Postpartum Dissecting Aneurysm of the Superior Cerebellar Artery
—Case Report—

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Abstract

A 37-year-old female with toxemia of pregnancy suffered sudden headache and loss of consciousness on the day following a cesarean delivery. Computed tomography revealed subarachnoid hemorrhage (SAH). Vertebral angiography revealed a fusiform dilatation near the origin of the right superior cerebellar artery (SCA) with distal luminal narrowing. She underwent surgery within 24 hours of the ictus. A SAH clot was carefully removed from the prepontine cistern, and subadventitial discoloration was seen in the wall of the right SCA just distal to the aneurysmal protuberance (rupture site). The dissecting aneurysm was treated with body clipping by directly clipping the rupture site and with additional wrapping of the proximal SCA, including the aneurysmal protuberance and discolored site. The postoperative clinical course was uneventful. Postoperative angiography revealed complete obliteration of the aneurysm and patency of the SCA. Therapeutic intervention should be considered for patients with ruptured dissecting aneurysm who present with recurrent SAH.

Key words: cerebral aneurysm, superior cerebellar artery, dissection, subarachnoid hemorrhage

Introduction

Dissecting aneurysm is recognized as a common cause of subarachnoid hemorrhage (SAH) or brainstem ischemia. Vertebrabasilar trunk lesions are the most common dissecting aneurysms in the posterior fossa, whereas lesions located in the peripheral arteries of the posterior inferior cerebellar artery and posterior cerebral artery (PCA) are less common. Dissecting aneurysms of the superior cerebellar artery (SCA) are extremely rare, with only one such case reported. Surgical treatment has been successful in many cases of dissecting aneurysms of the posterior circulation. We treated a postpartum female with a ruptured dissecting aneurysm of the SCA by surgery.

Case Report

A 37-year-old female became pregnant by in vitro fertilization and embryo transfer. At 35 weeks of gestation, she was admitted to our hospital due to intrauterine growth retardation of the fetus. At 37 weeks of gestation, edema of the lower extremities, proteinuria, and hypertension of over 160/80 mmHg were present, and a cardiotocogram revealed fetal distress. She underwent cesarean section under low spinal epidural anesthesia and delivered a female baby weighing 1898 g. During the delivery, she complained of headache but was awake, alert, and coherent, with no focal neurological deficit. The next day, her blood pressure was around 160/80 mmHg. In the evening, she suddenly complained of severe headache and lost consciousness. Her blood pressure rose to 180/90 mmHg.

Computed tomography revealed SAH, primarily in the suprasellar, prepontine, and right ambient cisterns (Fig. 1). Just afterwards, she suddenly entered a deep comatose state and rapidly developed bilateral dilated and non-reactive pupils and decerebrate posture with only the bilateral corneal reflex. The next day, she recovered consciousness and became stuporous only with right oculomotor nerve paresis (Hunt and Kosnik grade IV). Vertebral angiography revealed a fusiform dilatation of the
right SCA just beyond its origin, with distal luminal narrowing (Fig. 2 left). Carotid angiography revealed an infundibular dilatation of the right internal carotid artery at the origin of the posterior communicating artery (ICPC). The tentative diagnosis was a ruptured dissecting aneurysm of the SCA.

On the same day, exploratory surgery using the right pterional approach was performed within 24 hours of the second ictus. A catheter was inserted to reduce intracranial pressure and enable ventricular drainage. Opening of the dura disclosed a thin subdural hematoma, and the sylvian fissure and suprasellar and prepontine cisterns filled with a massive SAH clot. No aneurysm was found in the ICPC portion, and only an infundibular dilatation of the PCA. After the SAH clot in the prepontine cistern was carefully removed through the carotid-oculomotor triangular space, subadventitial discoloration was seen in the wall of the right SCA, just distal to the aneurysmal protuberance and directly behind the right oculomotor nerve. The aneurysmal protuberance of the SCA was considered to be the rupture site. The dissecting aneurysm was treated with body clipping by directly clipping the rupture site, and with additional wrapping.

Fig. 1 Computed tomography scan showing subarachnoid hemorrhage in the basal cisterns after the first ictus.

Fig. 2 Vertebral angiograms (oblique view) before the operation (left) revealing a fusiform dilatation of the right superior cerebellar artery (SCA) with distal luminal narrowing (arrow) indicating a dissecting aneurysm, and after (right) showing complete obliteration of the aneurysm and the patent SCA (arrow).

Fig. 3 Schematic drawing showing the dissecting aneurysm treated with body clipping by directly clipping the rupture site, and with additional wrapping.
and by additional wrapping of the proximal SCA, including the aneurysmal protuberance and discolored site (Fig. 3). A Doppler flowmeter was used for intraoperative monitoring of the blood flow to confirm the patency of the SCA.

Her postoperative clinical course was uneventful except for a delayed ischemic neurological deficit of transient left hemiparesis. Postoperative angiography revealed complete obliteration of the aneurysm and the patent SCA (Fig. 2 right). She was discharged on foot with no oculomotor nerve paresis 2 months after the ictus. Her child survived and is in good health.

**Discussion**

Postpartum rupture of a dissecting aneurysm as in the present case has only been reported in a case of postpartum vertebrobasilar artery dissection, although intracranial hemorrhage from cerebral aneurysms during pregnancy and puerperium are well known.

The etiology of dissecting aneurysms remains unclear, but is known to be related to atherosclerosis and degenerative disease of the vessel wall. Defects of the internal elastic lamina may be the origin of dissection in the wall of the intracranial artery, particularly if congenital or acquired medical abnormalities are present. Other contributing factors may be involved in the development of this lesion, including hypertension, delivery, various kinds of arterial disease, and anatomical characteristics of the artery. Although dissecting aneurysms occur primarily in the intracranial vertebrobasilar trunk, the changes undergone by the vertebral artery after penetrating the dura may be important, such as the reduction in the thickness of the adventitial and medial coat, and the very significant reduction or loss of elastic fibers in the medial and external lamina. Dissecting aneurysms involving the intracranial peripheral arteries predominantly occur in relatively young patients, in whom no underlying abnormalities are present in most cases to explain the appearance of the dissection of an artery (Table 1). This vascular abnormality has been related to migraine, fibromuscular dysplasia, mixed connective-tissue disease, angitis, and trauma.

In our case, the cause of the SCA dissection was unclear, but probably a congenital wall defect or acquired angiopathy was present triggered by an initiating factor such as the hemodynamic changes that occur during pregnancy and probably enhanced by the associated toxemia of pregnancy, dramatic but short-lived hemodynamic stress brought on by hypertension occurring during labor and delivery, or other physiological changes, such as elevation of progesterone and human chorionic gonadotropin and secretion of relaxin (a gestational hormone that relaxes the pelvic ligaments during delivery).

Dissecting aneurysms in the peripheral arteries of the posterior circulation are primarily located in the proximal portion of arterial branches (Table 1). Preaneurysmal changes, areas of arterial wall thinning and small arterial evagination, may be accentuations of the degenerative changes naturally occurring in the region of the branching. These degenerative lesions are likely to be the most important factor in the formation of a saccular aneurysm if evagination progresses, and also in the development of dissection if arterial wall disruption occurs.

The indications for surgical treatment of the dissecting aneurysms remain controversial. Dissecting aneurysms of the anterior circulation tend to manifest as ischemic symptoms, while those of the posterior circulation are more likely to lead to SAH. Direct and endovascular surgical treatment is recommended because of the high rate of recurrent hemorrhage and the extremely poor outcome in patients with ruptured dissecting aneurysm of the posterior circulation. However, there are some arguments against surgical treatment. Spontaneous evolution of a dissecting aneurysm of the PCA indicates the need for conservative management in the absence of neurological deterioration. However, surgical intervention was required in the acute stage in our case because of the recurrent hemorrhage. We consider that surgery should be considered for patients who present with recurrent SAH or angiographic progression of the dissection and that the initial decision to administer surgical treatment may be unadvisable in neurologically stable patients who present with ischemia or no recurrent hemorrhage as such cases may heal spontaneously and/or with anticoagulant therapy.

Surgical treatment of dissecting aneurysms includes excision, trapping, proximal ligation, and reinforcement with or without intracranial-extracranial bypass but the optimal surgical procedure has not yet been established. Aneurysmal excision or trapping seems to be the only effective means of managing of ruptured dissecting aneurysms, but revascularization procedures or preservation of the blood flow of the parent artery were necessary in our case, as the aneurysm was located in the proximal region of the parent artery and there was poor collateral circulation to the ar-

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terial territory distal to the aneurysm. We repaired the ruptured dissecting aneurysm with body clipping by directly clipping the ruptured site of the aneurysm,\(^\text{10}\) and with additional reinforcement by wrapping. The most appropriate surgical procedures should be selected for patients who present with recurrent SAH or angiographic progression of the dissection based on the aneurysm location, the anatomical relation to the parent arteries, the range of the dissection, and the collateral circulation.

This case of postpartum ruptured dissecting aneurysm of the SCA was successfully treated by surgery. Therapeutic intervention should be considered for patients with a ruptured dissecting aneurysm who present with recurrent SAH.

References

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