial-intracranial bypass trial (JET)-2 study]. Neurol Med Chir (Tokyo) 2015;55:460-8.
10. Katsuta T, Abe H, Miki K, Inoue T. Reversible occlusion of donor vessel caused by mouth opening after superficial temporal artery-middle cerebral artery anastomosis in adult moyamoya patients. J Neurosurg 2015;123:670-5.
11. Miyamoto S, Ogasawara K, Kuroda S, Itabashi R, Toyoda K, Itoh Y, *et al.* Japan stroke society guideline 2021 for the treatment of stroke. Int J Stroke 2022;17:1039-49.
12. Newell DW. Superficial temporal artery to middle cerebral artery bypass. Skull Base 2005;15:133-41.
13. Yasaka M, Omae T, Tsuchiya T, Yamaguchi T. Ultrasonic evaluation of the site of carotid axis occlusion in patients with acute cardioembolic stroke. Stroke 1992;23:420-2.

How to cite this article: Tsukada T, Masuoka T, Kubo M. A case of temporary occlusion of donor artery after secondary generalized seizure in a patient with superficial temporal artery-middle cerebral artery bypass. Surg Neurol Int 2023;14:330.