Thrombosis of the Superior Petrosal Vein Mimicking Brain Tumor
—Case Report—

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Abstract

A 77-year-old woman was admitted to a local hospital with a 7-day history of vertigo and nausea, followed by gait disturbance. Magnetic resonance imaging showed extensive brain edema with a hemorrhagic component in the right cerebellum. The lesion was heterogeneously enhanced after administration of contrast medium. The presumptive diagnosis was malignant glioma based on these findings, as well as the presence of mass effect and abnormal enhancement. She was referred to our hospital. However, cerebral angiography did not reveal tumor stain or arterial occlusion, but confirmed corkscrew-like venous collaterals and absence of opacification of the superior petrosal vein (SPV) and superior petrosal sinus. Topography of the brain edema was consistent with the drainage territory of the SPV. These findings suggested that the lesion was vasogenic edema caused by thrombosis of the SPV. The patient was conservatively treated without anticoagulation therapy, and the neurological and imaging abnormalities resolved spontaneously. To avoid unnecessary biopsy, thrombosis of the SPV should be considered in the differential diagnosis of infratentorial lesion mimicking brain tumors. Knowledge of the posterior fossa venous anatomy is essential to achieve the correct diagnosis.

Key words: superior petrosal vein, cerebral venous thrombosis, venous infarction, cerebellar infarction, brain tumor

Introduction

Cerebral venous thrombosis commonly involves the transverse sinus, superior sagittal sinus, and straight sinus, and accounts for less than 1% of all strokes. Subsequent venous infarction usually occurs in the supratentorial region. The infratentorial region has much more collateral circulation than the supratentorial region, which indicates that venous infarction rarely occurs in the posterior fossa. Cases of venous infarction in the posterior fossa have resulted from thrombosis of the transverse sinus, sigmoid sinus, or straight sinus, but venous infarction caused by thrombosis of the superior petrosal vein (SPV) is extremely rare. Brainstem or cerebellar involvement in spontaneous cerebral venous thrombosis is also very rare.

We report a case of thrombosis of the SPV and superior petrosal sinus (SPS) manifesting as cerebellar edema mimicking brain tumor on neuroimaging.

Case Report

A 77-year-old woman was admitted to a local hospital with a 7-day history of vertigo and nausea, followed by gait disturbance. She had no previous history of stroke, trauma, or other illness. Fluid-attenuated inversion recovery (FLAIR) magnetic resonance (MR) imaging showed an extensive hyperintense area in the right cerebellum (Fig. 1A), which appeared hyperintense on diffusion-weighted (DW) imaging, but was much less hyperintense compared to acute infarction, suggesting vasogenic edema (Fig. 1B). The lesion included a strikingly hyperintense component on FLAIR and DW imaging that suggested a small hemorrhage, with heterogeneous enhancement on T1-weighted MR imaging with contrast medium (Fig. 1C). The patient was referred to our hospital 2 weeks after the onset, under the presumptive diagnosis of malignant glioma.

On admission, the patient was alert with cerebellar ataxia in the right upper and lower limbs. Laboratory examination found no abnormalities, including antithrombin III, protein C, protein S, lupus anticoagulant, anticardiolipin antibodies, and antinuclear antibodies. Cerebral angiography did not
Fig. 1 Fluid-attenuated inversion recovery magnetic resonance image showing an abnormal hyperintense area in the right cerebellum (A), which appears slightly hyperintense on the diffusion-weighted image (B), and contains a strikingly hyperintense component (arrow), suggesting a small hemorrhage. T1-weighted image with contrast medium showing heterogeneous enhancement within the lesion (C). Coronal T2-weighted image demonstrating a wedge-shaped hyperintense area (arrowheads) (D).

reveal tumor stain or arterial occlusion, but confirmed corkscREW-like venous collaterals and absence of opacification of the right SPV and SPS (Fig. 2). The transverse, sigmoid, and straight sinuses were patent. Retrospective examination of the coronal T2-weighted images obtained at onset identified a wedge-shaped hyperintense lesion corresponding to the drainage territory of the SPV (Fig. 1D). Based on these findings, thrombosis of the SPV and SPS was suspected.

The patient was conservatively treated without anticoagulation therapy, the symptoms progressively improved, and the patient was able to walk without assistance 2 months after the onset. Follow-up MR imaging, 3 months after the onset, demonstrated spontaneous resolution of the cerebellar lesion (Fig. 3).

Discussion

Only one previous case has described SPV thrombosis as a possible cause of venous infarction, in which the transverse sinus, sigmoid sinus, and SPS were occluded.7 The topography of the lesion was similar to that in our case.

The SPV is an important venous drainage system in the posterior fossa that drains the anterior aspect of the brainstem and cerebellum into the SPS. The most common tributaries of the SPV are the vein of the cerebellopontine fissure, the vein of the middle cerebellar peduncle, the transverse pontine veins, the pontotrigeminal vein, and the veins draining the lateral cerebellar hemisphere.9,11

The diagnosis of venous thrombosis by cerebral angiography may be difficult because of marked individual variations in the venous outflow patterns.
Venous infarction is sometimes difficult to differentiate from neoplastic disorders. Our initial MR imaging findings were strikingly tumor-like, including mass effect and abnormal enhancement, which led to the incorrect initial diagnosis. Several patients have presented with cerebral venous infarction with MR imaging findings of abnormally enhanced tumor-like lesions,1,5 for which unnecessary biopsies were performed. Therefore, although thrombosis of the SPV is rare, it should be considered in the differential diagnosis of infratentorial lesion mimicking brain tumor.

References


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Fig. 4 Photographs of a cadaveric specimen demonstrating the veins in the right cerebellopontine angle, with the draining area of the vein of cerebellopontine fissure (VCPF) colored red (A), and showing the deep veins around the fourth ventricle (B). The vein of the cerebellomedullary fissure (VCMF) drains the periventricular area and empties into the VCPF. SPV: superior petrosal vein.

However, our case had some characteristic features. Firstly, the lesion appeared slightly hyperintense on DW imaging, suggesting vasogenic rather than cytotoxic edema. This phenomenon is known as the T2 shine-through effect, and should not be confused with the findings of acute infarction. Moreover, this lesion contained a hemorrhagic component, possibly caused by venous hypertension, which is an indirect sign of venous thrombosis. Secondly, the topography of the lesion was not consistent with any known arterial territory, but was compatible with the drainage territory of the SPV, especially the vein of the cerebellopontine fissure (Fig. 4A). This vein is the largest of the tributaries of the SPV, and drains both the petrosal surface of the cerebellum and the dentate nuclei or periventricular white matter through its tributaries such as the vein of the cerebellopontine fissure (Fig. 4B).1,11) Thirdly, the neurological and imaging abnormalities of the patient resolved spontaneously. Such reversibility supports the diagnosis of vasogenic edema without tissue necrosis. All these findings suggested vasogenic edema caused by thrombosis of the SPV as the predominant pathology in our case.

The clinical features of the thrombosis of SPV remain unclear, but several cases have shown the development of neurological deficit following sacrifice of the SPV during surgery.3,5,12) These postoperative changes vary from minor complications such as transitory cerebellar edema to major complications such as extensive hemorrhagic infarction in the brainstem and cerebellum. The complication rate may depend on individual variations in the anatomy of the SPV and on the degree of compensation by venous collaterals. We believe that an analogous situation exists in patients with thrombosis of SPV, as the severity of the symptom is attributable to individual variations in the anatomy of the SPV.