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## Isolated Cortical Venous Thrombosis Associated With Intracranial Hypotension Syndrome

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**The association of intracranial hypotension syndrome with cerebral venous thrombosis is rare. We report our experience with isolated cortical venous thrombosis, which developed after unsuccessful epidural anesthesia. Magnetic resonance imaging showed characteristic imaging findings of intracranial hypotension syndrome, such as dural thickening and brain sagging. We also detected right parietal venous hemorrhagic infarction secondary to right-sided cortical venous thrombosis. After the treatment of intracranial hypotension via epidural blood patch, heparin was used to treat cortical venous thrombosis.**

**Key words:** intracranial hypotension, cortical venous thrombosis, epidural anesthesia, magnetic resonance imaging

It is well-known that intracranial hypotension may be seen following spinal interventions including surgery and lumbar puncture (LP) or it may develop spontaneously. Diagnosis of this condition became easier after the introduction of magnetic resonance imaging (MRI).<sup>1</sup> A few reports published recently emphasize the tendency of intracranial venous thrombosis in patients with intracranial hypotension.<sup>2</sup> In this case report we aim to present our experience of isolated cortical venous thrombosis and venous ischemia in a 25-year-old patient with intracranial hypotension secondary to unsuccessful epidural anesthesia.

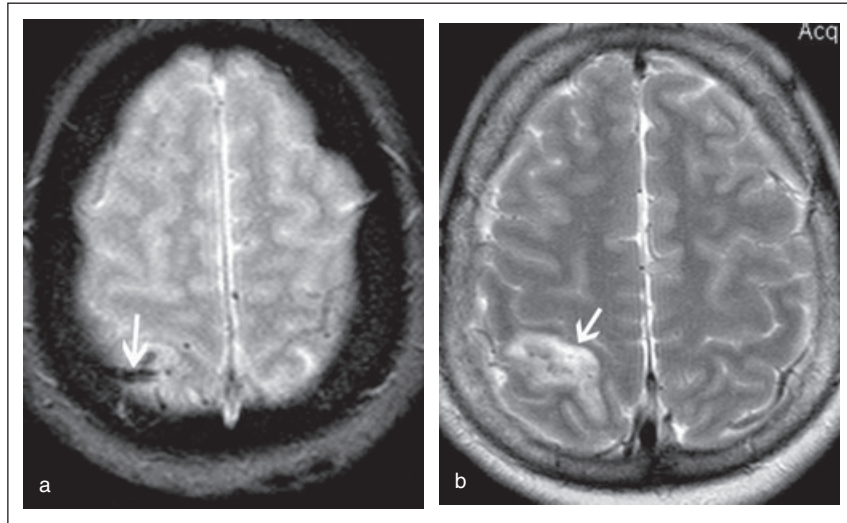
### CASE REPORT

The patient was a 25-year-old woman suffering from pelvic pain and vaginal bleeding during the 36th gestational week. During her admission to an outer institution, she was

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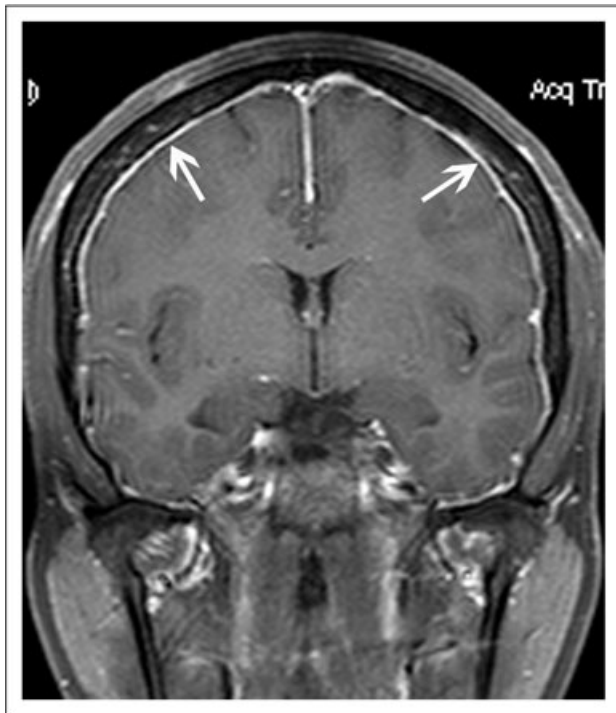
diagnosed with ablation placentae and scheduled for emergency caesarean section. The procedure was planned under epidural anesthesia; however, according to the patient's statement, epidural access could not be achieved after several painful attempts and the section was performed under general anesthesia. The operation was uneventful, but on the following day she began to suffer from a headache with moderate intensity. The headache developed when the patient stood up and subsided when she lied down. After symptomatic treatment for headaches, she was discharged on the third day postoperatively without any complaints. On the following day she was, however, admitted with a headache, dizziness, vertigo, and nuchal rigidity. This time, the headache was diffuse, had marked intensity, and was not exacerbated by erect posture. She was discharged with suggestions of oral hydration and caffeine consumption. On the sixth day postoperatively, she developed grand mal seizures 3 times followed by unconsciousness. Computed tomography revealed acute right parietal hemorrhage and the patient was referred to our institution. Nonenhanced MRI examination, which was performed at our institution, showed venous hemorrhagic ischemia adjacent to right central sulcus (Fig. 1). Contrast-enhanced images revealed bilateral dural thickening and brain sagging (Fig. 2). In mag-



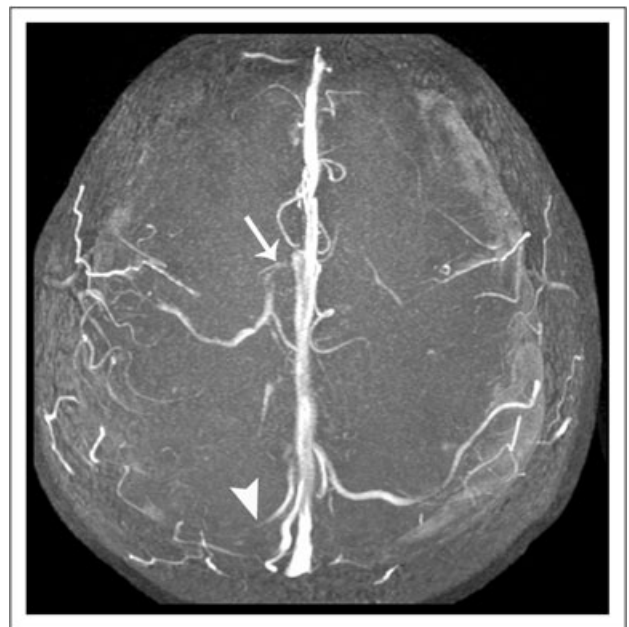
**Fig 1.—**The thrombosis of a right parietal vein (white arrow) is seen in axial gradient echo image (a). Axial T2-weighted image shows venous hemorrhagic ischemia adjacent to right central sulcus (white arrow) (b).

netic resonance venography, dural venous sinuses and deep venous structures were unremarkable. However, thrombosis of the right parietal vein and narrowing of a superficial cortical vein anterior to this parietal vein were detected (Fig. 3). These findings were found to be consistent with

intracranial hypotension secondary to epidural intervention and isolated thrombosis of the right parietal vein was considered a complication of intracranial hypotension. The patient was treated with epidural blood patch and followed by heparinization for cortical venous thrombosis. On the third day following the epidural blood patch, the patient was mobilized. Headaches and other symptoms were resolved



**Fig 2.—**Coronal contrast enhanced T1-weighted image depicts bilateral dural thickening and contrast enhancement (white arrows) secondary to intracranial hypotension syndrome.



**Fig 3.—**Magnetic resonance venography demonstrates thrombosis of a right parietal vein (white arrowhead) and anterior to this, narrowing of a superficial cortical vein (white arrow) is seen.

gradually and she was discharged symptom-free on the sixth day. Follow-up MRI in the third week showed complete resolution of the dural thickening and brain sagging. The right parietal venous ischemia resulted in encephalomalacia.

## DISCUSSION

The wide availability of MRI enables an increased rate of detection of intracranial hypotension. It is also accepted nowadays that what was previously believed to be an orthostatic headache after LP, or spinal interventions, are instances of intracranial hypotension syndrome. Intracranial hypotension is presented with a decreased volume of cerebrospinal fluid (CSF) resulting in venous engorgement according to Monro-Kelli law. This venous engorgement is seen as dural thickening and dilatation of venous structures in contrast-enhanced cranial MRI.<sup>1</sup>

To date, published experience with dural sinus thrombosis related to intracranial hypotension is limited to several case reports, with no studies of larger patient populations on this topic. These few anecdotal reports showed that both spinal interventions (including LP) and spontaneous intracranial hypotension may cause dural sinus thrombosis. The main trigger in the development of this rare complication is the venous engorgement because of a CSF leak through the spontaneous or iatrogenic dural tear. Compensatory dilatation of the venous sinuses and traction of cortical veins and venous sinuses in intracranial hypotension syndrome lead to stasis of blood flow and subsequent tendency for thrombosis.<sup>1,3,4</sup> A change in the characteristics of the headache, such as an increase in the intensity or loss of its orthostatic nature, should alert the clinician about venous thrombosis. Dural sinus thrombosis following LP was reported previously in the literature.<sup>3,4</sup> Canhão et al performed transcranial Doppler ultrasound study to register the mean blood flow volume (BFV) of the straight sinus (SS) before, during, and after LP.<sup>5</sup> They concluded that LP induced a sustained decrease of mean BFV in the SS. The decrease of venous blood flow is a possible mechanism contributing to the occurrence of cerebral venous thrombosis. The first report of dural sinus thrombosis after epidural anesthesia came from Schou et al in 1986.<sup>3</sup> Kapessidou et al detected superior sagittal sinus thrombosis after subarachnoid analgesia for labor.<sup>4</sup> These anecdotal reports emphasized that spontaneous intracranial hypotension syndrome may also cause dural sinus thrombosis.

Isolated cortical vein thrombosis has only been reported sporadically and was not seen in large series. The etiology of this entity remains unknown in a considerable number of patients. In previous reports of cortical vein thrombosis, different underlying diseases have been

described. These include Hodgkin's disease, systemic lupus erythematosus, postpartum state, decreased antithrombin III-level, protein C-resistance, bacterial meningitis, polycythemia, and nephrotic syndrome.<sup>6</sup> Mouraux et al reported cortical venous thrombosis in a patient with Behçet's syndrome after LP. They treated the patient only with heparinization.<sup>7</sup> Lai et al observed isolated cortical venous thrombosis in a patient with spontaneous intracranial hypotension. The patient was treated with epidural blood patch followed by heparinization. This management prevented the development of venous ischemia.<sup>8</sup> Wang et al detected isolated cortical venous thrombosis and subarachnoid hemorrhage in a patient with intracranial hypotension, which was treated with epidural blood patch only.<sup>9</sup>

In our case, the etiology of isolated cortical venous thrombosis is thought to be related to unsuccessful traumatic epidural anesthesia. It might be possible that the postpartum state played a role in causing the cortical venous thrombosis in our case. However, the initial headache was orthostatic in its character and later development of diffuse, nonpostural headache with increased intensity led us to think that the patient suffered from intracranial hypotension syndrome first and then she was complicated with cortical venous thrombosis.

To our knowledge, there is no publication in the literature about isolated cortical venous thrombosis in intracranial hypotension secondary to spinal anesthesia. Cortical venous thrombosis was also accompanied by venous hemorrhage. As the underlying pathology of the cortical venous thrombosis was intracranial hypotension, we preferred to treat it first. Immediately after the procedure, the patient experienced lessening of her headache, which led us to think that the dural tear occluded successfully and the leak of CSF ceased. Therefore, we initiated systemic heparin for the treatment of venous thrombosis on the next day.

In conclusion, unintended dural penetrations during epidural anesthesia may cause intracranial hypotension syndrome, which can result in dural sinus thrombosis because of compensatory dilatation and stasis of blood flow in venous structures. As was evident in this case, it is also possible to detect isolated cortical venous thrombosis and venous hemorrhage in these patients.

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## Dentigerous Cyst Presenting With Sinus Headache as the Primary Symptom

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The issue of whether headaches and facial pain are caused by sinusitis has generated much discussion in the literature. Nevertheless, headaches associated with facial pain and pressure are commonly given the nonspecific diagnosis of sinus headache.<sup>1</sup> A sinus headache is a secondary headache associated with sinusitis that occurs when the sinuses become congested and obstructed because of paranasal sinus pathology. Typically sinus headache refers to episodes of pain over the sinus area of the face or around the eyes, often associated with nasal congestion, rhinorrhea, lacrimation, and nausea.<sup>2</sup>

However, occasionally sinonasal disease can be manifested solely as headache.<sup>3</sup> On these occasions, sinus headache is often confused with primary headache such as migraine or tension headache. Thus, sinus headache, in the absence of sinonasal symptoms, usually delays its diagnosis.

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Here we report a case in which a dentigerous cyst in the maxillary sinus proved to be a clinically significant cause of sinus headache.

### CASE REPORT

A 20-year-old, previously healthy woman presented complaining of acute continuous headache of 3 days' duration. The pain, which was nonthrobbing and severe pressure-like, was located over the antral area and radiated to her upper teeth and to the forehead. It was not associated with facial swelling, and remained unchanged by position (bending forward or lying down or sitting up). Conventional analgesics were prescribed by a primary physician but had no effect. She was referred to our department for evaluation of continuous severe headache.

The patient denied hypertension, photophobia, visual disturbances, paresthesias, nausea, fever, or history of headaches. She had no history of any sinusitis, seasonal allergies, or other sinus problems and also denied sinonasal symptoms such as nasal discharge, postnasal drip, and nasal congestion.

On examination of the nasal cavity, the nasal septum was mildly deviated to the left, but there were no purulent discharge, nasal polyps, or masses. There was tenderness, without increased warmth or erythema, over her left max-

*Conflict of Interest:* None