



The Distribution of Subarachnoid and Intraventricular Hemorrhage on Computed Tomography Suggests the Location of an Idiosyncratic Vascular Lesion

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The incidence of aneurysmal subarachnoid hemorrhage (SAH) associated with intraventricular hemorrhage (IVH) varies. In general, the anterior communicating artery and posterior circulation aneurysms cause such IVHs. A 48-year-old man visited the Neurosurgical Department for the evaluation of a severe thunderclap headache that had awakened him from sleep. Brain computed tomography revealed diffuse, but asymmetric, SAH. Digital subtraction cerebral angiography (DSA) showed multiple vascular lesions, including an obvious saccular aneurysm on the left anterior choroidal artery. After surgical clipping, rebleeding was noted, and repeat DSA demonstrated that the bleeding site was on the right posterior cerebral artery. The distribution of SAH associated with the preserved Liliequist membrane may suggest the origin of the bleeding. Treatment of the correct bleeding site is effective for preventing rebleeding.

Key words: Aneurysm, intraventricular hemorrhage, Liliequist, subarachnoid hemorrhage

INTRODUCTION

The incidence of aneurysmal subarachnoid hemorrhage (SAH) associated with intraventricular hemorrhage (IVH) is 28-50%.^{1,2} In general, anterior communicating artery and posterior circulation aneurysms tend to cause such IVHs. The Liliequist membrane, which separated from the supratentorial cisterns, is an arachnoidal structure and could not be detected on computed tomography (CT). However, a preserved Liliequist membrane may affect the distribution of SAH and correctly indicate the bleeding site. Here, we present a rare case involving an intriguing posterior cerebral artery (PCA) lesion that was difficult to detect due to the superimposition of other vascular lesions.

CASE REPORT

History

A 48-year-old man, who was a chronic hepatitis C virus carrier with a history of hypertension and gout, experienced a severe thunderclap headache that awakened him from sleep a

few hours before arriving at the emergency department of our hospital. Upon presentation, he was lethargic with a Glasgow Coma Scale (GCS) of E3M6V5 and had isocoric and reactive pupils.

The patient did not demonstrate any specific neurological deficit. A brain CT revealed hyperdense SAH at the central cisterns and hydrocephalus [Figure 1]. He was assessed with a Hunt and Hess grade of 2 and grade 2 World Federation of Neurosurgical Societies SAH. Digital subtraction cerebral angiography, performed an hour later, revealed a saccular aneurysm on the left anterior choroidal artery (AChA) and infundibular morphology of both posterior communicating arteries (PCoAs) [Figure 2].

Operation

Two hours after the examination, due to suspected rupture of the AChA aneurysm, the patient underwent a craniotomy for aneurysm clipping and external ventricular drainage (EVD) via the left pterional approach. An aneurysm was successfully clipped, with an intentional residual neck, sparing the AChA and avoiding premature intraoperative rupture.

Postoperative course

Following the procedure, the patient's consciousness returned to a GCS of E4M6V6 (e: Endotracheal intubation), 3 h after arrival in the Intensive Care Unit (ICU). He was sedated, overnight, with extubation planned for the next morning. A weaning protocol commenced early the following morning (postoperative day 1), and he was extubated after

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Liliequist's membrane associated with intraventricular hemorrhage following

completing the protocol. Seconds after the extubation, he complained of a severe headache and the EVD system filled with fresh blood. He became comatose, with clinical findings associated with increased intracranial pressure (ICP). A brain CT revealed a new IVH, acute hydrocephalus, and a peculiar finding resembling intracerebral hemorrhage (ICH) at the right hippocampus [Figure 3b]. Digital subtraction cerebral angiography was performed, revealing an intriguing vascular lesion with active bleeding from the right PCA [Figure 4]. The clipped AChA aneurysm was also surveyed, without any signs of rebleeding. Right-sided EVD was performed due to obstruction of the left-sided one. During the operation, abrupt hypotension occurred and was successfully managed using vasopressors. The patient's ICP was >50 mmHg when he

was brought back to the ICU. Soon, both pupils became fully dilated, with negative brainstem reflexes. After discussion with his family, a decision was made to palliate the patient; he died 36 h after arriving at the hospital.

DISCUSSION

The distribution of SAH in the preoperative CT did not usually involve the interpeduncular and prepontine cisterns [Figure 3a], suggesting an intact Liliequist's membrane. The Liliequist's membrane is a complex and variable arachnoidal structure. In most cadaveric dissection, Liliequist's membrane was composed of two distinct leaves: A diencephalic leaf directed toward the diencephalon (separated chiasmatic and interpeduncular cisterns) and a mesencephalic leaf directed

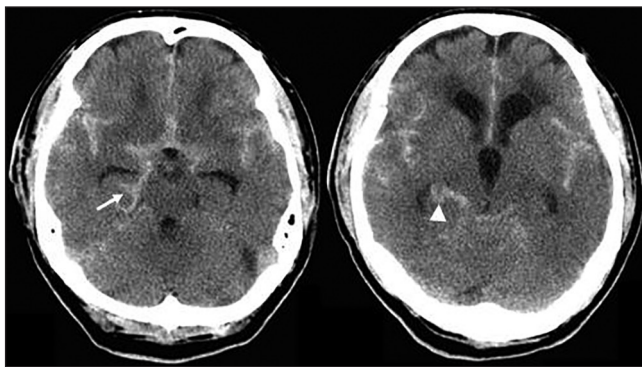


Figure 1. Brain computed tomography showing typical subarachnoid hemorrhage and aneurysm rupture. There was asymmetrically more subarachnoid hemorrhage at the right ambient cistern (arrow), which was not believed to be meaningful until the patient began re-bleeding. An intracerebral hemorrhage was found in the right hippocampus (arrowhead), without intraventricular hemorrhage

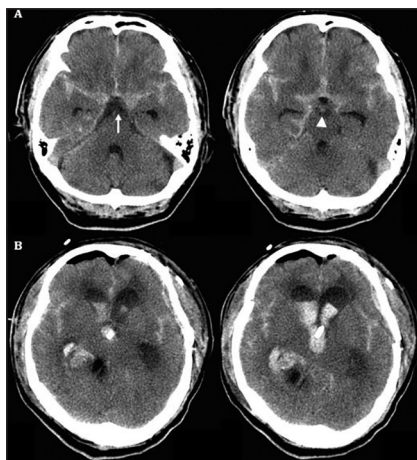


Figure 3. Subarachnoid hemorrhage is not present in the prepontine (arrow) and interpeduncular (arrowhead) cisterns in the preoperative computed tomography images (a) Postoperative computed tomography (b) shows more profound intracerebral and intraventricular hemorrhage in the right hippocampus than in the first computed tomography

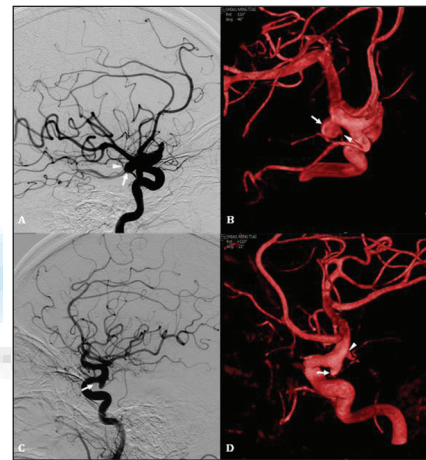


Figure 2. Digital subtraction angiography (a) and a three-dimensional reconstructed lateral-view image (b) of the left internal carotid artery. The anterior choroidal artery (arrowhead) arising from the proximal neck of an aneurysm (arrow). Right-sided traditional (c) and reconstructed (d) digital subtraction cerebral angiography (lateral view). Both the posterior communicating artery (arrow) and anterior choroidal artery (arrowhead) have infundibular morphologies, with their respective vessels exiting from the dome

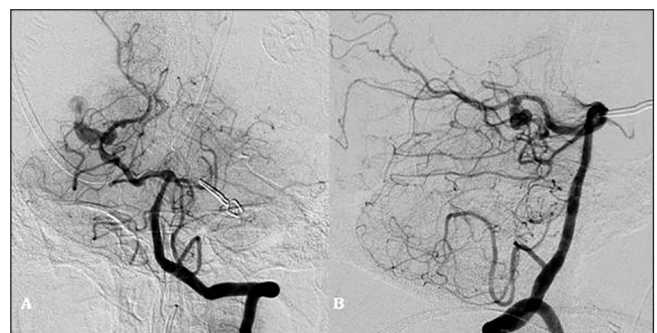


Figure 4. Postoperative digital subtraction cerebral angiography. Anteroposterior (a) and lateral (b) views showing contrast medium extravasation from the irregularly engorged P2-P3 segment of the right posterior cerebral artery

toward the mesencephalon (separated interpeduncular and prepontine cisterns). It could obstruct the flow of blood, confining the hemorrhage to the interpeduncular cistern, especially in low-pressure hemorrhages or small-volume hemorrhages.³ In general, aneurysmal SAH due to high-pressure arterial hemorrhage around the circle of Willis should indicate a damaged Liliequist membrane and present with diffuse SAH on the CT scan. Schwartz and Solomon suggested the importance of the Liliequist membrane for the distribution of blood.⁴ In our case, preservation of the Liliequist membrane seemed to be due to the low flow of blood in the brain parenchyma and small-volume hemorrhages into the subarachnoid space, instead of a left AChA aneurysm rupture. These results suggest that careful attention should be paid to the distribution of peculiar SAHs in patients with suspected aneurysmal SAHs in imaging studies.

In general, two major mechanisms of IVH, after an aneurysm rupture, are recognized. One involves blood directly entering the ventricles via the ventricle wall, similar to IVH caused by the rupture of an anterior communicating artery aneurysm through the lamina terminalis. The second mechanism involves large amounts of aneurysmal SAH regurgitating into the fourth ventricle via the foramina of Luschka. In this case, the source of the IVH was considered to be through the disrupted medial wall of the right temporal horn of the lateral ventricle.

In the present patient, the PCA features, demonstrated in the posterior circulatory system during the second angiography, were suggestive of an arterial dissection [Figure 4]. Reinspection of the preoperative CT demonstrated the presence of asymmetrical blood clots with ICH in the right hippocampus [Figure 1]; these features were more profound in the repeated CT [Figure 3b]. Retrospectively, the first episode of bleeding occurred at the site of the PCA dissection. The dissection was composed of a long segment extending from P1 to P3. Determination of whether the false lumen involved the PCoA or any perforators, based on angiography findings, was extremely difficult. Because the ICP was high, the open surgical intervention was impossible.

Ideally, the bleeding site should have been located by the first CT. However, the angiography clearly demonstrated an AChA aneurysm, about 6.10 mm long, 7.10 mm wide, and

4.16 mm into the neck. In such cases, the complication rate and the likelihood of incomplete occlusion by coil embolization are increased due to the ratio of the neck diameter to the largest aneurysm being >0.5 .⁵ Hence, management by surgical clipping and prevention of rebleeding was chosen. Intraoperatively, the blood clot around an aneurysm appeared to indicate the one that had ruptured. This was very difficult to distinguish intraoperatively, and we did not imagine, preoperatively, that an aneurysm to be clipped was not the one that bled.

CONCLUSION

Identification of the origin of bleeding is important in patients with SAH and multiple cerebrovascular lesions. The distribution of SAH and the putative mechanism of the possible IVH are crucial clues. The preservation of the Liliequist membrane seems to suggest that the hemorrhage results from low-pressure or small-volume blood flow.

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