

Unilateral Carotid and Vertebral Artery Dissections and Contralateral Subarachnoid Hemorrhage in a Postpartum Patient

Peiyuan F. Hsieh^{1,2,3}, Yi-Chung Lee^{1,2}, and Ming-Hong Chang^{1,2}

Abstract- Postpartum arterial dissection combined with subarachnoid hemorrhage (SAH) is rare and its mechanism is uncertain. A 32 year-old woman had a delivery by cesarean section 12 days prior to admission to our hospital. From the first day of delivery, she breast-fed her baby, sitting with her head always turned to the right. Each feeding lasted around 2 hours. A bilateral throbbing headache began two days after childbirth, and intermittent numbness of the right face, chest and hand as well as weakness of the right hand developed nine days after giving birth. A physical examination revealed transient mild hypertension and right hemiparesis. Her cholesterol ranged from 204 to 263 mg/dl. Computed tomography, magnetic resonance angiography and duplex ultrasound disclosed left fronto-parietal junction SAH and dissections of the right internal carotid (ICA) and vertebral arteries. Our patient demonstrated (1) that postpartum arterial dissection was not limited to natural delivery, (2) postpartum SAH could occur with dissections of the contralateral extracranial carotid and vertebral arteries, and (3) that turning one's head always to the same side during breast-feeding might be a risk factor for this unusual stroke pattern.

Key Words: Postpartum, Breast-feeding, Arterial dissection, Subarachnoid hemorrhage

Acta Neurol Taiwan 2008;17:94-98

INTRODUCTION

During pregnancy and the puerperium, the incidence of stroke increases threefold to 13-fold⁽¹⁾. The estimated risk of a peripartum stroke is 13.1 cases per 100,000 deliveries⁽²⁾. Of approximately 50,700 admissions for delivery, 34 patients with a diagnosis of stroke were identified (21 infarctions and 13 hemorrhages)⁽³⁾.

Possible explanations for this increased incidence

include hormonal changes, dehydration and intrinsic hypercoagulation during pregnancy, vascular trauma during delivery, and the contraction of the blood volume postpartum.

Although stroke and carotid artery dissection are not frequent causes of headache during pregnancy and the postpartum period, they should be included in the differential diagnosis. We report on the combined carotid and vertebral artery dissections superimposed with contralat-

From the ¹Division of Neurology, Taichung Veterans General Hospital, ²Department of Internal Medicine, National Yang-Ming University, and ³Graduate Institute of Biomedicine and Biomedical Technology, National Chi-Nan University, Taiwan. Received May 4, 2007. Revised October 3, 2007. Accepted January 7, 2008.

Reprint requests and correspondence to: Peiyuan F. Hsieh, MD. Division of Neurology, Taichung Veterans General Hospital. No. 160, Sec. 3, Taichung-Kang Rd., Taichung, Taiwan. E-mail: pfhsieh@vghtc.gov.tw

eral subarachnoid hemorrhage (SAH) in a postpartum patient.

CASE REPORT

A 32 year-old woman had an uneventful delivery by cesarean section under general anesthesia, due to an 8-cm uterine myoma, at a local hospital 12 days prior to her admission to our hospital. She started breast-feeding on the day of delivery. Each feeding lasted around 2 hours and occurred once every 2-4 hours. However, a progressive bilateral headache started two days after childbirth. The headache was throbbing, located over her bilateral temporal areas, sometimes sub-occipital, and persisted all day. Mild photophobia and nausea were also noted. Then intermittent numbness developed nine days after childbirth and proceeded from the right side of the face to the chest and down to the right hand, accompanied with right hand weakness. The frequency of the symptoms was 1-2 times a day initially, which pro-

gressed to 3-4 times per day. A detailed history revealed that she always kept her head turned to the right to watch her baby during breast-feeding. Due to the above symptoms, she was referred to our emergency room (ER) for further medical care. At the ER, a blood pressure 152/86 mm Hg was noted. Computed tomographic (CT) of the brain disclosed minimal subarachnoid hemorrhage (SAH) over the left central sulcus and a mild effacement of the left sylvian fissure with adjacent tissue edema (Fig. 1A). Other laboratory data were normal. She was then admitted for further evaluation and treatment.

Throughout the course of the illness, there was no fever, chills, or ecchymosis. She also denied any history of head trauma, taking oral contraceptives, arthritis or photosensitivity. She did not abuse drugs nor did she have an allergy history.

She had been healthy, without any systemic disease, except for resection of a uterine myoma 8 years before and ultrasound treatment for a right ureteral stone 2 years previously. She had a pregnancy history of gravida

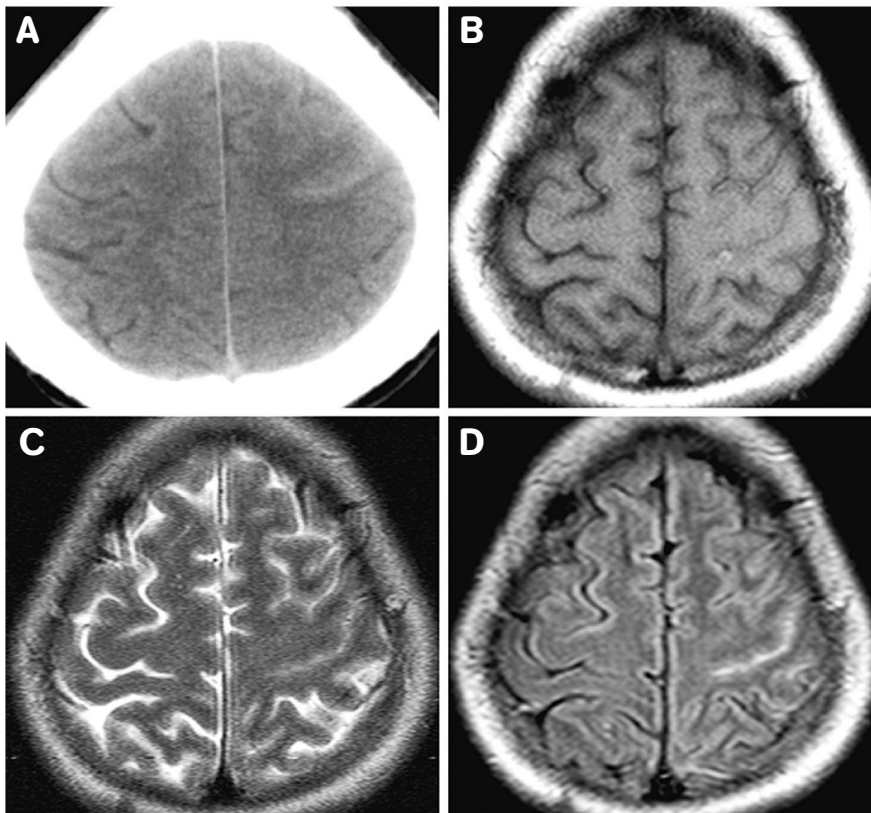


Figure 1. (A) CT shows minimal SAH at the left central sulcus. MRI discloses left fronto-parietal junction SAH: (B) T1-weighted, (C) T2-weighted, (D) fluid-attenuated inversion recovery.

3, para 2, and one spontaneous abortion.

Her family history revealed a cerebrovascular accident (CVA) in her grandfather, a transient ischemic attack in her father, and 5 otherwise normal brothers and sisters.

A physical examination revealed a body weight of 64 kg and height of 157 cm. Her vital signs were: blood pressure 174/98 mmHg, pulse rate 96/min, respiration rate 20/min, body temperature 37.2°C on admission. Blood pressure normalized during hospitalization and was 120/80 mmHg without any anti-hypertensive medication at discharge. A contrast medium-related mild generalized military itchy skin rash lasted 2 days. There was no abnormality of the head, eyes, ears, throat, neck, chest, heart, abdomen, back, spine, genitals or anus. The only abnormality revealed on neurological examination was right hemiparesis with muscle power grade 4. Consciousness, judgment, orientation, memory, abstract thinking, calculation, language, cranial nerves, deep tendon reflexes, plantar reflexes, coordination, equilibrium, gait and sensation were all normal.

Cholesterol was 204 mg/dl on admission and 263 mg/dl 1 week later. Routine laboratory data including complete blood counts, glucose, triglyceride, liver function, blood urea nitrogen, creatinine, electrolytes, pro-

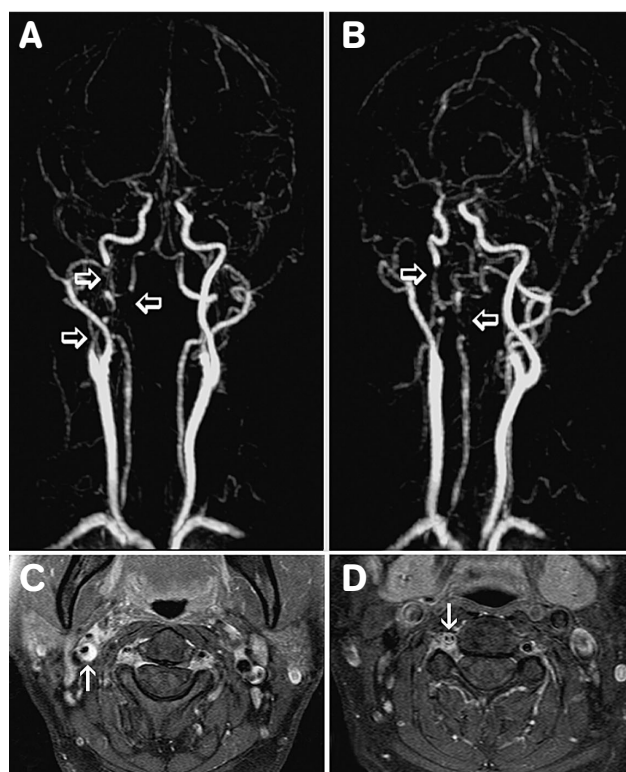


Figure 2. MRA shows irregular surface and severe stenosis over right internal carotid artery and vertebral artery due to dissections: (A) antero-posterior view, and (B) oblique view. Post-contrast T1-weighted MRI shows double lumens of (C) the right internal carotid artery and (D) the right vertebral artery.

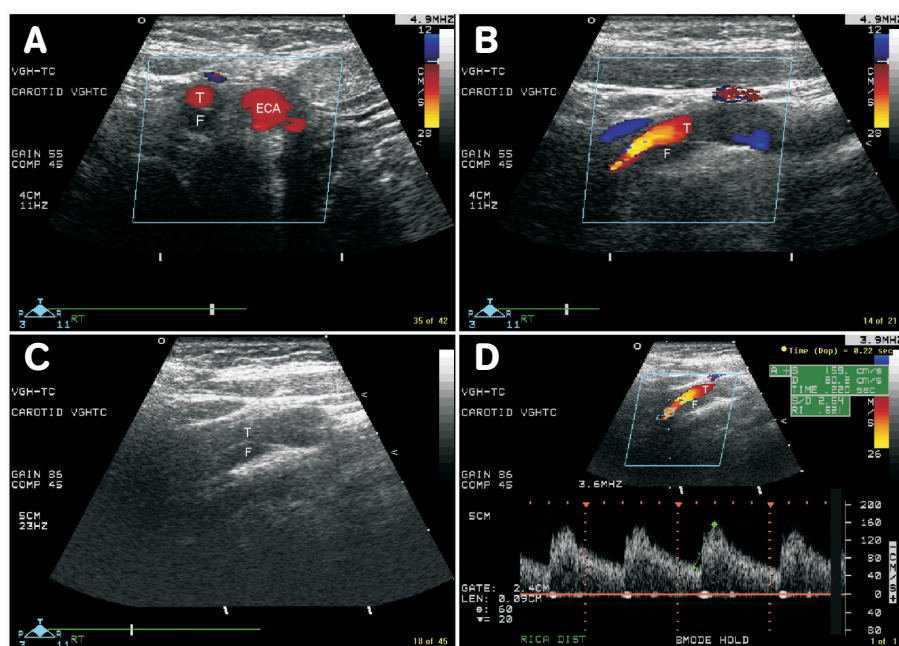


Figure 3. Carotid duplex shows the homogenous, smooth surface, hypoechoic false lumen caused eccentric 60 to 70 % diameter stenosis of the right proximal ICA. (A) Cross section. (B-D) Longitudinal section. (D) The flow velocity was 159/60 cm/sec in the true lumen. ECA: external carotid artery. F: false lumen. T: true lumen.

thrombin time, activated partial thromboplastin time, bleeding time and homocysteine were normal. Rapid plasma reagin, anti-nuclear antibody, anticardiolipin antibody (IgG, IgM), anti-neutrophil cytoplasmic antibody, lupus anticoagulant and anti-beta 2-glycoprotein I (IgG & IgM) were negative. Her chest X-ray and electrocardiogram were normal. A gynecological ultrasound showed a uterine myoma measuring 8 cm in diameter.

Brain and neck magnetic resonance angiography (MRA) disclosed a left fronto-parietal junction SAH, irregular surface and severe stenosis of the right internal carotid artery (ICA) and vertebral artery due to dissection (Figs. 1-2). Neither vascular malformation nor aneurysm was detected. A carotid duplex showed a homogenous, smooth surface, hypoechoic false lumen caused eccentric 60 to 70 % diameter stenosis of the right proximal ICA (flow velocity 159/60 cm/sec), compatible with arterial dissection (Fig. 3).

After admission, the patient was administered nimodipine to prevent vasospasm. Glycetose was used to decrease the intracranial pressure. Acetylsalicylic acid and Cerenin (extract Ginkgo Biloba) were prescribed instead of an anti-coagulant due to the presence of SAH. Atorvastatin was given to lower serum cholesterol. Cardiologists were consulted but they did not suggest carotid stenting due to technical difficulties. Also, the patient refused digital-subtraction angiography or interventional procedures because she just experienced contrast medium allergy and her symptoms improved rapidly in a few days. She recovered completely by the time she was discharged on the 9th day of hospitalization.

DISCUSSION

There are three important things to note in our patient. First, she always turned her head to the same side during each 2-hour period of breast-feeding. Secondly, she had dissections of carotid and vertebral arteries as well as a contralateral SAH. Thirdly, her postpartum arterial dissection occurred after a pre-planned cesarean section.

Another peculiar point worth mentioning in our patient is that her SAH was contralateral to the dissec-

tion of the carotid and vertebral arteries. There have been no reports of similar cases so far as we are aware. We cannot explain why our patient had SAH contralateral to the carotid and vertebral arteries, and, as our patient was allergic to the contrast medium, a digital-subtraction angiography was not performed. Hypercholesterolemia is a risk factor of stroke but it cannot explain the whole picture of our patient. Further observational studies are needed to answer this question.

Painful spontaneous cervical artery dissection occurs in 72% of patients with frontal and parietal localizations being significantly associated with internal carotid artery dissection, whereas occipital and nuchal pain occurring with vertebral artery dissection⁽⁴⁾. The headache of our patient was similar to that report and was located at the temporal and sub-occipital areas due to involvement of both carotid and vertebral arteries.

In a pregnancy and stroke survey, 13 patients with infarction were arterial and 8 were venous. Nine of 13 arterial events occurred in the third trimester or puerperium. Seven of 8 venous occlusions occurred postpartum. An etiologic diagnosis was made in 7 of 13 patients with arterial territory infarction, including cardiac emboli, coagulopathies, and carotid artery dissection. Of patients with hemorrhage, 7 were subarachnoid and 6 were intracerebral. The etiology was identified in 10 patients: 3 were due to ruptured aneurysms, 5 were associated with arteriovenous malformations, and 2 were associated with disseminated intravascular coagulation⁽³⁾.

The occurrence of spontaneous internal carotid or vertebral artery dissection after childbirth remains rare. There have been 13 previously reported cases of postpartum arterial dissections associated with stroke; 8 were of the internal carotid artery, 2 were of the basilar artery, 2 were of the vertebral artery, and 1 was of both the internal carotid and the vertebral arteries^(1,5-13).

In the medical literature of 2003, a 37-year-old woman was reported who had a cesarean section due to 20 hours of unsuccessful labor. Nine days later, she suffered from cerebral infarction in the territory of middle cerebral artery due to a dissection of the left internal carotid artery. A few days after the stroke, however, the patient developed additional dissections of the right

internal carotid artery and both vertebral arteries. Pregnancy, childbirth, and a history of rheumatoid arthritis in this patient may have contributed to the dissections⁽¹²⁾.

And in a 2004 case report, a 35-year-old woman presented to the emergency ward complaining of headaches for 3 days. Her medical history included a delivery of a full-term infant by cesarean section 9 days before presentation due to an arrest of descent after 3 hours of labor. Symptoms were later attributed to a parietal lobe infarction and bilateral internal carotid artery dissection. Intimal injury related to the Valsalva maneuver and straining during vaginal delivery was presumed to be the cause of carotid artery dissection, because no additional contributing factors were identified⁽¹³⁾.

It is well known that neck manipulation can cause arterial dissections. Our patient demonstrated an association between the sustained head position and arterial dissections as well as SAH that has not, until now, been reported in the literature. Further clinical studies are warranted in order to affirm whether a sustained head position in breast-feeding is a risk factor for postpartum arterial dissection and/or SAH.

Vascular trauma may occur in natural deliveries because the mother has to perform a Valsalva maneuver to push the fetus from the uterus. This may explain some patients with postpartum arterial dissections. However, this possibility can be ruled out in our patient who delivered the baby through pre-planned cesarean section because of a uterine myoma.

Treatment of carotid artery dissection includes intravenous heparin to prevent thrombus formation on the injured endothelial surface and to avoid secondary thrombosis and embolism. However, anticoagulant treatment is contraindicated in patients with intracranial hemorrhage, as in our patient.

A long-term follow-up study after a carotid artery dissection showed 50% of patients were neurologically normal, 21% showed mild deficits, and 25% showed moderate to severe deficits. Four percent of the reported patients died⁽¹⁴⁾.

In conclusion, physicians should consider the possibility of arterial dissection in women presenting with acute ischemic stroke in the postpartum period. At pre-

sent, we would also like to suggest that new mothers be advised to change their head positions frequently during breast-feeding.

REFERENCES

1. Sharshar T, Lamy C, Mas JL. Incidence and causes of strokes associated with pregnancy and puerperium. A study in public hospitals of Ile de France. Stroke in Pregnancy Study Group. *Stroke* 1995;26:930-6.
2. Lanska DJ, Kryscio RJ. Risk factors for peripartum and postpartum stroke and intracranial venous thrombosis. *Stroke* 2000;31:1274-82.
3. Jaigobin C, Silver FL. Stroke and pregnancy. *Stroke* 2000;31:2948-51.
4. Campos CR, Calderaro M, Scaff M, et al. Primary headaches and painful spontaneous cervical artery dissection. *J Headache Pain* 2007;8:180-4.
5. Gasecki AP, Kwiecinski H, Lyrer PA, et al. Dissections after childbirth. *J Neurol* 1999;246:712-5.
6. Kittner SJ, Stern BJ, Feaser BR, et al. Pregnancy and the risk of stroke. *N Engl J Med* 1996;335:768-74.
7. Bruninx G, Roland H, Matte JC, et al. Carotid dissection during childbirth. *J Mal Vasc* 1996;21:92-4.
8. Lepojärvi M, Tarkka M, Leinonen A, et al. Spontaneous dissection of the internal carotid artery. *Acta Chir Scand* 1988;154:559-66.
9. Perier O, Cauchie C, Demanet JC. Intramural hematoma caused by parietal dissection ('dissecting aneurysm') of the basilar trunk. *Acta Neurol Belg* 1964;64:1064-74.
10. Wiebers DO, Mokri B. Internal carotid artery dissection after childbirth. *Stroke* 1985;16:956-9.
11. Van de Kelft E, Kunnen J, Truyen L, et al. Postpartum dissecting aneurysm of the basilar artery. *Stroke* 1992;23:114-6.
12. Oehler J, Lichy Ch, Gandjour J, et al. Dissection of four cerebral arteries after protracted birth. *Nervenarzt* 2003;74:366-9.
13. Abisaab J, Nevadunsky N, Flomenbaum N. Emergency department presentation of bilateral carotid artery dissections in a postpartum patient. *Ann Emerg Med* 2004;44:484-9.
14. Stapf C, Elkind MS, Mohr JP. Carotid artery dissection. *Annu Rev Med* 2000;51:329-47.