Clinical Case Report

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Cerebral venous thrombosis originating from internal jugular vein outflow impairment

A case report

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Abstract

Rationale: Cerebral venous thrombosis (CVT) comprises a group of cerebral vascular diseases resulting from cerebral venous outflow obstruction caused by various etiologies. The etiology of CVT is complex, including infectious and noninfectious factors. The diagnosis is difficult. As a result, many patients are misdiagnosed or never diagnosed. This patient was diagnosed with CVT due to unilateral internal jugular vein compression.

Patient concerns: In this report, we present a case of acute onset CVT in a 15-year-old female patient who presented with a headache, nausea, and vomiting as the main clinical manifestations.

Interventions: This patient was administered with conventional anticoagulants and treated for dehydration, but the effect of conventional therapy was not obvious.

Outcomes: We recommended that this patient undergo left local decompression of the internal jugular vein to inhibit the thrombosis. But regretfully, due to economic reasons and surgical risk, the patient and her mother refused operation.

Lessons: This case report demonstrates the importance of considering jugular vein lesions as an etiology of CVT. Furthermore, computed tomography venography of the jugular vein and jugular vein ultrasound were instrumental in detecting the abnormal structure of the jugular vein and hemodynamic changes.

Abbreviation: CVT = cerebral venous thrombosis.

Keywords: cerebral venous thrombosis, jugular vein compression, jugular vein outflow impairment

1. Introduction

Cerebral venous thrombosis (CVT) comprises a group of cerebral vascular diseases resulting from cerebral venous flow obstruction caused by various etiologies.[1] CVT patients usually present with a worsening headache, visual loss, confusion, seizures, and altered consciousness. The etiology of CVT is complex, including infectious and noninfectious factors.[2] In addition, the diagnosis is difficult. The exact cause cannot be identified in approximately 20% to 35% of patients. Here, we present a case of CVT caused by unilateral internal jugular vein local compression and cerebral venous system hemodynamic alterations in a 15-year-old female patient.

2. Methods

Since the images presented in this case report are entirely unidentifiable and there are no details regarding the patient reported within the manuscript, the Ethics Committee of our institution waived the requirement for approval of this single case study with medical records. Oral informed consent was obtained from the patient's parents.

3. Case report

A 15-year-old female was referred to the Department of Neurology, First Hospital of Jilin University, after 2 months of intermittent headaches. In the last 4 days, her headache episodes worsened and were accompanied by nausea and vomiting. Upon admission, the patient had an unremarkable medical and surgical history and no evidence of infection or high blood coagulation. Overall, her medical history was normal. She had no family history or physical history including co-morbidities. Furthermore, she did not present with fever, limb activity disorders, convulsions, or disturbance of consciousness. The general physical exam revealed nothing remarkable, and she had a normal blood pressure (120/80 mmHg). The neurological examination was also normal, the patient was conscious and maintained verbal fluency, and no abnormalities were observed upon cranial nerve inspection. The muscle strength in the limbs was level 5, the muscle tension was normal, the limb tendon reflex

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CXL and LS equally contributed to this study.

CXL and YZ conceived the idea, and CXL, LS, XZZ, and MQZ wrote most of the paper. All authors have read and approved the final manuscript.

Consent: Informed consent was obtained from the patient's parents for publication of this case report and any accompanying images. In cases where images are entirely unidentifiable and there are no details on individuals reported within the manuscript, consent for publication of images can be obtained orally.

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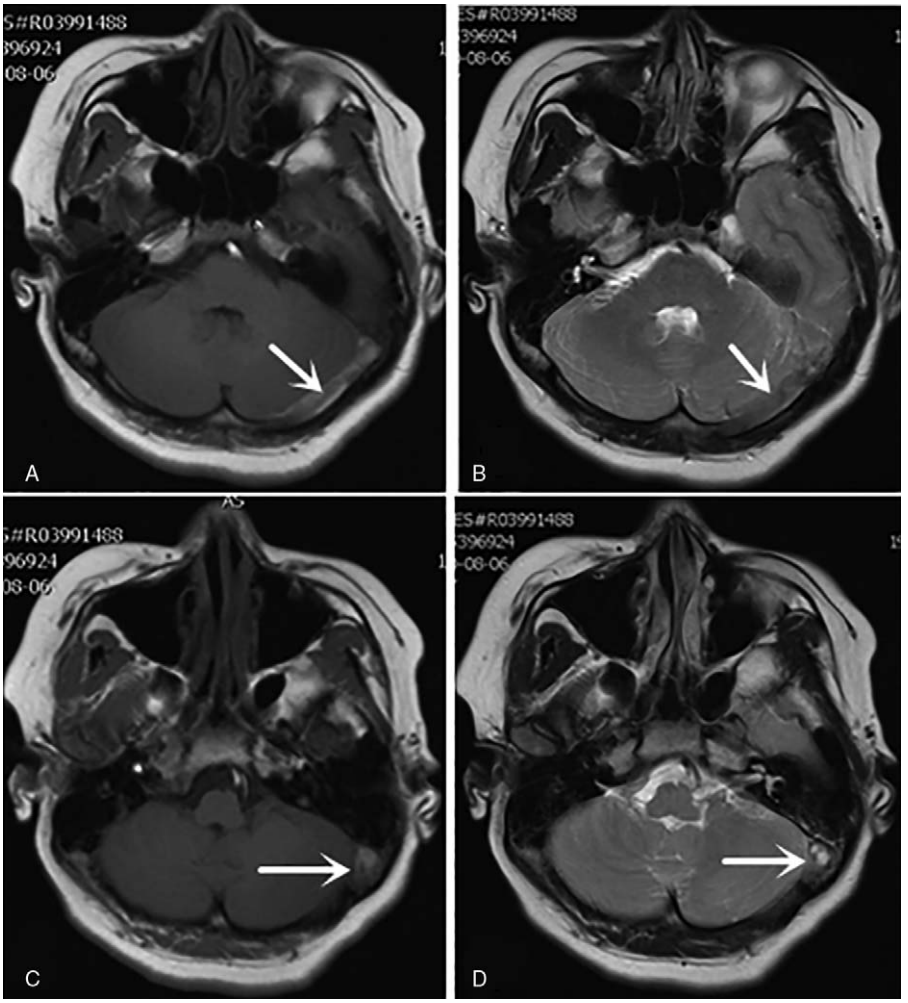

Figure 1. Head magnetic resonance imaging. (A–B) Arrows indicate abnormal T1/T2 signals in the left transverse sinus. (C–D) Arrows indicate abnormal T1/T2 signals in the sigmoid sinus.

was normal, the Barbinski reflex was negative, there were no signs of neck stiffness, and the Kernig sign was negative.

The auxiliary examination showed that the lumbar puncture pressure was 330 mmH_2O, which was significantly increased from normal levels (normal range: 80–180mmH_2O). The number of white blood cells (8×10^6/L) and the chlorine concentration (131.4 mmol/L) in the cerebrospinal fluid were normal. The immunoglobulin G level in the cerebrospinal fluid was 17.90 mg/L. The patient had an elevated D–dimer level of 696.00 μg/L (normal range: 0–232 μg/L). Moreover, the routine blood test results, protein C, protein S, anticardiolipin antibody, erythrocyte sedimentation rate, blood coagulation, antistreptolysin O, autoantibodies involved in rheumatism and immune diseases, antinuclear antibodies, and thyroid function were all normal.

Head magnetic resonance imaging revealed the absence of abnormal findings in the brain or the ventricular system. However, an abnormal T1/T2 signal was observed in the left transverse sinus, sigmoid sinus, and straight sinus, suggesting thrombosis in the transverse sinus, sigmoid sinus, or straight sinus, respectively (Fig. 1).

According to the head magnetic resonance venography, we were not able to detect the left transverse sinus, sigmoid sinus, or straight sinus, thus indicating thrombosis. Furthermore, the superior sagittal sinus was pale, which could be attributed to dysplasia (Fig. 2).

Computed tomography venography of the jugular vein revealed a narrow space between the left atlas transverse process and the left cranial skull styloid process. The local jugular vein

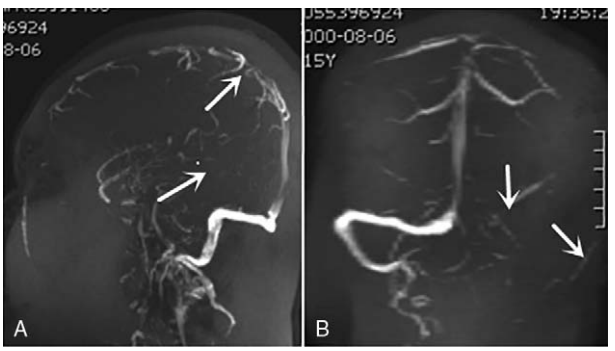

Figure 2. Head magnetic resonance venography. (A) The superior sagittal sinus is pale, and the straight sinus cannot be detected (arrows). (B) The left transverse sinus and sigmoid sinus (arrows) are not detected, respectively.

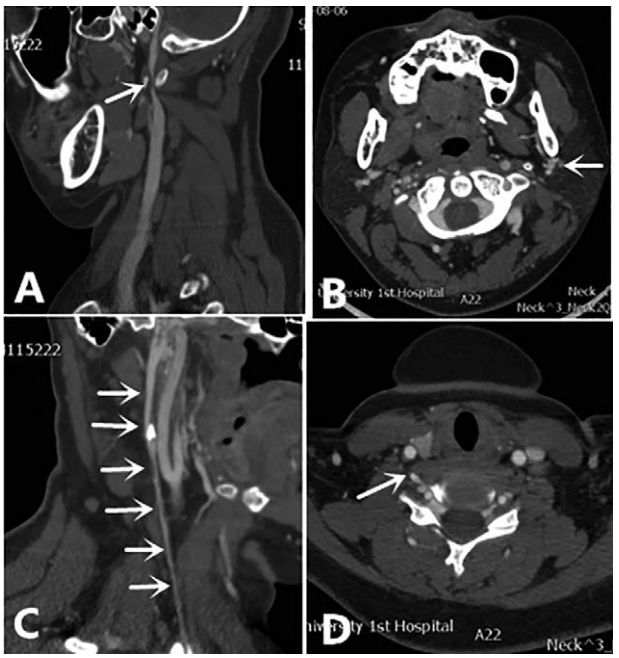

Figure 3. Computed tomography venography of the jugular vein. (A) Sagittal and (B) axial views: the distance between the left side of the atlas transverse process and the cranial skull styloid process was narrow (indicated by the arrow). (C) Sagittal and (D) axial views: the entire lumen of the right jugular vein was narrow, and multiple calcified and conglobate circuitous blood vessels were observed in the cranial exit (indicated by the arrows).

became narrow. Moreover, the lumen of the entire right jugular vein between the jugular foramen and the right subclavian vein was slightly narrower than normal; in addition, we observed multiple calcifications. Conglobate circuitous blood vessels were observed in the cranial exit of the right jugular vein (Fig. 3).

The ultrasound findings of the jugular vein indicated asymmetric diameters of the bilateral jugular vein. The venous outflow was hindered in the J2 (midpiece) segment of the bilateral jugular vein. During deep inspiration, the blood flow velocity of the left jugular venous J2 segment became slower along with pipe diameter narrowing. The J2 segment changes on the left side could have been caused by local compression, whereas the whole right jugular venous diameter was narrow. The blood flow velocity of the right jugular venous J2 segment did not change under quiescent conditions or deep inspiration; however, the blood flow of the J3 (distal) segment changed bidirectionally during deep inspiration along with a subsequent faster backflow velocity. These changes could have been caused by congenital birth defects combined with compensatory mechanisms (Fig. 4).

4. Discussion

In this report, we present a case of acute onset CVT in a 15-year old female patient who presented with a headache, nausea, and vomiting as the main clinical manifestations. The lumbar puncture pressure significantly increased to 330 mm H_2O, and we observed elevated D-dimer levels (696.00 μg/L) in the acute stage. Head magnetic resonance imaging and venography indicated intracranial venous thrombosis in the left transverse sinus, sigmoid sinus, and straight sinus. The patient had no history of surgery or trauma, no evidence of infection or high blood coagulation, and no abnormal overall medical history. Laboratory examination regarding the formation of thrombosis was normal. At that time, we had not found the cause of thrombosis formation. So, we performed computed tomography venography of the jugular vein. The jugular vein between the left atlas transverse process and the left cranial skull styloid process was found to be narrower than normal, where the jugular vein was compressed. In addition, the entire lumen of the right jugular vein was slightly narrower than normal, and we observed multiple calcifications as well as conglobate circuitous blood vessels in the cranial exit. Therefore, we concluded that the bilateral internal jugular vein was abnormal. Next, we investigated the cause of thrombosis on one side of the intracerebral vein. We performed jugular vein ultrasound and observed that the diameters of the bilateral jugular vein were asymmetric and that the bilateral jugular venous outflow was hindered. Furthermore, the entire right jugular vein was narrow, and the J3 segment appeared to reflux briefly in the deep inspiration state. Given the above-mentioned findings, we speculated that the right jugular vein narrowing might have been due to hypoplasia and that the collateral circulation in the blood supply might avoid the right lateral CVT. On the other hand, on the left side, the local compression of the left jugular vein with the cerebral venous outflow impairment resulted in thrombosis in the left intracerebral transverse sinus and sigmoid sinus.

Following diagnosis, we administered 10 mg of low-molecular-weight heparin by subcutaneous injection, twice daily, to inhibit continued thrombosis. 20% Mannitol 250 mL was administered, 4 times a day, to reduce the high lumbar puncture stress. Two weeks later, the symptoms were only slightly improved. Re-examination revealed that the lumbar puncture pressure was still

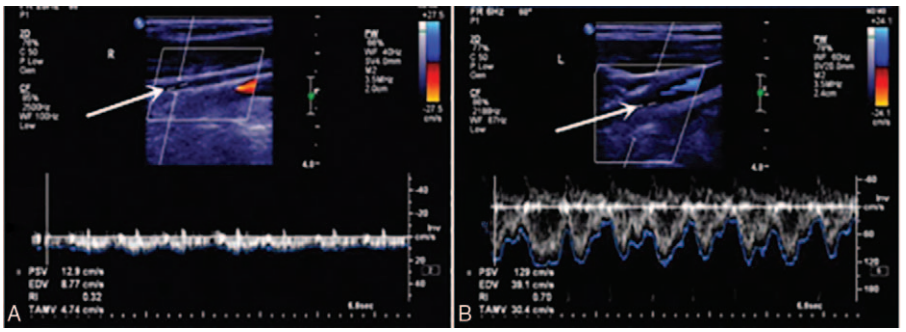

Figure 4. Ultrasound of the internal jugular vein. The diameter of the right (A, arrow) and left (B, arrow) internal jugular vein is asymmetric, and the right one is hypoplasic (A, arrow). The bilateral jugular venous outflow is impaired during inspiration, expiration, and the valsalva maneuver test.

higher than normal. Furthermore, we recommended that the patient should undergo left local decompression to inhibit the thrombosis. But regretfully, due to economic reasons and surgical risk, the patient and her mother refused operation.

In a study investigating the correlations between jugular vein abnormities and the risk of CVT, Jia et al[3] have reported that pathological changes of the jugular vein, including annulus stenosis, hypoplasia, thrombosis, and anomalous valve, indeed increase the risk of CVT. In addition, Gurley et al[4] once reported a case of CVT due to jugular vein thrombosis. In our case, the local compression of the left jugular vein resulted in hemodynamic changes that influenced venous return, ultimately leading to venous thrombosis formation.

5. Conclusion

In this case, local compression of the left jugular vein resulted in hemodynamic changes that influenced cerebral venous return, ultimately leading to venous thrombosis formation. To the best of our knowledge, reports of CVT associated with jugular vein compression are rare. This case reminds clinical doctors that jugular vein lesions may be a cause of CVT. Furthermore, we realized that computed tomography venography of the jugular vein combined with jugular vein ultrasound is useful means to find jugular vein lesions.

Acknowledgments

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References

[1] Saposnik G, Barinagarrementeria F. Diagnosis and management of cerebral venous thrombosis: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2011;42:1158–92.
[2] Stam J. Cerebral venous and sinus thrombosis: incidence and cause. Adv Neurol 2003;92:225–32.
[3] Jia LY, Hua Y, Ji XM, et al. Correlation analysis of internal jugular vein abnormalities and cerebral venous sinus thrombosis. Chin Med J (Engl) 2012;125:3671–4.
[4] Gurley MB, King TS, Tsai FY. Sigmoid sinus thrombosis associated with internal jugular venous occlusion: direct thrombolytic treatment. J Endovasc Surg 1996;3:306–14.