Introduction

Intracerebral hematomas complicate up to 40% of ruptured intracranial aneurysms, and there is a close correlation between the location of the intracerebral hematoma (ICH) and that of the ruptured aneurysm. Ruptured anterior communicating artery (ACoA) aneurysms frequently accompany hematomas in the frontal lobe or in the ventricles. A few ruptured middle cerebral artery (MCA) or internal carotid artery (ICA) aneurysms cause basal ganglia ICH's. The rupture of MCA aneurysms frequently associates frontal and/or temporal ICH's. Usually a direct extension route from the sylvian subarachnoid hemorrhage (SAH) to the ICH can be identified intraoperatively. The authors report a rare case with an idiopathic subcortical ICH superimposed on the SAH caused by a ruptured ipsilateral MCA bifurcation aneurysm and discuss on the clinical significance of the peculiar situation.

Case Report

This 59-year-old man complained of sudden headache on the day of admission. He had been alert initially when he visited an other hospital where his brain CT showed diffuse SAH, notably thicker in basal and left Sylvian cisterns (Fig. 1). When he arrived at the emergency room of our hospital several hours later, he was drowsy (GCS 10). The vital signs were blood pressure (BP) 130/70, pulse rate 62, respiratory rate 18, and body temperature 36.6°C. About 2 hours thereafter, he became stuporous and showed right-sided hemiplegia and left-sided pupillary dilatation; aneurysmal rebleeding was highly suspected. Immediate follow-up brain CT revealed a huge (145cc) left frontal subcortical ICH, the anteroinferior part of which looked in contact with the knee portion of the left MCA (Fig. 2); the ICH exerted so severe mass effect to cause 14mm midline shift to the right side and the effacement of cisterns around the upper brainstem, but no notable changes was evident in the volume and distribution of the SAH.

A rare case of idiopathic subcortical intracerebral hematoma superimposed on the subarachnoid hemorrhage due to the rupture of an ipsilateral middle cerebral artery bifurcation aneurysm is reported and pertinent literatures are reviewed.

KEY WORDS: Idiopathic intracerebral hematoma · Superimposition · Subarachnoid hemorrhage.
Ruptured middle cerebral artery aneurysm.

Subarachnoid Hemorrhage Due to a Ruptured Middle Cerebral Artery Bifurcation Aneurysm Superimposed by an Idiopathic Intracerebral Hematoma

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Fig. 1. Initial brain computed tomography showing diffuse subarachnoid hemorrhage notably thicker in basal and left sylvian cisterns.
Emergency transfemoral cerebral angiography showed an anterolaterally-directed small (5mm) saccular left MCA bifurcation aneurysm as well as a remarkable square shift of the anterior cerebral artery to the contralateral side (Fig. 3). Emergency operation was performed under the pre-operative diagnosis of a ruptured left MCA bifurcation aneurysm associated with an early rebleeding to cause a huge ipsilateral frontal ICH.

Operation
A large left frontotemporal craniotomy and dural incision disclosed tense reddishly-discolored brain. Partial ICH removal through a small mid-frontal corticectomy made the brain slack enough to expose and dissect the small MCA bifurcation aneurysm without much difficulty. The aneurysm sac was narrow-necked and directed anterolaterally. Its fundus was in contact with the temporal cortex; unexpectedly the aneurysm sac had no contact with the frontal lobe at all. The neck was clipped with a 7mm-long straight blade Sugita’s aneurysm clip. A route of extension of sylvian SAH into the ICH could not be identified at all even after a thorough search; it was concluded that the ICH was not correlated etiologically with the rupture of the MCA aneurysm. The residual ICH was removed; neither abnormal vessels nor tumorous tissues were found at the surrounding cerebral parenchyme. The operation wound was closed by expansion duroplasty and decompression craniectomy for a generous external decompression.

Post-op Course
Immediate post-operative intracranial pressure (ICP) was 15-20mmHg. He was semi-comatose with bilateral pupillary dilatation and absence of light reflex. On post-operative day (POD) 2, controlled mandatory ventilation mode mechanical ventilator was applied, and the ICP was ca 30mmHg. Neurologic status was stationary. Since POD 3, he was comatose with the ICP about 35mmHg. Around POD 4, he showed signs of the acute renal failure. On POD 7, cardiac arrest developed; immediate cardiorespiratory resuscitation had been unfruitful and he died.

Discussion
Pre-operatively, the ICH in this case was thought to have been caused by the rebleeding of the MCA bifurcation aneurysm. However, in spite of the intra-operative thorough search, the ICH had no connection with the sylvian SAH or the aneurysmal sac, and it was concluded that the ICH had not been caused by the rebleeding of the MCA bifurcation aneurysm, but was an idiopathic ICH. On close post-operative review, the subcortical ICH was somewhat separated from the aneurysm on the preoperative brain CT (Fig. 2). The etiology of the ICH in the patient is still undetermined. Such common causes of spontaneous ICH’s as arteriovenous malformation (AVM), brain tumor bleeding, amyloid angiopathy can be ruled out, and the most probable diagnosis is the so-called hypertensive ICH. Theoretically, an incidental superimposition of an ICH on an intracranial aneurysmal rupture or vice versa is not impossible. The authors could find out three previous case reports of concurrent aneurysmal SAH and hypertensive ICH. In addition, a case of the putaminal ICH associated with...
ipsilateral ICA posterior communicating artery (PCoA) aneurysm and frontotemporal parasagittal arteriovenous malformation has been reported. In those cases, the ICH's were located at the thalamus or basal ganglia, remote from the aneurysms, and there was probably no much difficulty in identifying the ICH's as hypertensive ICH's concurrent with ruptured aneurysms.

Pre-operative differential diagnosis between the ICH caused by an aneurysmal rebleeding and a concurrent hypertensive ICH might be difficult, if the ICH is close to the aneurysm as in this case. Silver et al wrote that it was difficult to distinguish the cause of the ICH in concurrent SAH and ICH at the basal ganglia. Such case reports as the hypertensive putaminal ICH accompanying SAH in the basal cisterns and the peripheral MCA aneurysmal rupture presenting with a putaminal hemorrhage make the differentiation furthermore confusing.

Of the three previous case reports of concurrent aneurysmal SAH and hypertensive ICH, the reporters of one case described that the rise in the BP at the onset of putaminal ICH probably caused the rupture of the ICA-PCoA aneurysm, and those of another case suggested that the rupture of the ACoA aneurysm probably caused a rapid increase in the BP and subsequently provoked the development of the hypertensive putaminal ICH. It could not be clearly determined in those previously reported cases which one of the two lesions had occurred before or whether they occurred simultaneously. As for the question, it is evident in this case that the aneurysmal rupture occurred ahead of the ICH, i.e. the aneurysmal SAH may have possibly provoked the development of frontal subcortical ICH.

Principles of surgical managements of the two conditions, i.e. the hypertensive ICH superimposed on an aneurysmal SAH and the ICH caused by an aneurysmal rebleeding, may not differ. It has not been clearly defined whether the prognosis of those conditions are different. Generally speaking, management outcomes depend both on the volume and location of the ICH's and on the clinical grade of the SAH and the surgical configuration of the aneurysms. In this case, the huge ICH was fatal, causing irreversible brain herniation. In the previously reported three cases, the aneurysms was secured by delayed surgical neck clipping.

The authors hopefully intend by this case report to inform neurovascular neurosurgeons of a rare confusing clinical situation.

Conclusion

Aneurysmal SAH complicated by an idiopathic ICH is not a common clinical setting. Pre-operative differential diagnosis of this particular situation from aneurysmal rebleeding would be difficult if the two lesions are close to each other. In order for cerebrovascular neurosurgeons to be prepared and to achieve better management outcome in these patients, this clinical situation should be taken into considerations.

References