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Cerebral venous thrombosis presenting with subarachnoid hemorrhage after spinal anesthesia

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Abstract

A 22-year-old male had a meniscopathy operation using spinal anesthesia. After the operation, the patient reported a throbbing headache. His brain computed tomography (CT) showed subarachnoid hemorrhage (SAH) and hyperdense dural venous sinuses suspicious for thrombosis. Filling defects were observed in the superior sagittal and right transverse sinuses on the contrast-enhanced magnetic resonance images. The patient was diagnosed with cerebral venous sinus thrombosis (CVST). On the tenth day of his admission, his clinical findings progressed and heparin therapy was initiated after resorption of hemorrhage was observed in a second non-contrast CT scan. The patient developed decreased consciousness the day after heparin administration. A subsequent brain CT revealed intraparenchymal hemorrhage in the right anteroinferior frontal region. Heparin therapy was discontinued, and anti-edema therapy was started. The presentation of CVST with SAH is a rare condition. Furthermore, development of CVST after spinal anesthesia is very rare. In this case, CVST developed after spinal anesthesia, and its first clinical presentation was SAH. To our knowledge, this is the first case CVST presenting with SAH after spinal anesthesia.

Key words: Postdural puncture headache; spinal anesthesia; subarachnoid hemorrhage; cerebral venous sinus thrombosis; intraparenchymal hemorrhage.

Introduction

Cerebral venous sinus thrombosis (CVST) is a rare form of thrombosis characterized by headache, nausea, vomiting, motor or sensory deficits, impaired consciousness, seizures, and even coma. Few reports of CVST concomitant with postdural puncture headache (PDPH) (1) and of CVST presenting with subarachnoid hemorrhage (SAH) have been published (2). Here, we report a case of CVST presenting with SAH after spinal anesthesia.

Case

A 22-year-old male had a meniscopathy operation using spinal anesthesia. Two days after the operation, the patient reported a throbbing headache, especially in the frontotemporal area. The headache was severe when the patient was standing and subsided when he lay down. He used analgesics during this period. He was admitted to our department because of generalized tonic-clonic epileptic seizures. His first neurological examination revealed limited cooperation. He had neck stiffness, left hemiparesis, and mild central left facial paralysis.

His non-contrast brain computed tomography (CT) showed hyperdense acute subarachnoid hemorrhage in the right supratentorial area, sylvian fissure, anteroinferior temporal, frontal and insular sulci, and hyperdense superior sagittal and right transverse sinuses (cord sign) suspicious for CVST (Fig. 1). The patient underwent contrast-enhanced magnetic resonance (MR) imaging. Defects were observed in the superior sagittal and right transverse sinuses on the contrast enhanced T1-weighted MR images (Fig. 2). Patient was diagnosed with CVST. The diagnosis of CVST was confirmed by conventional angiography. Defects were observed in the superior sagittal and right transverse sinuses during venous phase of the angiography.

Whole blood count test and routine biochemical analysis including sedimentation rate revealed normal results. Protrombin time, activated partial thromboplastin time, Lyme serology, VDRL, Wright agglutination, vasculitis markers, homocysteine, protein C, and protein S levels, antitrombin III activity, and Von Willebrand Factor antigen were within the normal range.

On the tenth day of his admission, left hemiparesis progressed and heparin therapy was initiated after

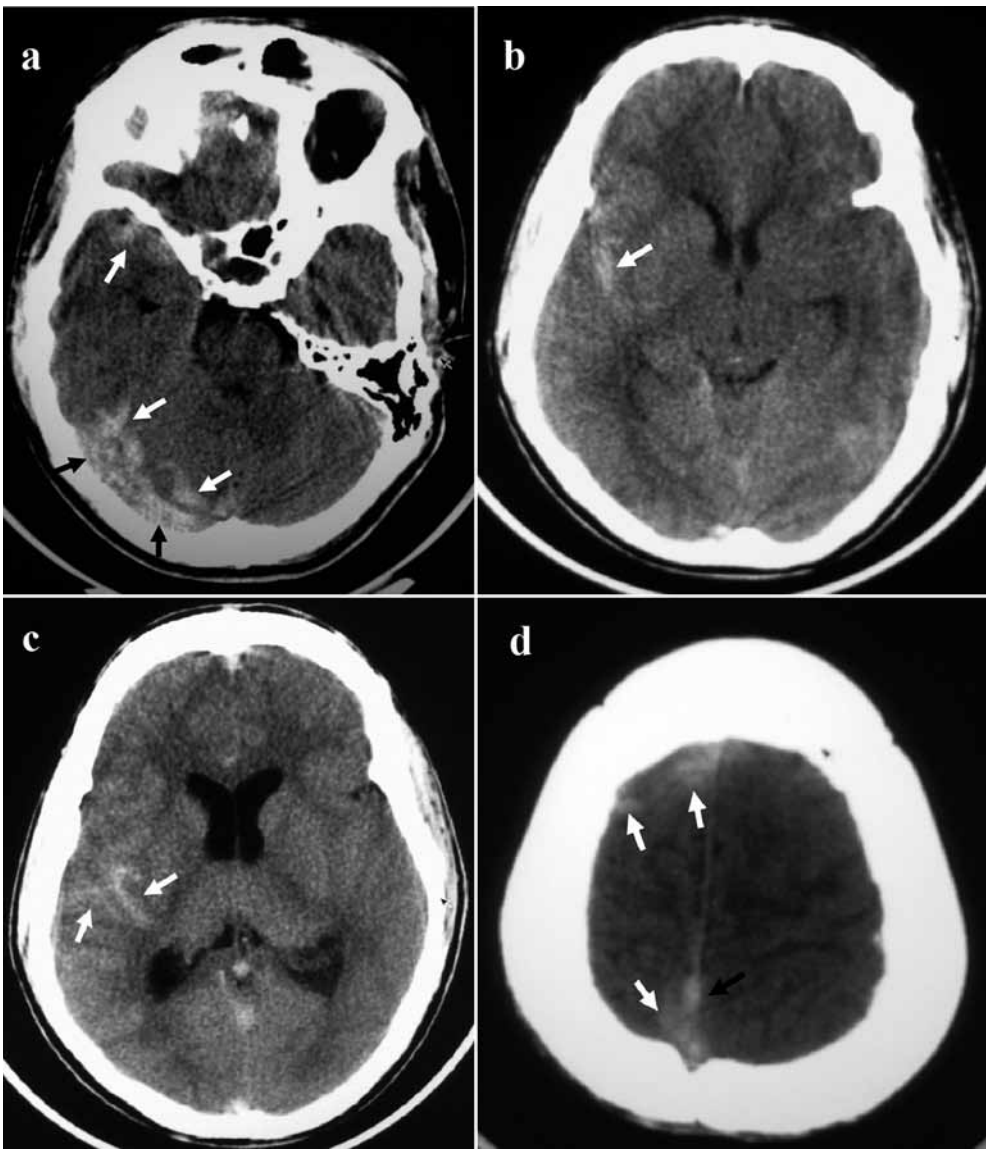

FIG. 1. — Axial computed tomography images without contrast (a-d) show hyperdense acute subarachnoid hemorrhage in the right supratentorial area, sylvian fissure, anteroinferior temporal, frontal and insular sulci (white arrows), and hyperdense right transverse sinus and superior sagittal sinus representing acute thrombosis (black arrows).

resorption of hemorrhage was observed in a second non-contrast CT scan. The patient developed decreased consciousness the day after heparin administration. A subsequent brain CT revealed intraparenchymal hemorrhage in the right anteroinferior frontal region (Fig. 3). Heparin therapy was discontinued, and anti-edema therapy was started. A day later, the patient fell unconscious. During the follow-up period, approximately 3 weeks after admission, the patient died due to secondary infections and metabolic changes.

Discussion

Headache is the most common symptom in CVST (3). Headache due to CVST can present symptoms similar to that following a dural puncture (1). PDPH characteristically presents with frontal or occipital headache that becomes intense when the patient is in the upright position and disappears when the patient returns to the supine position (4). Cerebrospinal fluid leakage may be responsible for headaches after dural puncture (5).

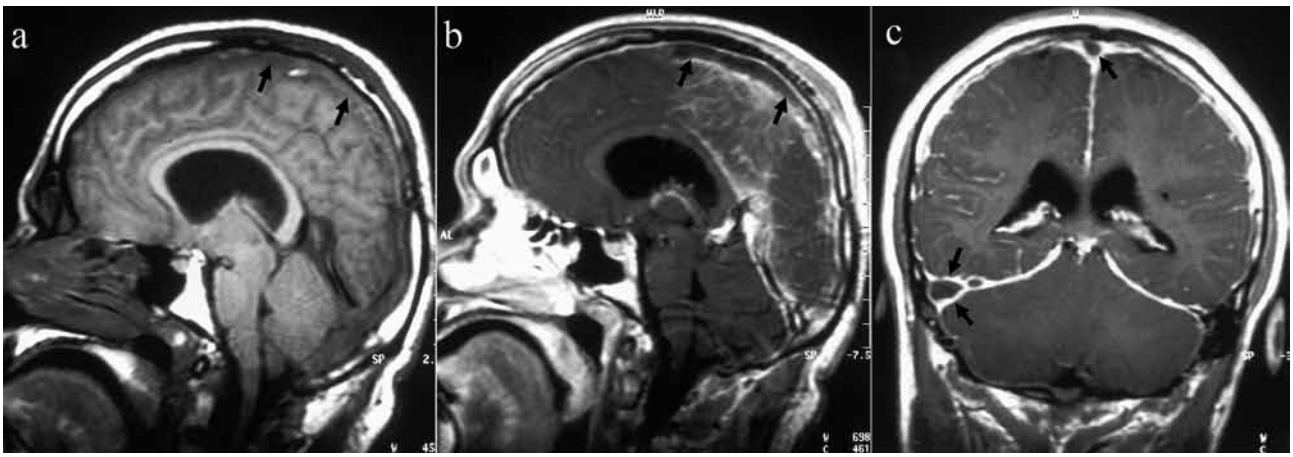

FIG. 2. — Sagittal T1-weighted (a), contrast-enhanced sagittal (b) and coronal (c) T1-weighted magnetic resonance images show filling defect in the superior sagittal sinus and empty delta sign (black arrows) in the superior sagittal and right transverse sinuses consistent with dural venous sinus thrombosis.

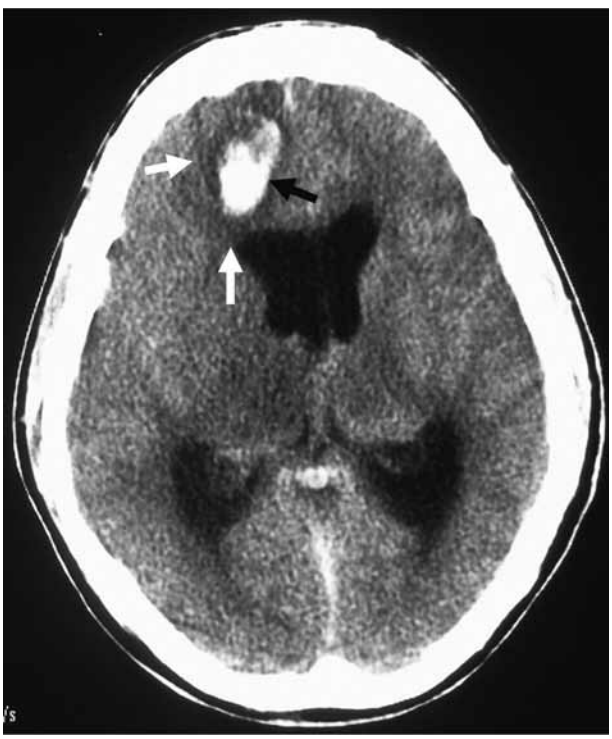

FIG. 3. — Axial contrast enhanced computed tomography image shows acute hyperdense parenchymal hematoma (black arrow) in right anteroinferior frontal region with hypodense peripheral edema (white arrows).

Caudal displacement of intracranial structures and disruption of the veins and sinuses may be seen, and all these may result in venous dilatation and thrombosis. Thrombosis may occur due to stasis as a result of compensatory dilatation and traction of cortical veins and venous sinuses (6).

Non-traumatic SAH develops most often due to the rupture of an intracranial aneurysm. Arteriovenous malformations, tumors, vasculitides, amyloid angiopathy, and coagulopathies are other causes of SAH. CVST is generally thought to be a rare cause of SAH. The claim that SAH occurs only rarely in patients with CVST is based on autopsy results, and most patients with CVST do not die (7). Some authors reported that cerebral spinal fluid (CSF) in patients with CVST is often bloody. Bousser's study demonstrated that in lumbar puncture in 32 patients, 62.5% had more than 20 erythrocytes per cubic mm of CSF with a mean value of 1716 and a maximum of 21000 (8, 9). As noticed by Benabu *et al*., there is a trend, which reports SAH with underlying CVST to be increasing. The authors conclude that the explanation for this trend is that advances in diagnostic radiology have increased the ability to diagnose underlying CVST (10).

In the first CT scan, SAH and CVST were observed at the same time. Brain MR findings confirmed CVST in a descriptive way. Typically SAH in CVST is focal and situated over the convexity (8). The exact mechanism of development of SAH in patients with CVST remains unknown. Benabu *et al*. reviewed the literature and found 3 theories, which have been proposed to explain this mechanism: one of the theory indicates that increased vascular permeability as a local inflammatory response in CVST allows extravasation of blood into the subarachnoid space, the other theory suggests that venous parenchymal hemorrhagic infarct can rupture into the subarachnoid space, and the third one represents that extension of the dural sinus thrombosis into the superficial veins causes localized venous hypertension and this may result in rupture of the

fragile-walled cortical veins into the subarachnoid space (10).

The treatment of CVST remains a controversial issue. A randomized and placebo-controlled trial of 20 patients showed the benefits of heparin (11). Another placebo-controlled trial, in which 60 patients were randomized to either low-molecular weight heparin followed by warfarin, or placebo, suggested that anticoagulated patients achieve better outcomes than controls, but the difference was not statistically significant (12). Khan *et al*. published a review about controversies of treatment modalities for cerebral venous thrombosis and they reported that most of the reviews suggest that anticoagulation is safe even in patients who have evidence of intracranial hemorrhage, be it intracranial or subarachnoid. And they underlined that anticoagulation with heparin is the only modality with reasonable evidence to support its use in CVST, even in patients with cerebral hemorrhage (13).

In our case, initially, we didn't use anticoagulation because there was intracranial hemorrhage. When symptoms worsened, we made a risk-benefit analysis and started anticoagulation. The risks of anticoagulation include progression of prior hemorrhage or new hemorrhage. He had new hemorrhage, intraparenchymal hemorrhage developed and the patient died due to secondary infections and metabolic changes. Therefore, profit-loss situation should be carefully evaluated prior to the treatment. There is not enough data about heparin therapy to administer it to patients with CVST presenting with SAH.

The presentation of CVST with SAH is a rare condition. Furthermore, development of CVST after spinal anesthesia is very rare. In this case, CVST developed after spinal anesthesia, and its first clinical presentation was SAH. To our knowledge, this is the first case CVST presenting with SAH after spinal anesthesia.

In conclusion, CVST can present with a headache pattern similar to that of PDPH. Changing headache patterns, such as loss of its orthostatic nature or increase in the intensity, and neurological signs may be considered factors indicating CVST. Especially, in the presence of risk factors, examinations should be conducted to eliminate possible CVST when a typical PDPH case loses its postural component.

CVST should be considered in the differential diagnosis of hemorrhagic cerebrovascular diseases developing after puncture. Early diagnosis of CVST by numerous clinical and imaging findings is important; delay in diagnosis may increase morbidity and mortality rates. Clinicians should be meticulous while using heparin in such cases.

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