

## Simultaneous Occurrence of Subarachnoid Hemorrhage due to Ruptured Aneurysm and Remote Hypertensive Intracerebral Hemorrhage : Case Report

Simultaneous occurrence of aneurysmal subarachnoid hemorrhage (SAH) and hypertensive intracerebral hemorrhage (ICH) is very rare and only two cases have been previously reported in the literatures. We present a case of 68-yr-old man with a history of untreated hypertension, who suffered from sudden onset of headache followed by right hemiparesis. Computed tomographic (CT) scan revealed SAH in the basal cistern and remote ICH at the left putamen. Cerebral angiography showed a saccular aneurysm at the anterior communicating artery. No other vascular anomaly could be found at left putaminal area. Nine days after the ictal attack of SAH, the neck of aneurysm was clipped via the left frontotemporal craniotomy. Because of the ICH at the left frontal lobe and intraventricular hematoma on postoperative CT, we performed hematoma removal and external ventricular drainage 3 hours after the first operation. Postoperative neurological status had been improved to be drowsy and he was discharged in a severely disabled state 4 weeks after surgery. We suggest that the rupture of aneurysm possibly caused a rapid increase in blood pressure and subsequently resulted in hypertensive ICH.

**Key Words :** Aneurysm; Cerebral Hemorrhage; Hypertension; Subarachnoid Hemorrhage

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Received : 23 November 2000  
Accepted : 2 April 2001

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### INTRODUCTION

Bleeding into the brain parenchyma occurs in the cases of up to 40% of ruptured aneurysms and the presence of ICH caused by ruptured aneurysms are associated with much higher mortality rates (1). On the other hand, simultaneous occurrence of aneurysmal SAH and hypertensive ICH is very rare with only two cases having been previously reported in the literatures (2, 3). To our knowledge, the case presented here would be the third report of aneurysmal SAH accompanied by hypertensive ICH in the remote area from the site of the ruptured aneurysm.

### CASE REPORT

#### History

A 68-yr-old man with a history of untreated chronic hypertension experienced a sudden onset of headache followed closely by loss of consciousness on January 24, 1998. He recovered over time but suffered from right hemiparesis. Computed tomographic (CT) scan checked at a local hospi-

tal showed both SAH in the basal cistern, intraventricular hemorrhage at both lateral ventricles, and ICH at the left putamen (Fig. 1). After several days of conservative management, aggravated headache and seizure attack followed by neurological deterioration led him to be transferred to our hospital on January 29.

#### Examination

On admission, the patient was drowsy with right hemiparesis and his pupils were equal in size and reactive to light. The blood pressure was 250/150 mmHg and other laboratory data were unremarkable. Cerebral angiography demonstrated a 7 mm sized saccular aneurysm at the anterior communicating artery (Fig. 2). There were no other vascular lesions, especially at left putaminal area.

#### Operation

Nine days after the ictal attack of SAH, the aneurysmal neck was successfully clipped via the left frontotemporal craniotomy without removing the left putaminal hematoma. On postoperative neurological examination, he was stuporous

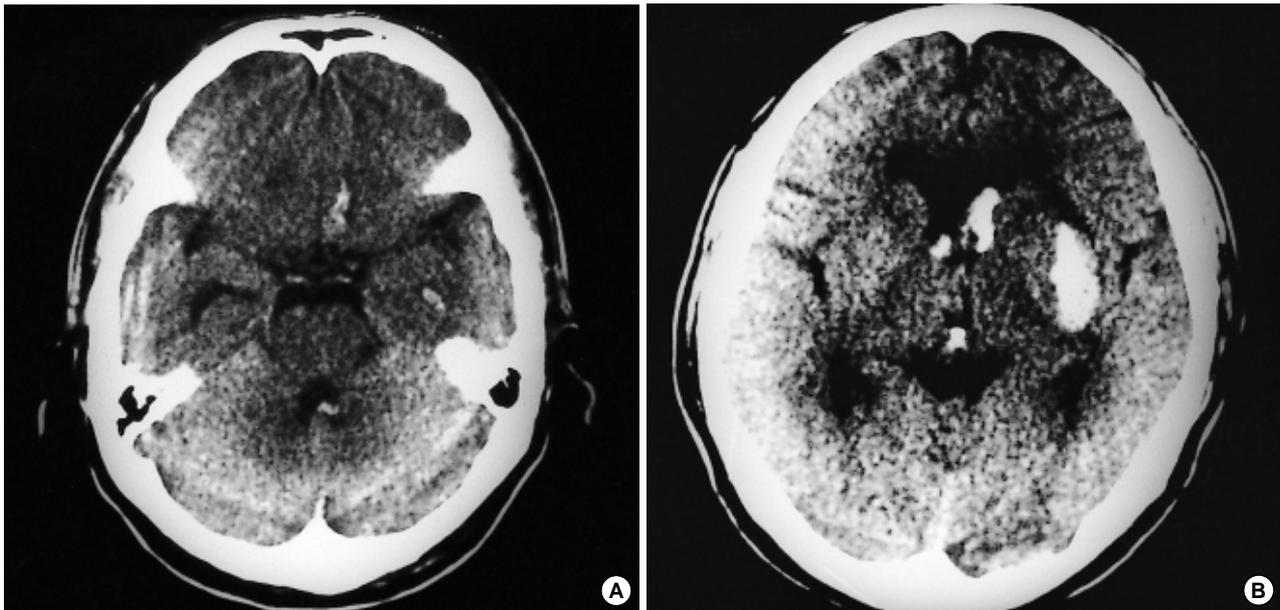


Fig. 1. CT scans showing a subarachnoid hemorrhage at interhemispheric cistern (A) and a left putaminal intracerebral hematoma and intraventricular hemorrhage on both frontal horns of the lateral ventricle (B).



Fig. 2. An oblique view of left carotid angiograms demonstrates a saccular aneurysm arising at the anterior communicating artery.

and CT showed ICH at the left frontal lobe and intraventricular hematoma at the lateral ventricle 3 hr after the first operation. Therefore, the patient underwent emergent hematoma removal and external ventricular drainage.

#### Postoperative Course

Postoperative neurological status had been gradually im-

proved up to Glasgow Coma Scale score 14 (E4V4M6). Post-operative angiography showed complete resolution of the aneurysm. He was transferred to a local hospital in a severely disabled state 4 weeks after surgery.

#### DISCUSSION

It is well known that there is a close correlation between the site of hematoma and that of the ruptured aneurysm, which may allow relatively accurate prediction of the location of the underlying aneurysm before cerebral angiography is performed. According to the previous reports, most hematomas secondary to ruptured anterior communicating aneurysms were localized at the frontal lobe and ventricular system (1, 4, 5). Although a few cases of basal ganglia hemorrhages are caused by middle cerebral artery aneurysms or internal cerebral artery bifurcation aneurysms, they are not easily caused by ruptured anterior communicating aneurysms (1, 6). According to Silver et al. (6), if there are concurrent SAH and ICH at the basal ganglia, it was difficult to distinguish the cause of hematoma, whether ruptured aneurysm or hypertension. In our patient, there was no continuity between ICH and SAH on the CT scans. Therefore, the origins of the two intracranial hemorrhages would be independent.

Regarding to the pathogenesis, three possible mechanisms could be assumed: 1) both lesions might have randomly occurred simultaneously; 2) the rupture of the aneurysm could have caused a reflex increase in blood pressure and increased intracranial pressure, resulting in hypertensive ICH due to the rupture of a penetrating artery which had

been weakened by chronic arterial hypertension in a relatively short time; 3) sudden rapid increase in blood pressure could have caused hypertensive ICH initially, and then resulted in rupture of remote aneurysm. However, there is a slight chance of the independent concurrence of aneurysmal rupture and ICH, making the other two mechanisms more likely.

In patients with chronic hypertension, acute changes in blood pressure and blood flow lead to the rupture of small penetrating arteries (7). The more sudden and the more severe the change is, the more likely is the risk of rupture. Furthermore, SAH is often accompanied by increased intracranial pressure. After ictal attack of SAH, the formation of cerebral edema, increase in intracranial blood volume, and accumulation of cerebrospinal fluid are all significant contributing factors of increased intracranial pressure (8-10). Yanaka et al. (3) described left thalamic ICH and concurrent rupture of left superior cerebellar artery aneurysm. Sugita et al. (2) also reported hypertensive ICH in the left corona radiata and the posterior limb of internal capsule associated with a rupture of left posterior communicating artery aneurysm. In the previous two cases, right sensory disturbance and/or right hemiparesis preceded a disturbance in consciousness, which suggested that the rapid rise in blood pressure after the hypertensive ICH caused the rupture of the aneurysm. In our case, however, a sudden onset of headache and loss of consciousness preceded right hemiparesis. Therefore, it is reasonable to think that the rupture of aneurysm possibly caused a rapid increase in blood pressure and subsequently resulted in hypertensive ICH. For further clarification of characteristic clinical features and pathogenesis of coincidental concurrent aneurysmal SAH and hypertensive ICH, more cases should be accumulated and analyzed in the future.

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