CASE REPORT

Posterior reversible encephalopathy syndrome with documented hyponatraemia

Puneet Aulakh, Eduard Fatakhov, Christopher Francis Koch Jr, Shikha Kapil

Department of Internal Medicine, Georgia Health Sciences University, Augusta, Georgia, USA

Correspondence to
Christopher Francis Koch Jr,
ckoch2250@gmail.com

SUMMARY

We present a case of a 78-year-old African-American man with a history of hypertension, and with no prior history of seizure. The patient presented with hypertensive urgency as well as stroke such as symptoms of confusion, seizure, postictal confusion, left hemianopsia and hyponatraemia. MRI findings were suggestive of posterior reversible encephalopathy syndrome (PRES). The patient was treated with appropriate medications with the resolution of his stroke-like symptoms. This case report discusses a patient with PRES, in the setting of hyponatraemia, and how prompt recognition may prevent permanent neurological sequela such as epilepsy.

BACKGROUND

Posterior reversible encephalopathy syndrome (PRES), better known as reversible posterior leukoencephalopathy syndrome is a rare neurological disorder, diagnosis of which is based on clinical as well as neuroradiological imaging studies.^{1–3} PRES is characterised by confusion, headache, seizures and vision loss. Acute hypertension appears to play a major role at the time of symptom onset.⁴ An MRI of the brain is used to visualise the vasogenic oedema characteristic for PRES. Impaired cerebrovascular autoregulation is thought to be the underlying mechanism for the oedema. The vasogenic oedema usually involves the subcortical white matter composed of myelinated-fibre tracts in a cellular matrix of glial cells, arterioles and capillaries.¹ It is important to note that the oedema may also involve the grey matter of the brain. In addition, there can be substantial variability in the location of the lesions.¹

CASE PRESENTATION

We present the case of a 78-year-old African-American man with a medical history of hypertension (HTN), benign prostatic hypertrophy, hyperlipidemia (HLD) and no prior history of seizures, who was transferred from an outside hospital to rule out cerebrovascular accident (eg, stroke). The patient had initially presented to the emergency department with sudden loss of vision along with an episode of seizure-like activity lasting approximately 5 min associated with urinary incontinence and postictal confusion. Patient was found to have a systolic blood pressure >190 mm Hg and diastolic blood pressure >90 mm Hg. The patient was treated appropriately with antihypertensive agents. With a subsequent reduction in BP, the patient regained sight, however his vision

continued to be blurry. A head CT was unremarkable. However due to a high suspicion for ischaemic stroke, the patient was transferred to Georgia Health Sciences University (GHSU).

On presentation to GHSU, the patient continued to be confused and had headache and blurry vision. The patient also reported polydipsia, polyuria and nocturia. A review of systems was positive for poor appetite, with a 30-pound weight loss over the last month, along with night sweats, fever and chills. On physical examination the patient was alert and oriented to name and place only but not to time. On neurological examination, the patient was found to have left hemianopsia, CN III–XII were grossly intact. Vision check performed by finger counting method was accurate.

Labs on admission, revealed serum sodium of 122 mEq/L (table 1). The patient's seizure was initially attributed to hyponatraemia, however the paroxysmal, transient loss of vision could not be explained by the electrolyte abnormality. Serum sodium was corrected overnight in the emergency department with normal saline infusion. Urine electrolytes and serum osmolality were obtained to establish the aetiology of the patient's euvolemic hyponatraemia. Differential diagnoses included a syndrome of inappropriate secretion of antidiuretic hormone (SIADH), hypothyroidism and glucocorticoid excess. The patient was on citalopram 10 mg daily, a selective serotonin reuptake inhibitor that is known to cause SIADH.⁵ However SIADH is characterised by high urine sodium and urine osmolality, whereas measured values in the patient were low and therefore inconsistent with this diagnosis. Additionally, the patient's thyroid stimulating hormone (TSH) was low which is in consistent with hypothyroidism and serum cortisol was within normal limits, which ruled out glucocorticoid excess. No other organic causes of hyponatraemia could be identified. As the patient had admitted to drinking an excessive amount of fluids over the past 2–3 months, the hyponatraemia was attributed to psychogenic polydipsia.⁶

INVESTIGATIONS

Further workup included an MRI of the brain which showed hyper-resonance in the occipital and parietal lobes bilaterally. As the name implies, the posterior (parieto-occipital) area of the brain is involved in our patient. However, it is important to note that there is substantial variability in the location of the lesions in the PRES patients.^{7–9} Vasogenic oedema associated with PRES most often involves the parieto-occipital lobes followed closely

To cite: Aulakh P, Fatakhov E, Koch Jr CF, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-009311

Table 1 Patient's relevant lab values

Lab values	Results	Reference ranges
Admission		
Serum Na (mEq/L)	122	136–145
Following morning		
Serum Na (mEq/L)	138	136–145
Serum Osmolality (mOsm/kg)	279	275–295
Serum TSH (mU/L)	0.304	0.5–5
Serum Cortisol (µg/dL)	23.7	5–25
Urine Na (mEq/L)	88	100–260
Urine osmolality (mOsm/kg)	246	500–800

Including at the time of admission and the following morning.
TSH, thyroid stimulating hormone.

by frontal and temporal lobe lesions. Subcortical and deep white matter is also commonly involved.² Additionally, underlying comorbidities seem to influence where lesions are most likely to occur. For example, patients with sepsis were significantly more likely to sustain cortical injuries in comparison to non-septic patients.⁸ Patients who presented with seizures (like our patient) most commonly had lesions in the frontal and temporal lobes in contrast to the typical parieto-occipital involvement.^{2–9}

DIFFERENTIAL DIAGNOSIS

Differential diagnoses included SIADH, hypothyroidism, psychogenic polydipsia, central pontine myelinolysis and glucocorticoid excess. Other possibilities included alcoholism, and cirrhosis for any cause. After a thorough workup and several different laboratory values that were drawn including both serum cortisol and the urine osmolality, the two most likely diagnosis including both

glucocorticoid excess as well as SIADH were excluded. This is because as glucocorticoid excess implies there should be a serum increase in the level of cortisol, which was within the normal range. Another possibility included SIADH was possible because of the aforementioned citalopram that the patient was taking is known to cause this condition. SIADH was excluded based on the low urine osmolality because there would be an expected rise in the urine osmolality if SIADH were indeed the cause of the hyponatraemia. Hypothyroidism was excluded because of the normal value of TSH as was seen in figure 1. However, after the patient was recovering it was revealed that a prodigious amount of water was being consumed on a daily basis. As a result of this information the hyponatraemia was therefore most likely due to the psychogenic polydipsia. The final diagnosis that was urgently considered was central pontine myelinolysis frequently referred to as osmotic demyelination syndrome. This condition is typically considered because of a rapid correction in the serum sodium that is, far too rapid. Moreover this condition presents with a loss of consciousness, and other neurological symptoms including visual disturbances such as diplopia, seizures and possible quadriplegia. This condition generally gets worse as the sodium is corrected and is most commonly iatrogenic. In the present case however the patient rather than get worse with the increased sodium, continued to improve indeed regaining consciousness, and become more lucid as his electrolytes were corrected. When this coupled with the profound HTN that the patient was experiencing on admission made the final diagnosis of PRES syndrome the most likely diagnosis when the patient's symptoms were looked at globally.

OUTCOME AND FOLLOW-UP

The patient was treated with appropriate medications with resolution of his stroke-like symptoms. The patient was then

Figure 1 MRI of the brain in the given patient showing subcortical areas of hyperintense (white) signal involving the occipital lobes bilaterally.

discharged 2 days after admission, with a serum sodium of 132 mEq/L, and advised to restrict fluid intake.⁹ However, based on the patient's recovery and the financial constraints that the patient had additional imaging follow-up was refused.

DISCUSSION

PRES varies in onset and acuity and is often non-specific in nature. Headaches are usually typical on presentation and usually resolve with normalisation of blood pressure (BP). In addition, patients may present with symptoms of confusion, decreased level of consciousness, seizures and visual disturbances, which were all present in our patient. The cardinal systemic sign of PRES is an acute rise in diastolic blood pressure, which usually precedes symptom onset.^{3–5} Our patient became symptomatic hours before presentation. He was admitted with a BP of 198/98 mm Hg, which fluctuated through his hospital stay with a peak of 218/100 mm Hg. (table 1) Although there are many proposed mechanisms for the pathophysiology of PRES, the most widely accepted is impaired autoregulation of cerebral blood flow combined with some component of endothelial dysfunction.³ Symptoms associated with PRES usually resolve in hours to days after the onset, as was the case with this patient where the symptoms subsided in 6–8 h. The patient was discharged 2 days after admission, with a serum sodium of 132 mEq/L, and advised to restrict fluid intake.⁹

Contributors PA was involved in the management of this patient and the preparation of this manuscript. EF, CFK and SK were intimately involved in all stages of the interpretation and drafting and alterations made to this case report. All the authors of the aforementioned manuscript have been equally involved in the authorship, editing and design of the case report.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Hinchey J, Chaves C, Appignani B, *et al.* A reversible posterior leukoencephalopathy syndrome. *N Engl J Med* 1996;334:494–500.
- 2 Fugate JE, Claassen DO, Cloft H, *et al.* PRES: associated clinical and radiologic findings. *Mayo Clinic Proc* 2010;85:427–32.
- 3 Rabinstein A, Mandrekar J, Merrell R, *et al.* Blood pressure fluctuations in posterior reversible encephalopathy syndrome. *J Stroke Cerebrovasc Dis*, 2012;21:254–8.
- 4 Pugliese S, Finocchi V, Bozzao A, *et al.* Intracranial hypotension and PRES: case report. *J Headache Pain* 2010;11:437–40.
- 5 Barclay TS, Lee AJ. Citalopram-associated SIADH. *Ann Pharmacother* 2002;36:1558–63.
- 6 Gleason V, Martin N. Intracranial hypertension secondary to psychogenic polydipsia. *J Emerg Trauma Shock* 2012;5:193–5.
- 7 Wright KL, Polito H, French AE. PRES a case study. *Am J Nurs* 2012;112.
- 8 Hagemann G, Ugur T, Witte OW, *et al.* Recurrent posterior reversible encephalopathy syndrome (PRES). *J Hum Hypertens* 2004;18:287–9.
- 9 Lee VH, Wijidicks EF, Manno EM, *et al.* Clinical spectrum of reversible posterior leukoencephalopathy syndrome. *Arch Neurol* 2008;65:205–10.

Learning points

- Posterior reversible encephalopathy syndrome (PRES) syndrome presents with an acute rise in diastolic blood pressure.
- The most widely accepted mechanism of PRES syndrome is impaired auto regulation of cerebral blood flow.
- Untreated PRES syndrome frequently leads to the development of permanent neurological sequela such as epilepsy.

Copyright 2013 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- Submit as many cases as you like
- Enjoy fast sympathetic peer review and rapid publication of accepted articles
- Access all the published articles
- Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow