



Clinical letter

Electroencephalographic manifestations of intracranial hypertension in a patient with ventriculoperitoneal shunt malfunction

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ABSTRACT

Background: Differentiating epileptic seizures from manifestations of intracranial pressure (ICP) fluctuations can be challenging in patients with a ventriculoperitoneal shunt (VPS). We report a case of intermittent VPS malfunction presenting with focal motor events initially misdiagnosed as refractory status epilepticus.

A 27-year-old man with childhood-onset hydrocephalus treated with a right VPS and a previous history of focal epilepsy—managed with levetiracetam, perampanel, and lacosamide—presented with increased seizure frequency and intermittent headaches. Initial treatment included continuous infusions of valproate, lacosamide, and levetiracetam under the presumption of status epilepticus. However, prolonged (15-hour) video-EEG monitoring during the clinical events showed diffuse delta activity with suppression of the background rhythm rather than ictal discharges. These EEG changes coincided with episodes of arterial hypertension, profound bradycardia (as low as 20 bpm), and papilledema on fundoscopic examination. Revision of the shunt system resulted in complete resolution of symptoms and withdrawal of antiseizure medications.

Conclusion: Extended video-EEG monitoring can be a useful tool to distinguish epileptic seizures from paroxysmal manifestations of paroxysmal intracranial hypertension due to VPS malfunction.

1. Introduction

Patients with ventriculoperitoneal shunts (VPS) are at risk of intermittent or chronic shunt dysfunction, leading to episodes of raised intracranial pressure (ICP). Clinical signs—headache, focal neurological deficits, and altered consciousness—may be confused with epileptic seizures, especially in those with a pre-existing epilepsy diagnosis. Misinterpretation may prompt aggressive antiseizure therapy without clinical improvement increase of adverse effects.

In the present report, we describe the case of a young man with intermittent VPS dysfunction initially interpreted as epilepsy. Key to diagnosis was a prolonged video EEG capturing electro-clinical events suggestive of cerebral hypoperfusion rather than true epileptic activity.

2. Case presentation

We present the case of a 27-year-old man with a history of infantile communicating hydrocephalus treated by VPS implant at 18 months and normal neurodevelopment. Since the age of 22, the patient experienced episodes of right upper limb stiffness with preserved awareness, alongside episodes of impaired awareness and urinary incontinence. These clinical features were deemed consistent with left frontal lobe seizures and treatment with levetiracetam was instituted; however, due to sub-optimal seizure control, perampanel and lacosamide were added to the therapeutic regimen. Eventually, he consulted because of a 5-week history of increased seizure frequency, experiencing three to four daily episodes of right-sided motor phenomena, intermittent bifrontal headaches, and transient blurred vision. Neurological examination between events was unremarkable. Once in the emergency room, the patient

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presented multiple clinical events characterized by unresponsiveness, right arm extension and right sided oculocephalic preference, as well as other episodes mimicking left hand automatisms lasting up to 15 min. The episodes persisted despite treatment with midazolam, levetiracetam, valproate and lacosamide.

On admission, routine blood analysis was unremarkable. Interictal EEG recordings failed to demonstrate epileptiform discharges. Repeated cerebral MRIs showed neither ventricular system dilatation, nor transependymal oedema, nor acute structural lesions. Some periventricular hyperintensities on T2 series were found, without any diffusion restriction nor contrast enhancement. Such lesions were already known and had been studied in a previous hospitalization to rule out demyelinating disease (oligoclonal bands, anti-MOG and anti-AQP4 antibodies being negative). Cerebrospinal fluid (CSF) work-up was unremarkable (normal cell count and biochemistry, negative microbiology) and opening pressure was not significantly increased, although no objective measurement was performed.

Despite treatment, focal events persisted and were associated with paroxysmal arterial hypertension (systolic up to 180 mmHg) with severe sinus bradycardia (as low as 20 bpm). Peripheral paralysis of the right abducens nerve was observed on day 4 of hospitalization, and the fundoscopic exam right after one such episode revealed bilateral papilledema. However, shunt reservoir puncture was ineffective.

A continuous video-EEG study was conducted over 15 hours. During the clinical events, the EEG did not show any ictal pattern. Instead, episodes were associated with diffuse delta waves followed by suppression of background activity, an EEG pattern known to reflect cerebral hypoperfusion [1]. EEG alterations occurred simultaneously with hypertensive and bradycardic crisis, and—when considered alongside the clinical findings of papilledema, abducens nerve palsy, and bitemporal headache—provided supportive evidence for transient elevations in ICP. Additionally, a carotid Doppler ultrasound performed during one of the episodes demonstrated postictal absence of diastolic flow, consistently with elevated ICP.

Ultimately, the patient underwent shunt revision by the neurosurgical team. Intraoperative assessment revealed significant calcification of the ventricular and peritoneal catheters and reservoir. VPS was completely replaced. Postoperatively, the patient experienced complete resolution of motor events, autonomic fluctuations, and papilledema. Antiseizure medication was tapered off over five days, and his maintenance regimen was de-escalated to levetiracetam monotherapy without recurrence of events. Finally, levetiracetam was discontinued during the following months and the patient remains seizure free at 1-year follow up.

3. Discussion

VPS malfunction can rarely present with paroxysmal neurological symptoms that closely mimic seizures [2]. In particular, the striking asymmetry of motor symptoms can be quite misleading. However, diffuse delta activity and suppression of the background in EEG may point to global cortical hypoperfusion due to raised ICP. When coupled with transient arterial hypertension and bradycardia, these changes reflect Cushing's response and support the diagnosis of intracranial hypertension. Additional findings such as cranial nerve palsies and transient papilledema provide further support. The exact cause of such a focal semiology in the context of a global hemodynamic failure is poorly understood, but some authors have suggested that release from cortical inhibition and the resulting partial decerebrate posture could be an explanation. One alternative reason may be transient uncus herniation with compression of the ipsilateral cerebral peduncle. Such a semiology

has been described in acute herniation crisis, which notably can affect cranial nerves as well [3].

The case we presented is illustrative of the diagnostic challenges posed by fluctuating VPS dysfunction. Neither reservoir puncture was under elevated pressure nor signs of LCR pressurization were observed during lumbar puncture. White matter lesions involving periventricular and callosal regions posed a diagnostic challenge by raising the differential diagnosis of a demyelinating disease (as initially suspected) versus a hypoxic-ischemic etiology, the latter being more frequently encountered in older patients with chronic normal pressure hydrocephalus. Such changes are thought to be secondary to prolonged hypoperfusion of the small penetrating arterial branches. Interestingly, classic radiological signs of hydrocephalus, such as an elevated Evan's index, are less reliable in patients with long-standing VPS, since they may develop a peculiar form of slit syndrome characterized by impaired ventricular compliance, possibly due to subependymal gliosis or altered venous drainage [4]. Thus, exclusive reliance on imaging and lumbar or reservoir puncture to confirm ICP elevation may be misleading. The Doppler finding of absent diastolic flow provides an underutilized but valuable non-invasive clue to intracranial hypertension, reinforcing the benefit of multimodal assessment.

In conclusion, this case underscores the critical role of extended video-EEG monitoring in distinguishing between epileptic seizures and paroxysmal manifestations of intermittent intracranial hypertension due to VPS dysfunction, especially when interictal EEGs are unremarkable, and even with normal imaging. Awareness of characteristic EEG hypoperfusion patterns and atypical presentations of intracranial hypertension can guide timely neurosurgical intervention, avoid misdiagnosis and prevent inappropriate antiseizure therapy. A multidisciplinary approach including neurology, neurosurgery, and neurocritical care is essential for rapid diagnosis and management.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Supplementary materials

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