Case Studies

Report of Dramatic Improvement after a Lumboperitoneal Shunt Procedure in a Case of Anticoagulation Therapy-Resistant Cerebral Venous Thrombosis

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Cerebral venous thrombosis (CVT), which typically progresses from either acute or subacute onset, presents with symptoms related to intracranial hypertension (e.g., headache and papilledema) and brain parenchymal lesions (e.g., aphasia and hemiplegia). Anticoagulation therapy is generally accepted as a treatment for CVT and often leads to good clinical outcomes. However, we experienced a case of CVT with an uncommon clinical course. The patient was a 63-year-old man who presented with headache, papilledema, visual loss, and diplopia; his condition gradually deteriorated, and he was diagnosed with CVT via cerebral angiography. The sinus thrombus was extensive and resistant to anticoagulation therapy, and lumbar puncture revealed a progressive increase in cerebrospinal fluid (CSF) pressure. We performed a lumboperitoneal (LP) shunt procedure, which yielded marked improvement in the symptoms. The main mechanism of neurological dysfunction in CVT is venous outflow obstruction caused by venous thrombus, which results in brain edema, and/or venous infarction, which induces focal neurological signs. Another mechanism is impaired CSF absorption in the thrombosed sinuses, resulting in intracranial hypertension. We speculated that the latter mechanism strongly influenced our case, thus explaining the uncommon clinical course and effectiveness of the LP shunt procedure. Although LP shunting is not a common treatment for CVT, this case report could indicate the usefulness of this procedure for CVT with chronic progression and resistance to anticoagulation therapy. **Key Words:** Cerebral venous thrombosis—extensive thrombus—impaired cerebrospinal fluid absorption—resistance to anticoagulation—lumboperitoneal shunt.

Introduction

Cerebral venous thrombosis (CVT) is a rare type of stroke (.5%-1.0% of all strokes).[1] CVT often exhibits an acute or subacute onset with various symptoms. Ferro et al[2] reported that more than 90% of CVT cases exhibited acute or subacute onset, with less than 10% of cases featuring chronic onset. The clinical manifestations of CVT may include symptoms related to intracranial hypertension, such as headache and papilledema, or focal neurological symptoms, such as aphasia, hemiplegia, and seizure, caused by brain parenchymal lesions (e.g., venous infarction and/or hemorrhagic infarction).[3] To date, various treatments for CVT have been reported; of these, anticoagulation is a therapeutic mainstay, and its usefulness has been supported by 2 previous randomized controlled trials.[4,5] Ferro et al[2] indicated the use of anticoagulation therapy in most cases of CVT and reported a relatively good prognosis with approximately

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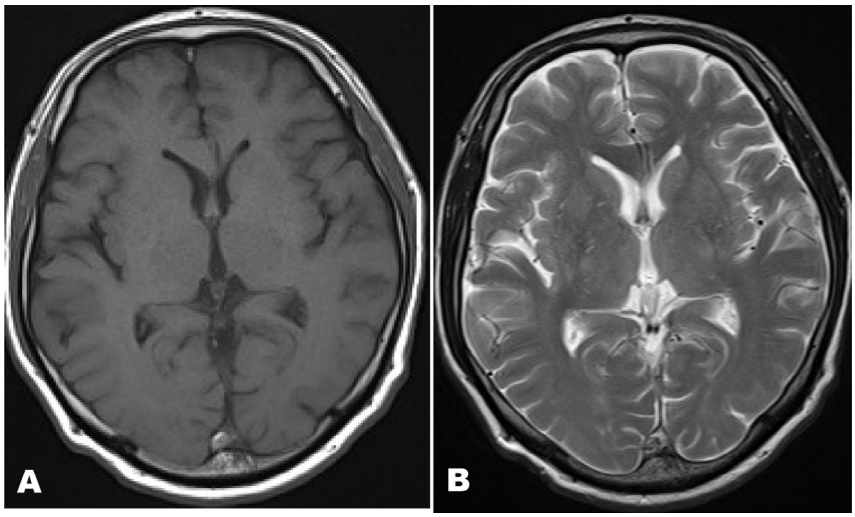

Figure 1. *T1-weighted magnetic resonance image (A) and T2-weighted image (B). These images indicated signal intensity alternation in the posterior aspect of the superior sagittal sinus (A and B).*

80% of patients achieving a complete recovery. More recently, endovascular treatments for CVT have been frequently reported. Some case series have indicated direct catheter thrombolysis and mechanical thrombectomy as effective therapies for CVT. Siddiqui et al[6-9] reported that more than 70% of CVT cases achieved near to complete recanalization, and more than 80% achieved a good outcome (modified Rankin Scale score 0-2). Notably, no relevant large prospective studies have been conducted. However, the American Heart Association guideline recommends considering endovascular intervention if deterioration occurs despite intensive anticoagulation treatment.[10]

Herein, we describe our experience with a case of chronically progressing CVT that exhibited resistance to anticoagulation therapy. As endovascular treatment was not available, we selected a shunt procedure, an uncommon treatment for CVT that was effective in our case and led to a good clinical outcome.

Case Report

A 63-year-old man with a history of diabetes mellitus, hypertension, and chronic otitis media was admitted to the department of internal medicine in our hospital with complaints of headache, nausea, and mild dizziness of 5 days' duration. He did not present with paralysis or disturbed consciousness. Plain computed tomography (CT) did not indicate any abnormal signs, but magnetic resonance imaging (MRI) demonstrated altered signal intensity in the superior sagittal sinus (Fig 1). This finding suggested a CVT, and the patient was subsequently referred to our department 7 days after symptom onset.

Cerebral angiography revealed a long segment thrombus from the superior sagittal sinus to both transverse sinuses. Collateral venous drainage, however, was relatively abundant, with principal drainage via the right sylvian vein into the jugular vein and the left vein of Labbe into the sigmoid sinus (Figs 2, 3). Although

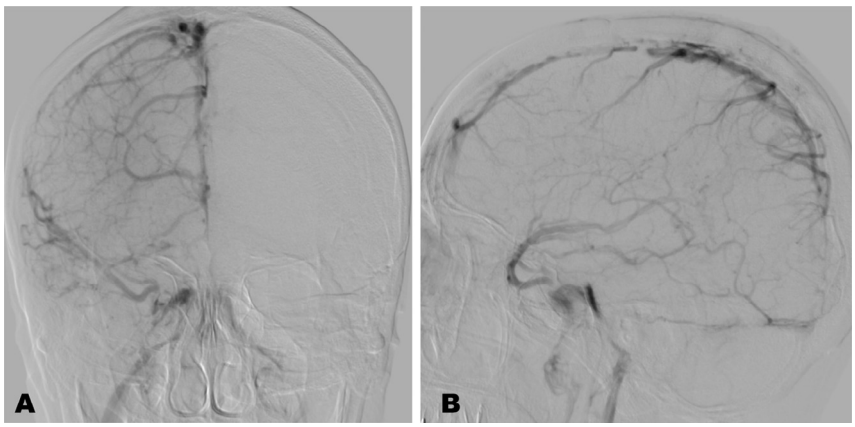

Figure 2. *Venous-phase right carotid angiography (anteroposterior view: A, lateral view: B) shows a filling defect in the posterior aspect of the superior sagittal sinus, the right transverse sinus, and the right sigmoid sinus, which caused a slight delay in the visualization of cerebral venous structures (not shown). The flow from the right sylvian veins to the cavernous sinuses and into the jugular veins is the main collateral venous drainage route and is relatively enlarged because of the large amount of venous drainage.*