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Case Report

Pseudohypoxic brain swelling following elective lumbar laminectomy: A rare case report and review of literature ☆,☆☆

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ABSTRACT

Pseudohypoxic brain swelling, also known as postoperative intracranial hypotension-associated venous congestion, is an intriguing complication following routine neurosurgical interventions. We report a case of a 73-year-old female patient who exhibited this rare complication following an elective L4–L5 laminectomy, without evidence of intraoperative cerebrospinal fluid leakage. Initially presenting with clinical features suggestive of anoxic/hypoxic brain injury, the case deviated from typical pseudohypoxic ischemic venous hypertension (PIHV) patterns, leading to a challenging diagnostic process. The patient's remarkable recovery, contrary to the initial grim prognosis, emphasizes the critical need for considering PIHV in differential diagnoses when postoperative symptoms mimic anoxic/hypoxic brain injuries. This case contributes to the evolving understanding of PIHV, particularly in scenarios lacking conventional risk factors like cerebral spinal fluid (CSF) leakage, and underscores the importance of comprehensive postoperative surveillance and management. It also highlights the imperative for continued research into the pathophysiology and treatment strategies of PIHV to enhance patient outcomes in complex surgical contexts.

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Introduction

Pseudohypoxic brain swelling, otherwise known as postoperative intracranial hypotension-associated venous congestion, is an uncommon and potentially fatal complication that can occur after an elective spine or brain surgery [1,2]. Clinical presentation, including imaging, is strikingly similar to anoxic brain injury. It manifests on CT brain as a hypodensity of the basal ganglia and thalami, and on MRI brain as edema and restricted diffusion in the basal ganglia and thalami, similarly to hypoxic-ischemic brain injury [3]. The mechanism of pseudohypoxic ischemic venous hypertension (PIHV) is postulated to be due to acute cerebral spinal fluid (CSF) loss which is followed by an acute increase in cerebral blood volume overcoming the autoregulatory mechanisms causing diffuse cerebral vasogenic edema [4]. Few cases have been reported without obvious intraoperative CSF leakage. Functional outcomes vary from remarkable neurological recovery to death. The reason for deterioration in some patients versus improvement in others is not well understood. However, if detected and treated early and aggressively by looking for leakage defects and correcting them, PIHV may be reversible [5]. In this case report, we are presenting a 73-year-old female patient who had PIHV following an elective, uncomplicated L4–L5 laminectomy without reported CSF leakage. Fortunately, our patient managed a full recovery after an initially predicted fatal prognosis.

Case presentation

A 73-year-old female, without notable medical history, presented initially at an outpatient neurosurgical facility due to cervical and lumbar pain. An MRI of the lumbar spine revealed severe L4–L5 lumbar stenosis with a grade 2 spondylolisthesis and notable right-sided nerve root compression. Subsequently, she underwent ACDF (anterior cervical discectomy and fusion) at C5–C6 with cages to address her cervical complaints. Following this, she underwent physical therapy for her lower extremity symptoms. However, upon returning for follow-up, she reported worsening pain instead of improvement. Hospitalization ensued, and neurosurgery recommended a laminectomy at L4–L5, coupled with pedicle screw fixation and fusion. The surgical procedure was uneventful and devoid of intraoperative complications or indications of cerebrospinal fluid leakage. A 7 Jackson Pratt (JP) drain was inserted and subsequently removed postoperatively. Fig. 1 shows postoperative X-ray of the lumber spine. The patient was extubated in the operating room upon surgery's completion.

In the postanesthesia care unit, the patient exhibited shallow breathing. Her vital signs were within normal limits, with oxygen saturation at 99% on a nonrebreather mask. Sugammadex was administered to counteract possible residual muscle blockade, followed by naloxone due to unresponsiveness to normal stimulation, which indicated potential residual opioid effects. While her condition remained stable, her shallow breathing persisted. An arterial blood gas analysis and stat chest X-ray yielded unremarkable results. Electrolyte levels

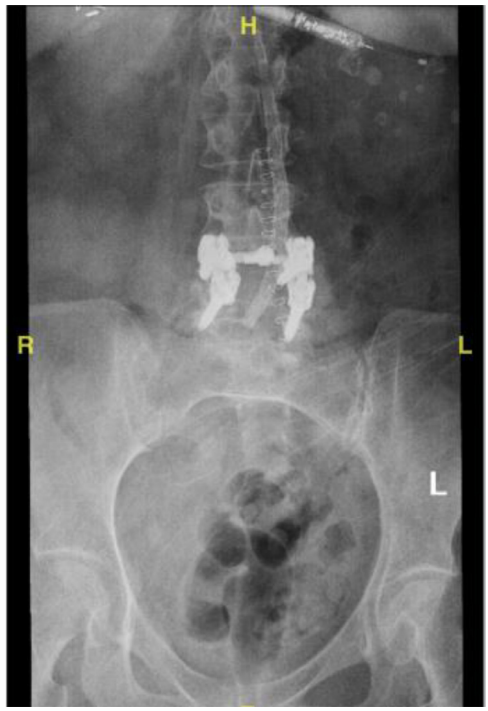

Fig. 1 – Postoperative lumber X-ray.

were within normal range, and the patient received ventilation via bag-mask along with 100% oxygen supplementation. Bi-level positive airway pressure (BiPAP) was introduced, yet the etiology of her deep sedation remained unclear. At that point, her Glasgow Coma Scale (GCS) score was 6 (eyes closed - 1, no verbal response - 1, withdrawal to painful stimuli - 4).

Shortly thereafter, the patient had emesis in the BiPAP apparatus, triggering the rapid response team's involvement. Flumazenil was administered to reverse the effects of midazolam. A brief seizure lasting under 1 minute was witnessed and managed with 2mg of lorazepam. Subsequently, she was intubated to protect her airway and was taken for a head CT.

The initial head CT (Fig. 2) demonstrated diffuse early cerebral edema and indicators of hypoxic/anoxic brain injury. CTA venogram revealed normal intracranial arterial and venous circulation. She was subsequently admitted to the ICU and underwent follow-up imaging, including another CT on the night of admission (Fig. 3), and a CT and an MRI the following morning (Figs. 4 and 5). The CT results remained consistent with the initial finding of cerebral edema. By the second day in the ICU, the patient began displaying signs of neurological improvement. Her condition continued to improve, and by the fourth ICU day, she had returned to her baseline with a GCS of 15. As her condition stabilized, she was transferred to the medical floors on ICU day 5 and was discharged 7 days postoperatively (Fig. 6).

Discussion and literature review

This case highlights the intricate nature of postoperative complications and underscores the importance of vigilant moni-

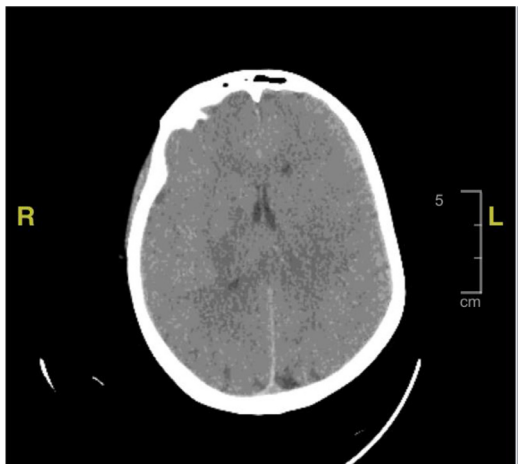

Fig 2 – POD#0: CT brain without contrast. Axial view. Early, diffuse cerebral edema. There is no evidence of herniation or intraparenchymal hemorrhage.

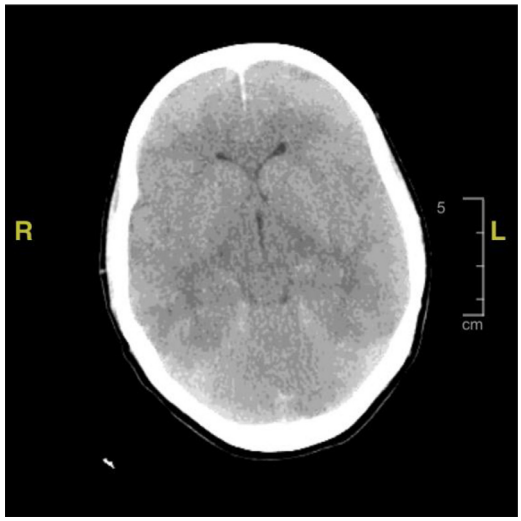

Fig 3 – POD#0: CT brain without contrast 6 hours later. Axial view. Mild, cerebral edema similar to the study performed earlier.

toring and prompt intervention in the context of complex surgical procedures.

The initial presentation of the patient was misleading, resembling anoxic/hypoxic brain injury, a phenomenon well-documented in literature [6,7]. The reported patients all undergo routine surgery, then have initial clinical presentations commensurate with anoxic/hypoxic brain injury without periods of hypotension, or any events where the brain would have been devoid of oxygen. However, our patient's progressive neurological improvement and the diagnostic limitations of initial CT imaging cautioned against anchoring bias [8]. The significance of our patient's clinical scenario lies in the rare presentation of PIHV, which deviated significantly from the standard clinical deterioration seen in patients with anoxic brain injuries. This case thereby warrants a reassessment of

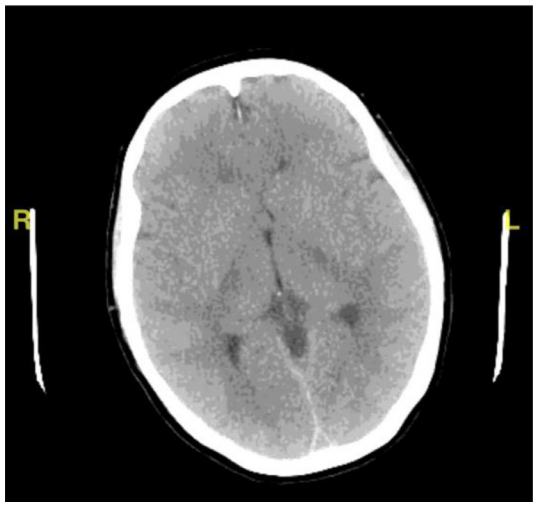

Fig 4 – POD#1: CT brain without contrast. Axial view. Stable mild cerebral edema shown the following day.

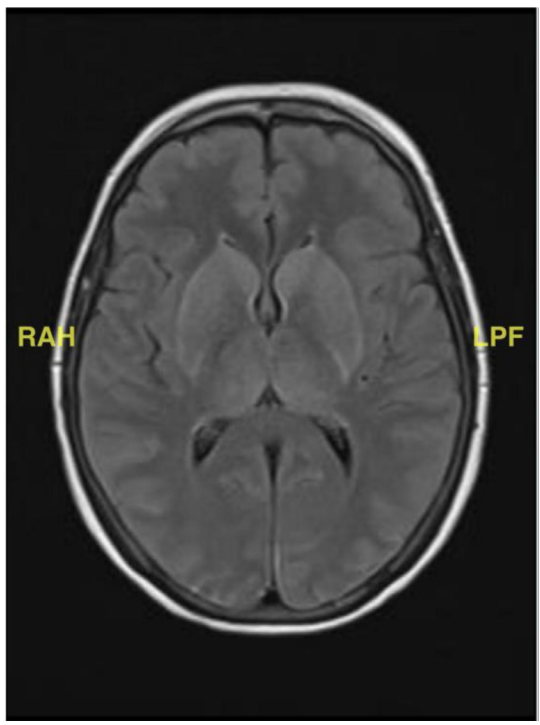

Fig. 5 – POD#1: MRI brain without contrast. Axial view. Increased signal with enlargement through the basal ganglia surrounded by a hyperintense rim delineating the lentiform nucleus.

initial diagnostic presumptions. Although appropriate medical interventions were made, the poor prognostic indicators from her postoperative presentation were discussed with her family. This is a call to action for furthering our knowledge of PIHV, specifically the early recognition and differentiation from anoxic brain injury, though prognostic factors for patients diagnosed with PIHV are yet to be fully identified.

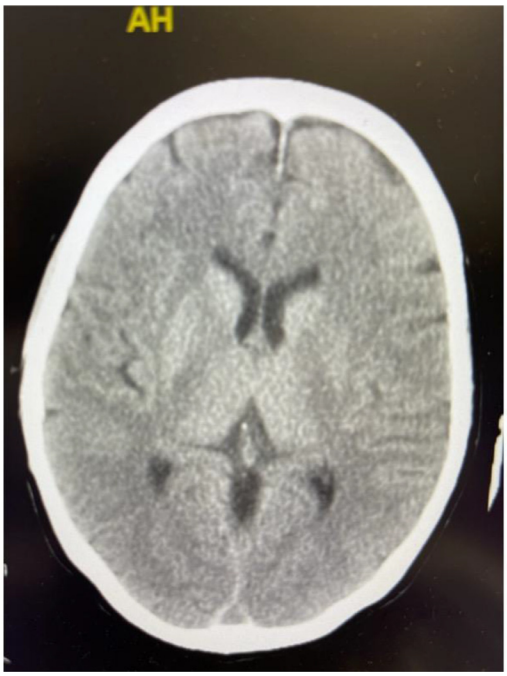

Fig. 6 – POD#6: CT Brain revealed decreased cerebral edema compared to prior exams with better visualization of the cortical sulci and decreased mass effect on the ventricles.

A notable feature in this case was the absence of any documented CSF. leakage during the surgery, which is often considered a significant factor in the development of PIHV secondary to durotomies and violation of the subarachnoid space at the surgical site [9,10]. There are few cases of PIHV in literature without obvious intraoperative CSF leakage and it was first reported in 2019 by Chidambaram et al [11]. Their case presents a patient who underwent an uneventful lumbar decompression and fusion with postoperative complications characteristic of pseudohypoxic brain swelling with full neurological recovery similar to our patient. In 2022, the case of an uncomplicated unilateral burr hole drainage for a patient with a subdural hematoma (SDH) with postoperative complications characteristic of PIHV was reported [12]. Of note, this case also resulted in a favorable recovery for their patient which may suggest a positive prognostic factor in PIHV after neurosurgical cases without obvious intraoperative CSF leakage.

The underlying pathophysiological mechanism of venous pooling and cerebral edema in PIHV is usually attributed to CSF leakage leading to intracranial hypotension, but several other mechanisms have been identified. These include intracranial decompression, brain re-expansion, change in autoregulation, and hyperperfusion resulting in deep venous congestion (Nakamura et al). These mechanisms, however, raise more questions than answers in our case of an elective uncomplicated laminectomy without a CSF leak.

There are unique features reported in the literature that might allow differentiation of PHBS from hypoxic-ischemic encephalopathy. Sotoudeh et al [13] concluded that the lentiform rim (or “fork”) sign can be helpful in differentiating pseudohypoxic brain swelling from hypoxic-ischemic encephalopathy. The lentiform fork sign has been described on MRI and is seen as bilateral symmetrical hyperintensities in the basal ganglia surrounded by a hyperintense rim delineating the lentiform nucleus. This finding can be appreciated in our patient’s MRI.

Our patient’s presentation and subsequent clinical course add a layer of complexity to the existing medical literature, particularly concerning immediate postoperative management and the understanding of PIHV. The case encourages a multidisciplinary approach involving immediate interventions like mechanical ventilation and pharmacological support, which may be indispensable in similar scenarios [14,15]. This report also reiterates the need for a broad differential diagnosis in cases of postoperative cerebral edema, opening avenues for further research into alternative mechanisms of PIHV and implications for anesthetic protocols.

While the case provides valuable insights, it is limited by the absence of definitive etiological data for PIHV and a need for broader clinical studies. Future research should focus on the complexities of diagnosing PIHV, especially when no CSF leakage is evident, and explore the potential implications for anesthetic and postoperative care protocols [16]. This case offers an enriching contribution to the sparse literature on PIHV, its differential diagnosis, and its potential complications, thereby aiding clinicians in managing similar challenging postoperative scenarios.

Conclusion

In conclusion, this case report underscores the complexity of postoperative complications and the need for vigilant monitoring during complex surgeries. Despite initially resembling anoxic/hypoxic brain injury, the patient’s unique clinical course cautioned against diagnostic bias, emphasizing the importance of considering alternative diagnoses. Notably, this case deviates from the typical pattern of PIHV as there was no documented CSF leakage during surgery, challenging our understanding of PIHV’s pathophysiology, which still remains unclear.

The report also highlights the potential utility of the lentiform fork sign in differentiating PIHV from hypoxic-ischemic encephalopathy, offering a valuable diagnostic tool.

While this case provides some relevant insights, it calls for further research into PIHV, especially when no CSF leakage is evident, and the implications for anesthetic and postoperative care protocols. In essence, it emphasizes the need for a multidisciplinary approach and continued research to better manage challenging postoperative scenarios like PIHV.

Patient consent

Appropriate informed consent was obtained.

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