



Case report

Acute cerebral infarction of posterior circulation in a patient with vertebral artery fenestration deformity: A case report

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ABSTRACT

Introduction: Cerebrovascular fenestration malformation is a rare congenital vascular variation. Cerebrovascular fenestration malformation rarely directly leads to cerebral infarction, and the mechanism of cerebral infarction is not clear. Cases of young patients with vertebral artery fenestration malformation who suffered from acute cerebral infarction of posterior circulation are rare and have not been reported widely.

Patient concerns: A 36-year-old male patient, who had been in good health and without a family history of stroke, was admitted to our hospital with a 6-h history of dizziness and unstable walking.

Diagnosis: Brain MR examination showed multiple irregular high signal lesions in the left thalamus, left occipital lobe and left cerebellum. Brain MR enhancement examination confirmed multiple cerebral infarction in left thalamus, left occipital lobe and left cerebellum. CT angiography of head and neck showed fenestration deformity of V2–V3 segment of left vertebral artery.

Interventions: Considering that the patient was suffering from acute cerebral infarction of posterior circulation, he was treated with antiplatelet, lipid-lowering and plaque stabilization, etc.

Outcomes: After receiving our treatment, the patient's symptoms were relieved. At 3 and 6 months after discharge, there was no dizziness, unstable walking, no acute cerebral infarction, which meant that the patient recovered well.

Conclusion: In the absence of traditional risk factors and other evidence of cryptogenic stroke, the cerebral infarction in the blood supply area of fenestration malformation should be considered to be related to fenestration malformation, but its pathogenesis is not clear. Antiplatelet therapy, lipid-lowering and plaque stabilization, etc. are effective in prevention of new infarction for such patients.

1. Introduction

Cerebrovascular fenestration is a rare congenital vascular development abnormality. It refers to the limited repetition that the blood vessels are divided into two branches in the process of walking, and then merged into one branch after walking for a certain length [1]. The shape of cerebrovascular fenestration malformations can be divided into silk-like shape, convex-lens-like shape, duplication shape and irregular shape [2]. Studies have reported that cerebrovascular fenestration deformity may be related to aneurysms, moyamoya disease, transient ischemic attack, cerebral infarction and other diseases [3, 4, 5]. For young patients with acute cerebral infarction, we must do a good job in differential diagnosis with other etiologies, which allows us to treat patients more accurately [6]. However, cerebrovascular fenestration deformity rarely

leads to acute cerebral infarction, and the specific pathogenesis of cerebral infarction caused by cerebrovascular fenestration deformity is still unclear [7, 8]. We report a case of acute cerebral infarction of posterior circulation in a patient with vertebral artery fenestration deformity.

2. Case presentation

A 36-year-old male patient having no cigarette smoking history or sedentary lifestyle, with a BMI of 24.2 and with no special medical history was admitted to the hospital with a 6-h history of dizziness, vomiting and unstable walking. Physical examination showed that he was conscious, his speech was clear, the left finger nose test and heel knee tibia test were slightly inaccurate, and the Romberg sign was positive. The NIHSS (National Institute of Health stroke scale) score was 1 point.

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No hemorrhage was found in emergency brain CT (Computed Tomography) examination. Emergency brain MR (magnetic resonance) examination at onset showed multiple irregular high signal hyperintensity in left thalamus, left occipital lobe and left cerebellum (Fig. A). According to the patient's symptoms, signs and auxiliary examination, it was preliminarily considered that the patient may have acute cerebral infarction, and the patient shall be treated with antiplatelet (Aspirin enteric coated tablets, 100mg, once a day), lipid-lowering and plaque stabilization (Atorvastatin calcium tablets, 20mg, once a night), circulation improvement (Xueshuantong for injection, 500mg, once a day), nerve nutrition (Cerebroprotein hydrolysate for injection, 60mg, once a day) and relieving dizziness (Betahistine for injection, 20mg, once a day) (see Figures 1, 2, 3, 4).

There were no traditional risk factors for cerebral infarction of the patient, such as hypertension, diabetes and coronary heart disease. Blood routine examination showed no increase in eosinophils, which did not support eosinophilia. The serum anti neutrophil cytoplasmic antibody MPO-ANCA, anti neutrophil cytoplasmic antibody PR3-ANCA and anti glomerular basement membrane antibody GBM were negative, which did not support vasculitis. There was no abnormality in blood lipid, blood glucose, rheumatism, autoimmune index, systemic lupus erythematosus, homocysteine, hepatitis B, hepatitis C, syphilis, human immunodeficiency virus and other blood test results. The cerebrospinal fluid pressure was 270mmH₂O, the cerebrospinal fluid protein was 53mg/dl, and the red blood cell and white blood cell counts were normal. Cerebrospinal fluid and serum anti-ampar1-antibody, anti-ampar2-antibody, anti-caspr2-antibody, anti-gababr-antibody, anti-LGI1-antibody and anti-NMDAR-antibody were negative, which did not support autoimmune encephalitis. Cerebrospinal fluid pressure which was high may be related to stress. No atrial fibrillation or other arrhythmias was found in 24h-dynamic-electrocardiogram, which did not support cardiogenic cerebral embolism caused by atrial fibrillation. Transcranial Doppler foam test showed no abnormal embolism and did not support cerebral infarction caused by abnormal embolism of patent foramen ovale or pulmonary artery fistula. No obvious abnormality was found in the cardiac color ultrasound examination of the patient. Therefore, this patient had no evidence of cardiogenic cerebral infarction. Further examination of brain MR enhancement seven days after onset showed multiple subacute lacunar infarction in left thalamus, left occipital lobe and left cerebellar hemisphere (Fig. B). Reexamination of DWI sequence seven days after

onset showed that multiple diffusion restriction in left thalamus, left occipital lobe and left cerebellar hemisphere disappeared (Fig. C).

The head and neck CTA original drawing and reconstructed drawing of the patient showed that the fenestration deformity of V2–V3 segment of the left vertebral artery and the type of it was duplication shape (Fig. D). According to the patient's medical history and all laboratory and examination results, the patient's cerebral infarction site was just located in the blood supply area of fenestration deformity. At the same time, the patient was asked about the situation before the onset. The patient complained that he had no history of neck massage before the onset, but he had a large rotation range of the head and neck, which could not rule out the occurrence of thromboembolism due to the traction of fenestration deformity blood vessels. At present, the etiology of acute cerebral infarction in posterior circulation may be caused by local thrombosis and thromboembolism from the fenestration deformity of left vertebral artery. The patient's symptoms were basically relieved after 1 week of treatment, the left finger nose test and heel knee tibia test were basically stable, the Romberg sign was negative, and the NIHSS score decreased to 0. During the follow-up of 3 months and half a year after discharge, the patient did not have dizziness, vomiting, unstable walking and other symptoms. No new acute emerging cerebral infarction occurred to him which meant that the patient recovered well.

3. Discussion

Cerebrovascular fenestration malformation is a rare congenital vascular developmental abnormality. Its formation is closely related to the process of embryonic development. The incidence of cerebrovascular fenestration malformation ranges from 0.03% to 1%. Cerebrovascular fenestration deformity is most common in basilar artery, followed by vertebral artery [4]. Wu X et al. divided cerebrovascular fenestration into four types: silk-like shape, convex-lens-like shape, duplication shape and irregular shape [4]. Cerebrovascular fenestration deformity is usually found during head and neck CT or MR examination [9]. The fenestration deformity of this patient we reported was located in the vertebral artery, which was found by head and neck CT angiography examination. According to the morphological characteristics of the patient's fenestration deformity, it was considered as duplication shape.

Studies have reported that cerebrovascular fenestration malformation may be related to aneurysms, moyamoya disease, transient ischemic

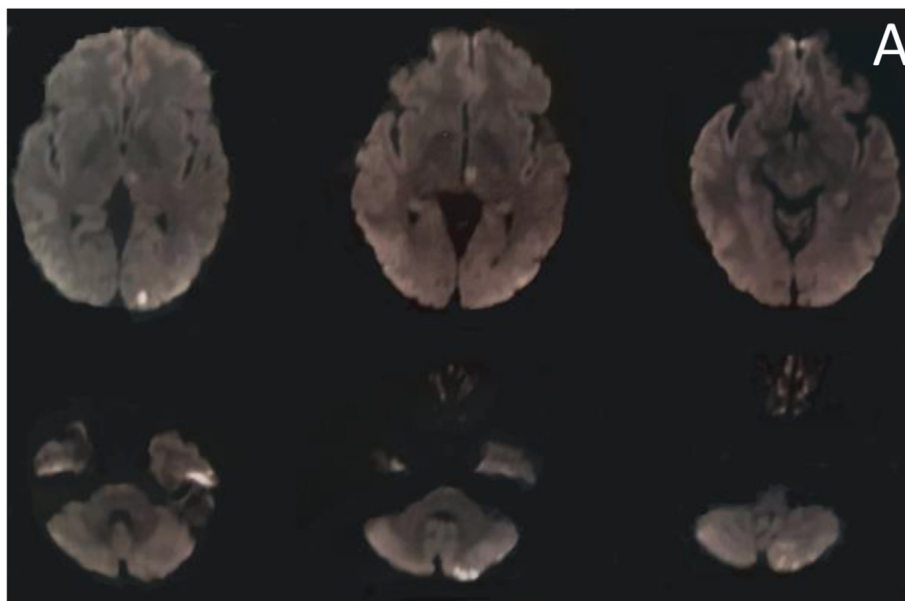


Figure 1. Brain MR at onset showed multiple irregular high signal lesions in the left thalamus, left occipital lobe and left cerebellum.

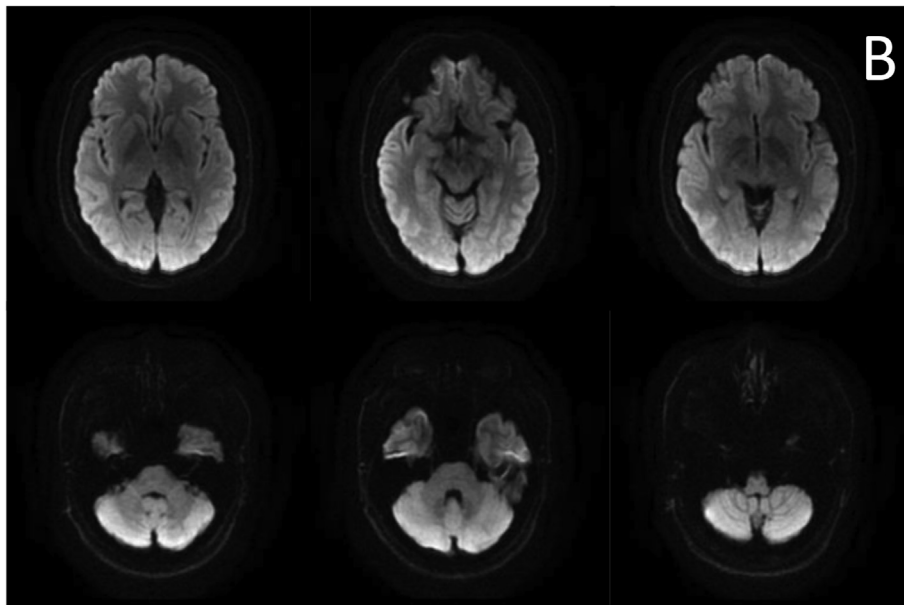


Figure 2. Brain MR reexamination seven days after onset, DWI sequence showed that multiple high signal lesions in left thalamus, left occipital lobe and left cerebellar hemisphere disappeared.

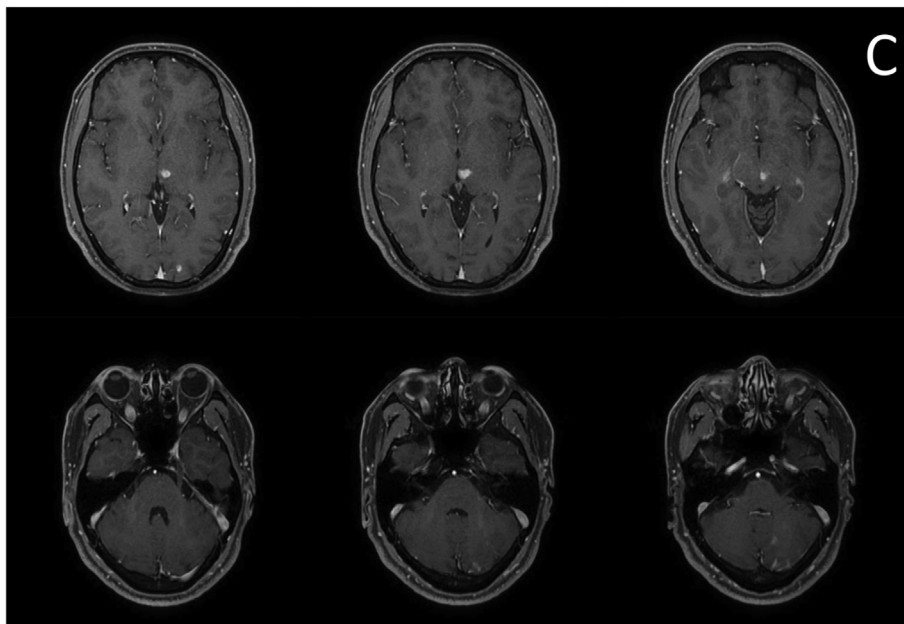


Figure 3. Brain MR enhancement examination seven days after onset confirmed multiple subacute lacunar infarction in the left thalamus, left occipital lobe and left cerebellar hemisphere.

attack, cerebral infarction and other diseases [4]. However, few literatures have reported that cerebrovascular fenestration malformation can directly lead to acute cerebral infarction, and the specific pathogenesis of cerebral infarction caused by cerebrovascular fenestration malformation is not clear [4]. The patient we reported had fenestration deformity in V2–V3 of vertebral artery, and there were no traditional risk factors of cerebral infarction such as hypertension, diabetes, coronary heart disease, atrial fibrillation, etc. Acute cerebral infarction was proved after brain MR enhancement examination. However, the etiology of acute cerebral infarction of posterior circulation caused by fenestration deformity of vertebral artery was not clear, and it was not excluded as cryptogenic stroke. For young patients, attention should be paid to exclude the existence of other etiologies or diseases such as patent foramen ovale, potential tumors, hematological diseases and autoimmune

diseases. Further tests and examinations did not support eosinophilia, autoimmune encephalitis, vasculitis, hematological diseases, cardiogenic cerebral embolism caused by atrial fibrillation, cerebral infarction caused by abnormal embolism, etc [10]. The original and reconstructed CT angiography images of the head and neck of the patient suggested that the V2–V3 segment of the vertebral artery was fenestrated and did not support the diagnosis of vascular dissection, so cerebral digital subtraction angiography was not performed. Maybe the only clue of cerebral infarction was the fenestration deformity of the left vertebral artery. According to the location of the patient's cerebral infarction included the left thalamus, left occipital lobe, left cerebellum, etc, the infarct focus was just located in the blood supply area of fenestration malformation. We comprehensively considered that the patient's acute cerebral infarction was the most likely to be related to the fenestration malformation of left



Figure 4. Original drawing and reconstructed drawing of CT angiography of head and neck indicated that the fenestration deformity of V2–V3 segment of left vertebral artery (as shown by the arrow) and the shape was duplication shape.

vertebral artery, but the specific pathogenesis was not clear. Some studies have analyzed the mechanism of cerebral infarction caused by fenestrated vessels, which may be due to hemodynamic changes in fenestrated vessels, such as turbulence caused by local blood flow disorder of fenestrated vessels, thrombosis, etc. [4, 5, 7, 11] As for this patient, he had no traditional risk factors for cerebral infarction. The fenestration deformity of the left vertebral artery was duplication shape, and the length of the fenestration vessels was relatively long. The rotation of head and neck by a large margin of the patient may pull the fenestration deformity vessels, impact the blood vessel wall, damage the intima, generate thromboembolism and finally resulted in acute infarction of posterior circulation. Unfortunately, this guess could not be confirmed by CT angiography or MR angiography examination. In addition, whether patients with fenestration of duplication shape deformity are more prone to cerebral infarction than patients with silk-like shape or convex-lens-like shape fenestration deformity needs to be examined in more cases.

After treated with antiplatelet, lipid regulation and plaque stabilization, etc. after admission, the symptoms of the patient were basically relieved and no new infarction was occurred. After 3 months and half a year of follow-up after discharge, the patient no longer had symptoms such as dizziness, vomiting and unstable walking, and no new acute cerebral infarction occurred to him, suggesting that antiplatelet treatment, lipid regulation and plaque stabilization, etc. were effective in the prevention of new cerebral infarction that caused by cerebral vascular fenestration malformation [12, 13]. Further more, this case suggests that for the patient with acute cerebral infarction caused by cerebrovascular fenestration malformation, the focus shown by DWI sequence has the opportunity to be reversible. We may think that the prognosis of the patient whose focus shown by DWI sequence could disappear may be better than those who could not. The symptoms of this patient were mild at onset, and the lesions of DWI sequence of brain MR reexamination disappeared seven days after onset. It is considered that it may be due to the rapid autolysis of most of the emboli generated by fenestration deformity after the reaching of the affected vessel.

4. Conclusion

In the absence of traditional risk factors, cardiogenic, blood, rheumatism, immunity and other detremined diseases for cerebral infarction and other evidence of cryptogenic stroke, cerebral infarction in the blood supply area of fenestration malformation should be considered to be related to fenestration malformation. However, the pathogenesis of cerebral infarction caused by cerebrovascular fenestration malformation is not clear, which may be related to abnormal local vascular

hemodynamics. Antiplatelet therapy, lipid-lowering and plaque stabilization, etc. are effective in prevention of new infarction for such patients. For patients with fenestration deformity who suffer from acute cerebral infarction, if the cerebral infarction lesions disappear in DWI sequence in a relatively short time such as within 7 days on follow-up MRI, the prognosis may be better.

Declarations

Author contribution statement

All authors listed have significantly contributed to the investigation, development and writing of this article.

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Data availability statement

Data will be made available on request.

Declaration of interest's statement

The authors declare no conflict of interest.

Additional information

No additional information is available for this paper.

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