CASE REPORT

Postpartum vertebral artery dissection

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SUMMARY

We report a case of a right vertebral artery dissection in a 35-year-old woman, 3 weeks post partum, with manifestations of vertebrobasilar disease. She was 3 weeks out from the uneventful delivery of her fourth child, with presentation of acute neurological symptoms, predominantly intractable vertigo. Vertigo can have many non-specific generalised symptoms and clinical findings. Postpartum women have a lengthy list of possible aetiologies of vertigo not limited to our initially suspected preeclampsia, dural venous thrombosis and vertebral dissection.

BACKGROUND

Vertebral artery dissections (VADs) have shown to be a known common cause of stroke in young adults. Most VADs occur spontaneously, and can occur secondary to minor or severe trauma.¹ The symptoms of vertebral dissections are pain, most often in the posterior part of the neck or occiput, spreading into the shoulder. Dizziness, diplopia and signs of lateral medullary or cerebellar infarctions can also manifest as symptoms of VAD.

Because the vertebrobasilar system is involved owing to posterior circulation and involves the vertebral artery, posterior circulation ischaemia rarely causes only one symptom and, rather, is responsible for a collection of symptoms depending on the area of ischaemia. Most commonly, vertebrobasilar disease is from ischaemia or embolism.²

CASE PRESENTATION

A 36-year-old G5P5 woman, 3 weeks after the uneventful delivery of her fifth child, was found lying on the floor by her daughter, on the morning of the patient's admission date. The patient's husband had been summoned and he had attempted to arouse the patient. After about 3 min, the patient was finally aroused enough to communicate that she could not feel her arms and that she had a terrible headache. She described her headache as 'an ice pick is in my brain'. Shortly afterwards, she developed intractable nausea and vomiting. Therefore, she was brought to our hospital via emergency medical services. The patient had a history of migraines for greater than 10 years and was not currently on any medications. She noted that her migraines were associated with an aura and photophobia, but she had never had nausea or vomiting. Her medical history also revealed hypothyroidism secondary to a thyroidectomy due to a benign tumour.

Family history showed that the patient's father had Guillain-Barré syndrome and her mother and sister both had a history of hypertension.

The patient was on no known medications, and had no known drug allergies. She had a history of smoking for 5 pack-years, but had quit 10 years prior. She had no history of alcohol abuse or illicit drug use.

Of note, the patient's family did relate that every time the patient had undergone an epidural for delivery, she had developed a 'bells palsy', which they described as left facial numbness and droop, and left upper extremity weakness. This had, each time, spontaneously resolved with discontinuation of the epidural.

During initial evaluation, the patient's vitals were stable apart from hypertension at 150/100 mm Hg. Complete blood count, comprehensive metabolic panel, urine drug screen and urine analysis were essentially normal. CT of the head was obtained in the emergency room and was negative.

The patient was unsuccessfully treated with antiemetics and non-steroidal anti-inflammatory drugs in the emergency room. On evaluation on the medicine floor, the patient was found to have no focal deficits; she was moderately photophobic but could open her eyes on command. Her primary symptoms were of severe headache and vertigo. To rule out preeclampsia, urine analysis was carried out, which was negative for protein on two concurrent dipstick tests. Glomerular filtration rate was calculated as >60 mL/min, and normal platelet values were noted at 290 000/μL.

On initial physical examination, there were no changes in speech, and the rest of the examination was relatively normal, apart from the active nausea and vomiting. Kernigs and Brudzinski's signs were both negative.

INVESTIGATIONS

A MRI/MR venography was obtained. There were noted diffusion defects within the cerebellar hemispheres bilaterally, with the left worse than the right. There was also a small acute infarction within the cerebellar vermis, as seen with isotropically diffusion-weighted imaging in [figure 1](#).

Neurology was consulted and the decision was made to start the patient on heparin and transfer her to the intensive care unit for closer monitoring. The patient began to show signs of increasing intracranial pressure with worsening nausea, vomiting, somnolence, hypertension and temperature elevation.

Therefore a MR angiography was obtained, which did show signs of a possible vertebral artery thrombosis.

DIFFERENTIAL DIAGNOSIS

On the basis of presentation, the differential was noted and not limited to what is listed in [table 1](#).

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Figure 1 MRI diffusion-weighted imaging.

TREATMENT

The patient was transferred to a neurosurgical facility, due to concerns of further neurological sequelae. She was transferred via helicopter with heparin drip, intravenous acetaminophen and a cooling blanket.

CT angiogram of the head and neck (figure 2) showed right VAD at the level of C1 extending to the dural entry with no signs of dural venous sinus or internal jugular vein thrombosis. There was also a noted variation of the patient's left vertebral artery (LVA), which originated from the proximal descending aorta. This was best visualised with three-dimensional reconstructed images, shown in figure 3.

On review of imaging by the multidisciplinary team, the patient was also noted to have right vertebral artery flow dominance. There was also a filling defect noted in the right vertebral artery, shown in figure 4.

OUTCOME AND FOLLOW-UP

The remainder of the patient's hospital stay was uneventful. Her anticoagulation was bridged to oral anticoagulation with warfarin. The patient was transitioned successfully to inpatient

Figure 2 Three-dimensional CT reconstruction imaging (dissection).

rehabilitation and then sent home. Given the nature of her infarcts, she underwent aggressive physical and occupational therapy for the next few months. To date, she still has minor right-sided deficits including difficulties with coordination, but has otherwise regained a functional status and is able to perform all activities of daily living, including taking care of her five children. Follow-up imaging was performed at 3 and 6 months following the case and showed no further evolution of dissection or ischaemia.

DISCUSSION

While the exact incidence of cervical artery dissection, including carotid artery dissection and VAD, is unknown, it is estimated

Table 1 Differential diagnosis

Presentation

Most common

Large artery atherosclerosis	Brief transient ischaemic attacks, often symptoms appear with situations that reduce blood pressure and positive orthostatics
Penetrating small artery disease	Pure motor strokes of the paramedian pons and/or weakness of the face and extremities without visual, sensory or cognitive changes
Embolism	Most often embolic strokes are located within the intracranial vertebral arteries, which leads to cerebellar infarcts. Symptoms include dizziness, vertigo, blurred vision, difficulty in walking and vomiting. Often the patient will continually drift, or turn to one side and will have nystagmus
Arterial dissection	Most common presenting symptom is posterior neck or occiput pain. If dissection extends into the intracranial vertebral artery then dizziness and diplopia are often present

Less common

Migraine	Presentation is variable but classically patient progresses through phases including prodrome, aura, headache, and the postdrome
Fibromuscular dysplasia	Headache, pulsatile tinnitus, severe or persistent lightheadedness. If ischaemia is present it can have amaurosis fugax, Horner's syndrome, transischaemic attack and stroke
Coagulopathies	Uncommon, but to be considered in younger patients who present with neurological symptoms who are on oestrogen therapy, oral contraceptives, postpartum and postoperative states or who have known cancer
Drug abuse	Suspect when social history is suggestive
Giant cell arteritis	Uncommon, but should be considered in patients with tender nodular or pulseless temporal arteries. Can affect carotids, vertebral and intracranial arteries

Source: Caplan *et al*² and Greenberg *et al*³.

Figure 3 Three-dimensional CT reconstruction imaging (variant anatomy).

to have an annual incidence of 2.6–3.0/100 000, with carotid artery dissection being 3–5 times more common than VAD.⁴ Postpartum VAD is a rare condition with limited presentation in the literature, and an unknown incidence. Epidemiological data suggest that women are 2.5× more likely than men to have a VAD, and it is thought that hormonal factors stemming from pregnancy, and migraine, could be implicated in the pathogenesis of peripartum dissection.^{5 6}

There have been similar cases presented, most notably from Mas⁶ and Arnold⁷ in 2008. Mas⁶ reviewed 13 cases of VAD in men and women, and described two peripartum cases of VAD, one that involved cervical manipulation and the other thought to be complicated by preeclampsia and hypertension. Arnold

Figure 4 CT angiogram of filling defect in right vertebral artery.

reviewed 245 patients with cervical artery dissection and found 6 patients who were post partum. Four of the six had only vertebral artery involvement and the other two had the carotid artery involved. The majority of patients reviewed had a history of hyperlipidaemia and migraine and one had a history of cervical manipulation.⁷ VADs in the young have been noted to be associated with mechanical conditions such as bone fusions and mechanical compression of the vertebral artery, as in bow hunter's stroke.⁸

Another report of a similar presentation of postpartum VAD was described by Drazin *et al.*⁹ They reported a postpartum 37-year-old woman 1 h after delivery, with severe headache, who was found to have bilateral extracranial VADs. While the correlation between their case and ours, with presentation at 3 weeks post partum, is unclear, it can, nevertheless, be directly associated with strain and trauma associated with delivery.

Our case is unique because of her peripartum presentation, rare anatomical variation, absence of any neck manipulation, and bilateral cerebellar involvement. We found several anatomic case reports that described LVA originating from the aortic arch. There is an estimated incidence of variant LVA ranging from 2.4% to 6.9%.¹⁰ It is crucial to have knowledge of these possible variations in anatomy, as there is potential for unanticipated consequences during surgical and angiographic procedures.¹¹ According to Komiyama,¹² LVA originating from the aortic arch were associated with a significantly higher incidence of dissection. Although our patient had a right VAD, it does highlight the possibility of future complications.

When examining our patient's case fully, we had several questions regarding her diagnosis and what potential physiological and anatomic processes could be contributing. It is known that vertebral arteries that have variant origins are most commonly affected with atherosclerosis at the origin, which leads to altered cerebral haemodynamics.^{10 13 14} We believe that our patient's variant anatomy likely predisposed to her VAD.

Our patient's LVA appeared to essentially be non-functional secondary to its smaller calibre and calcifications at its origin. In fact, her cerebellar infarcts were actually greater on the left than the right, which questions what type of collateralisation to her cerebellum was present. The LVA was hypoplastic causing the regions perfused by the vertebral arteries to be perfused by the right dominantly. This could account for why the patient's dissection in the right vertebral artery resulted in brainstem and cerebellar infarction.

Reviewing the patient's chronic medical history is also interesting to relate her chronic migraines in relation to her vascular event. Her chronic headaches were described with aura and photophobia, which might suggest they were basilar migraines. Our patient presented with very broad symptoms but was found to have some very unique radiological and anatomic findings that likely contributed to her symptoms.

Learning points

- ▶ Differential of vertigo with a cerebellar cerebrovascular accident.
- ▶ Peripartum dissection might be more prevalent in women with history of migraines and vascular variations.
- ▶ The key to treating vertebral artery dissection is quick identification with prompt anticoagulation.

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Competing interests None declared.

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