Case Studies

Hyperperfusion after Clipping of Aneurysm: A Rare Entity

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Background: Cerebral vasospasm is an uncontrollable and sometimes fatal complication occurring after subarachnoid hemorrhage. However, cerebral hyperperfusion syndrome is a rare complication after subarachnoid hemorrhage. Although plain computed tomography of cerebral hyperperfusion syndrome looks similar to cerebral infarction induced by cerebral vasospasm, they should be distinguished from each other because they require completely different treatments. *Case Description:* A 65-year-old man complained of severe headache and vomiting. A computed tomography scan of his head showed subarachnoid hemorrhage with acute hydrocephalus caused by intraventricular hematoma and aneurysm of the left middle cerebral artery. After endoscopic irrigation of the ventricular hematoma to decrease the intracranial pressure, we performed neck clipping for the ruptured aneurysm. We used a temporary clip to the proximal M1 segment twice for a total of 15 minutes. Five days after the clipping, a computed tomography scan of the patient's head showed a large low-density area in the left cerebral hemisphere. Although cerebral infarction caused by cerebral vasospasm was suspected, his perfusion computed tomography demonstrated a state of hyperperfusion corresponding to the low-density area. We started treatment to prevent vasodilation and excessive cerebral blood flow instead of treatment for cerebral vasospasm. After changing the treatment, the patient's symptoms gradually improved, and his perfusion computed tomography (8 days after surgery) showed no excessive increased blood flow. *Conclusions:* We present a case of cerebral hyperperfusion syndrome and its successful treatment, distinct from that of cerebral vasospasm, after ruptured aneurysm clipping. In addition, we discuss the mechanism of this rare syndrome based on previous reports. **Key Words:** Hyperperfusion syndrome—clipping—subarachnoid hemorrhage—temporary occlusion—endoscopic irrigation—perfusion CT.

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Introduction

Cerebral vasospasm (CVS) after subarachnoid hemorrhage (SAH) is one of the major targets of management during postoperative intensive care. Uncontrollable CVS often causes severe cerebral infarction and consequently results in a poor prognosis. To prevent pathophysiological vasoconstriction, vasodilator drugs and antiplatelet agents are usually used in addition to triple H (hypertension, hypervolemia, hemodilution) therapy. However, although cerebral hyperperfusion syndrome (CHS)

following SAH is rare, it can result in severe brain edema, intracerebral hemorrhage or SAH, and death.[1,2] The mechanisms underlying CHS involve dysregulation of the cerebral vascular system and hypertension in the setting of an acute and excessive increase of cerebral blood flow (CBF). The treatment strategies of CHS that focus on limiting the rise in CBF by regulating blood pressure and inhibiting vasodilation conflict with the therapeutic approaches for CVS.[1-6]

We report successful treatment of a case of CHS after clipping of a ruptured aneurysm. Differential diagnosis followed by the proper treatment is indispensable to control excessive blood flow and avoid a serious outcome.

Case Report

A 65-year-old man complained of sudden severe headache and vomiting accompanied by a progressive disturbance in consciousness. On arrival at the emergency department, his Glasgow Coma Scale was E1 V1 M5. Incomplete hemiplegia of the right side was also observed. A computed tomography (CT) scan of the patient's head showed a small SAH with intracerebral hematoma in the left temporal lobe and an acute hydrocephalus caused by intraventricular hematoma (Fig 1, A,B). Three-dimensional CT angiography (3D-CTA) showed 2 aneurysms on the anterior communicating artery and bifurcation of the left middle cerebral artery (MCA) (Fig 1, C). Because of the size of the aneurysm and location of the hematoma, rupture of the MCA aneurysm was suspected. We planned neck clipping of the aneurysm combined with endoscopic irrigation of the ventricular hematoma to decrease the intracranial pressure. After the left frontotemporal craniotomy, we punctured the left frontal lobe at Kocher's point before opening the dura, and maintained the sheath with no damage to the superficial vein. We irrigated the bilateral ventricular hematoma by septostomy using a rigid endoscope (2.7 mm diameter). After insertion of a ventricular drainage tube, the Sylvian fissure was opened without sacrificing any of the cerebral veins. Intermittent cerebrospinal fluid drainage was effective to control brain tension. During the left MCA aneurysm clipping, we used a temporary clip for the proximal M1 segment twice. To avoid the ischemic and reperfusion injury, edaravone was administered before application of a temporary clip. The first and second temporary occlusions took 7 minutes, 15 seconds, and 9 minutes, 14 seconds, respectively. The interval between each temporary occlusion was 3 minutes, 45 seconds. Neck clipping of the anterior communicating artery, the unruptured aneurysm, was also performed without using a temporary clip. We confirmed the complete obstruction of the aneurysms and normal brain circulation without abnormal arterial dilation by administering indocyanine green. Through all surgical procedures, the brain was always treated gently using soft retraction with a spatula.

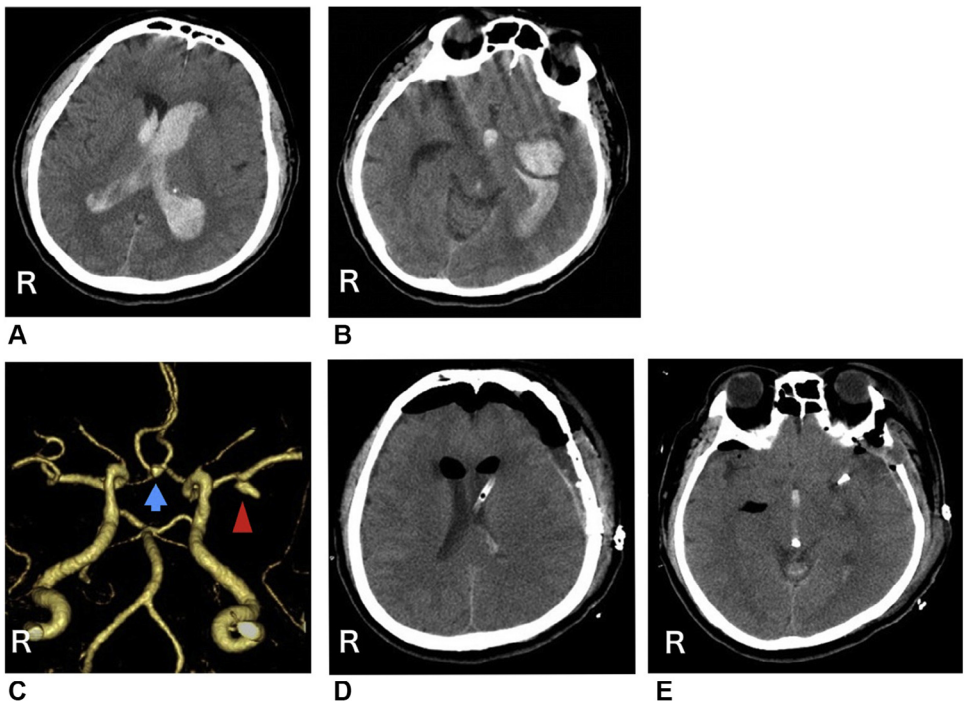

Figure 1. *Pre- and postoperative computed tomography (CT). (A) Preoperative head CT shows hydrocephalus caused by intraventricular hematoma. (B) Preoperative head CT shows subarachnoid hemorrhage with intracerebral hematoma. (C) Three-dimensional CT angiography shows aneurysm of the anterior communicating artery (3 × 3 mm, blue arrow) and left middle cerebral artery aneurysm (3 × 7 mm, red arrowhead). (D and E) Postoperative day (POD) 1 CT did not show a low-density area. (Color version of figure is available online.)*