—Case Reports—

Atypical Radiological and Intraoperative Findings of Acute Cerebral Hemorrhage Caused by Ruptured Cerebral Aneurysm in a Patient with Severe Chronic Anemia

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Abstract

Acute intracerebral hemorrhage (ICH) associated with mild anemia is commonly observed on radiological examination, and there are several reports of ruptured aneurysms occurring with ICH but without accompanying subarachnoid hemorrhage. However, the relationship among computed tomography (CT), magnetic resonance imaging (MRI), and intraoperative findings of ICH caused by ruptured cerebral aneurysm in patients with severe chronic anemia has been rarely reported and is poorly understood. Here, we report atypical radiological and intraoperative findings of acute ICH caused by ruptured cerebral aneurysm in a patient with severe chronic anemia. A 64-year-old man with anemia was admitted to our hospital after he experienced left hemiparesis and a disturbance of consciousness. At a referring institution, he showed evidence of macrocytic anemia (white blood cell count, 9,000/μL; red blood cell count, 104×10^4/μL; hemoglobin, 4.0 g/dL; hematocrit, 12.2%; and platelet count, 26.6×10^4/μL). Both CT and MRI showed a right frontal ICH. The outer ring of the hematoma appeared as low-density area on CT, a low-intensity area on T1-weighted MRI, and a high-intensity area on T2-weighted MRI with a serous component. The patient received a blood transfusion and underwent surgical removal of the hematoma the following day. The white serous effusion visualized with CT and MRI was identified as a blood clot in the hematoma cavity. The blood that leaks from blood vessels appears as a high-intensity area on CT because it undergoes plasma absorption in a solidification shrinkage process, and is, therefore, concentrated. Although we did not examine the white effusion to determine if serous components were present, we speculated that the effusion may have contained serous components. Therefore, we removed the part of the effusion that appeared as a low-density area on CT. The presence of ICH without subarachnoid hemorrhage suggested the possible adhesion and rupture of a previous aneurysm. Therefore, ICH appeared as a mixed density area on CT because bleeding may have occurred several times. Because radiological findings of ICH caused by ruptured cerebral aneurysm in patients with severe chronic anemia are similar to those of ICH and cerebral edema, we suggest that the atypical radiological findings of ICH caused by ruptured cerebral aneurysm in patients with severe chronic anemia should be carefully evaluated, especially when surgery is indicated.
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Key words: intracerebral hemorrhage, anemia, intraoperative finding, computed tomography, magnetic resonance imaging

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Introduction

Acute intracerebral hemorrhage (ICH) associated with slight anemia is commonly observed on radiological examination, and there are several reports of ruptured aneurysms occurring in ICH without accompanying subarachnoid hemorrhage (SAH). However, the relationship among findings of computed tomography (CT) and magnetic resonance imaging (MRI) and intraoperative findings in cases of acute ICH caused by ruptured cerebral aneurysm in patients with severe chronic anemia has been rarely reported and is poorly understood.

Here, we report atypical radiological and intraoperative findings in a case of acute ICH caused by ruptured cerebral aneurysm in a patient with severe chronic anemia.

Case Report

A 64-year-old man with anemia was admitted to our hospital after experiencing left hemiparesis and a disturbance of consciousness. Anemia had been noted in periodic medical examinations but had not been evaluated. The patient was discovered in an unconsciousness state at his residence; however, his wellness had been confirmed earlier. The episode of headache with aura was unclear.

Physical examination showed a blood pressure of 110/59 mmHg. At a referring institution, hematologic examinations showed evidence of macrocytic anemia (white blood cell count, 9,000/μL; red blood cell count, 104×10^4/μL; hemoglobin, 4.0 g/dL; hematocrit, 12.2%; and platelet count, 26.6×10^4/μL), but other results were normal. Both CT **(Fig. 1)** and MRI **(Fig. 2a, 2b, 2c)** almost 12 hours after the ictus revealed a right frontal ICH without SAH. The outer ring of the hematoma showed low density on CT, low intensity on T1-weighted MRI, and high intensity on T2-weighted MRI with a serous component. Magnetic resonance angiography **(Fig. 2d)** and 3-dimensional CT angiography **(Fig. 2e)** revealed a right middle cerebral artery aneurysm. On the basis of these images, a ruptured aneurysm was believed to have caused the ICH.

The patient received a blood transfusion and underwent surgical clipping and removal of the

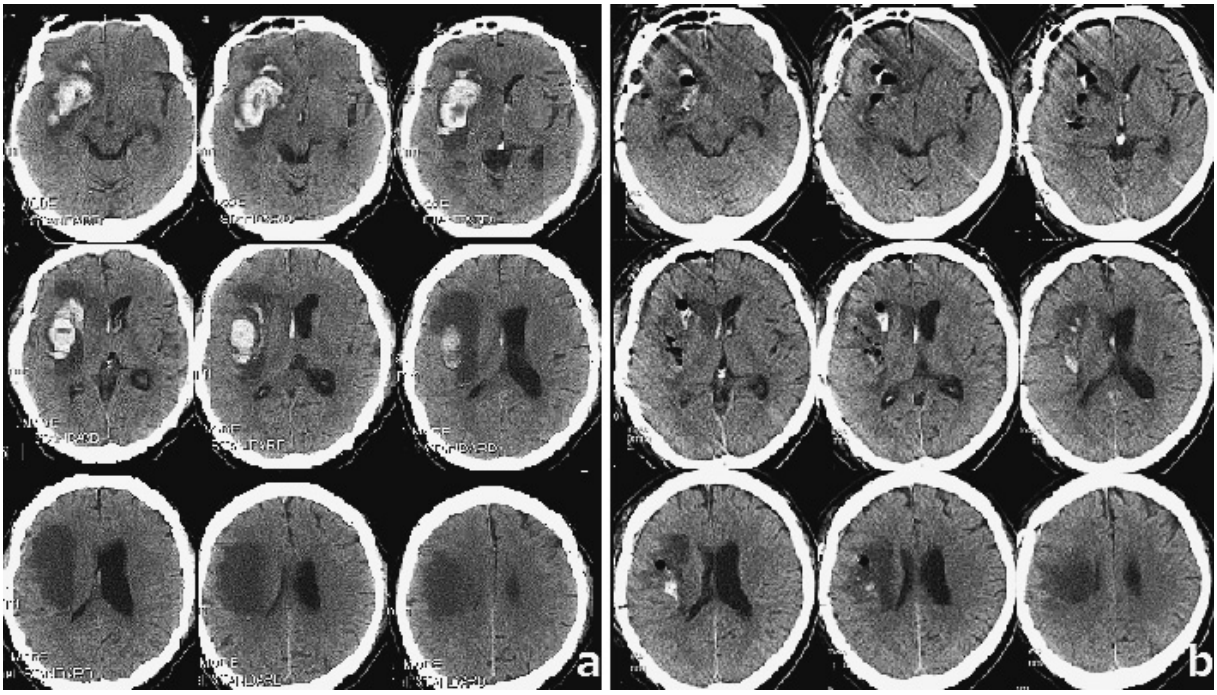

Fig. 1 **a**: Computed tomography reveals a right frontal intracerebral hemorrhage.
b: Postoperative computed tomography reveals almost complete removal of the hematoma and the presence of an outer ring of the hematoma, which was a low-intensity lesion.

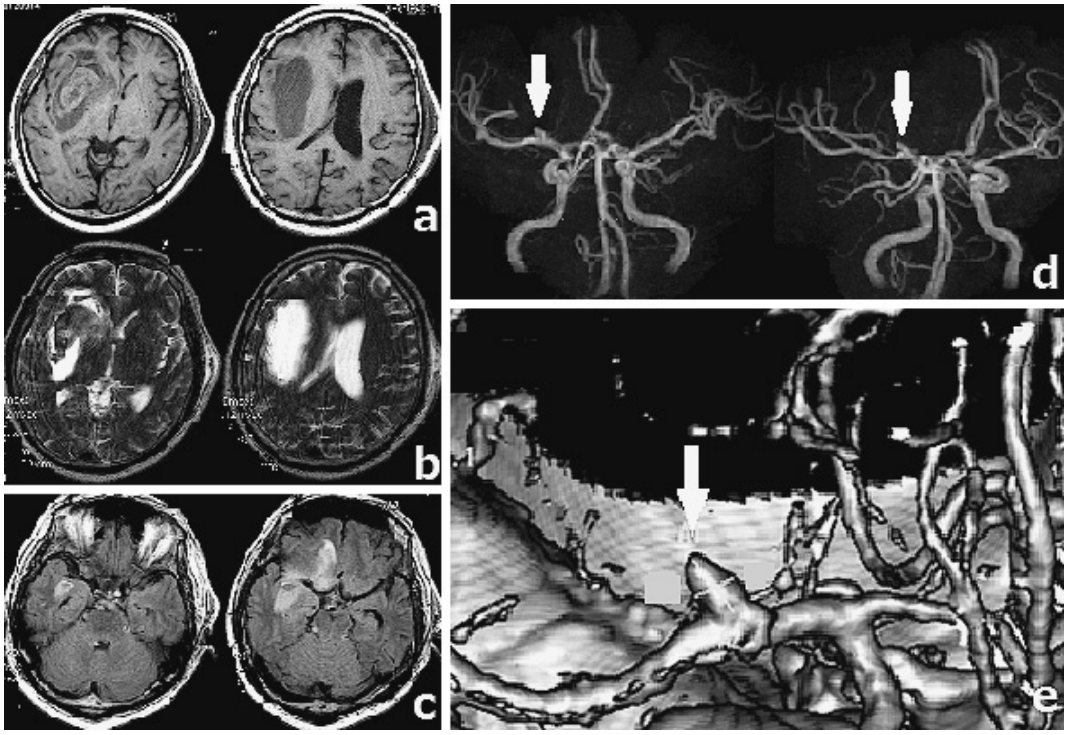

Fig. 2 **a**: T1WI magnetic resonance imaging reveals a right frontal intracerebral hemorrhage.
b: T2WI magnetic resonance imaging reveals a right frontal intracerebral hemorrhage.
c: Fluid-attenuated inversion recovery magnetic resonance imaging reveals no subarachnoid hemorrhage.
d: Magnetic resonance angiography reveals a right middle cerebral artery aneurysm **(white arrow)**.
e: Three-dimensional computed tomography angiography reveals a right middle cerebral artery aneurysm **(white arrow)**.

hematoma the following day. A right frontotemporal craniotomy was performed with a pterional approach to expose the aneurysm. The aneurysmal neck was successfully clipped, and the hematoma was evacuated with a transcortical approach. A white serous effusion in the hematoma cavity **(Fig. 3a)** was identified as a blood clot **(Fig. 3b)**. Postoperative CT revealed near-complete removal of the hematoma and the absence of any low-density area around the hematoma **(Fig. 1b)**.

The patient's recovery after surgery was uneventful. He showed improvement of consciousness and could communicate; however, the hemiparesis showed no improvement. Digital subtraction angiography performed after surgery showed that the aneurysm had been clipped completely. After the patient's general condition improved, we consulted the hematology department of internal medicine regarding bone marrow aspiration, but the cause of anemia still could not be determined. The appropriate management of anemia is a blood transfusion as suggested by a hematologist. The patient was transferred to another hospital for rehabilitation.

Discussion

In this study, we have reported atypical radiological and intraoperative findings in a case of acute ICH caused by ruptured cerebral aneurysm in a patient with severe chronic anemia. The potential reasons for these findings are discussed below.

Rupture of a cerebral aneurysm resulted in SAH. In 35% of cases of SAHs caused by ruptured cerebral aneurysms, ICH is also present[1]. Ruptured aneurysms resulting in ICH often do not cause SAH[2,3]. However, the relationship among the findings of CT and MRI and intraoperative findings in cases

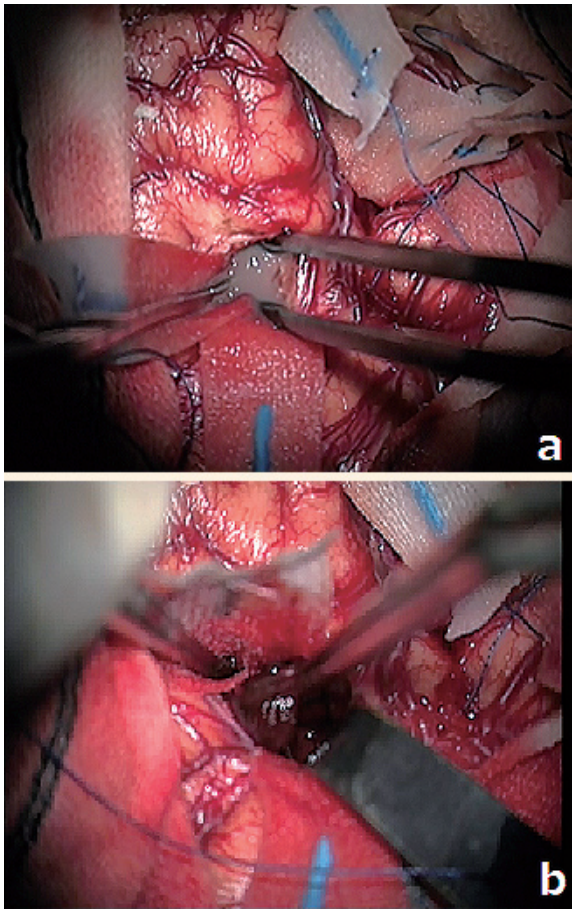

Fig. 3 **a**: Intraoperative observation of the white effusion indicates a peripheral hematoma that appeared as a low-density area on computed tomography.
b: Intraoperative observation showed a hematoma that appeared as a high-density area on computed tomography.

of acute ICH caused by ruptured cerebral aneurysm in patients with severe chronic anemia has been rarely reported and is poorly understood.

During the acute phase ICH appears as a high-density area on CT and as a isointensity area on both T1-weighted and T2-weighted MRI; brain edema appears as a low-density area on CT, as a low-intensity area on T1-weighted MRI, and as a high-intensity area on T2-weighted MRI[4,5]; and perihematomal edema volume increases during the first 24 hours after hyperacute spontaneous ICH[6]. Findings in the present case suggest that coagulated blood cell components (blood clots) in the acute phase of ICH appear as high-density lesions on CT and as isointensity lesions on T1-weighted and T2-weighted MRI and that the perihematomal serum component of the acute phase of ICH appears as a low-density area on CT, as a low-intensity area on T1-weighted MRI, and as a high-intensity area on T2-weighted MRI. In the present case, the presence of ICH without SAH suggested the possible adhesion and rupture of a previous aneurysm[7]. Therefore, ICH showed mixed density because bleeding may have occurred several times. The image intensity of the outer ring of the acute hematoma was similar to that of the perihematomal edema, which exhibited low density on CT, low intensity on T1-weighted MRI, and high intensity on T2-weighted MRI. However, the morphology of the outer ring of the hematoma differed from that of the brain edema around the acute phase of the ICH because of the well-circumscribed and round outer ring of the hematoma. In addition, the intraoperative observation revealed a low-density area in the outer ring of the hematoma on CT, which suggested that the low-density area was white effusion of the hematoma with a serous component. New and Aronow[8] have concluded that the high-density areas of hematomas on CT are dependent on hemolysis, hemoglobin, and hematocrit. Furthermore, New and Aronow have reported that hematomas appear as isodensity lesions on CT in patients with anemia (hemoglobin, 9–11 g/dL). Bergstrom et al.[9] have reported that areas of higher and lower attenuation may exist within a single hematoma, and the existence of both types of attenuation may be due to clot retraction or the age of the hematoma. In other words, the blood that leaks from blood vessels appears as a high-intensity lesion on CT because it undergoes plasma absorption in a solidification shrinkage process, and therefore, is concentrated[10–12]. Although we did not examine the white effusion to determine whether serous components were present, we speculate that the effusion may contain these components. Postoperative CT revealed decreasing low-density areas, and because usually there is no edema immediately after ICH, we concluded that the low-density area on CT was not due to brain edema but rather due to the serum components; thus, we removed this area.

Because radiological findings of ICH caused by ruptured cerebral aneurysm in patients with severe

chronic anemia are similar to those of ICH and cerebral edema, we suggest that atypical radiological findings of ICH caused by ruptured cerebral aneurysm in patients with severe chronic anemia need to be carefully evaluated, particularly if surgery is indicated. A study with larger numbers of patients with ICH caused by ruptured cerebral aneurysm and with severe chronic anemia would be helpful to obtain additional evidence in support of these findings.

Conflict of Interest: The authors declare no conflict of interest.

References

1. Pasqualin A, Bazzan A, Cavazzani P: Intracranial hematomas following aneurysmal rupture: experience with 309 cases. Surg Neurol 1986; 25: 6–17.
2. Quoc-Anh T, Shaan MR, Rafael JT: Aneurysmal rupture without subarachnoid hemorrhage: Case series and literature review. Neurosurgery 2005; 57: 225–229.
3. Park J, Hwang YH, Park SH: Angiographic examination of spontaneous putaminal hemorrhage. Cerebrovascular Disease 2007; 24: 434–438.
4. Alemany Ripoll M, Stenborg A, Sonninen P, Terent A, Raininko R: Detection and appearance of intraparenchymal haematomas of the brain at 1.5 T with spin-echo, FLAIR and GE sequences: poor relationship to the age of the haematoma. Neuroradiology 2004; 46: 435–443.
5. Goren O, Monteith SJ, Hadani M, Bakon M, Harnof S: Modern intraoperative imaging modalities for the vascular neurosurgeon treating intracerebral hemorrhage. Neurosurg Focus 2013; 34: E2. doi: 10.3171/2013.2.FOCUS1324.
6. Gebel JM Jr, Jauch EC, Brott TG, et al.: Natural history of perihematomal edema in patients with hyperacute spontaneous intracerebral hemorrhage. Stroke 2002; 33: 2631–2635.
7. Takeuchi S, Takasato Y, Masaoka H, et al.: Case of ruptured middle cerebral artery bifurcation aneurysm presenting as putaminal hemorrhage without subarachnoid hemorrhage. Brain and Nerve 2009; 61: 1171–1175.
8. New PF, Aronow S: Attenuation measurements of whole blood and blood fractions in computed tomography. Radiology 1976; 121: 635–640.
9. Bergstrom M, Ericson K, Levander B, Svendsen P: Computed tomography of cranial subdural and epidural hematomas: variation of attenuation related to time and clinical events such as re-bleeding. J Comput Assisted Tomogr 1997; 1: 449–455.
10. Mauersberger W, Lanksch W, Kazner E, Grumme T: Computerized tomography in head injuries. Zentralbl Chir 1978; 103: 501–511.
11. Nishizawa S, Ninchoji T, Izumiya Y, Uemura K: Atypical CT findings of acute epidural hematomas. CT kenkyu 1982; 4: 349–354.
12. Stone JL, Schaffer L, Ramsey RG, Moody RA: Epidural hematomas of the posterior fossa. Surg Neurol 1979; 11: 419–424.

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