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Case Report

Mutism due to a massive hematoma after rebleeding of an aneurysmal subarachnoid hemorrhage in the territory of the distal anterior cerebral artery

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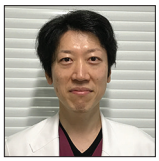

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ABSTRACT

Background: The mutism caused by hematoma after subarachnoid hemorrhage (SAH) is extremely rare, and the details of its clinical course have not been clarified.

Case Description: A 75-year-old woman who presented with transient loss of consciousness and a subsequent severe headache was transferred to our hospital. She was diagnosed with the World Federation of Neurosurgical Societies Grade II SAH due to the rupture of an aneurysm at the A2–3 junction in the left anterior cerebral artery (ACA). Endovascular coil embolization was successfully performed; however, postoperative computed tomography (CT) confirmed a massive hematoma in the corpus callosum and expansion into the cingulate gyrus, which was suspected to be due to preoperative or intraoperative rebleeding. The patient remained completely mum, which was considered as mutism due to a hematoma in the ACA territory. The postoperative clinical course was favorable, and the patient had fully recovered speech fluency with the disappearance of hematoma on CT scan at 44 days after the occurrence of SAH.

Conclusion: This is a rare case of mutism caused by an interhemispheric hematoma due to rebleeding after SAH. No radical evacuation of the hematoma may be desirable for the improvement of mutism because additional structural damage to the ACA territory by surgical stress should be avoided.

Keywords: Distal anterior cerebral artery, Hematoma, Mutism, Subarachnoid hemorrhage

INTRODUCTION

The term mutism refers to a clinical state characterized by the complete or almost complete absence of speech that is not associated with other aphasic symptoms or deteriorated consciousness. The condition of mutism tends to be transient and is rarely observed in conscious neurological patients. The areas responsible for mutism include the supplementary motor area, corpus callosum, anterior cingulate gyrus, ventral lateral thalamus, periaqueductal gray region, and dentate nucleus.[5,7,8]

Mutism is normally observed in cases of head trauma, brain neoplasia, and ischemic stroke; however, it is rarely confirmed in hemorrhagic stroke. In contrast to the relatively high incidence of aneurysmal subarachnoid hemorrhage (SAH) in the territory of the anterior cerebral artery (ACA), mutism complicating vasospasm-related cerebral infarction is uncommon, much less due

to the massive hematoma after SAH within the supplementary motor area, corpus callosum, and anterior cingulate gyrus.[3,6]

In this report, we describe a rare case of mutism due to a massive hematoma after rebleeding of aneurysmal SAH in the territory of the distal ACA and consider the optimal treatment strategy for SAH patients with this uncommon symptom.

CASE DESCRIPTION

A 75-year-old woman presented with transient loss of consciousness and subsequent severe headache at home and was transferred to the emergency department of our hospital. She had a Glasgow Coma Scale score of 13/15 (E3V4M6) and no obvious focal neurological symptoms on admission. Head computed tomography (CT) revealed a Fisher Group 3 SAH uncomplicated with acute hydrocephalus [Figures 1a and b]. Digital subtraction angiography revealed the presence of a saccular aneurysm at the A2–3 junction in the left ACA with the formation of a daughter sac [Figure 2a and b]. Therefore, she was diagnosed with the World Federation of Neurosurgical Societies Grade II SAH due to the rupture of an aneurysm at the left ACA.

On day 1, endovascular coil embolization was successfully performed [Figures 2c and d]; however, postoperative CT revealed a massive hematoma in the corpus callosum with expansion into the cingulate gyrus, which was compatible with the site of the aneurysm [Figure 3a]. The occurrence of rebleeding was suspected at some points, although there was no obvious sign of rebleeding, such as a sudden increase in blood pressure and/or a decrease in consciousness preoperatively and intraoperative angiographic extravasation. On day 2, the patient presented with spontaneous eye opening and some movements of the upper and lower limbs. However, she remained completely mum but was able to perform some reactions according to the instructions of intense verbal and/or auditory stimulation. The patient underwent conservative treatment for the hematoma, and gradually regained her ability to speak. Forty-four days after SAH onset, head CT demonstrated that the hematoma in the corpus callosum had disappeared for the most part [Figure 3b], and the patient had recovered fully from mutism. On day 7, magnetic resonance (MR) imaging demonstrated secondary multiple ischemic lesions in the left putamen, corona radiata, and frontal lobes [Figure 4a-c] and MR angiography revealed mild vasospasm of the ACA, although it was indistinct due to the presence of a hematoma [Figure 4d and e]. Fortunately, the patient did not show obvious neurological deficits due to these ischemic lesions. After that, advanced vasospasm and ischemic stroke were not confirmed until day 14. Finally, she

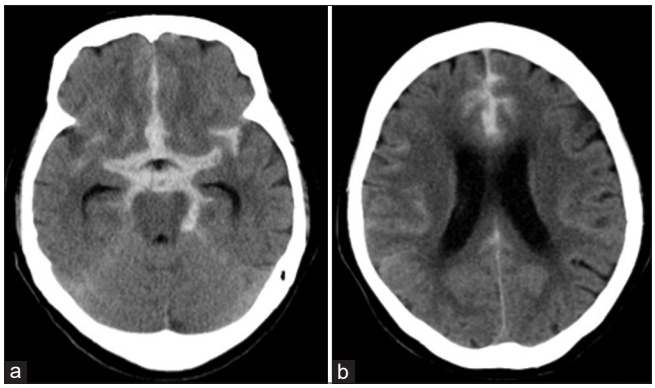

Figure 1: Computed tomography on admission showing a Fisher Group 3 subarachnoid hemorrhage uncomplicated with acute hydrocephalus (a and b).

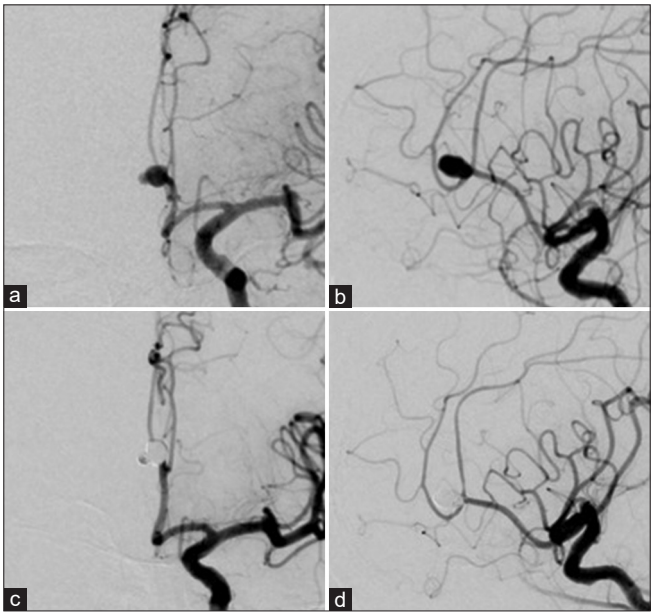

Figure 2: Digital subtraction angiography (a) anteroposterior (AP) projection, (b) lateral projection showing a saccular aneurysm at the A2–3 junction of the left anterior cerebral artery with a daughter sac. Endovascular coil embolization is executed successfully (c) AP projection, (d) lateral projection.

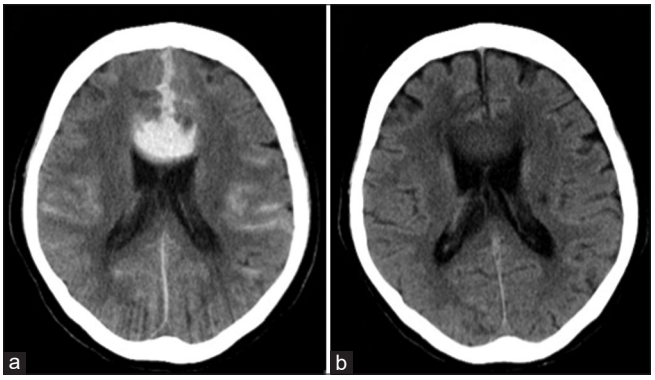

Figure 3: Postoperative computed tomography (CT) showing massive hematoma on the corpus callosum expanding into the cingulate gyrus (a). Forty-four days after subarachnoid hemorrhage onset, CT demonstrates that the hematoma on the corpus callosum has mostly disappeared (b).

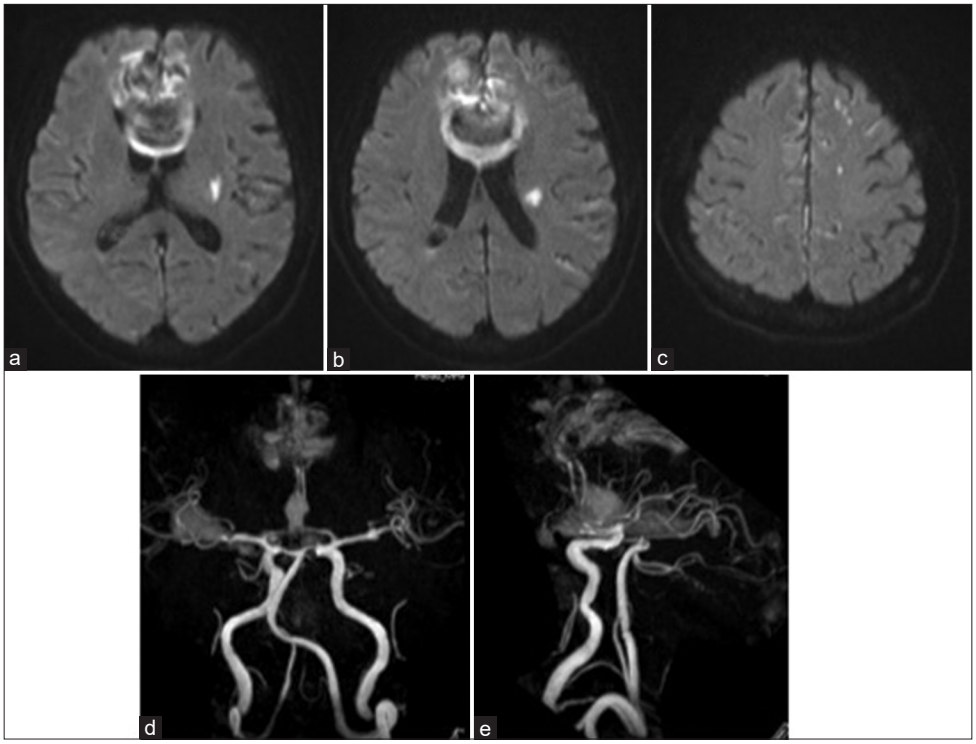

Figure 4: Magnetic resonance (MR) imaging demonstrates multiple ischemic lesions at the left putamen, corona radiata, and frontal lobes (a-c), and MR angiography shows mild vasospasm of anterior cerebral artery, although it is indistinct due to the presence of hematoma (d and e).

was transferred to a rehabilitation hospital because of residual diffuse muscle atrophy of the lower limbs.

DISCUSSION

Mutism due to vascular disease is commonly caused by cerebral ischemia of the basilar artery, ACA territories, or the bilateral thalamic region. A literature review demonstrated that the most common cause of mutism in patients with SAH is secondary infarction in the distal ACA territory.[1-3] Some patients with hydrocephalus after SAH are considered to be associated with delayed mutism.[4] It is unusual for mutism to be linked to the occurrence of aneurysmal SAH.

To the best of our knowledge, three cases of mutism caused by hematoma due to SAH have been reported, which showed a massive interhemispheric subpial clot located dorsal to the corpus callosum splaying the cingulate gyri without the confirmation of secondary bilateral infarction in these territories.[3,6] Our case also presented with sudden mutism after the formation of a massive interhemispheric hematoma due to rebleeding. Although mutism is a rare neurological condition, neurosurgeons should keep in mind that a massive hematoma in the ACA territory after SAH could be one of the causes.

The prognosis of mutism due to hematoma after SAH has not yet been clarified. The case of existing permanent damage to the cingulate gyri is an unfavorable outcome compared to that of reversible compression. Choudhari *et al*. suggested that acute surgical or endovascular intervention might worsen the neurological state of patients who already had mutism and that the postponement of surgical treatment may be better for weeks following the onset. They believe that delayed intervention would maximize the chances of spontaneous neurological improvement.[3]

On the other hand, early intervention for aneurysmal SAH is desirable to avoid the danger of rebleeding with fatal consequences. In the previous three cases of mutism due to hematoma after SAH that underwent surgical intervention in the acute phase, one showed a slight improvement of symptoms after 3 months in spite of efforts to evacuate the clot,[3] while the others had almost fully recovered speech frequency at the 6-month follow-up without removal of the hematoma.[6] Furthermore, our patient fully recovered from mutism without surgical evacuation of the hematoma 44 days after the onset of the condition. Based on these findings, early intervention for SAH patients with mutism due to hematomas in the ACA territory could be performed safely without aggravating neurological symptoms, and radical evacuation of the hematoma is not desirable in the management of mutism because additional structural damage to the ACA territory by surgical stress should be avoided.

CONCLUSION

We described a rare case of mutism caused by an interhemispheric hematoma due to rebleeding after SAH. Acute surgical intervention for these patients could be performed safely, and radical evacuation of hematoma is not necessary for favorable outcomes of mutism. However, the etiology and prognosis of this uncommon neurological state are not fully understood; therefore, further investigations are needed to clarify this in the future.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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