Case Studies

Association of Cerebral Venous Thrombosis and Intracranial Hypotension: Review of 3 Cases

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Cerebral venous thrombosis is a rare complication of intracranial hypotension. We describe 3 cases in which this phenomenon occurred, as a result of a lumbar puncture or due to a spontaneous cerebrospinal fluid leak. We emphasize the importance of early detection of the intracranial hypotension syndrome, the most common clinical manifestation being orthostatic headache. It is not an innocent condition as it is associated with other potential complications such as subdural hygroma/hematoma, cranial nerve palsies, cerebellar tonsillar descent, and even brainstem manifestations. Any change in the typical features of the syndrome should lead to further investigation. Repeat cerebral imaging is important in that situation, including ruling out cerebral venous thrombosis. **Key Words:** Liquor hypotension—intracranial hypotension—cerebral venous thrombosis—venous sinus thrombosis—orthostatic headache.
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Introduction

The most common manifestation of intracranial hypotension is orthostatic headache. Typically, the headache worsens over a 10-15 minute period after the subject moves upright. Other, but less pathognomonic, symptoms are tinnitus, neck pain, nausea, and hearing changes. The low pressure status is the result of a depletion of the intracranial cerebrospinal fluid (CSF) volume and can be due to spontaneous intracranial hypotension or secondary to a trauma or procedure, including a lumbar puncture. The incidence is estimated to be 2-5/100,000 and women are more affected than men (ratio 2:1).[1,2]

The diagnosis is based on patient history. Measurement of low intracranial pressure by lumbar puncture can confirm the diagnosis. Typical magnetic resonance imaging (MRI) changes are pachymeningeal enhancement after intravenous gadolinium contrast administration (present in 85%-90% of cases), subdural collections, downward displacement of the cerebral tonsils, enlargement of the venous structures, and pituitary hyperemia.[3] It is not unusual for milder cases that imaging remains normal.[4] In the case of otorrhea or rhinorrhea, CSF leakage can be proven by the detection of beta-trace protein. The site of leakage can be demonstrated by a variety of imaging techniques. Computed tomography (CT) myelography and cisternography are widely used for the detection of the leakage site, have a high sensitivity, and are currently the standard techniques. The resolution and sensitivity of MRI myelography and cisternography after off-label intrathecal administration of gadolinium are even better than CT myelography, but widespread introduction is hampered by concerns over

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gadolinium deposition in the brain, which is of unknown clinical significance[5,6] Radionuclude cisternography has become less important because of better resolution of the aforementioned techniques but can have additional value when both CT and MRI myelography remain negative.[7]

Persistent low pressure status can lead to several complications, such as cranial nerve palsies, subdural hygroma or hematoma, cerebellar tonsillar descent, and brainstem symptoms, including coma.[8] About 2% of the cases are complicated by venous thrombosis.[9] Several cases have been described in the past[10-20] and intracranial hypotension is briefly mentioned in a recent review on risk factors for cerebral venous thrombosis.[21] In this report, we describe 3 patients who were diagnosed with cerebral venous sinus thrombosis secondary to the intracranial hypotension syndrome. We hope this will help the stroke physician to recognize this clinical scenario.

Case Descriptions

Case 1

A 21-year-old woman with headache, which had started 1 day before, presented to the emergency department. She complained of occipital headache, nausea, and photophobia when upright, with complete relief when lying down. She recalled a minor trauma to the head several days before, without loss of consciousness or any sign of rhinorrhea or otorrhea. The clinical examination was normal, including absence of neck stiffness or fever. Laboratory testing was unremarkable. A non–contrast-enhanced brain CT was normal. Gadolinium-enhanced MRI of the brain was performed that same day and was normal as well. Despite the absence of meningeal enhancement, we made the diagnosis of a spontaneous intracranial hypotension syndrome. She was admitted and received an autologous 25-cc lumbar epidural blood patch, after which she remained pain-free for a short period. During further observation, the headache redeveloped but persisted even in the horizontal position. A repeat non–contrast-enhanced brain CT was suggestive of bilateral cortical vein thrombosis, which was confirmed by MRI (Fig 1). She was taking an oral contraceptive drug at that time. Screening for thrombophilia revealed a heterozygous Factor V Leiden mutation. Anticoagulant therapy with a vitamin K antagonist was initiated for a period of 6 months, after which further treatment with acetylsalicylic acid was continued. After discharge, she remained free of headache.

Case 2

A 22-year-old woman was referred for urgent neurologic evaluation because of worst headache ever. Eight days before, when she gave birth to her son, she was given epidural anesthesia. A day after she gave birth, she complained of an orthostatic headache. The diagnosis of postdural puncture headache was made, and she was successfully treated with a single autologous 25-cc lumbar epidural blood patch. However, after a pain-free interval of 1 day, the headache recurred in a gradual manner and was not relieved by lying down. The headache had a throbbing quality and was accompanied by nausea, vomiting, and visual blurring. The neurologic examination was unremarkable. There was no fever or neck stiffness. An urgent non–contrast-enhanced CT of the brain showed no abnormalities. Laboratory testing showed no signs of infection, but revealed elevated D-dimers (7480 ng/mL; normal value <500 ng/mL). She was admitted for further diagnostic testing and symptomatic treatment. A gadolinium-enhanced brain MRI was scheduled for the day after, and showed impressive meningeal enhancement, a narrow aspect of the ventricles, and cisterns and a small subdural hematoma in the caudal posterior fossa (Fig 2). Besides these hallmarks of intracranial hypotension, a cortical vein thrombosis was seen in the right rolandic area. There were no parenchymal lesions. No papilledema was seen during funduscopic examination. The diagnosis of a cortical vein thrombosis, secondary to intracranial hypotension, was made. No underlying thrombophilia was found. Treatment with oral antico-

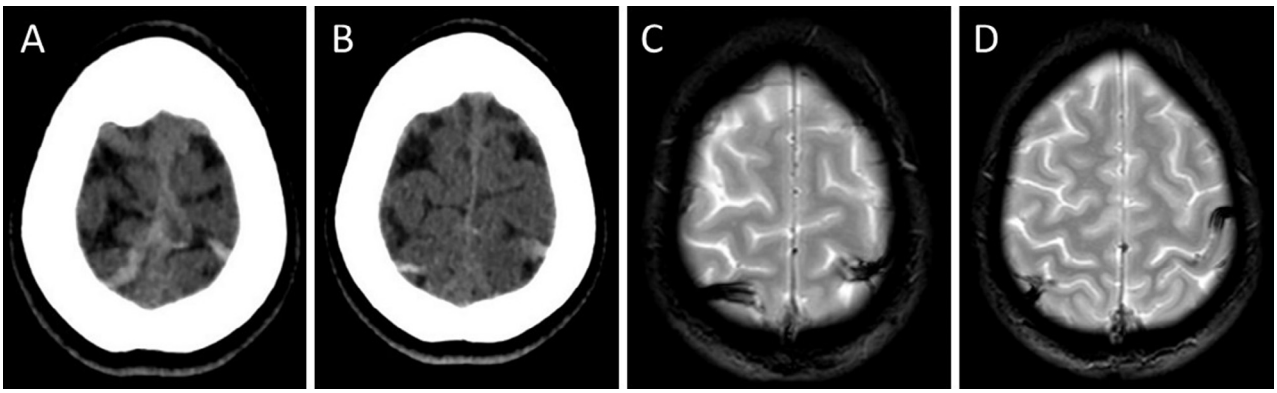

Figure 1. *(A and B): Unenhanced brain CT showing bilateral cortical hyperdensities, most likely thrombosis of cortical veins. (C and D): T2* weighted MRI sequences showing bilateral cortical vein thrombosis in the parietal region. Abbreviations: CT, computed tomography; MRI, magnetic resonance imaging.*

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