CASE REPORT

Postpartum Acute Basilar Artery Occlusion Secondary to Vertebral Artery Dissection. Case Report and Literature Review

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Abstract

Female patients in the peripartum and postpartum periods have an increased risk of stroke than nonpregnant women. Cerebrovascular complications of pregnancy represent a significant cause of maternal mortality and morbidity and are potentially disabling. Acute basilar artery occlusion secondary to spontaneous vertebral artery dissection in the postpartum period is an infrequent entity and a major diagnostic and treatment challenge. In the present case, a 37-year-old female patient, eight weeks after caesarean delivery, presented with a history of sudden cervical pain, followed by headache and dizziness. Some hours later, she was found unconscious by her family and was transferred to the emergency department, where a neurological status assessment suggested vertebrobasilar stroke. The imagistic workup revealed right vertebral artery dissection and basilar artery occlusion without constituted ischemic lesions. The patient underwent endovascular intervention with dilation of the narrowed vertebral artery and stent retriever basilar artery thrombectomy, with a favourable clinical outcome. This report first presents the details of this case and the relevant literature data on postpartum arterial dissections and the subsequent ischemic complications and available treatment options.

Keywords: basilar artery occlusion, vertebral artery dissection, postpartum period, stroke

■ Introduction

The basilar artery is a vital vessel with a major role in the posterior circulation of the brain. It is formed by the confluence of the dual vertebral arteries near the junction of the medulla oblongata and the pons and directly supplies blood to the brainstem and cerebellum. After its division into the paired posterior cerebral arteries, it transfers blood to the thalami and the medial temporal and occipital lobes through its cortical branches [1].

The main causes of posterior circulation ischemia include cardio-embolism, large artery atherosclerotic disease, small-vessel disease, and vertebral artery dissection [2].

Acute ischemic stroke due to basilar artery occlusion is found in 20% of posterior circulation strokes and 1% to 4% of all ischemic strokes in the general population [3, 4]. A neurological evaluation is essential for establishing the site of cerebral ischemia. However, though this assessment is typically carried out at the early onset of symptoms, it may mislead the clinician in establishing the correct diagnosis. Neurological symptoms due to vertebrobasilar ischemia can be either unspecific at the beginning or very severe, leading to rapid deterioration and coma.

The dissection of the cervical and cerebral arteries is the leading cause of stroke in young adults. The dissection mechanism is not fully understood, although damage to the arterial wall layers triggered by minor trauma or infectious or autoimmune mechanisms seems to be implicated in the pathogenesis [5–9].

The pregnancy and postpartum periods are marked by an increased risk for cerebrovascular complications.

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Stroke is a significant cause of maternal morbidity and mortality [10-12].

This article aims to present a complex case of postpartum basilar artery occlusion secondary to vertebral artery dissection treated with endovascular intervention.

■ CASE PRESENTATION

The case of a 37-year-old female, who had three previous pregnancies and three live births, with no other remarkable medical history, apart from autoimmune thyroid disease with hypothyroidism under substitution therapy, and who had experienced a recent uneventful pregnancy is reported.

She did not have any complications during her first two pregnancies nor her caesarean operation for the third child. Eight weeks after the birth of her third child, she experienced cervical pain suddenly, followed by headache and dizziness. Some hours later, the patient was found unconscious by her family and was transferred to the emergency department, Mureş County Clinical Emergency Hospital, Târgu Mureş, Romania more than six hours following the onset of symptoms. A neurological examination revealed symmetrical intermediary pupils with preserved pupillary light reflex, right conjugate gaze palsy, tetraplegia, diminished deep tendon reflexes, indifferent plantar response. On admission, a *National Institutes of Health Stroke Scale (*NIHSS) score of 25 points was recorded and a Glasgow Coma Scale (GCS) of six points. The main stroke timelines in her case history are presented in Table 1.

The patient was intubated and mechanically ventilated. Non-contrast brain computer tomography (CT) was performed with no description of ischemic or haemorrhagic lesions. The CT angiogram revealed a 10-mm-long filling defect of the basilar artery within its distal segment and severe luminal narrowing of the distal V2 segment of the right vertebral artery, suggestive for dissection. At this point, the decision to pursue per primam endovascular treatment was made. The angiography confirmed a right distal V2 dissection, revealing a 16 mm long eccentric, irregular stenosis (string sign) (Figure 1A) and a basilar artery occlusion eight mm from the point of origin (Figure 1B).

The dissected vertebral artery was dilated, and after two attempts of stent-retriever thrombectomy, complete revascularization of the basilar artery was achieved (Figure 1C).

During the endovascular intervention, the patient remained intubated and mechanically ventilated; her mean arterial pressure (MAP) values varied between 95 and 110 mmHg. For proper oxygen delivery to cerebral tissue, the fraction of inspired oxygen (FiO) was titrated to maintain peripheral arterial oxygen saturation (SpO2) > 92%. A control brain CT scan did not show any lesion. After the control CT scan, the patient was admitted to the intensive care unit.

Post-procedure antiplatelet therapy with 100 mg of acetylsalicylic acid (Aspirin; Bayer, Leverkusen, Germany) and anticoagulation therapy with enoxaparin 2x0.4 ml were initiated (Clexane; Sanofi, Le Trait, France). The patient was extubated six hours after the intervention. A neurological examination performed after 24 hours revealed horizontal nystagmus, severe dysarthria, tetraparesis with right-sided grade 1/5 *Medical Research Council (*MRC) hemiparesis and left-sided grad 4/5 MRC hemiparesis, bilateral Babinski sign, a GCS of 12 points.

The following day, brain magnetic resonance imaging was performed, showing acute bilateral pontine infarction with a left predominance (Figure 2).

Table 1. The primary stroke timelines in the patient's case history

The main events in the patient's stroke history	Time
the appearance of the first symptoms: cervical pain, headache, dizziness	19:00
the patient was last seen without neurological deficit	23:30
the patient was found unconscious with difficult respiration	24:00
presentation to the emergency unit	01:48
emergency laboratory workup	01:55
cerebral CT scan	01:56
cervico-cerebral angio CT scan	02:24
groin picture	03:47
recanalization	04:50
the patient was transferred to the intensive care unit	05:50
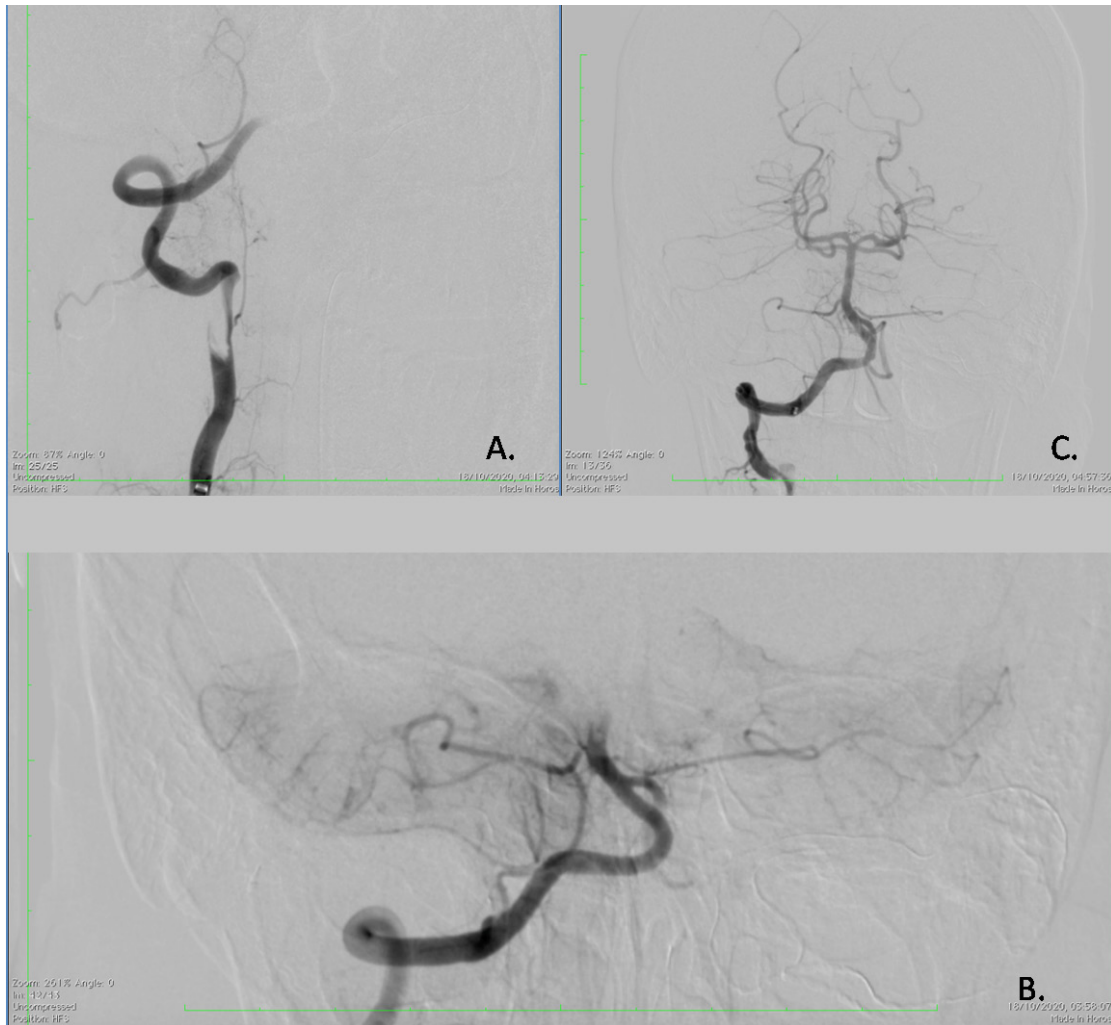
Fig. 1. Digital subtraction angiography. A. Long, eccentric, irregular stenosis (string sign) at the distal V2 segment of the right vertebral artery; B. Basilar artery occlusion 8 mm from the origin; C. Complete recanalization of the basilar artery after thrombectomy
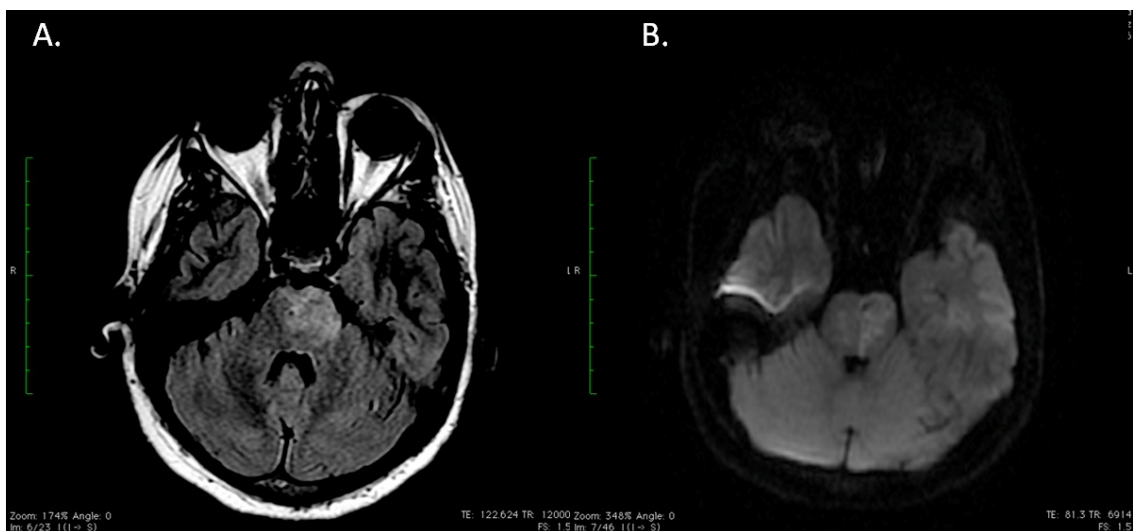
Fig. 2. A. Axial FLAIR weighted; B. Axial Diffusion-weighted MRI images showing bilateral, predominantly left-sided pontine infarction.

After 48 hours, the patient was transferred to the stroke unit.

To rule out another possible aetiology of stroke, the patient was tested for autoimmune disease and infectious disease, with negative results for both. Her thyroid hormone levels were in a normal range, but her anti-thyroid peroxidase antibody (TPO) antibody level was elevated (797,2 UI/mL; normal range <60). Transthoracic echocardiography and agitated saline transthoracic contrast echocardiography revealed no pathological findings.

During hospitalization, she undertook daily rehabilitation therapy sessions; her neurological status improved, with tetraparesis with right hemiparesis grad 4/5 MRC, left hemiparesis grad 4+/5 MRC, and moderate dysarthria.

She was discharged from the clinic on antiplatelet therapy with 100 mg of acetylsalicylic acid (Aspirin; Bayer, Leverkusen, Germany) and a thromboprophylaxis dose of subcutaneous low-molecular-weight heparin with enoxaparin 2x0.4 ml (Clexane; Sanofi, Le Trait, France).

One month after discharge, her neurological status was significantly improved, presenting with only grade 4 MRC right-sided hemiparesis.

■ DISCUSSION

Cervical artery dissection (CAD) has an approximate annual incidence of 2.6 to 3.0 per 100,000 individuals, with carotid artery dissection three to five times more common than vertebral artery dissection. CAD during the postpartum period is rare and requires immediate recognition and adequate treatment to prevent severe disability. Although the exact incidence of postpartum vertebral artery dissection is unknown, women are 2.5 times more likely than men to develop vertebral artery dissection due to pregnancy-related hormonal changes [13–18].

Extracranial segments of the cervical arteries are more exposed to dissection because of their greater mobility and high risk of injury due to their closer proximity to bone structures. The leading cause is a tear in the intimal layer of the artery, followed by blood accumulation under arterial pressure. The result is either stenosis/occlusion of the arterial lumen or thrombosis at the dissection site, which can cause downstream emboli [19, 20]. In our patient, the distal V2 segment of the vertebral artery was dissected. According to stud-

ies in the literature, the most common sites of vertebral dissection are the V2 and V3 segments [21].

The mechanism of CAD during the postpartum period is not fully understood. A proposed hypothesis is dissection in the presence of an underlying structural defect of the arterial wall in the context of a connective tissue disorder, but most of the young women included in studies were not reported to present with such diseases. There is, however, evidence to support that the physiological changes that occur during pregnancy might contribute to artery dissection: that is, hemodynamic forces are intensified by increased blood volume and cardiac output, which can cause additional stress to the vessel walls, hormone-mediated changes concerning vasoactive substances, and collagen degradation [13, 22–25]. A report by Salehi Omran et al. (2020) offered results of an investigation into this area, which proved that pregnancy is an independent risk factor for CAD. Therefore, CAD is regarded as a possible cause for pregnancy-related stroke, and symptoms like neck pain or headache during pregnancy and postpartum period should alert clinicians [26].

The main comorbidity in the reported case was autoimmune thyroid disease. Pezzini et al. (2013) reported a higher prevalence of thyroid autoimmunity among patients with spontaneous CAD than those with non-CAD ischemic stroke. Though the exact pathological mechanism is yet to be explained, it is believed that chronic vascular damage leading to dissection results from immunological mechanisms. Another supporting argument is the histopathological findings in dissecting arteries. Inflammatory infiltrates have been discovered in many cases of spontaneous coronary dissections [27, 28].

Carotid and vertebral arteries are not the only arteries affected by dissection in the postpartum period. Brantley et al. (2012) reported a rare case of spontaneous multi-arterial dissection, encompassing the coronary artery, the vertebral artery, and the internal mammary artery, in the postpartum period. Persistent neck and chest pain were the only symptoms, and the diagnosis of dissection was confirmed only after computer tomographic angiography was performed. The coronary angiogram revealed arterial dissection in the proximal portion of the left anterior descending coronary artery, and dissection of the left internal mammary artery was found incidentally during operation for coronary artery bypass grafting with a saphenous vein graft. She was treated with dual an-

tiplatelet therapy and anticoagulation. It is believed that spontaneous coronary artery dissection is due to the hormonal and hemodynamic changes resulting in intimal weakness [29].

Regarding medical treatment, antithrombotic therapy consisting of antiplatelets and anticoagulants is the main option because of the high risk of microemboli events. Antiplatelet treatment is represented by single or dual therapy, whereas anticoagulation is initiated by unfractionated or low-molecular-weight heparin, followed by oral anticoagulation [30]. According to existing guidelines, the duration of antithrombotic therapy varies between three and six months, but there is no clear consensus at this time [5,14]. Although antiplatelet therapy is preferred due to its convenience and cost, there is not enough evidence to demonstrate which of these two treatment options are more efficient in preventing recurrent stroke. Therefore, the choice of antithrombotic treatment and the duration should be adapted individually for each case [31,32].

Acute ischemic stroke caused by CAD treated with intravenous thrombolysis within 4.5 hours of onset is a safe bet for a good outcome. There is, however, a general concern that recombinant tissue-type plasminogen activator may worsen the vascular injury and increase the risk of bleeding. However, a recent meta-analysis proved no difference regarding safety and prognosis in CAD related-stroke treated with thrombolysis than a stroke caused by other aetiologies [33].

Endovascular treatment, consisting mainly of stent implantation, is a popular alternative in patients with CAD who have failed antithrombotic treatment or who have contraindications for anticoagulation therapy. Because thromboembolism was reported to be the primary mechanism of CAD-related stroke, antithrombotic treatment has been preferred over endovascular treatment [34]. However, recent studies have also demonstrated that patients presenting with both embolism and hypoperfusion due to local stenosis benefit more from endovascular treatment, decreasing the risk for secondary stroke events [35-37].

Ischemic stroke due to acute basilar artery occlusion (BAO) is a devastating subtype of stroke, leading to severe and persistent neurological deficits. The achievement of a good clinical outcome depends upon the timing of recanalization. Because of the high risk of a poor outcome, endovascular treatment can be attempted beyond six hours, up to a maximum of 12 to 14 hours from the onset of symptoms [38]. Recanalization of acute BAO is associated with a two-fold reduction in mortality and a 1.5-fold reduction in death or dependency [39].

There is no clear guideline for decision-making algorithms regarding acute BAO revascularization; therefore, scientific research has sought to investigate parameters that might predict the outcome.[40]. In their study, Ravindren et al. (2019), investigated the validity of the six-hour window and concluded that, regarding functional outcome at 90 days, the six-hour time-to-treatment and postinterventional haemorrhage played a major role as predictive factors. On the other hand, collateral status and posterior communicant artery (PCOMA) status are strong predictive factors of mortality. According to Ravindren, patients who experienced recanalization within the first six hours have a better outcome [40]. Separately, a large multicentre study including 592 patients presenting with ABAO demonstrated that a worse outcome would likely be achieved if recanalization were to take more than nine hours [41-43].

There are, however, contrary findings from a single-centre report including 184 patients, which suggest that time to treatment does not predict outcome [40].

Patients with good collaterals showed a greater possibility for successful recanalization even beyond six hours from the onset of symptoms. Particular attention has been given to the patency of the posterior communicating artery. A good functional outcome is less likely to be achieved if this artery is absent due to distal occlusion of the basilar artery, as the odds of a good functional outcome were shown to be reduced by 70%. The pathophysiological explanation for this is the occurrence of reverse blood flow from the anterior circulation. Postinterventional haemorrhage was also associated with a poor outcome, leading to a greater than the four-fold probability of dying. NIHSS is regarded as a powerful tool to predict functional outcome over 90 days [40].

A retrospective study conducted by Alemseged et al. (2019) investigated the benefit of a time-extended therapeutic window for endovascular treatment in acute basilar artery occlusion by evaluating thrombus burden in the basilar artery and the collaterals' patency. The Basilar Artery on Computed Tomography Angiography score evaluated thrombus burden and collaterals,

and the Posterior Circulation Collateral score, which evaluates collaterals, were used to assess patients.

The study aimed to establish whether favourable imaging characteristics (low thrombus burden and good collaterals) had an impact on the association between revascularization, time to treatment, and functional outcome [44]. Although it is believed that a better clinical outcome is achieved in the first six hours, this study suggested good collaterals and low thrombus burden are predictors for successful endovascular treatment beyond six hours from the onset of symptoms [44].

The use of collateral flow at angiography as a predictor for an eventual endovascular success was previously investigated by Bang et al. (2011), who encouraged clinicians to include collateral status in decision-making for mechanical thrombectomy. Patients with a good collateral flow should benefit from repeated endovascular attempts, whereas the absence of collaterals should be regarded as an unfavourable predictor of successful recanalization [45].

Proper anaesthesia significantly influences the success of the endovascular procedure. General anaesthesia (GA) was preferred in our case over monitored anaesthesia care or conscious sedation because of the severe neurological status of our patient and altered level of consciousness, which involves a high risk of aspiration and impending respiratory failure [46].

According to the literature data, there is no significant difference regarding the neurological outcome between the patient groups receiving general or local anaesthesia or conscious sedation. A meta-analysis published in 2017, including data from six endovascular stroke treatment studies performed during the newer-generation stent-retrievers era, proved no difference regarding the 90-day mortality and good neurological outcome between the general and non-general anaesthesia groups. [46]. A 2019 meta-analysis proved even less disability evaluated by the modified Rankin Scale among patients receiving GA versus non-GA [47]. The main advantages of GA are the less risk of patient movements during the intervention, improved comfort for both the patient and interventional specialist, controlled ventilation and secured airway [47].

After the endovascular treatment, the patients should be extubated as soon as possible. Some centres extubate most patients immediately after the post-interventional control CT scan; other centres routinely transport the patients to the intensive care unit before extubation. This procedure is at the discretion of the

anaesthesia team and the local protocols [46, 47]. After the interventional treatment, the management of ventilation, temperature, blood glucose level, blood pressure and other hemodynamic and physiological parameters are of significant importance because they are important determinants of outcome [46, 47].

The present patient had two possible aetiologies for vertebral artery dissection-related stroke: the physiological hemodynamic changes in the postpartum period, predisposing to vascular injury, and the immunological mechanism related to autoimmune thyroid disease. Importantly, our case is a reminder of the chance for ischemic stroke among young people, and its report is intended to raise awareness among clinicians of the need to consider CAD as a possible cause of stroke in young, including women soon after childbirth.

■ CONCLUSIONS

Spontaneous vertebral artery dissection followed by acute basilar artery occlusion is a rare cause of stroke during the postpartum period. Although the pathophysiological mechanism remains unclear, the pregnancy and postpartum periods are risk factors for CAD and stroke, which require rapid recognition and appropriate treatment to prevent a devastating outcome. Endovascular treatment in acute BAO is life-saving and can be extended beyond the first six hours from the onset of symptoms.

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