Clinical Case Study

Consideration of intracranial venous thrombosis in cerebrovascular disease: a case study emphasizing diagnosis and treatment awareness

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Abstract

When discussing cerebrovascular diseases, we will undoubtedly think of intracranial arterial system diseases, such as cerebral infarction and cerebral embolism. It is easy to ignore intracranial venous system diseases. By describing the diagnosis and treatment of a case of intracranial venous thrombosis, we emphasize the need for physicians to consider the formation of intracranial venous thrombosis and the formation of arterial thrombosis in diagnosing cerebrovascular diseases.

Keywords: Neurology, cerebral vascular disease, intracranial venous thrombosis, cerebral embolism, stroke, Labbe venous thrombosis, cerebral infarction.

1. Introduction

The clinical incidence rate of intracranial venous thrombosis is much lower than that of intracranial arterial thrombosis. The early symptoms are often atypical and can be manifested as headache, focal neurological deficit, seizures, nausea, vomiting and mental symptoms. It is easy to simulate the clinical manifestations of other diseases (Ge et al., 2021; Ferro et al., 2004), and some are related to pneumonia (Klok et al., 2020; Cavalcanti et al., 2020), resulting in delayed diagnosis or misdiagnosis. Imaging diagnosis mainly depends on cranial CT and MRI (Canedo-Antelo et al., 2019; Leach et al., 2007).

The characteristics of cerebral infarction are as follows: 1. The area of cerebral infarction is consistent with the drainage vein and mostly occurs in the cortex and subcortical. 2. The lesions were irregular in shape, patchy or gyrus-like. DWI was in the corresponding parts of MRI, with large areas of obvious high signal, and ADC signal was not significantly reduced. 3. Cellular edema is often accompanied by massive vascular edema. 4. The lesion was not seriously damaged and had great recovery potential. Venous thrombosis includes cortical venous thrombosis, deep cerebral venous thrombosis and venous sinus thrombosis. Clinical manifestations include aphasia, psychosis and other neurological deficit symptoms.

2. Case report

A 59-year-old male was admitted to the hospital for more than 20 days due to right-hand weakness and 10 days due to speech disability and right lower limb weakness, which were aggravated for 20 hours. The patient had no obvious inducement of right-hand weakness, was unable to grasp, accompanied by right-hand numbness during the disease, and did not care.

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There was no obvious inducement for speech failure, accompanied by weakness of the right lower limb, poor activity, and mental behavior abnormalities that the family could not understand. The patient was treated in the local hospital and was given "*an aspirin enteric coated tablet*" and "atorvastatin calcium tablet" orally. At the same time, the patient was given an intravenous drip of circulation-improving drugs. The symptoms did not improve significantly. To seek further diagnosis and treatment, the patient was treated in the outpatient department of our hospital and admitted to the hospital.

1.1 History

The history of hypertension, diabetes and heart disease, history of surgery and blood transfusion, history of hepatitis tuberculosis and history of food and drug allergy were denied. Smoking for more than 30 years, seven cigarettes/day, occasional drinking history.

1.2 Physical examination

The vital signs are stable. Clear mind, mixed aphasia (hearing comprehension disorder, naming disorder, difficulty finding words), and lack of cooperation in physical examination. The muscle strength of the distal end of the right upper limb was grade 2, the muscle strength of the right lower limb was grade 4, and the muscle strength of the remaining limbs was grade 5. The muscle tension was normal. No obvious positive signs were found in the rest of the nervous system.

1.3 Auxiliary examination

ldl:4.88mmol/l, hcy:95.59umol/l. No obvious abnormality was found in rheumatoid factor, anti-chain, ESR, C-reactive protein, antinuclear antibody, vasculitis autoantibody and thyroid function. Coagulation series tips: pt-s:14.5 s, fib:1.44g/l, pt%:71.5%, inr:1.20r, tt:15.7s, d-dimer:0.5mg/l feu.

After admission, a complete cranial MRI and perfusion examination was given. The results showed multiple subacute infarcts in the left frontotemporal lobe and semioval area, accompanied by small bleeding. CBV and CBF in both cerebral hemispheres are symmetrical, no obvious abnormal perfusion area is found, and no delay in TTP and MTT is found. Head and neck CTA indicates mild sclerosis: non-calcified plaque at the beginning of the left internal carotid artery and mild stenosis of the lumen.

The patient had no risk factors for cerebrovascular diseases such as hypertension and diabetes. CTA of the head and neck showed mild sclerosis: non-calcified plaque at the beginning of the left internal carotid artery and mild lumen stenosis. Cranial perfusion: the distribution of CBV and CBF in both cerebral hemispheres is symmetrical, no obvious abnormal perfusion area is found, and no delay in TTP and MTT is found. These do not support the continuous occurrence of infarction in a short time. Homocysteine 95 fifty-nine μ Mol/l.

Hyperhomocysteine is not only a risk factor for acute ischemic cerebral infarction but also a risk factor for venous system infarction. According to the infarct location and pathogenesis of the patient, there have been limb weakness, aphasia, mental symptoms, etc. in combination with DWI and ADC, patchy T1WI low signal T2WI high signal shadow can be seen in the semi-oval area, flair is a high signal, DWI is a slightly high signal, and ADC is a high signal, Considering the infarction of the arterial system in the center of the semicircular oval and beside the lateral ventricle, the secondary focus of the patient is mostly concentrated in the left frontotemporal lobe, and considering the infarction of the venous system: Labbe vein (see **Fig. 1**). No obvious abnormality was found in detecting perfect protein C and protein S. Complete head MRV showed that the left transverse sinus and sigmoid sinus were fine. Therefore, the possibility of intracranial venous thrombosis is considered high.

2. Discussion

The Labbe vein (see Silvis et al. (2017)) is a part of the intracranial superficial venous system. It starts from the lateral fissure and flows into the transverse sinus backward and downward to drain the lateral side of the temporal lobe. It is the anastomosis between the middle and lower cerebral veins. The communicating vein Labbe venous thrombosis of the superior sagittal sinus and transverse sinus is rare in the clinic, accounting for

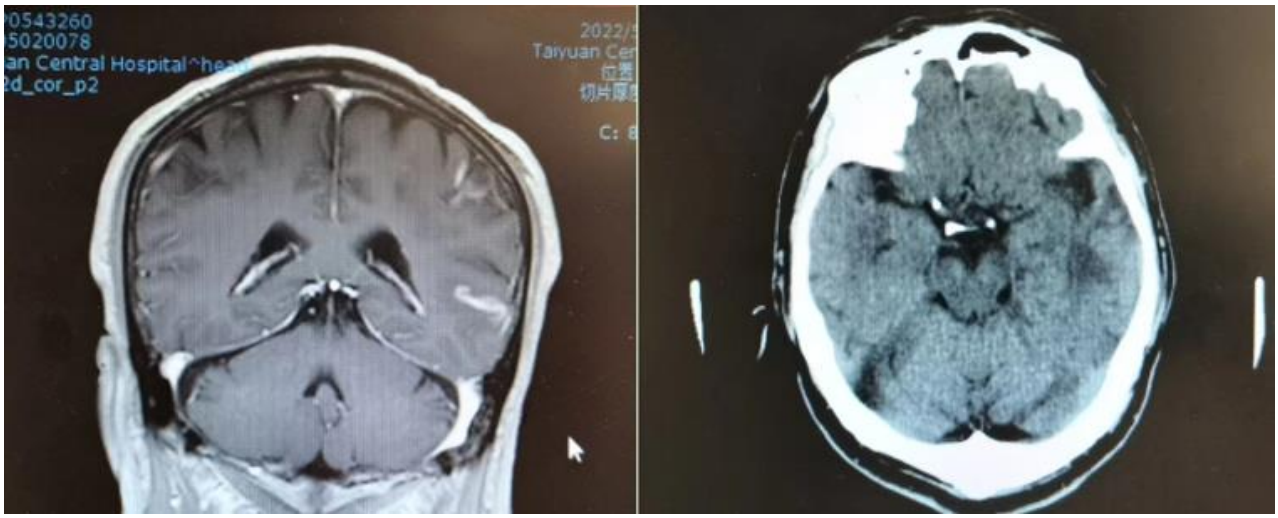

Figure 1 Temporal lobe hemorrhagic cerebral infarction and dense transverse sinus. The secondary lesions of the patients were mostly concentrated in the left frontotemporal parietal lobe, considering the venous system: infarction of Labbe vein. Coronal view LHS, transverse RHS**.**

about 6% of intracranial venous thrombosis. Isolated Labbe venous thrombosis is prone to occur in some people at risk of hypercoagulability. The peak of the disease is 20-30 years old, and the clinical manifestations are diverse. It often manifests as subacute headache and focal neurological dysfunction (epilepsy, hemiplegia, semi-blindness, etc.)

Combined with the patient's imaging lesions and clinical manifestations, considering the formation of cortical venous thrombosis, Labbe venous thrombosis is the most common, and its main temporal lobe blood supply is reflux, which corresponds to the symptoms, signs, and infarct site of the patients. There was no obvious optic papilledema in the fundus of the patients. Superficial cortical venous thrombosis rarely occurs in intracranial hypertension and optic disc edema. Among the numerous risk factors, no obvious abnormality was found in the related indexes of immunology, rheumatism, inflammation, D-dimer, and homocysteine was high: 95 fifty-nine μ Mol/l. It is one of the factors closely related to venous thrombosis. After receiving anticoagulant therapy in a hospital, the patient's physical and mental symptoms improved significantly.

3. Conclusion

In conclusion, the patient was diagnosed with venous thrombosis. After diagnosing this case, we strengthened the diagnosis and treatment of venous thrombosis. At the same time, be vigilant and identify the problem as soon as possible to avoid delaying diagnosis and treatment. During hospitalization, 5000iu low molecular weight heparin calcium was injected subcutaneously for q12h. After discharge, rivaroxaban anticoagulation therapy was continued for 6-8 months. At the same time, atorvastatin calcium tablets were given to reduce lipids and stabilize plaque. Quetiapine fumarate tablets improved the mental symptoms of the patients. Dynamic observation of related blood indexes and imaging examination.

Conflict of Interest:

The authors declare no conflict of interest.

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