Postoperative Intracranial Hypotension-Associated Venous Congestion after Spinal Surgery Managed with Multiple Blood Patches: Case Report

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Abstract

Background Postoperative intracranial hypotension-associated venous congestion (PIHV) is a rare event. The authors report the case of a patient presenting with PIHV after spinal surgery following the sudden loss of cerebrospinal fluid (CSF) induced by suction drainage.

Methods A 69-year-old patient underwent uneventful revision surgery for wound dehiscence after lumbar surgery with placement of a subfascial suction drain.

Results Postoperatively, the patient presented with fluctuating consciousness and a generalized tonic–clonic seizure. Computed tomography (CT) and serial magnetic resonance imaging (MRI) were performed showing convexity subarachnoid hemorrhages (SAHs), diffuse swelling of the brain and thalami and striatum bilaterally without diffusion restriction, and signs of intracranial hypertension resulting in pseudohypoxic brain swelling in PIHV. A dural leak at L3–L4 was treated with several CT-guided patches combining autologous blood and fibrin glue injections. The patient recovered without neurologic deficit and follow-up MRI revealed progressive complete reversal of brain swelling, and re-expansion of CSF spaces.

Conclusion PIHV is a rare but potentially fatal entity. Awareness of PIHV after cranial or spinal surgery leads to early treatment of CSF hypovolemia and possibly better clinical outcome. Following acute CSF volume loss, an acute elevation of cerebral blood volume overcoming autoregulatory mechanisms seems a likely explanation for diffuse cerebral vasogenic edema and SAH in PIHV.

Keywords
- CSF loss
- hypoxic brain swelling
- intracranial hypotension
- postoperative intracranial hypotension-associated venous congestion
- suction drain

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Introduction

Postoperative intracranial hypotension-associated venous congestion (PIHV),[1] also known as pseudohypoxic brain swelling,[2] due to sudden loss of cerebrospinal fluid (CSF) is described after cranial surgery[2–4] and more rarely after spinal[1,4–6] surgery with the placement of suction drainage. On imaging, PIHV shares common findings with hypoxic-ischemic encephalopathy.[1,2,4–6] The clinical outcome spans from complete recovery to fatal outcome.[1,3–6]

We describe the clinical and radiologic course of a patient who presented with PIHV after an uneventful revision surgery for wound dehiscence.

Case Report

History

A 69-year-old patient known for auricular fibrillation was transferred to our hospital with necrotizing pancreatitis and wound dehiscence after lumbar decompression and fusion surgery in another institution.

Operation

The patient underwent an uneventful surgery for wound dehiscence in our hospital with the placement of a subfascial suction drain.

Postoperative Course

In the recovery room, he presented with fluctuating consciousness and had a generalized tonic–clonic seizure treated with levetiracetam.

CT scan performed 4 hours later showed convexity subarachnoid hemorrhages (SAHs), diffuse brain swelling, and signs of intracranial hypertension with uncal, superior vermian, and cerebellar tonsillar herniation. The drain with 340 ml of sanguinolent fluid was removed. Magnetic resonance imaging (MRI) 5 hours after surgery (►**Fig. 1**) confirmed findings of diffuse cerebral and cerebellar swelling and swelling of the thalami and striatum bilaterally. Restricted diffusion was conspicuously absent on diffusion-weighted imaging (DWI), consistent with vasogenic edema. MRI ruled out intracranial venous thrombosis, though the dural venous sinuses and pial veins were dilated. Pseudohypoxic brain swelling was suspected.

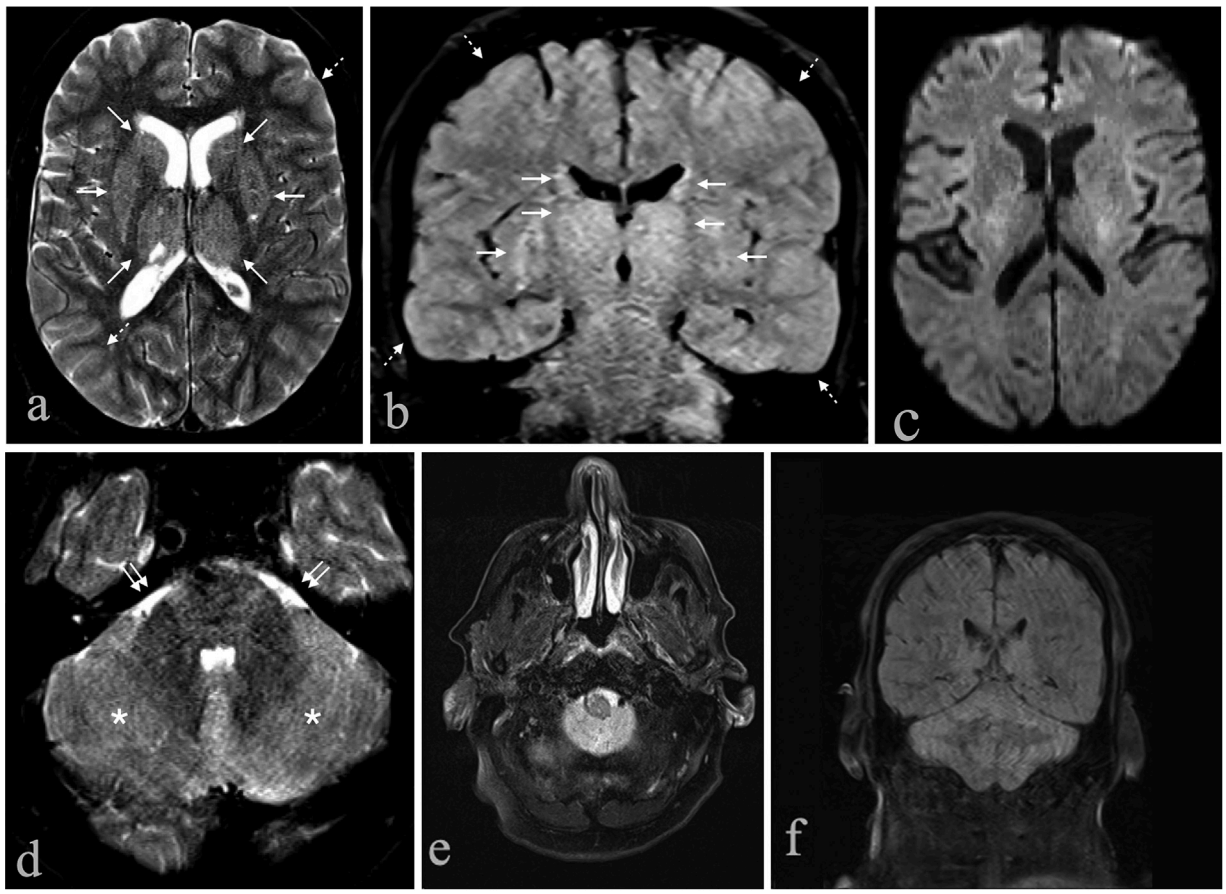

Fig. 1 Magnetic resonance imaging (Siemens 1.5-T Aera scanner, Erlangen, Germany) performed 5 hours after surgery and 1 hour after noncontrast computerized tomography. (**a**,**d**,**e**) Axial turbo spin echo (TSE) T2-weighted images, (**b**,**f**) coronal T2 turbo inversion recovery magnitude (TIRM) dark fluid, and (**c**) diffusion-weighted imaging (DWI), axial B1000 s/mm^2. (**a**,**b**) Symmetrical increased signal of the caudate nuclei, thalami, and putamina (*arrows*) and cortical ribbon (*dotted arrows*). There is also effacement of cerebral sulci. (**c**) restricted diffusion is conspicuously absent on B1000 and ADC maps (not shown). (**d**) Signs of posterior fossa exploration showing effacement of all cerebrospinal fluid spaces (*double arrows*) from cerebellar swelling and cortical edema in high signal (*asterisks*). (**e**,**f**) Cerebellar tonsillar herniation. Uncal and superior vermian herniation were present (not shown).

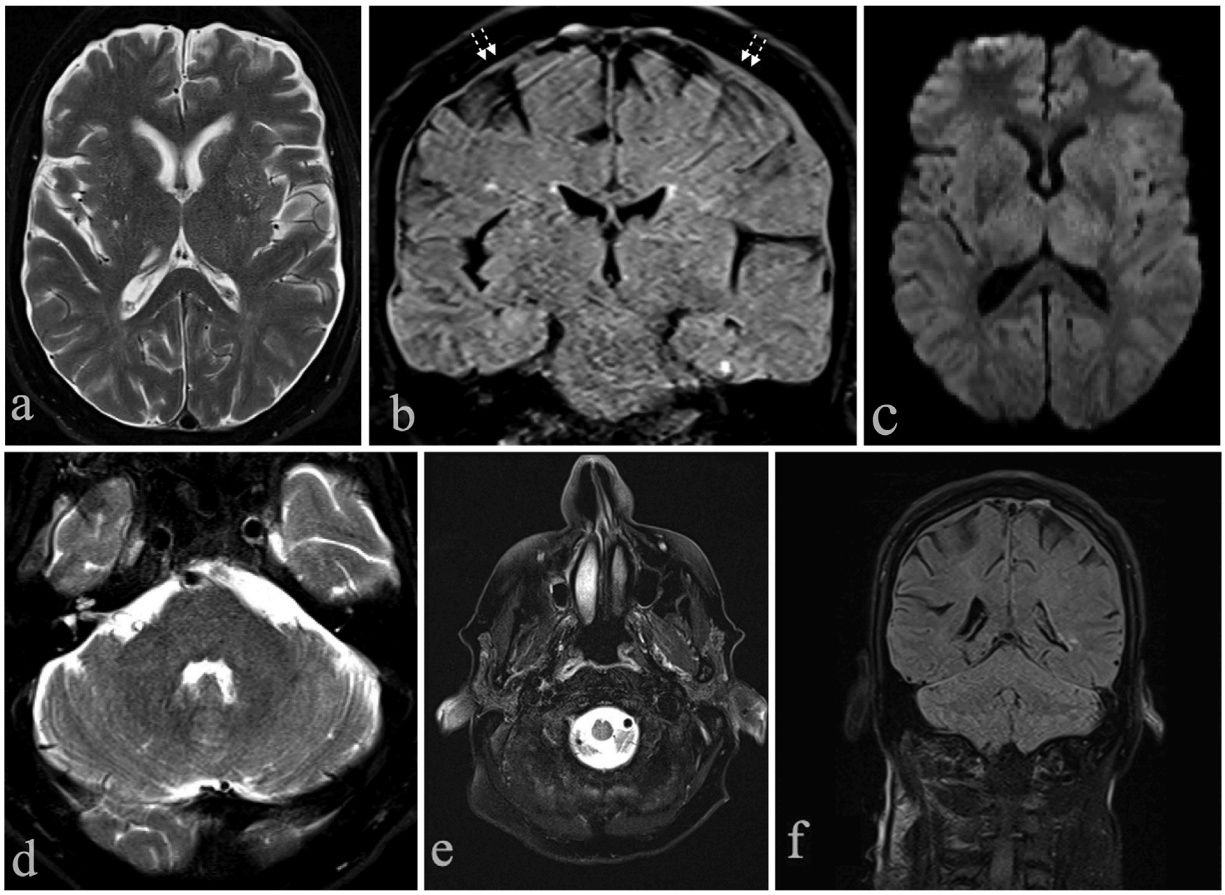

Fig. 2 Magnetic resonance imaging (Siemens 1.5-Tesla Aera scanner) performed at day 11 after surgery. (**a**,**d**,**e**) Axial turbo spin echo (TSE) T2-weighted images, (**b**,**f**) coronal T2 turbo inversion recovery magnitude (TIRM) dark fluid, (**c**) diffusion-weighted imaging (DWI), axial B1000 s/mm^2. Complete resolution of brain swelling, cerebellar tonsillar herniation, and signal abnormalities and signs of intracranial hypertension. Note narrower lateral ventricles and bilateral convexity subdural effusions (*double dotted arrows*) consistent with cerebrospinal fluid hypovolemia.

The patient was placed on strict bed rest for 72 hours. He recovered consciousness within 24 hours but remained confused without focal neurologic signs.

MRI at days 4 and 11 revealed progressive complete reversal of brain swelling, re-expansion of CSF spaces, and disappearance of intracranial hypertension (►**Fig. 2**).

A myelo-CT performed on day 2 revealed a high-flow, large neck dural leak at L3–L4. The patient refused further surgery. A selective CT-guided blood patch was performed (35 mL of autologous blood). Due to persistence of the dural leak and signs of intracranial hypovolemia on MRI, repeat CT-guided patches were performed at 2, 4, and 6 weeks, combining autologous blood and fibrin glue injections. The patient's neurologic status returned to presurgical baseline. He refused further neuroimaging control and was discharged. The clinical follow-up examination 5 weeks later was unremarkable. Head and spine MRI performed at 9 months in the setting of newly diagnosed pulmonary neoplasia showed persistent collected CSF leak at the surgical site without signs of CSF hypovolemia.

Discussion

We report a case of PIHV in an elderly male patient immediately after spinal surgery and placement of a subfascial suction drain, presenting with seizure and conscious impairment related to an acute periprocedural CSF leak. The patient made full clinical recovery following suction drain removal and repeat CT-guided blood and fibrin glue patches. Pseudohypoxic brain swelling signs showed rapid and complete reversal in keeping with the neurologic recovery.

According to the Monro–Kellie doctrine, CSF hypovolemia results in a compensatory increase in cerebral blood volume (CBV).[3,4] Acute CBV elevation induced by acute loss of CSF as observed in the present patient could have led to the observed SAH and diffuse brain swelling. It has been suggested that in PIHV brain and brain stem sagging[1] could lead to venous outflow obstruction of the great vein of Galen[4,5] at its junction with the straight sinus at the tentorium cerebelli. This explains the vasogenic edema of the basal ganglia and thalamic nuclei. This hypothesis does not account for the cerebral and cerebellar cortical edema observed in PIHV or the SAH in the present case and seems contrary to the venous dilatation observed in CSF hypovolemia. Acute elevation of CBV overcoming autoregulatory mechanisms seems a likely explanation for the observed diffuse cerebral vasogenic edema and SAH. SAH has been reported in a case of PIHV,[7] but it is a rare presentation of spontaneous intracranial hypovolemia.

The primary goal of the treatment of a documented large neck dural leak is surgical closure. The patient, however, refused any further surgery.

Conclusion

A rapid rate of CSF depletion rather than the volume of CSF lost may be the major contributing factor to an unfavorable clinical outcome in patients with PIHV.[2,3] As in our case, it is likely that if the CSF depletion is corrected rapidly before cytotoxic edema and venous infarction occur, a favorable clinical outcome may ensue. For this reason, recognition of signs of pseudoanoxic brain swelling in the right clinical setting will allow for diagnosing PIHV and introducing measures to correct further CSF depletion, including lumbar drain removal and surgical or percutaneous closure of the CSF leak.

Statement of Ethics

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Ethical approval by the local Ethics Committee was not necessary.

Informed Consent

The patient has consented to submission of this case report.

Funding

None.

Conflict of Interest

None declared.

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