

Intracerebral Hematoma Due to Ruptured Nontraumatic Middle Meningeal Artery Aneurysm

—Case Report—

Hitoshi KOBATA, Hideo TANAKA, Yuichi TADA, Kentaro NISHIHARA,
Akira FUJIWARA, and Toshihiko KUROIWA*

Osaka Mishima Critical Care Medical Center, Takatsuki, Osaka;

**Department of Neurosurgery, Osaka Medical College, Takatsuki, Osaka*

Abstract

A 77-year-old woman suddenly lost consciousness and presented with right hemiparesis. Computed tomography showed a large subcortical hematoma in her left frontal lobe associated with subarachnoid hemorrhage. The first impression was hemorrhage due to a ruptured aneurysm of the middle cerebral or the internal carotid artery on the left. Left internal carotid angiography showed no aneurysm or vascular anomaly, but back flow of contrast medium into the external carotid artery disclosed two saccular aneurysms arising from the anterior branch of the left middle meningeal artery (MMA). Emergent surgical intervention confirmed that the hematoma was due to ruptured MMA aneurysm. Nontraumatic MMA aneurysm should be recognized as a possible causative lesion of intracranial hemorrhage despite the extremely low incidence. External carotid angiography should be performed in patients with intracranial hemorrhage in whom no vascular cause can be detected in the internal carotid system.

Key words: cerebral angiography, intraparenchymal hematoma, middle meningeal artery aneurysm, subarachnoid hemorrhage

Introduction

Traumatic pseudoaneurysm of the middle meningeal artery (MMA) is a known cause of intracranial hemorrhage. Delayed onset of intracranial hemorrhage following previous head injury indicates rupture of a MMA pseudoaneurysm. Nontraumatic MMA aneurysm is uncommon, and rupture resulting in intracranial hemorrhage is extremely rare,^{7,8,15)} so might be overlooked especially in patients without a history of previous head injury. The aneurysm may not be recognized until the second operation after rebleeding.⁸⁾ We report an unusual case with a misleading hemorrhagic pattern of intracerebral hematoma (ICH) and subarachnoid hemorrhage (SAH), which mimicked a ruptured aneurysm at the middle cerebral artery (MCA) or the internal carotid artery (ICA). Urgent left carotid angiography failed to depict an aneurysm of the left internal carotid system; however, overflow of contrast medium into the external carotid artery demonstrat-

ed ruptured MMA aneurysm fortunately.

Case Report

A 77-year-old woman suddenly lost consciousness and fell down at another hospital on April 24, 1999, and was transferred to our hospital 30 minutes later. She had undergone mitral valve replacement (Carpentier-Edwards bioprosthesis; Baxter, Irvine, Calif., U.S.A.) for mitral valve regurgitation 18 years prior to admission, but cardiac insufficiency persisted due to combined mitral stenosis and tricuspid regurgitation. She had received no anticoagulant therapy. No head injury was documented within several weeks before admission. In addition, she had been taken thyroid hormone for hypothyroidism for 18 years.

On admission, her level of consciousness was 7 (E: 4, V: 1, M: 2) on the Glasgow Coma Scale and 3A on the Japan Coma Scale. She presented with right hemiparesis. Her blood pressure was 140/60 mmHg. External jugular and superficial temporal veins were engorged on both sides, suggesting severe cardiac failure. Pansystolic cardiac murmur was audible on

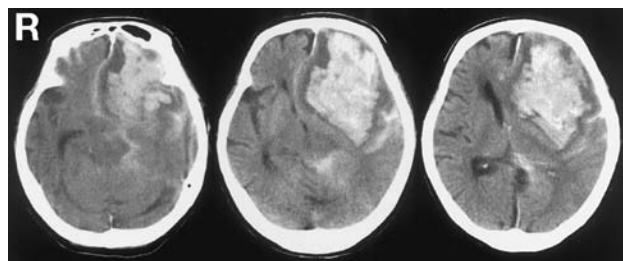


Fig. 1 Axial computed tomography scans showing an extensive intraparenchymal hematoma in the left frontal lobe associated with subarachnoid hemorrhage and a thin subdural hematoma. The intraparenchymal hematoma extends to reach the cortical surface.

chest auscultation. She was intubated and evaluated on an emergency basis.

Computed tomography showed a large subcortical hematoma that measured $8 \times 5 \times 6$ cm in her left frontal lobe. The hematoma reached the cortical surface and was continuous with a thin subdural hematoma (SDH). Associated SAH was predominant in the basal cisterns on the left side (Fig. 1). The first impression was hemorrhage due to a ruptured aneurysm of the left MCA or ICA. Skull roentgenography detected no fracture. Chest roentgenography exhibited marked cardiomegaly with cardiothoracic ratio of 76%. Echocardiography showed severe mitral stenosis and tricuspid regurgitation. No valvular vegetations were detected.

Urgent digital subtraction angiography was performed. The left selective internal carotid angiography showed displacement of the cerebral vessels, indicating hematoma in the left frontal lobe. No aneurysm or vascular anomaly was detected in the internal carotid system. However, overflow of contrast medium into the external carotid artery disclosed two saccular aneurysms, 0.8 cm and 0.3 cm in diameter, that had arisen from the anterior branch of the left MMA (Fig. 2). During the angiographic procedure, she developed anisocoria and lost light reaction. The angiography was discontinued and she underwent emergency surgical intervention.

A right frontotemporal craniotomy was performed. The dural vessels were unusually engorged and easy to bleed. The thin SDH was evacuated after dural incision. A small bulge associated with a clot of blood, consistent in location with the larger aneurysm of the MMA, was identified in the inner dural surface opposite to the destroyed cortical surface (Fig. 3). The MMA exposed in the craniotomy

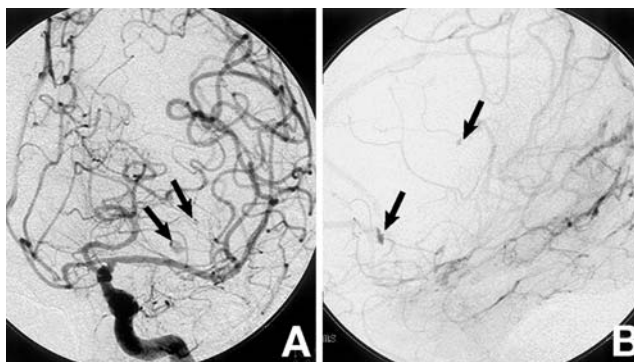


Fig. 2 Anteroposterior (A) and lateral (B) projections of digital subtraction angiograms demonstrating double saccular aneurysms arising from the middle meningeal artery (arrows). Marked displacement of the cerebral vessels is seen.

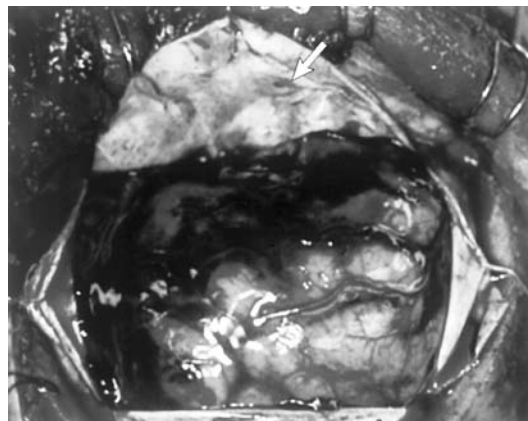


Fig. 3 Intraoperative photograph taken after subdural hematoma evacuation demonstrating the cortical surface destroyed by the hematoma. A small bulge associated with a clot of blood is seen identified in the inner dural surface opposite the destroyed cortical surface (arrow).

field was coagulated together with the two aneurysms arising from the MMA. The ICH was evacuated via the destroyed cortical surface. The hematoma cavity and contents were thoroughly inspected to detect any possible vascular anomalies, but no particular abnormal tissue was identified. No leptomeningeal anastomotic vessels were detected on the brain surface. Exploration of the ICA and the trifurcation of the MCA disclosed no aneurysm. Intraoperative findings indicated that rupture of an MMA aneurysm had caused the SAH, SDH, and ICH.

Table 1 Reported cases of nontraumatic middle meningeal artery (MMA) aneurysms

Author (Year)	Age	Sex	Associated disease	Presentation	Type of hemorrhage	Remarks
Berk (1961) ²⁾	73	F	Paget's disease, HT	headache	no	
Holland and Thomson (1965) ⁵⁾	49	F	none	headache	EDH	
New (1965) ¹⁰⁾	79	F	Paget's disease, HT	stroke (ICA occlusion)	no	
Zingesser et al. (1965) ¹⁹⁾	ND	ND	ICA occlusion	ND	ND	
New (1967) ¹¹⁾	57	F	Paget's disease, HT	headache	no	
Sanchis et al. (1975) ¹⁴⁾	59	F	dural angioma	headache	EDH	
Bollati et al. (1980) ³⁾	50	F	none	epilepsy	old SDH	
Takahashi (1980) ¹⁶⁾	10	F	moyamoya	incidental	no	double aneurysms
Jin et al. (1981) ⁶⁾	9	M	asthma, MMA angioma	headache, LOC	no	
Korosue et al. (1988) ⁸⁾	66	F	none	headache	recurrent SDH	
Ohta et al. (1991) ¹²⁾	47	M	cavernous hemangioma of skull	incidental	no	multiple aneurysms
O'Neill et al. (1995) ¹³⁾	82	F	meningioma	incidental	no	
Ushikoshi et al. (1996) ¹⁷⁾	69	M	HT, PCA occlusion	LOC	ICH, IVH	
Zubkov et al. (1998) ²⁰⁾	22	M	none	SAH	SAH	double aneurysms
Sandin et al. (1999) ¹⁵⁾	46	M	mild HT	headache	ICH	
Kähärä (1999) ⁷⁾	58	F	dural AVM	LOC	ICH*	
Present case	77	F	chronic heart failure (MS, TR)	LOC	ICH, SAH	

*Hemorrhage due to a ruptured cortical ectatic vein of the dural arteriovenous malformation (AVM). EDH: epidural hematoma, HT: hypertension, ICA: internal carotid artery, ICH: intracerebral hematoma, IVH: intraventricular hematoma, LOC: loss of consciousness, MS: mitral stenosis, ND: not described, PCA: posterior cerebral artery, SAH: subarachnoid hemorrhage, SDH: subdural hematoma, TR: tricuspid regurgitation.

Postoperative angiography confirmed the disappearance of both MMA aneurysms. She was referred to the rehabilitation institute for her motor aphasia and right hemiparesis.

Discussion

Only 16 cases of nontraumatic MMA aneurysms have been reported (Table 1).^{2,3,5-8,10-17,19,20)} Seven presented with intracranial hemorrhage: two cases of epidural hematoma,^{5,14)} two of SDH,^{3,8)} two of ICH,^{7,15,17)} and one of SAH.²⁰⁾ Double aneurysms were found in two cases^{16,20)} and multiple aneurysms in one.¹²⁾ The aneurysm was not detected at initial surgery in one of the cases with SDH, but was demonstrated by angiography after recurrent hemorrhage.⁸⁾ An aneurysm of the anterior communicating artery was suspected in the case with SAH. The MMA aneurysm was not visualized at the initial angiography and was only detected at repeat angiography.²⁰⁾

In the present case, the pattern of combined ICH and SAH was similar to that typically seen with ruptured MCA or ICA aneurysm. The patient's condition did not allow full angiographic study, so only left selective internal carotid angiography was performed on an emergency basis. Fortunately, the contrast medium overflowed into the external carotid artery to depict the MMA aneurysms. At surgery, the location of the MMA aneurysm was opposite the

impaired cortical surface and extended to the intraparenchymal hematoma. Angiographically occult vascular malformations, which are frequently the cause of lobar hemorrhage,^{9,18)} were excluded in the present case.

Dural vascular lesion can cause intracranial hemorrhage. Rupture of arterialized leptomeningeal veins results in intracranial hemorrhage in cases of dural arteriovenous malformation (AVM).¹⁾ However, nontraumatic MMA aneurysm is rare and could easily be overlooked as a cause of intracranial hemorrhage. Ruptured MMA aneurysm should be considered as a possible hemorrhagic origin if ICH reaches the cortical surface as in the present case. When standard four-vessel angiography yields negative results, external carotid angiography should be undertaken.

Necropsy study has found medial defects are common in the meningeal arteries. The elastic lamina and adventitia often showed increased thickness over the medial defect. However, the defects were sometimes thin-walled and separated from the subarachnoid space only by a thin, loose layer of collagenous tissue.⁴⁾ Pathological conditions associated with increased hemodynamic stress of the MMA could promote aneurysm formation at these thin-walled defects. Histological study has confirmed that most nontraumatic MMA aneurysms are true aneurysms like normal cerebral aneurysms.^{6,8,10-12,15,20)} Lack of media and internal elastic lamina

were present.^{15,20} Presumably, rupture at the projected dome into the subarachnoid space causes SAH, and in the presence of pial adhesion, ICH will develop, similar to hemorrhage caused by cerebral aneurysms.

Nontraumatic MMA aneurysms were associated with Paget's disease,^{2,10,11} dural angioma or AVM,^{6,7,14} cavernous hemangioma of the skull,¹² meningioma,¹³ moyamoya disease,¹⁶ and occlusion of ICA¹⁹ and the posterior cerebral artery.¹⁷ Aneurysm formation in Paget's disease is promoted by loss of normal support of the MMA due to cranial softening, increased blood flow in the MMA, and chronic systemic hypertension.^{10,11} The MMA is also exposed to increased blood flow as a feeding artery of vascular malformations or vascular-rich tumors. Development of leptomeningeal anastomosis is another cause of MMA overflow associated with cerebral ischemia. In the present case, whether chronic venous congestion was related to the formation of MMA aneurysms remains unclear. The reason for aneurysm multiplicity is also unknown. At present, little is known about the incidence and natural history of MMA aneurysms.

Rupture of a nontraumatic MMA aneurysm caused ICH and SAH. This uncommon aneurysm can cause intracranial hemorrhage of any type. The external carotid artery should be evaluated if internal carotid angiography does not demonstrate the source of hemorrhage. Radical surgery is warranted if a MMA aneurysm is found to be a possible cause of intracranial hemorrhage. Recognition of this uncommon entity is essential to avoid diagnostic and therapeutic problems.

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Address reprint requests to: H. Kobata, M.D., Osaka Mishima Critical Care Medical Center, 11-1 Minami Akutagawa-cho, Takatsuki, Osaka 569-1124, Japan.
e-mail: neu035@poh.osaka-med.ac.jp