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Subarachnoid venous hemorrhage in a patient with retrograde cerebral perfusion during surgery for a thoracic aortic aneurysm

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Introduction

Delayed awakening, transient cranial nerve disorders, and cerebral infarction have all been reported as complications of retrograde cerebral perfusion during repairs to the thoracic aorta [1,2]. However, subarachnoidal venous hemorrhage has not been reported to date. Furthermore, cerebral circulation during retrograde cerebral perfusion remains to be clarified in many respects. Under inappropriate perfusion conditions, the distribution of blood flow may be uneven and cerebral complications may occur [3,4]. We treated a patient in whom retrograde cerebral perfusion during surgery for a thoracic aortic aneurysm caused subarachnoidal venous hemorrhage. In this patient, conservative cerebral protection therapy improved the consciousness level, and thereafter the cerebral neurological deficit disappeared.

Case report

The patient was a 70-year-old male (height 167 cm; body weight 63 kg). Myocardial infarction developed at the age of 59. An artificial Y-type graft replacement was performed for an abdominal aortic aneurysm when the patient was 68 years old. During the follow-up study of the myocardial infarction, a thoracic descending aortic aneurysm and stenosis of coronary lesions were detected, and replacement of the thoracic descending aorta and a coronary artery bypass graft involving one branch were scheduled.

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Intraoperative course

Prior to surgery, a silicone subarachnoidal drainage tube [Silascon (Kaneka Medix, Osaka, Japan), 5 Fr] was inserted from L3 to L4 (5 cm on the cephalad side) to monitor cerebrospinal pressure and to facilitate the suction of cerebrospinal fluid. The patient was positioned for left posterolateral thoracotomy. The cardiopulmonary bypass was obtained by cannulating the right femoral artery and vein, and the pulmonary artery. The venous cannula was advanced to the right atrium, and then the patient was cooled until a blood temperature of 23.5°C and rectal temperature of 25.4°C were reached. Methylprednisolone (1 g) and pentobarbital (250 mg) were given during cooling. Upon circulatory arrest, the patient was placed in the Trendelenburg position and his head was wrapped in a cold ice bag. The circulation was arrested for 28 min. Simultaneously, retrograde cerebral perfusion was performed through the right atrium, and the flow rate was controlled at around 600 ml·min^{-1}. During the retrograde cerebral perfusion, the right atrial pressure was about 10 mmHg. Under additional circulatory arrest (11 min), aortocoronary bypass grafting involving one branch was performed. The blood supply to the upper body was resumed through the replaced thoracic aorta. The blood supply to the lower body was resumed through the femoral artery. Anastomosis on the peripheral side was performed. The duration of aortic clamping was 49 min. The duration of extracorporeal circulation was 2 h 19 min.

Postoperative course in the intensive care unit

When the patient was transferred to the intensive care unit (ICU), his cerebrospinal pressure was 10 mmHg. Positive reactions to calling the patient's name were observed 4 h after admission to the ICU.

On the first postoperative day, bloody cerebrospinal fluid from a spinal drainage tube was noted at 6:00 A.M.,

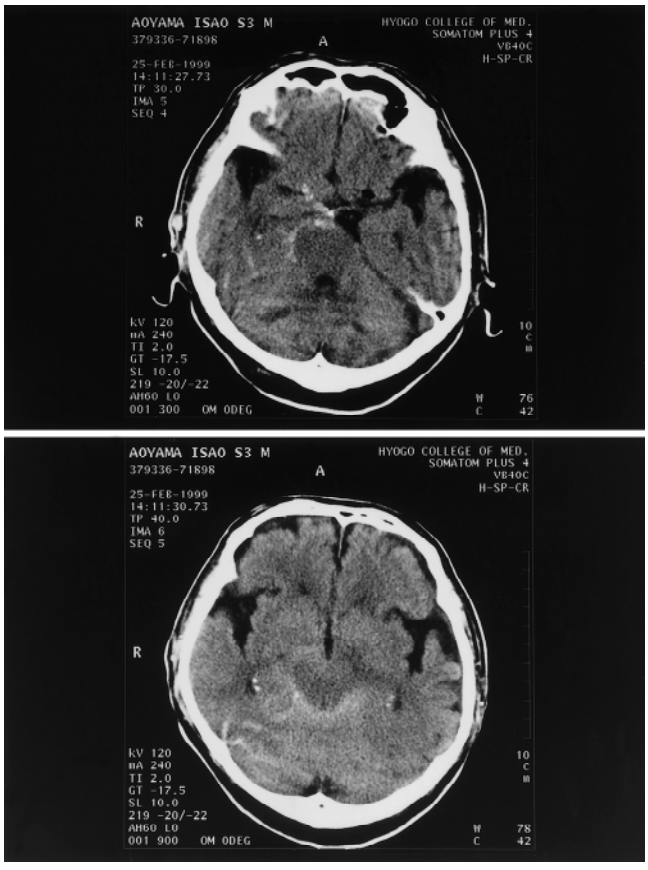

Fig. 1. Contrast enhancement of brain computed tomography findings. These findings revealed a low-density area in the left frontal lobe (*upper*). Furthermore, a high-density area was noted at the quadgeminal and ambient cistern (*lower*)

suggesting vascular injury in the spinal canal or subarachnoidal hemorrhage. The drainage tube was removed. Conjugate right upper deviation of the eyes appeared. In addition, systemic convulsive attacks with ventricular tachycardia developed. Treatment with a DC shock of 100 J achieved sinus rhythm. Immediately, 200 $mg \cdot h^{-1}$ continuous infusion of thiamylal sodium was started. Contrast-enhanced computed tomography (CT) of the brain (Fig. 1) performed on the same day revealed a low-density area suggesting air in the left cerebral hemisphere. Furthermore, a high-density area was noted at the quadgeminal and ambient cistern. However, CT at the spinal cord level did not reveal any remarkable changes. On the second postoperative day, brain CT combined with angiography did not reveal arterial hemorrhage, cerebral aneurysm, or cerebral arteriovenous malformation. Venous subarachnoidal hemorrhage was suggested. As surgical treatment was not indicated, cerebral pressure control therapy with thiamylal sodium and mannitol was initiated. On the fourth postoperative day, infusion of thiamylal sodium was discontinued. Thereafter, the consciousness level improved rapidly. On the sixth postoperative day, a tracheal tube was removed. The patient was discharged from the ICU on the eight postoperative day without neurological sequelae.

Discussion

With respect to the diagnosis of venous hemorrhage, it is difficult to detect venous hemorrhage by cerebral angiography from the perspective of dilution of contrast medium. Therefore, venous hemorrhage is suggested when the possibility of arterial hemorrhage is denied. Of course, even in patients with arterial hemorrhage, it is generally difficult to detect the hemorrhage by angiography when the volume of blood loss is 0.5 $ml \cdot min^{-1}$ or less. Therefore, the possibility of arterial hemorrhage cannot be completely excluded in our patient. However, if arterial hemorrhage in the subarachnoidal region had occurred during extracorporeal circulation with heparin, a more serious cerebral disorder would have developed than was evident in our patient. In our case, injury or air embolism related to excessive pressure on a portion of the venous wall during retrograde cerebral perfusion may have caused brain infarction due to venous hemorrhage.

Subarachnoidal venous hemorrhage is not common. In the neurosurgical field, traumatic subarachnoidal hemorrhage is detected on brain CT. However, angiography fails to demonstrate arterial hemorrhage. In some patients, subarachnoidal hemorrhage develops slowly, shows relatively mild symptoms, and follows a good prognosis. In our case, conservative cerebral protection therapy achieved a favorable course without neurological abnormalities.

It is difficult to prove the relationship between retrograde cerebral perfusion and subarachnoidal venous hemorrhage. With our patient, bloody cerebrospinal fluid and systemic convulsive attacks with ventricular tachycardia were noticed on the morning of the first postoperative day. A rise in intrathoracic pressure accompanied by convulsive attacks may have triggered subarachnoidal venous hemorrhage. Considering that the patient's consciousness was not clear on emergence from the anesthesia in the night of the operative day, and that a time lag can exist between subarachnoidal arterial hemorrhage and the finding of bloody cerebrospinal fluid, we believe that inappropriate retrograde cerebral perfusion caused subarachnoidal venous hemorrhage.

Retrograde cerebral perfusion is used during surgery of the aortic arch or the ascending thoracic aorta [2,5,6]. In some patients undergoing surgery of the descending thoracic aorta, retrograde cerebral perfusion is performed because this has the advantage that aortic cross-

clamping is not required on the proximal side of an aortic aneurysm. Complications of the central nervous system during the perioperative period in patients undergoing retrograde cerebral perfusion have been reported, including delayed awakening, dysphemia, quadriplegia, and cerebral infarction [1,2]. The safety of this perfusion procedure, the efficacy of cerebral protection, and the limit of perfusion time remain controversial [4,7]. In normal cerebral circulation, intracranial venous pressure is similar to intracranial pressure. However, when retrograde cerebral perfusion increases intracranial pressure beyond the limit of the compensatory mechanism, cerebral circulatory disorder may occur. In animal experiments [3,4,7–9], a marked deviation in the distribution of cerebral blood perfusion was demonstrated when the retrograde cerebral perfusion pressure was over 40 mmHg. A rapid increase in venous pressure causes dilatation of capacitance vessels beyond the permissible range, and increased intracranial pressure-compensating mechanisms reach their limits. In addition, the occlusive mechanism of the vascular bed, including venules, causes a blocking of blood flow in some areas, while perfusion pressure induces excessive blood flow in other areas, thus resulting in an uneven distribution of cerebral perfusion. In contrast, when perfusion pressure is 10 mmHg, effective cerebral perfusion may not be achieved. It is also speculated that the internal jugular venous valve is involved in this mechanism. Appropriate perfusion conditions remain to be clarified in many respects. In this patient, the perfusion method from the superior vena cava, which is used for retrograde cerebral perfusion during surgery of the aortic arch, was not performed, but an efferent blood vessel from the femoral vein was placed in the right atrium. The perfusion flow ($600\,ml \cdot min^{-1}$) was slightly larger than that of routine perfusion from the superior vena cava. However, considering blood flow to the subclavian vein or inferior vena cava, the flow was not too large for blood flow to the brain. However, in our case, neither internal jugular venous pressure nor brain tissue oxygen saturation was monitored during cerebral perfusion. Such monitoring might have been valuable in this patient.

Subarachnoidal venous hemorrhage may be a rare complication, but early recognition and treatment for possible cerebrovascular disorders is important in patients undergoing retrograde cerebral perfusion.

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