Ruptured Internal Carotid Artery Bifurcation Aneurysm Presenting With Only Intracerebral Hemorrhage Without Subarachnoid Hemorrhage

-Case Report-

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Abstract

A 55-year-old man presented with intracerebral hemorrhage (ICH) without subarachnoid hemorrhage (SAH) manifesting as acute onset of consciousness disturbance and right hemiparesis. Computed tomography showed ICH mainly localized in the left putamen, but no evidence of SAH. Magnetic resonance angiography demonstrated a cerebral aneurysm originating from the bifurcation of the left internal carotid artery, which was considered to be responsible for the ICH. The patient underwent emergent intravascular surgery for coil embolization of the aneurysm, and his neurological symptoms gradually recovered with rehabilitation after surgery. Although ICH without SAH is a rare presentation of cerebral aneurysm, ruptured cerebral aneurysm should be considered as a potential cause of ICH. The localization and extent of ICH may be suggestive of latent cerebral aneurysm in such cases.

Key words: cerebral aneurysm, intracerebral hemorrhage, subarachnoid hemorrhage, internal carotid artery

Introduction

Rupture of cerebral aneurysms most often results in subarachnoid hemorrhage (SAH), and SAH may occasionally accompany intracerebral hemorrhage (ICH) with an incidence of 4.5–31%. However, ICH without SAH is an extremely rare presentation of ruptured cerebral aneurysms, which potentially leads to a delay in the correct diagnosis. Here, we describe a case of ruptured internal carotid artery (ICA) bifurcation aneurysm presenting with only ICH without SAH.

Case Report

A 55-year-old right-handed man was admitted to our hospital after sudden onset of right hemiparesis and dysarthria. He did not complain of headache. His past history included hypertension. On admission, blood pressure was 145/86 mmHg and nuchal rigidity was absent. Neurological examination showed disturbance of consciousness (Japan Coma Scale I-2, Glasgow Coma Scale 14), slight central-type facial weakness on the right side, and moderate right hemiparesis (manual muscle test 4/5).

Computed tomography (CT) of the head on admission showed ICH with a volume of approximately 10 ml, which

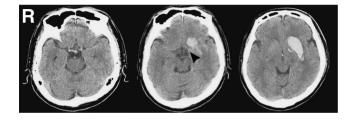


Fig. 1 Computed tomography scans showing intracerebral hemorrhage mainly localized in the left putamen and extending to the inferior frontal gyrus of the left frontal lobe (arrowhead). Subarachnoid hemorrhage was not seen.

was mainly localized in the left putamen and extended to the base of left frontal lobe. There was no evidence of SAH (Fig. 1). Magnetic resonance (MR) imaging and MR angiography performed the following day showed no apparent evidence of SAH on fluid-attenuated inversion recovery images, but revealed a left ICA bifurcation aneurysm measuring 6 mm. The aneurysm projected superiorly adjacent to the cavity of the ICH (Fig. 2). Accordingly, we suspected that the rupture of this aneurysm was the possible cause of ICH, although no direct evidence to support our speculation was found.

The patient underwent emergent coil embolization of

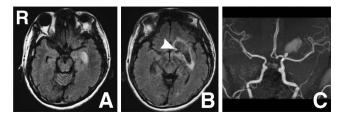


Fig. 2 A, B: Fluid-attenuated inversion recovery magnetic resonance (MR) images showing no evidence of subarachnoid hemorrhage, with a vascular flow void adjacent to the medial part of intracerebral hemorrhage (arrowhead). C: MR angiogram demonstrating an internal carotid artery bifurcation aneurysm adjacent to the cavity of the intracerebral hemorrhage.

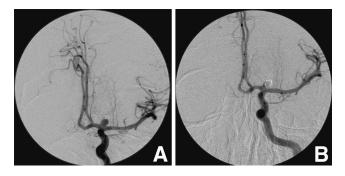


Fig. 3 A: Left carotid angiogram showing an aneurysm of 6 \times 4.9 mm originating from the bifurcation of the left internal carotid artery. B: Postoperative left carotid angiogram showing complete obliteration of the aneurysm.

the aneurysm. Angiography showed the aneurysm was 6 \times 4.9 mm and originated from the bifurcation area of the left ICA (Fig. 3A). The aneurysm was successfully obliterated using Guglielmi detachable coils (Boston Scientific Corp., Fremont, California, USA) with a volume embolization ratio of 27%. The operation was uneventful. Postoperative carotid angiography demonstrated complete obliteration of the aneurysm (Fig. 3B). The patient had no additional neurological deficits postoperatively, and was discharged home, walking unaided, one month after endovascular surgery.

Discussion

This rare case of ruptured cerebral aneurysm presenting with only ICH was caused by rupture of an ICA bifurcation aneurysm. Ruptured cerebral aneurysms are known to cause ICH, but are usually accompanied by SAH.⁶⁾ The most common sites of aneurysms causing ICH with SAH are the middle cerebral artery (MCA) and anterior communicating artery (AcomA), with incidence rates of 46% and 28%, respectively.⁶⁾ Another study also indicated that MCA and AcomA aneurysms were the most frequent with incidence rates of 46% and 30%, respectively.⁴⁾ The infratemporal lobe and the external capsule are the most

typical locations of ICH caused by ruptured MCA aneurysm.^{1-4,6)} Ruptured AcomA aneurysm causes ICH in the frontal lobe.⁷⁾ ICH in the frontal lobe and temporal tip from ICA aneurysms has been reported,⁴⁾ but these cases were also accompanied by SAH.

ICH without SAH is a rare presentation of ruptured aneurysm. In previous studies, 7 of 618 cases (1.1%)⁶⁾ and 1 of 492 cases (0.2%)¹⁾ of ruptured aneurysms presented with only ICH without SAH, most of which were associated with MCA aneurysms. Ruptured ICA dorsal wall aneurysms presenting with ICH in the frontal lobe without SAH were reported,^{5,7)} but ICA bifurcation aneurysm presenting only with ICH is extremely rare.

Differentiation of ICH caused by a ruptured aneurysm from hypertensive ICH is often difficult and can be a problem clinically. In the present case, ICH was localized in the putamen and misdiagnosed as hypertensive ICH. Hypertensive putaminal hemorrhage is usually distributed regularly and extends in the longitudinal directions. Extension of the hematoma to the middle fossa or temporal tip is rare. 1,6) Retrospective review of the CT in the present case found that the ICH was mainly located in the putamen but was also localized at the base of frontal lobe, appearing as if crossing the left sylvian fissure. Furthermore, ICH was accompanied by a small high density area adjacent to the medial-inferior part of ICH (Fig. 1). We considered these CT findings to be atypical for hypertensive ICH. Thus, radiological examinations, such as MR angiography or three-dimensional CT angiography, should have been performed without delay in the present case.

Several factors for ICH without SAH from ruptured aneurysms have been proposed, such as deeply buried dome of an aneurysm into the cerebral parenchyma, 1,3,4,6) or adhesion of the ruptured point of an aneurysm to the adjacent neural tissue intercepting SAH. 4,6) In the present case, the dome of the aneurysm was estimated to be buried in the frontal lobe, but the precise mechanism of ICH without SAH is unknown, as the patient was treated by endovascular surgery.

The present rare case of ruptured ICA bifurcation aneurysm that caused ICH without SAH indicates that a ruptured aneurysm should be considered as a potential cause of ICH without SAH. Atypical localization and extent of hematoma or an accompanying lesion may be helpful to suggest latent aneurysm in such cases.

References

- Hada H, Inagawa T: [A case of ruptured aneurysm which seemed putaminal hemorrhage]. No Socchu 14: 414-416, 1992 (Japanese)
- Hayward RD, O'Reilly GVA: Intracerebral haemorrhage. Accuracy of computerised transverse axial scanning in predicting the underlying aetiology. Lancet 1(7949): 1-4, 1976
- Inomori S, Ilu K, Ueda S, Shinsa P: [Ruptured aneurysms of the middle cerebral artery with intracerebral hematoma. CT findings and outcome]. Neurol Med Chir (Tokyo) 24: 573–579, 1984 (Japanese)
- Kawamura S, Suzuki A, Sayama I, Yasui N: [Clinical significance of intracerebral hematomas following aneurysmal rupture]. Neurol Med Chir (Tokyo) 27: 1158-1166, 1987

- (Japanese)
- 5) Nishikawa T, Ueba T, Kajiwara M, Yamashita K: Bilateral acute subdural hematomas with intracerebral hemorrhage without subarachnoid hemorrhage, caused by rupture of an internal carotid artery dorsal wall aneurysm. Case report. Neurol Med Chir (Tokyo) 49: 152-154, 2009
- 6) Torii T, Kajiwara H, Fujii S, Wada M, Yamamura K, Kawanishi M, Nakamura S: [Ruptured intracranial aneurysms presenting only intracerebral hematoma on CT in an acute stage]. Hiroshima Igaku 47: 1171-1175, 1994 (Japanese)
- 7) Yamada T, Tanaka Y, Kamada K, Inui T: [A case of ICA anterior wall aneurysm presenting with intracerebral hematoma]. No Shinkei Geka Journal 7: 37-41, 1998 (Japanese)

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