

# Delayed Intraparenchymal Hematoma Following Diagnostic Lumbar Puncture

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**Abstract:** Lumbar puncture is a safe and commonly performed procedure, with an overall complication rate of 0.1% to 0.5%. Well-known contraindications to lumbar puncture are an intracranial tumor, noncommunicating hydrocephalus, coagulopathy, and ruptured aneurysm with subarachnoid hemorrhage. We report a case of a young man with epilepsy who, after a lumbar puncture performed for research purposes, presented with an intracerebral hematoma and neurological deficits. To the best of our knowledge, post-tap intraparenchymal hematoma is extremely rare and only 1 case has been reported previously. In consideration, all patients undergoing a lumbar puncture should be informed about this possible rare complication, even in the absence of documented hemorrhagic risk factors.

**Key Words:** lumbar puncture, intracranial hemorrhage, complication  
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Lumbar puncture is a safe and commonly performed procedure, with an overall complication rate of 0.1% to 0.5%.<sup>1</sup> The most commonly reported complications are orthostatic headache or herniated spinal nerve root, spinal epidural hematoma, infection, and pneumocephalus. Very rarely, a lumbar puncture may cause a severe, life-threatening complication such as cerebral herniation or subdural, subarachnoid, and/or intraventricular hemorrhage. Well-known contraindications to lumbar puncture are an intracranial tumor, noncommunicating hydrocephalus, coagulopathy, and ruptured aneurysm with subarachnoid hemorrhage.<sup>2</sup>

Here, we report a case of a young man with epilepsy who, after a lumbar puncture performed for research purposes, presented with an intracerebral hematoma and neurological deficits. To the best of our knowledge, post-tap intraparenchymal hematoma is extremely rare and only 1 case has been reported previously.<sup>3</sup>

## CASE REPORT

In March 2011, a 31-year-old man with a history of epilepsy since the age of 12 years was recruited for a scientific study. He was being treated with two 200-mg carbamazepine tablets/day, and presented with focal cortical dysplasia (FCD) type I on the right frontal sulcus. His blood chemistry values were in the normal range and no

diseases were reported. In the setting of research on cerebrospinal fluid (CSF), the patient was hospitalized for 1 day and, after providing informed consent, underwent a lumbar puncture.

## METHODS

The patient was positioned in lateral decubitus position. After a careful skin site disinfection, the entry point was located between the third and the fourth vertebral lumbar spinal processes. A 20-G atraumatic spinal needle was inserted and, after 1 attempt, 20 mL of clear CSF was extracted. The opening pressure was 12 cm H<sub>2</sub>O and closing pressure was 11 cm H<sub>2</sub>O.

## Post Lumbar Puncture Course

The procedure was uneventful, and after 1 hour of bed rest the patient was asymptomatic, out of the bed, and eating his lunch.

Twelve hours after the spinal tap, the patient complained of an orthostatic headache, which resolved after administration of nonsteroidal anti-inflammatory drugs. The patient was therefore discharged. After 72 hours, he started to complain of acute language impairment and progressive numbness and motor impairment of his right superior limb and face. He was promptly admitted to the emergency department. During the neurological examination, he was awake, oriented in time and space, and cooperative. He presented with mild right faciobrachial hemiparesis, fluctuations with a tendency to the left on Romberg test, and motor aphasia.

Head computed tomography (Fig. 1) and brain magnetic resonance imaging (MRI) scans (Fig. 2) documented a left frontal intracerebral hematoma with no displacement of the intracranial structures. A conservative approach was adopted; therapy with osmotic diuretics was started. A cerebral angiography (Fig. 3) and thrombophilic screening were negative for any underlying disease. Six days after the event, the patient had partially recovered from the motor aphasia and limb disturbance. He was discharged on day 10. A brain MRI 3 months (Fig. 4) later showed resorption of the frontal hematoma. At the 4-year follow-up, the patient was fully employed, taking antiepileptic drugs with effect, and had no further neurological sequelae.

## DISCUSSION

Intracranial hematomas following lumbar puncture are an extremely rare complication. In the English literature, few cases following diagnostic lumbar puncture or accidental dural puncture with an epidural needle while performing spinal anesthesia, myelography, or discography have been reported. The most commonly reported intracranial bleeding modalities are subdural and intraventricular; subarachnoid hemorrhage is even rarer.<sup>1</sup>

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The authors declare no conflict of interest.

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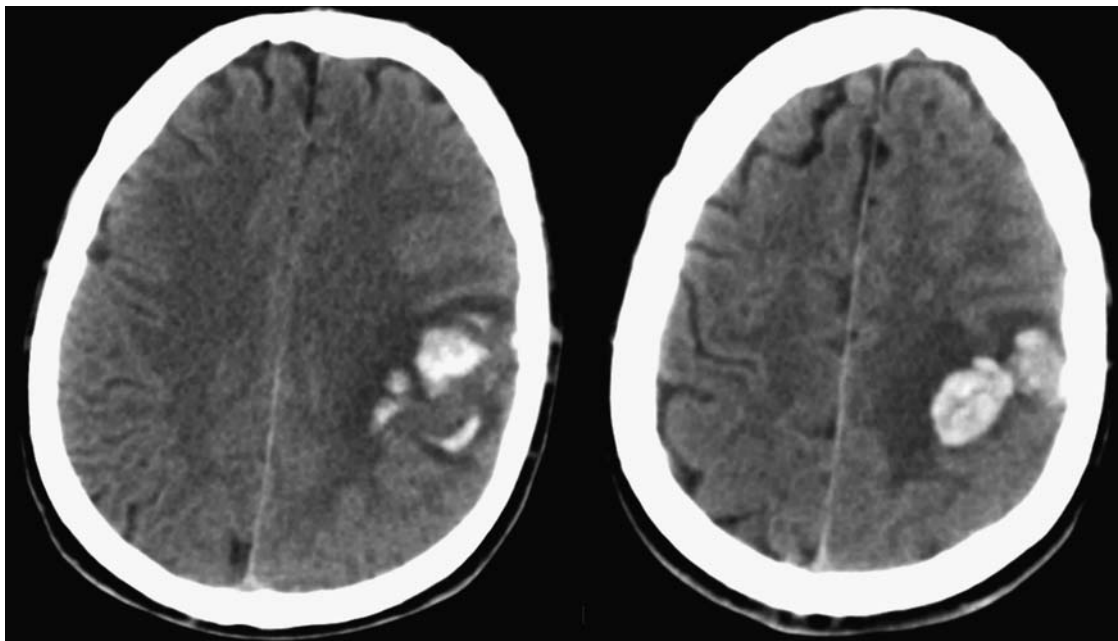


FIGURE 1. Head computed tomography scan showing an acute left frontal hemorrhage.

A single case of an adult patient in whom a CSF tap test was followed within days by an intracerebral hemorrhage is reported. The lumbar puncture was performed for diagnostic purposes in the setting of secondary chronic hydrocephalus.<sup>3</sup>

Intracranial hemorrhage (subdural and cerebellar) after CSF removal has been associated with spinal surgery.<sup>4</sup> Despite

that diagnostic CSF taps and dural leakage during spinal surgery are similar triggering events, namely loss of CSF, the difference in CSF volume lost suggests that the underlying mechanism for hemorrhage formation differs. It has been postulated that intracranial hemorrhage during spinal surgery could be caused by loss of CSF pressure and that excessive

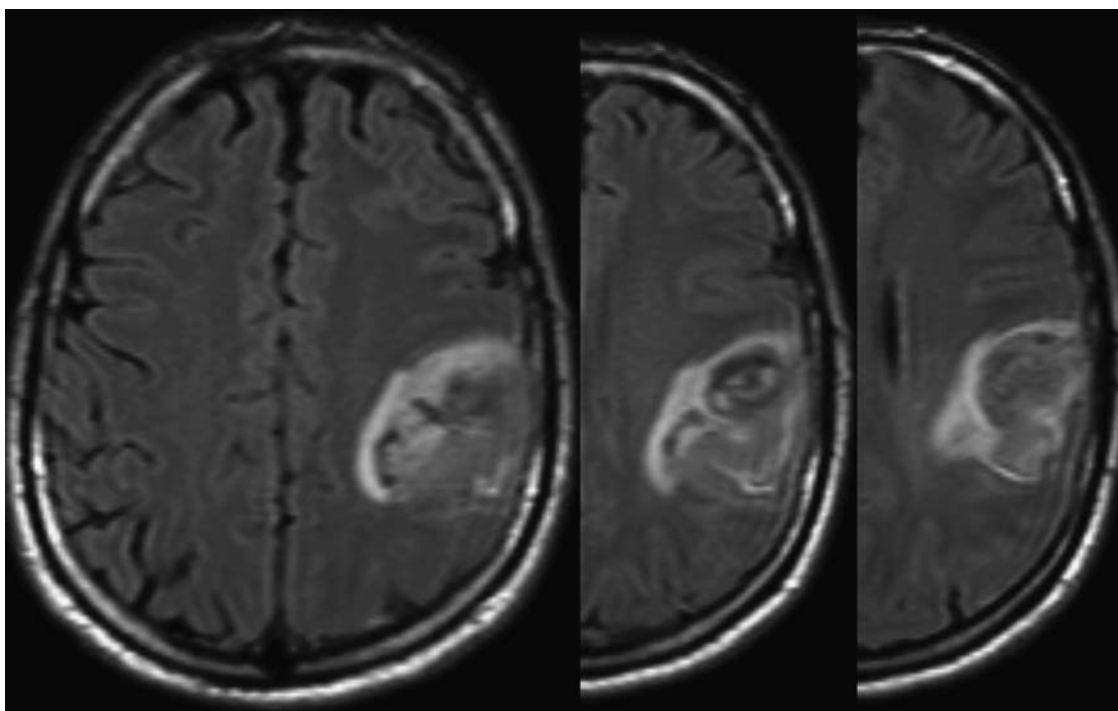
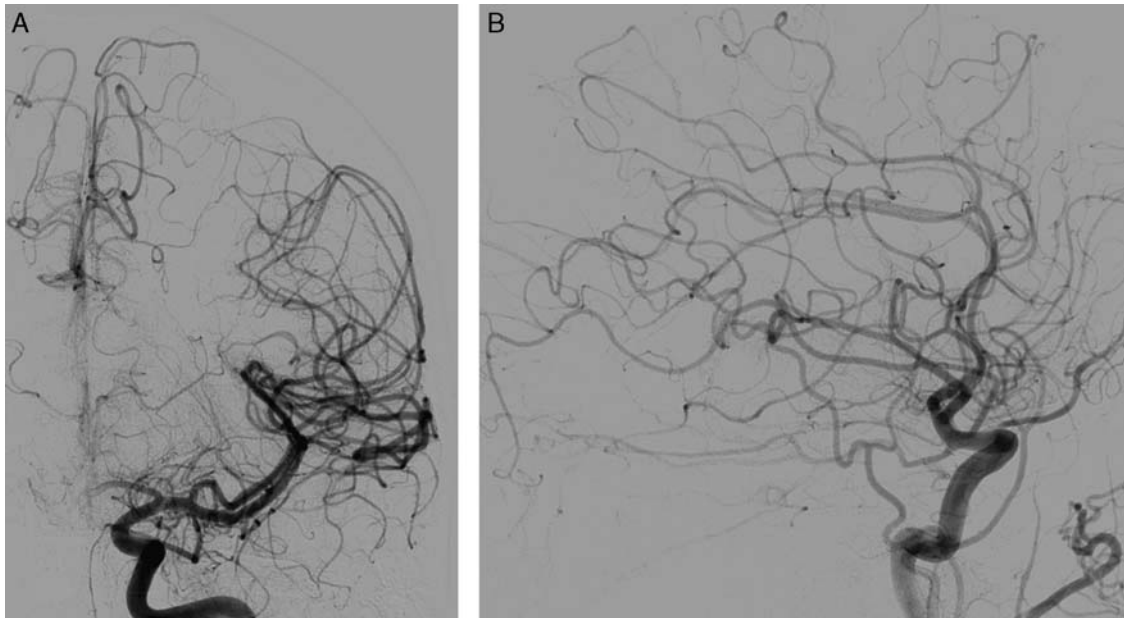


FIGURE 2. Brain MRI FLAIR images showing the left hemorrhage causing surrounding edema.

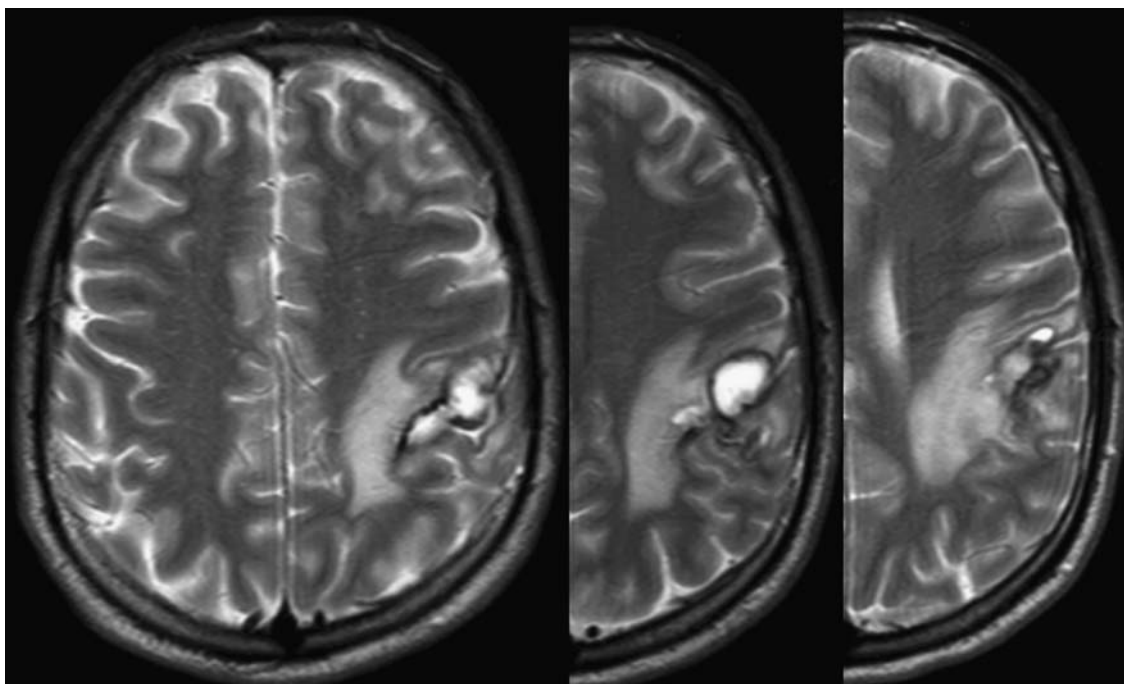


**FIGURE 3.** Cerebral angiography in anteroposterior (A) and lateral view (B) of the left hemisphere showing no sign of vascular malformation.

leakage ( $\geq 250$  mL) would lead to caudal displacement of intracranial structures, resulting in intracranial hematoma following vascular tearing and venous occlusion.<sup>5</sup>

This is unlikely to have been the case for our patient given the small amount of CSF extracted (20 mL). We believe that our case and a previous one,<sup>3</sup> reported by the senior author, share the same etiopathologic mechanism. CSF volume loss leads to intracranial CSF pressure reduction with a consequent increase in cerebral blood perfusion, which might

trigger an intracranial hemorrhage.<sup>6</sup> The mechanism is easily explained by the Monro-Kellie doctrine: the sum of the volumes contained in the skull (ie, brain parenchyma, CSF, and blood) remains constant to such an extent that increasing or decreasing 1 component causes a compensating volume change in the other components.<sup>7</sup> Moreover, the relationship between CSF removal and intracranial pressure is not linear, and even a small amount of CSF loss produces an important intracranial pressure reduction at the vertex.<sup>7</sup> Multiple



**FIGURE 4.** The brain T2-weighted MRI at 3 months showing partial absorption of the hemorrhage.

intracerebral hemorrhages, even in the supratentorial compartment, were reported in 1 case.<sup>8</sup> In this case, the spinal needle was erroneously left in place for >20 minutes leading to the acute loss of 250 mL of CSF. Despite the fact that other causes were not to be excluded, given the advanced age of the patient with multiple concomitant pathologies, the authors postulate that the acute lowering of the intracranial and intraspinal CSF pressure brought forth an increase in the transmural pressure gradient of the vessels and thus a secondary wall stress rupture. In this case, both multiple cerebellar hemorrhages, which are characteristic of acute and excessive CSF loss with brain sagging,<sup>9</sup> and intracerebral hematomas, which we suggest are related to the elevation of cerebral blood perfusion secondary to CSF hypovolemia, are reported.

In the present case, the intracranial bleeding was left frontal, despite the existence of a right FCD. Hashizume et al<sup>10</sup> analyzed the histopathology of FCD in patients treated for epilepsy and found that epileptogenic FCD are rich in small-sized vessels that are not present in nonepileptogenic FCD and in normal brains. It might be suggested that epileptogenic FCD are a possible site of intracranial bleeding due to the possible presence of an abnormal vasculature. Despite that, the hemorrhage was in a different location at a site where no abnormalities were visible on the previous MRI controls.

### CONCLUSIONS

Despite being a routine practice, CSF tapping rarely leads to acute hemorrhagic complications such as intraparenchymal, intraventricular, or subarachnoid and/or subdural hemorrhage. In consideration, all patients undergoing a lumbar puncture should be informed about this possible rare complication, even in the absence of documented hemorrhagic risk factors.

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