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Case Report

Unilateral Thrombosis of a Deep Cerebral Vein Associated with Transient Unilateral Thalamic Edema

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Symptoms of deep cerebral vein thrombosis (DCVT) are variable and nonspecific. Radiologic findings are essential for the diagnoses. In the majority of cases of deep internal cerebral venous thrombosis, the thalamus is affected bilaterally, and venous hypertension by thrombosis causes parenchymal edema or venous infarction and may sometimes cause venous hemorrhage. Intravenous injections of mannitol can be administered or decompressive craniectomy can be performed for reduction of intracranial pressure. The objectives of antithrombotic treatment in DCVT include recanalization of the sinus or vein, and prevention of propagation of the thrombus. Herein, the authors report DCVT which was successfully treated by low molecular weight heparin.

Keywords Intracranial thrombosis, Venous thrombosis, Cerebral infarction, Brain edema

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INTRODUCTION

Deep cerebral venous thrombosis is an uncommon but often fatal disease, which can result in possible stroke, coma, and local neuropathies, with a mortality rate of 8-14%.[1)6)] However, most patients, exhibit mild symptoms, such as headache or papilledema due to increased intracranial pressure.[4)] Of these phenomena, occurrence of the deep cerebral venous thrombosis is more uncommon.[1,4)] The lesions of deep venous origin tend to be bilateral, and spread to the adjacent basal ganglia and other white matter regions from both sides of the thalamus.[2)3)] Thus, cases involving unilateral deep venous thrombosis at the thalamus are rarely reported.[7)12)] This study reports on a case in which a unilateral thrombosis at the internal cerebral vein caused reversible changes of an edema in the unilateral thalamus region.

CASE REPORT

A 36-year-old female patient presented with dysarthria and right-sided hemiparesis. The patient had suffered a spell of nausea, vomiting, and headache two years before admission. Findings on brain computed tomography (CT), and a cerebrospinal fluid (CSF) examination during the time showed no abnormal findings. The same symptoms recurred one week before admission, however, brain magnetic resonance imaging (MRI) showed no abnormal findings (Fig. 1A). One week later, she was admitted again due to newly-developed dysarthria and hemiparesis of her right side. A T2 fluid attenuated inversion recovery (FLAIR) imaging study of brain MRI at this time showed left thalamic high signal intensity, suggesting edema (Fig. 1B). An MRI Susceptibility weighted im-

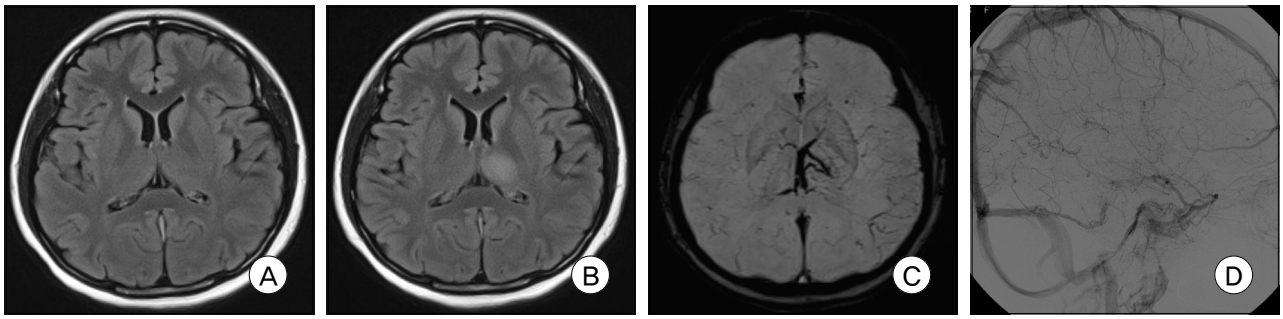

Fig. 1. Brain magnetic resonance images (MRI), computed tomography (CT) and digital subtraction angiography (DSA) obtained from a 36-year-old female patient with unilateral thrombosis of the left internal cerebral vein and thalamostriate vein. Initial T2 fluid attenuation inversion recovery (FLAIR) image shows normal finding in the thalamic area (A). Left thalamic edema is noted on a T2 FLAIR image obtained seven days after initial symptoms. The patient had dysarthria, hemiparesis, and a headache (B). Susceptibility weighted image (SWI) shows a diffuse susceptibility effect of thrombus in the left internal cerebral vein and thalamostriate vein (C). DSA shows occlusion in the left internal cerebral vein (D).

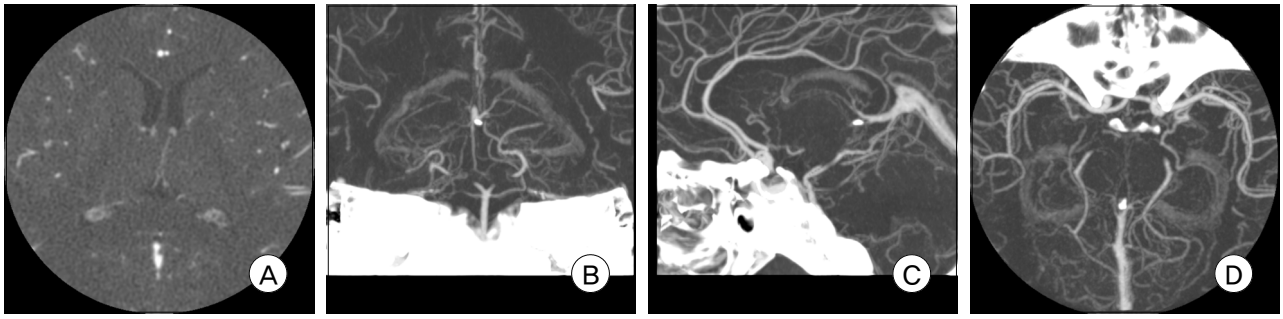

Fig. 2. After low molecular weight heparin therapy (60 mg subcutaneous injection, 120 mg per day), CT (A) and CT (B, C, D) angiography shows a recanalization in internal cerebral vein.

age (SWI) showed occlusions of the left internal cerebral vein and thalamostriate vein due to the thrombosis (Fig. 1C). Digital Subtraction Angiography (DSA) revealed a venous occlusion of the left internal cerebral vein and thalamostriate vein (Fig. 1D).

Low molecular weight heparin (LMWH) was administered subcutaneously 60 mg twice per day for one week without replacing LMWH to warfarin, and the dysarthria and right hemiparesis subsided completely. Findings on CT angiography one week after symptom onset revealed the left internal cerebral vein and the thalamostriate vein had regained normal blood flow (Fig. 2). Ten days after treatment, the patient was discharged with only a mild headache. The headache subsided over a period of several months, and a T2 FLAIR image of the brain MRI taken two months after the initial symptoms showed disappearance of pre-existing edema of the thalamus. SWI revealed decreased thrombotic stenosis of the internal cerebral and thalamostriate vein (Fig. 3).

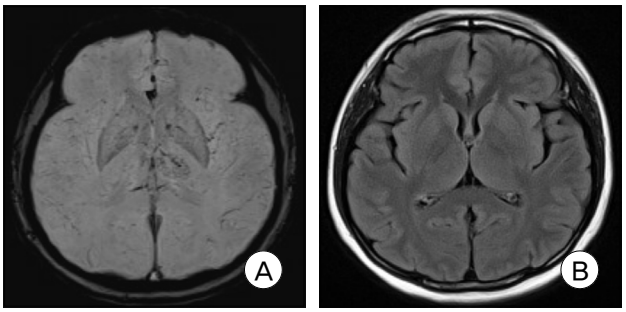

Fig. 3. Brain MRI 53 days after treatment. Susceptibility weighted image (SWI) shows resolved thrombus in the left internal cerebral vein and thalamostriate vein (A). T2 FLAIR image shows resolved left thalamic edema (B).

DISCUSSION

Pathogenesis and symptoms

Thrombosis of the cerebral veins is an uncommon disease, which arises from various causes, including infection, connective tissue disease, anemia, inflammatory bowel diseases, and trauma.[11] Women are afflicted three times more often than men.[6] Cerebral vein thrombosis often affects young pregnant or puerperal women, and those who are taking oral contraceptives or have coagulopathic disorders.[1]

When a superficial cerebral vein thrombosis occurs, the thrombosis shows slow progression, and the majority of patients complain of headache. Symptoms can also appear suddenly, and can be mistaken for subarachnoid hemorrhage.[5] As for cardiac arrhythmial thrombosis, it tends to occur in the bilateral thalamic region, and may result in delirium, amnesia, and mutism.[9] Cerebral vein thrombosis arises from either of two mechanisms, one being an ischemic nerve injury following venous edema caused by a thrombosis, and the other being an intracranial hypertension due to the decrease of CSF absorption following a venous occlusion.[14] In our patient, nausea and headache were followed by dysarthria and unilateral nerve palsy. The cause of nausea and headache appeared to be intracranial hypertension and neurologic deficit appeared to be ischemic nerve injury due to venous edema.

Diagnosis

Due to its non-specific symptoms, cerebral vein thrombosis can be easily over-looked. Because venous paths do not match with arterial paths, venous infarctions cannot be easily localized with symptoms. Thus, suspicion of cerebral vein thrombosis in high-risk patients is very important, and adequate radiologic study is needed.[13] A diagnosis can be made with an abnormal signal on MRI, and confirmation of loss of blood flow on MRA.[8] As for a thrombosis on the internal cerebral vein, the thalamus is bilaterally affected, causing vascular edema of parenchyma, which can induce hemorrhage or infarction. A vascular edema or infarction is hyperintense upon T2 of the MRI, and hypointense upon T1 imaging. A thrombosis at a deep cerebral vein, like our patient's finding, showed hyperintensity on T2 FLAIR, and a loss of flow signal intensity through an SWI.[2][3] Based on these radiological findings, our patient was confirmed as having a thrombosis of the left internal cerebral vein and left thalamic edema. Other than the MRI, a cerebral angiography, which can show a more precise vascular stature, can also be utilized. We were able to confirm the accurate location of the thrombosis of our patient using the MRI and DSA.

Treatment and prognosis

When the patient's neurologic status suggests imminent brain herniation, emergency treatment is needed. Intravenous mannitol and sometimes decompressive craniotomy may be required.[15] Other than immediate treatment for acute exacerbation, the general antithrombotic treatment using heparin is used for reperfusion of a thrombotic occluded vein or sinus. As reported by Pfefferkorn et al., of 32 patients with deep cerebral vein thrombosis, all patients were treated with dose-adjusted intravenous heparin (activated partial thromboplastin time (aPTT) 60-100 sec) or LMWH. Twenty-four patients (75%) were stabilized and later showed improvement. Heparin was then switched to oral anticoagulants. Eight patients (25%) showed rapid deterioration to coma over a period of 6-48 hours.[10] Our patient received subcutaneous administration of 60 mg of LMWH twice per day without replacing LMWH to warfarin, and neurological symptoms showed improvement. Urokinase or endovascular thrombolysis can also be considered for patients who show no improvement with this conventional treatment.[10] The outcome of deep cerebral vein thrombosis varies greatly, from complete recovery to death. As for our patient, neurological symptoms disappeared completely after treatment, and previous lesions were no longer seen.

Compared to superficial cerebral venous thrombosis,

deep cerebral venous thrombosis has a higher tendency to leave permanent neurologic symptoms and to show more rapid progression.[3] Prognosis of unilateral deep cerebral vein thrombosis is better than that of bilateral thrombosis. According to one report, a patient with a unilateral thrombosis showed complete recovery from neurologic symptoms.[7][12]

CONCLUSION

Due to its lack of specific symptoms, unilateral deep cerebral vein thrombosis is an uncommon disease that can be misdiagnosed. Therefore, careful suggestion is important. In addition, due to the fact a unilateral lesion shows better prognosis than a bilateral one, a quick diagnosis and treatment are needed. Due to the small volume of patients in previously reported studies, more research into the pathology and natural course of a unilateral deep cerebral vein thrombosis through treatment of more patients is needed.

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