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Case Report

A Case of Cerebral Venous Sinus Thrombosis Followed by Subdural Hematoma in a Pregnant Woman

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Abstract

Cerebral venous sinus thrombosis (CVST) is an uncommon cause of stroke and accounts for 0.5-1% of all stroke cases. A 23-year-old woman who was 33 weeks pregnant visited the hospital complaining of a right temporo-parietal headache which had lasted for 5 days. The headaches were new onset, unilateral, and non-throbbing and were associated with nausea and vomiting. She had no significant past medical history. There was no history of trauma, illicit drug abuse, smoking, or alcohol use. There was no family history of coagulopathy or thrombophilia. A neurological examination demonstrated bilateral grade 2 papilledema. Brain MRI and MR venography were performed that showed a left fronto-parietal subdural hematoma with left transverse sinus thrombosis. Genetic analysis showed a heterozygotic mutation of MTHFR and a homozygotic mutation for Angiotensin-Converting Enzyme (ACE). Screening for other hypercoagulable states were negative. Anticoagulant therapy with a therapeutic dose of heparin was administered intravenously immediately and her symptoms disappeared one week after admission.

Keywords: *Cerebral Venous Sinus Thrombosis, Subdural Hematoma*

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Introduction

Cerebral venous sinus thrombosis (CVST) is an uncommon cause of stroke and accounts for 0.5-1% of all strokes. The most common clinical manifestations are intracranial hypertension and hemorrhagic infarctions [1,2]. Nontraumatic subdural hematomas can occur secondary to various types of cerebrovascular lesions, such as ruptured aneurysms [3,4] or arteriovenous malformations [3], spontaneous intracerebral hemorrhages [4], or thrombosis of a cerebral vein or dural sinus [5]. Among these lesions, subdural hematoma associated with cerebral venous thrombosis has been rarely reported. A case in which thrombosis of the left transverse sinus was followed by a frontoparietal subdural hematoma has been observed.

Case Presentation

A 23-year-old woman who was 33 weeks pregnant presented with a new onset, unilateral, non-throbbing right temporoparietal headache, associated with nausea and vomiting, which had started 5 days prior to admission. There was no history of trauma, drug abuse, smoking, or alcohol use. There was no family history of coagulopathy or thrombophilia.

Neurological examination

Bilateral grade 2 papilledema, and other normal findings.

Laboratory studies

Complete blood count (including platelet) and biochemistry were normal.

Brain MRI and MR venography

Left frontoparietal subdural hematoma with left transverse sinus thrombosis (Figure 1). Intravenous heparin was administered to the patient immediately and her symptoms disappeared one week after admission. She underwent a caesarean section, and was referred to our hospital again for continuation of treatment. Follow-up MRI and MR venography were done and she was discharged without any symptoms.

Genetic analysis

A heterozygotic mutation for MTHFR and a homozygotic mutation for Angiotensin-Converting Enzyme (ACE) were detected. Screening for other hypercoagulable states was negative. Factor V Leiden and prothrombin levels were normal.

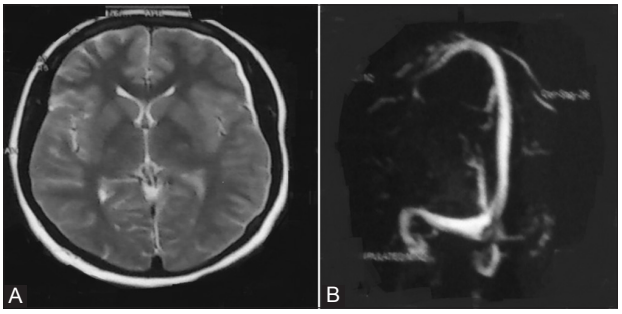

Figure 1. (A)T2 weighted brain MRI revealed high signal intensity at left frontal lobe of subdural hematoma . (B) Brain MR venography shows a .(filling defect in favor of left transverse sinus thrombosis

Discussion

The pathological changes described in the early stages of thrombosis of the cerebral veins and dural sinuses include cerebral edema, petechial hemorrhages, hemorrhagic infarction, subarachnoid hemorrhage (SAH), and intracerebral hematoma. In the acute stage of thrombosis, dilated cerebral veins are prone to rupture, causing subdural, subarachnoid, or intracerebral hemorrhage [6, 7]. However, subdural hematoma with cerebral venous sinus thrombosis are rarely reported in conjunction. Most patients with CVST are younger than 50 years. Systemic conditions which increase the risk of CVST include pregnancy, puerperium, hormone consumption, genetic coagulopathy, malignancy, polycythemia vera and anemia, myeloproliferative disorders, and dehydration.

Mechanical factors which reduce blood flow in the cerebral sinuses and cause thrombosis include adjacent infections (such as mastoiditis), neoplastic invasion of the sinus, trauma, and neurosurgical procedures. About one-third of patients with CVST do not have thrombophilia or other predisposing conditions [2]. The use of magnetic resonance imaging (MRI), MR venography (MRV) and computed tomography (CT) venography has improved the ability to detect other clinical manifestations of CVST, including subdural hematoma (SDH) [8] and cortical SAH [9]. Diagnosis of CVST is challenging due to the variability in presenting symptoms and signs. It is accepted that CVST may present with elevated intracranial pressure (ICP) and hemorrhagic venous infarcts [2]. There is increasing evidence that CVST can cause SDH and focal cortical SAH [9].

Many patients with SDH following CVST had underlying hypercoagulable states. The presence of thrombophilia is generally established based on personal and family history, as well as from selective laboratory testing. Our patient was at risk due to pregnancy. An evaluation of risk factors for cerebral venous sinus thrombosis should include screening for thrombophilia, including medications such as oral contraceptives, deficiencies in protein C, protein S, antithrombin III, genetic mutations in the prothrombin gene (G20210A mutation) and the Factor V gene (Leiden mutation), anticardiolipin, beta-2-glycoprotein, lupus anticoagulant, anti-dsDNA antibodies, ANA, homocysteinemia related to mutations in the methyltetrahydrofolate reductase gene (C677T and A1298C mutations), and hemoglobinopathies such as sickle cell disease [10]. The proposed mechanism of SDH following CVST is venous hypertension and associated venous engorgement, after which venous bleeding occurs. Treatment must be individualized. Once CVST is identified and treated using anticoagulants, satisfactory recovery occurs. The use of anticoagulation in patients with hemorrhagic infarct and CVST has gained acceptance [2]. In patients with small SDHs (with or without associated intracerebral hemorrhage), there are no clear guidelines for patient management. The patient in this case was successfully managed with systemic anticoagulation as well as frequent laboratory and imaging monitoring.

It is important to recognize that SDH and CVST may both arise as a rare complication of intracranial hypotension [8, 11]. Our patient had neither a clinical history nor a radiologic finding of intracranial hypotension.

Conclusion

Cerebral venous sinus thrombosis may present with spontaneous SDH and with or without associated hemorrhagic infarction. With easy to use MR and CT venography, the diagnosis of CVST complicated by SDH and cortical SAH will improve. The management of patients with SDH complicating CVST is complex, as a result of contraindications for anticoagulation in patients with symptomatic SDH.

Conflict of Interest

There is no conflict of interest regarding this manuscript.

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