Dissecting aneurysms arising from the vertebrobasilar complex are extremely rare, and little is known about their natural history.\textsuperscript{1,2} Few patients have been reported since the initial description by Scholefield in 1924.\textsuperscript{3-6} Patients present with headache, followed by nausea, vomiting, and unconsciousness. Occurrence of subarachnoid hemorrhage (SAH) was the most important reason for hospitalization.\textsuperscript{7}

The etiology of intracranial dissecting aneurysms remains obscure, but they have been associated with manipulation, trauma, migraine, oral contraceptive use, syphilis, polyarteritis nodosa, fibromuscular dysplasia, and mucoid degeneration of the media. Unusual exertion has been implicated as a factor in some cases. No case of immediate postpartum vertebrobasilar artery dissection has been reported before, although intracranial hemorrhage from aneurysms and arteriovenous malformations during puerperium is reported.\textsuperscript{8}

This case is reported not only to describe the clinical and pathognomonic radiological findings, but also to emphasize the spontaneous evolution and natural history of the lesion without surgery.

Case Report

A 31-year-old primigravida without specific medical history was admitted after 41 weeks of amenorrhea. She had never used oral contraceptives or any other medication. A spontaneous, uncomplicated vaginal delivery, during which a systolic blood pressure of 220 mm Hg was recorded, followed after 2 hours of labor. During this period she experienced double vision.

Four hours after delivery, she suddenly became nauseated and developed a constant occipital headache, without neck stiffness. Moderate confusion, cerebellar ataxia, and Horner’s sign on the right side were observed. The patient became more confused and was transferred to our hospital. By that time, she felt dizzy, had severe vertigo, and experienced difficulty talking and swallowing. Neurological examination revealed ipsilateral ataxia, cerebellar nystagmus, Horner’s sign on the right side, and hypalgesia on the right side of the face and the left side of the body. There was a left visual field deficit and a slight motor dysfunction on the left. We also noticed a right-sided facial weakness of the central type and a deviation of the soft palate to the left. These findings indicated major right brain stem dysfunction. Her optic discs were clear, without evidence of papilledema. Clear cerebrospinal fluid was obtained by lumbar puncture. Other findings included a blood pressure of 158/90 mm Hg, a regular pulse of 72 beats/min, a respiratory rate of 20 breaths/min, and a white blood cell count of 14,800/mm\textsuperscript{3}.

A computed tomographic scan of the brain was normal. Magnetic resonance imaging 1 day later revealed right-sided ischemic lesions in the thalamus, posterior limb of the internal capsule, and occipital lobe (Figure 1). It also indicated an ischemic lesion in the distribution of the right posterior inferior cere-
bilar artery. Bilateral vertebral angiography demonstrated a dissecting aneurysm of the basilar artery, with a double lumen as a pathognomonic finding, and a thrombosis of the intracranial segment of the right vertebral artery proximal to and at the origin of the right posterior inferior cerebellar artery (Figure 2).

The patient remained clinically stable, and the neurological examination was unchanged for the next 48 hours. She was treated conservatively with 500 mg ticlopidine daily.

One month later, she was discharged without neurological signs, but with slight left-sided hypesthesia. Arteriography 2 months after the dissection disclosed a normal basilar artery and an occluded, thrombosed right vertebral artery.

**Discussion**

Fusiform aneurysms in the vertebrobasilar system are reported to frequently cause SAH, without being specified as dissecting lesions. Dissection is believed to occur between the media and adventitia of the arterial wall. Gap defects in the internal elastic lamina, because of their unusual size and number, may be responsible for initiating dissection between the internal elastic lamina and the media. This initial event could be demonstrated in most other cerebral and extracranial vessel dissections. Arteries change morphology as they penetrate the dura. In the vertebrobasilar system, subsequent rupture into the subadventitial plane and subarachnoid space may occur. Some authors believe that intimal cushions, formed at branch points in the cerebral circulation by splitting of the internal elastic lamina, may serve as a starting point for dissection, especially if an underlying medial defect is present. Reported causative factors are manipulation of the neck as the most prominent factor in traumatic dissection, and congenital medial defects, syphilitic arteritis, cystic medial degeneration, arteriosclerosis, allergic arteritis, and variant periarteritis nodosa as factors in nontraumatic dissection. There is strong evidence that exertion, such as during delivery, can be regarded as an initiating factor. Ten case reports of intracranial hemorrhage from aneurysms of the vertebrobasilar system during pregnancy and puerperium have been described in the English literature, but no case of documented dissecting aneurysm has been reported.

Subarachnoid hemorrhage and posterior circulation ischemia are two major clinical presentations. Posterior fossa mass effect is rare. The most common initial symptom of vertebrobasilar dissection, before the onset of brain stem dysfunction, is headache. The pain is usually in the occipital and posterior cervical regions. No pathognomonic clinical sign or symptom of vertebrobasilar dissection has been identified, but rapidly evolving brain stem dysfunction is the most common clinical presentation. Occasionally, the stroke is not complete at its onset, but proceeds in a stuttering fashion. Indeed, our patient had a short period of double vision during delivery.

No common specific angiographic finding has been reported in vertebrobasilar dissection. The diagnosis has to be made in conjunction with the clinical presentation. In our case, however, a double lumen, representing the passage of contrast material into
both true and false channels, must be interpreted as the only pathognomonic, but extremely rare, finding of vertebrobasilar dissection. An occlusion of the right intracranial segment of the vertebral artery was noticed and may be interpreted as an intramural clot, large enough to occlude not only the vessel lumen itself but also the origin of the right posterior inferior cerebellar artery. The occlusion probably protected the aneurysm from further dissection or even rupture by reducing the blood flow in the basilar artery (the left vertebral artery was hypoplastic).

Most authors recommend exploration of vertebrobasilar dissecting aneurysms in patients with or without SAH who are clinically stable. Some vessels will heal spontaneously, but a review of the literature reveals that the dissection can progress. The Hunterian approach of ligating the artery proximal to the origin of the posterior inferior cerebellar artery or a dissecting aneurysm distal to its origin that does not involve the vertebrobasilar junction can be trapped intracranially to prevent collater al flow from cervical muscular branches. If the dissection involves the origin of the posterior inferior cerebellar artery, an extracranial bypass can be performed. If the dissection extends into the basilar artery, a proximal vertebral artery ligation may prevent further dissection, but should be discouraged if the contralateral vertebral artery is hypoplastic or not patent. This situation describes what happened in our patient without surgery and with good clinical outcome. Probably, this operative procedure is safe even with a contralateral patent but hypoplastic artery. Berger and Wilson recommend reinforcing (wrapping) as much of the basilar artery as possible. They reported a patient with cerebellar and brain stem infarction after a trapping procedure and a patient with a hemiparesis after excision of a distal posterior cerebral artery dissection. The ligation procedure seemed to us unjustified in a patient in stable clinical condition without SAH and with a perfect occlusion of the intracranial segment of the right vertebral artery protecting the aneurysm from further dissection or even rupture. The thrombosed right vertebral artery itself can be interpreted as a seal on the origin of the dissection plane and, as such, prevents further dissection. Angiography 2 months after the dissection demonstrated that the dissection had resolved but that the right vertebral artery still was occluded.

Anticoagulation therapy for intracranial dissection has not been widely advocated because of the potential danger of further dissection, rupture, or hemorrhage. Aspirin or ticlopidine may be useful in reducing platelet aggregation.

Our patient is the second reported case of apparent spontaneous healing with complete neurological recovery. The few previously reported cases with a morbid outcome may not be representative of the disease. For the lesions that occur on the vertebral artery, spontaneous proximal occlusion appears to be a safe and successful mechanism for producing oblitative thrombosis of the dissected segment. More of the natural history must be known before it can be decided whether surgical or conservative treatment is indicated.

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