Case Report

Subarachnoid Hemorrhage with Progressive Cerebral Steno-Occlusive Disease: Report of 2 Cases

Soichiro Takamiya, MD, Tetsuyuki Yoshimoto, MD, PhD, and Katsuhiko Maruichi, MD, PhD

Nontraumatic nonaneurysmal subarachnoid hemorrhage (SAH) is a rare condition. Among them, SAH with cerebral steno-occlusive disease is quite rare. Moreover there has been no report of SAH patient who had been diagnosed with steno-occlusive disease since before. We here report 2 cases of nontraumatic nonaneurysmal convexity SAH who originally had progressive cerebral steno-occlusive disease. Case 1, a woman in her 40s who had diagnosed left internal carotid artery (ICA) stenosis 6 years before complained of headache. She was diagnosed SAH and progressive ICA stenosis, then performed revascularization. Case 2, a woman in her 40s who had diagnosed right ICA stenosis 7 months before complained of headache. She was diagnosed with SAH and ICA occlusion. These 2 cases suggested that progressive cerebral steno-occlusive disease lead to SAH due to collapse of their fragile pial anastomoses.

Key Words: Convexity subarachnoid hemorrhage—cerebral steno-occlusive disease—pial anastomosis—revascularization.

Introduction

Nontraumatic subarachnoid hemorrhage (SAH) generally develops following the rupture of a cerebral aneurysm.[1] However, there have been reports of nonaneurysmal cases associated with either reversible cerebral vasoconstriction syndrome or cerebral amyloid angiopathy.[2,3] In recent year, there have been a small number of reports of SAH with cerebral steno-occlusive disease.[4–10] In all of these cases, SAH and steno-occlusive disease were diagnosed simultaneously. Additionally, to date, no reports are available on patients with SAH who had previously been diagnosed with steno-occlusive disease. In this study, we report 2 cases of patients with nontraumatic nonaneurysmal convexity SAH (cSAH) who were originally diagnosed with progressive cerebral steno-occlusive disease.

From the Neurosurgery, Kashiwaba Neurosurgical Hospital, Sapporo, Hokkaido, Japan.

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Address correspondence to Soichiro Takamiya, MD, Neurosurgery, Kashiwaba Neurosurgical Hospital, 7-20, 15-chome, Tsukisamu Higashi 1-jo, Toyohira-ku, Sapporo 062-8513, Hokkaido, Japan. E-mail: soichiro.tkmy@gmail.com.

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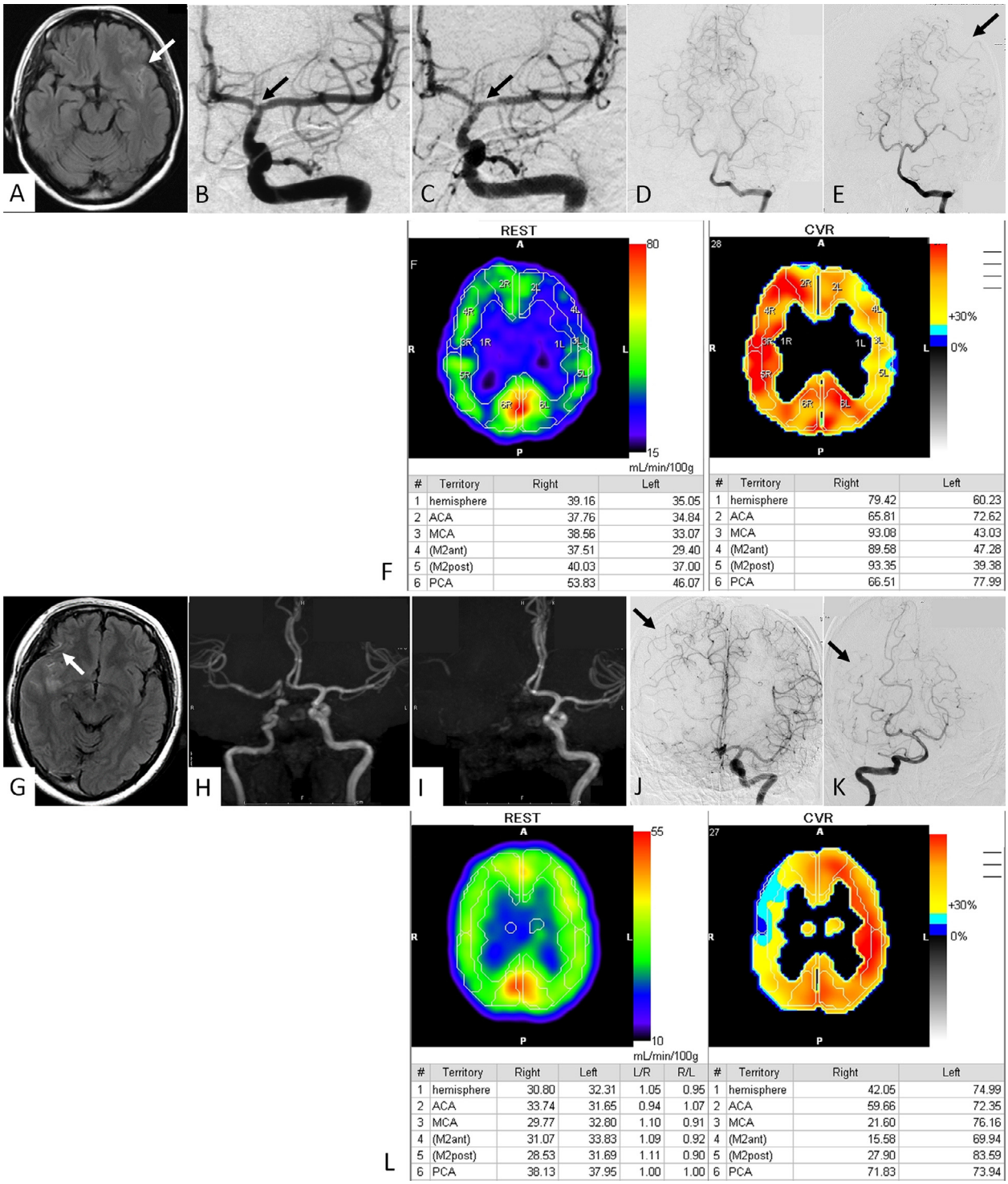
Case Presentation

Case 1

A 42-year-old woman visited our hospital with a headache. The patient had been diagnosed with cerebral infarction and left internal carotid artery (ICA) stenosis (Fig 1, B) 6 years prior to the complaint and had been provided antiplatelet therapy. Magnetic resonance image revealed left frontotemporal cSAH (Fig 1, A). Furthermore, digital subtraction angiography (DSA) demonstrated the absence of cerebral aneurysm and vascular malformation. However, DSA did show the progression of left ICA stenosis and middle cerebral artery (MCA) stenosis (Fig 1, C). Additionally, it showed the development of pial anastomosis from the left posterior cerebral artery (Fig 1, E) compared to the results obtained 6 years ago (Fig 1, D) and hypoplasia of the anterior communicating artery and left posterior communicating artery. An ^{123}I-IMP single photon emission computed tomography was performed. Through this, we



#	Territory	Right	Left
1	hemisphere	39.16	35.05
2	ACA	37.76	34.84
3	MCA	38.56	33.07
4	(M2ant)	37.51	29.40
5	(M2post)	40.03	37.00
6	PCA	53.83	46.07

#	Territory	Right	Left
1	hemisphere	79.42	60.23
2	ACA	65.81	72.62
3	MCA	93.08	43.03
4	(M2ant)	89.58	47.28
5	(M2post)	93.35	39.38
6	PCA	66.51	77.99

#	Territory	Right	Left	L/R	R/L
1	hemisphere	30.80	32.31	1.05	0.95
2	ACA	33.74	31.65	0.94	1.07
3	MCA	29.77	32.80	1.10	0.91
4	(M2ant)	31.07	33.83	1.09	0.92
5	(M2post)	28.53	31.69	1.11	0.90
6	PCA	38.13	37.95	1.00	1.00

#	Territory	Right	Left
1	hemisphere	42.05	74.99
2	ACA	59.66	72.35
3	MCA	21.60	76.16
4	(M2ant)	15.58	69.94
5	(M2post)	27.90	83.59
6	PCA	71.83	73.94

Fig 1. *Case 1. Magnetic resonance image at the time of the headache showed left frontotemporal convexity subarachnoid hemorrhage (arrow) (A). Digital subtraction angiography, performed 6 years ago, showed left internal carotid artery stenosis (arrow) (B), which worsened (arrow) (C). Additionally, compared to 6 years ago (D), we observed the development of pial anastomosis (arrow) from the left posterior cerebral artery (E). An* ^{123}*I-IMP single photon emission computed tomography showed a decline of cerebral blood flow and cerebrovascular reactivity of the left hemisphere (F). Case 2. Magnetic resonance image at the time of the headache showed right frontotemporal convexity subarachnoid hemorrhage (arrow) (G). Magnetic resonance angiography obtained 7 months ago showed right internal carotid artery stenosis (H), which then occluded (I). Additionally, Digital subtraction angiography revealed the development of pial anastomosis (arrow) from the right anterior cerebral artery and posterior cerebral artery (J, K). An* ^{123}*I-IMP single photon emission computed tomography showed a decline of cerebral blood flow and cerebrovascular reactivity of the right hemisphere (L).*

observed a decrease in cerebral blood flow and cerebrovascular reactivity of the left hemisphere (Fig 1, F). Because of the fact that the left frontal watershed area is where the cerebral infarction developed during conservative therapy, we proceeded with performing a left superficial temporal artery-MCA bypass. The patient did not experience any perioperative complication, and was discharged with no neurological deficit.

Case 2

A 46-year-old woman visited our hospital with a chronic left-sides paresthesia. At 15 years of age, the patient had received coating of right ICA aneurysm. Magnetic resonance angiography revealed right ICA stenosis (Fig 1, H). As a consequence, an antiplatelet therapy was administered. After 7 months, the patient returned to our hospital due to a headache. Magnetic resonance image showed right frontotemporal cSAH (Fig 1, G), and Magnetic resonance angiography showed right ICA occlusion (Fig 1, I). Additionally, by DSA, we identified the development of pial anastomosis from the right anterior cerebral artery and posterior cerebral artery (Fig 1, J, K) and hypoplasia of right A1 and right posterior communicating artery. An ^{123}I-IMP single photon emission computed tomography demonstrated a decrease of cerebral blood flow and cerebrovascular reactivity of the right hemisphere (Fig 1, L). We proposed the patient to undergo revascularization. However, she refused.

Discussion

We experienced 2 cases of nontraumatic nonaneurysmal cSAH, which is a rare entity. Specifically, it constitutes about 5% of all nontraumatic SAH[1]. The most common causes are reversible cerebral vasoconstriction syndrome (29.5%-37.9%); cerebral amyloid angiopathy (26.1%-34.5%);[2, 3] and cerebral steno-occlusive disease, of which only 12 cases been reported to date (excluding the cases presented here).[4–10] Our 2 cases were relatively younger age. We deem it to be a coincidence because the average age of the 12 cases was 58.6 years (from 28 to 77 years), and it is difficult to say that cSAH with steno-occlusive disease occurs frequently in younger age. As for the etiology of steno-occlusive lesion, we assume it of case 1 to be atherosclerosis, and it of case 2 to be inflammatory change due to coating of cerebral aneurysm. The latter was a unique etiology since all of them in reported 12 cases were atherosclerosis.

With the exception of our cases, in all the cases reported in literature, SAH, and steno-occlusive disease were diagnosed simultaneously. To the best of our knowledge, there have been no reports of patients with SAH previously diagnosed with steno-occlusive disease. Our cases suggest that the progression of cerebral artery stenosis and hemodynamic compromise may lead to the development of fragile pial anastomoses, and their collapse may cause cSAH. Actually, the sides of cSAH and the steno-occlusive lesion accorded in all 14 cases (including our cases). Additionally, there is a possibility that such a condition occurs easily when the collateral pathways across the circle of Willis are hypoplastic.

In 3 of the 14 aforementioned cases, patients underwent STA-MCA bypass with good clinical courses. Therefore, revascularization can be an option for the treatment as moyamoya disease, not only for ischemic but also for hemorrhagic cases.

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