Case Reports & Case Series

Idiopathic normal pressure hydrocephalus presenting with epileptic seizure as a cardinal symptom: A case presentation

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**ABSTRACT**

Idiopathic normal pressure hydrocephalus is usually regarded as a disease characterised by gait and balance disturbance, cognitive dysfunction and urinary symptoms. We report a rare case where iNPH should be considered as a cause of seizures.

1. Introduction

Idiopathic normal pressure hydrocephalus (iNPH) is usually regarded as a disease characterised by gait and balance disturbance, cognitive dysfunction, and urinary symptoms caused by a disturbance of the cerebrospinal fluid (CSF) dynamics [1]. The diagnosis of possible iNPH is based on clinical features, brain imaging, and CSF dynamics [2]. Nonetheless, many other symptoms have been described in patients with iNPH such as neuropsychiatric symptoms, parkinsonian symptoms, depression, bulbar dysfunction, olfactory palsy, gait apraxia, apathy, schizophrenia, immature personality and finally seizures [3]. We report a peculiar case of a patient with iNPH who presented with epileptic seizures as a first symptom.

2. Case report

The subject was a 60-year-old Caucasian woman, operated at the age of 53 for high-grade dysplastic rectum adenoma, and referred by ambulance to the emergency room (ER) of Linköping University hospital because of confusion. The same day, she underwent planned colonoscopy and laxation. She felt normal during the daytime but in the late evening her husband noticed some tics followed by uncontrollable jerking movements of the arms and legs and temporary confusion. At the ER her respiration and circulation were stable. Neurological assessment was normal within 30 min of observation. Because of hyponatraemia (sodium level at 125 mm/L) the primary diagnosis was laxative-induced hyponatraemia with seizure. The primary differential diagnosis was revised after an acute computer tomography (CT) which revealed obvious ventriculomegaly disproportionate to cerebral atrophy, Evans index of 0.46, corpus callosum thinning and elevation with a callosal angle of 62°, widening of the temporal horns (without hippocampal atrophy) and widening of the third ventricle to 20 mm, narrowing of the sulci and subarachnoid spaces over the high convexity and midline surface of the brain, and ballooning of the frontal horns (Fig. 1). A magnetic resonance image (MRI) of the brain demonstrated an aqueductal flow void and normal form of the third ventricle. There was no microangiopathy in the periventricular white matter. Radiological imaging fulfilled the criteria for iNPH. An acute electroencephalogram (EEG) with a total registration time of 20 min showed episodic, slow, and bi-temporal activity as a non-specific finding (Fig. 2a). An acute electrocardiograph (ECG) showed T-negativity at V1 to V3 and a suspect epsilon wave from V1 to V2 (Fig. 3). The patient was admitted to the neurological department for further monitoring and observation. Telemetry showed episodes of sinus bradycardia and some asystoles with maximum duration of 2.9 s without any atrioventricular block (AV) block. After consultation with a cardiologist, an ultrasound of the heart was performed showing a normal left ventricle with normal systolic and diastolic function, apical trabeculation of the right ventricle, but no findings for arrhythmogenic right ventricular dysplasia. Furthermore, a MR of the heart was normal without any
myocardium injury. Moreover, a loop recorder under 20 days was normal. In addition, an Ajamline provocation test was negative for Brugada syndrome. The ECG changes were reversible and no more arrhythmia was noticed, and the cardiological explanation was ECG and rhythm disorder because of hyponatraemia. Thus, the first seizure was provoked by hyponatraemia. No antiepileptic drug (AED) was prescribed. After corrugation of hyponatraemia the patient was dismissed with planned follow-up.

The patient was seen in follow-up and an iNPH investigation with a tap test was suggested to determine if she could be a positive subject for shunt surgery. Unfortunately, the patient was not motivated to undergo further examination.

Six months later the patient was referred to the ER by ambulance because of epileptic seizures with a duration of 40 min without sufficient effect of intravenous (iv) diazepam but with a satisfactory result with lorazepam 4 mg iv. An acute brain CT showed no change compared to the previous one, showing ventriculomegaly. Given the fact that the patient suffered from status epilepticus, she was intubated and connected to a Nicolet™ EEG monitor (Nervus), which demonstrated recurrent seizure activity with a typical evolving pattern that was parietal on the left side, and AED was started with Fosfenytoin i.v. (Fig. 2b). A lumbar puncture showed no pleocytosis, and the albumin and immunoglobulin G (IgG) index were normal. The next day, the EEG was seizure-free and the patient was extubated without any complications (Fig. 2c). She was started on Levetiracetam 500 mg twice a day. Under iNPH investigation, the patient described insidious balance impairment, and gait disturbance characterised by magnetic gait with a broader style without any sensory disability of the lower limbs. A Romberg test, start-stop test, reflexes, limb-power test, ataxia test, and Grasset test were all normal under neurological investigation. A gait test was compatible with iNPH disease with decreased step-height and length, decreased cadence, increased trunk sway during walking, turned-out toes on walking, widened standing base, turning bloc and retropulsion. A physiotherapist performed an evaluation and the patient needed nine seconds to complete a Timed Up and Go Test (TUG), 13 steps for TUG step-test, seven seconds for the 10-metre walking time test, and 13 steps for the 10-metre walking step test. A cognitive evaluation by the iNPH team’s occupational therapist showed normal results on a Mini Mental Statement test without any cognitive impairment. A lumbar puncture (LP) was performed with the patient lying in the lateral decubitus position and the opening pressure was 19 cm H2O. Drainage of 50 ml CSF was carried out. There was a motor improvement after the tap test and a ventriculoperitoneal shunt was put in place. Because of a shunt dysfunction the shunt was replaced and at the three- and six-month follow-ups the patient’s iNPH scale improved.

During the follow-up, the AED medication was switched from Levetiracetam to Lamotrigin 200 mg every day because of side effects, and during the transition period the patient experienced an episode of seizures. A new EEG revealed bi-temporal theta activity (Fig. 2d). A new brain MR with the epilepsy protocol revealed almost unmodified ventriculomegaly and no any epilepsy focus. After shunt surgery, the AED medication was stepped down without any seizure being experienced.

3. Discussion

Epilepsy is a common neurological condition characterised by recurrent seizures, at least two being unprovoked, taking place in a period of more than 24 h [4]. Hyponatraemia could be a provocative factor for seizures. In our case, the first seizure episode could be associated with hyponatraemia, but on the second occasion there was no any underlying factor except hydrocephalus. There are many possible triggers of epilepsy but ventriculomegaly is not so common. Common EEG characteristics in patients with iNPH is rhythmic slow waves [5]. The widening of the temporal horns should influence the function of the hippocampus area. The narrowing of high convexity sulci should influence parietal function and the bulging of the lateral ventricular roof could lead to disrupting parieto-occipital function, which could be a possible epilepsy trigger factor. After shunt insertion the ventriculomegaly decreases and the influence of adjacent ventricular parenchyma is reduced. Our patient responded to AEDs and after shunt insertion she was seizure-free for more than a year with non-therapeutic

Fig. 1. A brain CT revealed obvious ventriculomegaly with a callosal angle of 62°, widening of the temporal horns and widening of the third ventricle, narrowing of the sulci and subarachnoid spaces over the high convexity and midline surface of the brain, and ballooning of the frontal horns.
lamotrigine concentration.

4. Conclusion

Although iNPH is a disease with well characterised symptoms, in this rare case iNPH should be considered as a cause of seizures, and this is an issue that merits further research.

Authors’ contributions

Andreas Eleftheriou was the neurologist who performed the clinical and neurological evaluation. Andreas Eleftheriou was the major contributor in writing the manuscript. Salvador Amezcua was the neurophysiologist who performed the EEG investigations. Martin Nilsson was the neurosurgeon who performed the shunt operation.

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.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.inat.2019.100618.

References