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Vascular

Incidence and risk factors for symptomatic cerebral hyperperfusion after superficial temporal artery-middle cerebral artery anastomosis in patients with moyamoya disease

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Abstract

Background: Superficial temporal artery-middle cerebral artery anastomosis for moyamoya disease prevents cerebral ischemic attack by improving CBF, whereas recent evidence suggests that the temporary neurologic deterioration because of postoperative cerebral hyperperfusion could occur despite its low-flow revascularization. The present study investigates the incidence and the risk factors for symptomatic hyperperfusion after STA-MCA anastomosis in patients with moyamoya disease.

Methods: We prospectively performed *N*-isopropyl-p-[^{123}I]iodoamphetamine single-photon emission computed tomography 1 and 7 days after STA-MCA anastomosis on 80 hemispheres of 58 consecutive patients with moyamoya disease (approximately 2-62 years old, 34.4 years old in average). Mean follow-up period was 22.7 months. Symptomatic cerebral hyperperfusion was defined as the presence of the significant increase in CBF at the site of the anastomosis that is responsible for the apparent neurologic sign.

Results: Twenty-one patients (22 sides, 27.5%) temporarily had symptomatic cerebral hyperperfusion, who were subjected to intensive blood pressure control. Postoperative magnetic resonance imaging/angiography showed the thick high signal of bypass without ischemic changes in all 21 patients. Adult-onset ($P = .013$) or hemorrhagic-onset patients ($P = .027$) had significantly higher risk for symptomatic hyperperfusion. There was no difference in intraoperative temporary occlusion time between each group. No patients had permanent neurologic deficit because of hyperperfusion.

Conclusion: The STA-MCA anastomosis is a safe and effective treatment of moyamoya disease, although adult-onset and/or hemorrhagic-onset patients had higher risk for symptomatic hyperperfusion. We recommend routine CBF measurement especially for these patients because the management of hyperperfusion is contradictory to that of ischemia.

Keywords: Moyamoya disease; Cerebral hyperperfusion; Risk factor; Extracranial-intracranial bypass

Abbreviations: BBB, blood-brain barrier; CBF, cerebral blood flow; CT, computed tomography; DWI, diffusion-weighted images; EDMS, encephalo-duro-myo-synangiosis; ICH, intracerebral hemorrhage; ^{123}I-IMP-SPECT, *N*-isopropyl-p-[^{123}I]iodoamphetamine single-photon emission computed tomography; MCA, middle cerebral artery; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; ROS, reactive oxygen species; SAH, subarachnoid hemorrhage; STA-MCA, superficial temporal artery-middle cerebral artery; TIA, transient ischemic attack.

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1. Introduction

Moyamoya disease is a chronic, occlusive cerebrovascular disease with unknown etiology characterized by bilateral stenoocclusive changes at the terminal portion of the internal carotid artery and an abnormal vascular network at the base of the brain [20]. Surgical revascularization for moyamoya disease prevents cerebral ischemic attacks by improving CBF, and STA-MCA anastomosis with or without indirect pial synangiosis is generally used as the standard surgical treatment of moyamoya disease [4,8,9,15,17]. Despite its favorable long-term outcome, increasing evidence suggest that direct revascularization surgery for moyamoya disease could result in temporary neurologic deterioration owing to focal cerebral hyperperfusion at the site of the anastomosis during the acute stage [3-6,12,14]. Because the clinical manifestation of cerebral hyperperfusion in patients with moyamoya disease includes transient focal neurologic deficit mimicking cerebral ischemic attack [4-6], it is clinically important to make accurate diagnosis of symptomatic hyperperfusion and to conduct its adequate management such as intensive blood pressure control [4,5]. Furthermore, it would be of great value to clarify the predictive factors for postoperative symptomatic hyperperfusion in moyamoya disease, although the exact incidence and the risk factors of hyperperfusion are totally undetermined in moyamoya disease.

To address this issue, we retrospectively investigated the incidence and the risk factors of symptomatic cerebral hyperperfusion in 58 consecutive patients with moyamoya disease, who were all treated by STA-MCA anastomosis on 80 hemispheres and were examined by ^{123}I-IMP-SPECT 1 and 7 days after 80 consecutive surgeries.

1.1. Patients and methods

The correlation between postoperative changes in CBF and clinical course was investigated in 58 consecutive patients (approximately 2-62 years old; mean 34.4 years) with moyamoya disease operated on 80 hemispheres by the same surgeon (MF) in Tohoku University Hospital (Sendai, Japan) from March 2004 to May 2007. All patients were strictly followed-up in our institute with the mean follow-up period of 22.7 months. All patients satisfied the diagnostic criteria of the Research Committee on Spontaneous Occlusion of the Circle of Willis, of the Ministry of Health, Labor, and Welfare, Tokyo, Japan, except for 3 patients with "probable moyamoya disease" with unilateral involvement. All patients underwent STA-MCA anastomosis with or without EDMS and dural pedicle insertion. The CBF was routinely measured by ^{123}I-IMP-SPECT 1 and 7 days after surgery in all patients. The CBF was quantified by the autoradiographic method, the CBF in each subregion of the cerebral cortex was automatically calculated by Three-Dimensional Stereotactic Region of Interest Template (3D-SRT) software (version 2) provided by Daiichi Radio-Isotope (Tokyo, Japan), and the diagnosis of cerebral hemodynamics was made by 2 specialized radiologists. The 1.5 or 3 Tesla MRI and MRA were routinely performed 2 and 8 days after surgery. The MRI includes DWI, fluid attenuated inversion recovery, T1/T2-weighted images, and T2*-weighted images. The diagnostic criteria for symptomatic cerebral hyperperfusion include all of the following issues; (1) the presence of the significant increase in CBF at the site of the anastomosis that is responsible for apparent neurologic signs including focal neurologic deficit and/or severe headache because of hemorrhagic changes; (2) apparent visualization of STA-MCA bypass by MRA and the absence of any ischemic changes by DWI; and (3) the absence of other pathologies such as the compression of the brain surface by the temporal muscle inserted for indirect pial synangiosis, ischemic attack, and seizure. We evaluated the correlation between the occurrence of symptomatic cerebral hyperperfusion and patients' information including age, sex, side of the operated hemisphere, onset-type, and the period of temporary occlusion time of the recipient arteries during surgery. Statistical analysis was performed by χ^2 test or by Student t test.

2. Results

Among 58 consecutive patients with 80 surgeries, no patients had perioperative cerebral infarction, except for 3 patients (3.7%) presenting with pseudolaminar necrosis in a part of cerebral cortex supplied by STA-MCA bypass at the subacute stage, which did not affect their long-term neurologic status. All patients with the onset of TIA obtained disappearance or improvement of ischemic attack during the follow-up period. One hemorrhagic-onset patient had ICH on the contralateral side 3 months after surgery, which did not affect his neurologic status. The patency of STA-MCA bypass was confirmed in all 58 patients with 80 surgeries by MRA after surgery. Among the 58 consecutive patients with 80 surgeries, 21 patients (22 hemispheres, 27.5% of 80 operated hemispheres) had temporary neurologic deterioration because of postoperative cerebral hyperperfusion from 2 to 9 days after surgery, which sustained for several days (Table 1). Postoperative MRI/MRA showed no ischemic changes, and the thick high signal of STA on the operated hemisphere was evident in all 22 hemispheres except for one

Table 1
Incidence of symptomatic cerebral hyperperfusion in moyamoya disease

	No. of hemisphere sides (n = 80)	Initial symptom (d after surgery)	Permanent neurologic deficit
Symptomatic hyperperfusion	22 (27.5%)		
Focal neurologic deficit	18 (22.5%)	Approximately 2-7 d	None
SAH	3 (3.8%)	Approximately 1-2 d	None
ICH	1 (1.2%)	4 d	None