

## Postpartum Focal Seizure associated With Severe Progressive Hypotonic Hyponatremia and Pituitary Enlargement: A CARE Case Report

Jeyaram Nadarajan Saraswathy<sup>1</sup>, Geetha Guruswamy<sup>2</sup>

and Kabilesh Jothilingam<sup>3</sup>

<sup>1</sup>Department of Obstetrics and Gynaecology, Government Dindigul Medical College, Dindigul, Tamil Nadu, India.

ORCID 0009-0007-7670-0096 [jairam.cr@gmail.com](mailto:jairam.cr@gmail.com)

<sup>2</sup>Assistant Professor, Department of Obstetrics & Gynaecology, Government Vellore medical college, Tamil Nadu, India. ORCID: <https://orcid.org/0009-0007-1828-993X>, [dr.geethaguruswamy@gmail.com](mailto:dr.geethaguruswamy@gmail.com)

<sup>3</sup>Department of Medicine, BAU International Medical University, Batumi, Georgia.

ORCID 0009-0006-6460-6193; [drkabileshjothilingam@gmail.com](mailto:drkabileshjothilingam@gmail.com)

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### Article Info:

#### Corresponding Author:

Jeyaram Nadarajan  
Saraswathy

Department of Obstetrics and  
Gynaecology, Government  
Dindigul Medical College,  
Dindigul, Tamil Nadu, India

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### Abstract

**Background:** New-onset seizure in the postpartum period is a neurological emergency that requires prompt consideration of eclampsia as well as vascular, structural, infectious, endocrine, and metabolic causes. Severe hypotonic hyponatremia is a potentially reversible seizure precipitant, whereas syndrome of inappropriate antidiuresis (SIAD) remains a diagnosis of exclusion. The concurrent occurrence of a first postpartum seizure, severe progressive hypotonic hyponatremia, MRI-documented pituitary enlargement, and incompletely characterized adrenal function illustrates the difficulty of determining the cause of postpartum seizures.

**Case presentation:** A 24-year-old gravida 3 para 3 woman experienced a first focal convulsive seizure on postpartum day 8 after vaginal delivery. Serial investigations showed progressive hyponatremia from 142 mmol/L before delivery to a nadir of 112 mmol/L on postpartum day 11, measured serum osmolality of 256 mOsm/kg, urine sodium of 212 mmol/L, hypocalcemia to 6.3 mg/dL, and preserved renal function. The sodium concentration at the time of the seizure was unavailable. CT brain showed no acute intracranial abnormality. MRI demonstrated multifocal small bilateral subcortical parietal T2/FLAIR hyperintense lesions without diffusion restriction or susceptibility blooming, together with reported pituitary enlargement of approximately 10 x 11 mm. The treating teams considered hypotonic euvoletic hyponatremia and SIAD; however, urine osmolality was unavailable, the timing of urine sodium

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sampling relative to treatment was unclear, and a morning cortisol value of 5.05 had no recorded unit or confirmatory adrenal-axis testing. Management included close monitoring, electrolyte correction, calcium supplementation, fluid restriction, increased oral salt intake, sodium valproate, and multidisciplinary assessment. The patient's clinical condition improved, and specialist endocrine and pituitary evaluation was recommended. The sodium nadir occurred three days after the seizure; therefore, a

direct temporal association between the convulsive episode and a sodium concentration of 112 mmol/L could not be established.

**Conclusion:** Severe progressive hypotonic hyponatremia may have contributed to the seizure presentation, although a direct causal relationship was not established and SIAD could not be confirmed. Concurrent pituitary enlargement and incompletely characterized adrenal function warranted further endocrine assessment. Postpartum seizures require coordinated obstetric, metabolic, cerebrovascular, and endocrine evaluation. SIAD remained a provisional diagnosis because essential diagnostic criteria were incomplete, while postpartum eclampsia, posterior reversible encephalopathy syndrome (PRES), and cerebral venous sinus thrombosis (CVST) were not definitively excluded.

**Keywords:** postpartum seizure; hypotonic hyponatremia; syndrome of inappropriate antidiuresis; pituitary enlargement; acute symptomatic seizure; CARE case report

### Learning points

- A postpartum seizure should trigger immediate evaluation for eclampsia while metabolic and cerebrovascular causes are investigated in parallel.
- Profound hypotonic hyponatremia and hypocalcemia can lower the seizure threshold, but biochemical measurements close to the seizure are important when assessing causality.
- SIAD requires inappropriate urine concentration and exclusion of adrenal insufficiency, severe hypothyroidism, renal failure, and other causes; a high urine sodium value alone is insufficient.
- Pituitary enlargement in the early postpartum period may be physiological, but concomitant hyponatremia or uncertain cortisol status warrants dedicated pituitary imaging and endocrine assessment.
- The coexistence of a first postpartum seizure, severe progressive hypotonic hyponatremia, and pituitary enlargement requires careful interpretation of competing neurological and endocrine causes, particularly when adrenal-axis findings are incomplete.

### Introduction

New-onset seizures during the puerperium require urgent assessment. The assessment of this patient included competing obstetric, neurological, endocrine, and metabolic considerations, consistent with the comprehensive reporting recommended by CARE [1]. Postpartum preeclampsia or eclampsia may develop de novo after delivery, even after an apparently uncomplicated pregnancy, and may occur between 48 hours and 6 weeks postpartum [2]. Cerebral venous sinus thrombosis, posterior reversible encephalopathy syndrome (PRES), reversible cerebral vasoconstriction syndrome, ischemic or hemorrhagic stroke, infection, and other neurological disorders may resemble eclampsia clinically [3,4].

Hyponatremia is particularly important because severe hypotonic states may produce headache, nausea, confusion, encephalopathy, seizures, coma, and respiratory compromise. Evaluation should establish tonicity, assess extracellular volume status, measure urine osmolality and urine sodium, and exclude endocrine and renal mimics before SIAD is diagnosed [5,6]. Severe sodium

disturbances and hypocalcemia are recognized precipitants of acute symptomatic seizures [7]. Under International League Against Epilepsy recommendations, a seizure temporally linked to a severe metabolic derangement is classified as acute symptomatic rather than unprovoked, a distinction that influences prognosis and decisions about long-term antiseizure treatment [8].

Postpartum endocrine changes further complicate assessment. The pituitary enlarges during pregnancy because of lactotroph hyperplasia and can reach its greatest dimensions around delivery [9]. Contemporary volumetric imaging also confirms that anterior and total pituitary volumes are increased during pregnancy and may remain larger in the postpartum period [10]. At the same time, lymphocytic hypophysitis has a recognized association with pregnancy and the postpartum state and may present with pituitary enlargement and selective or multiple pituitary hormone deficiencies [11]. The concurrent hyponatremia, seizure, MRI signal abnormalities, and pituitary enlargement suggested several potentially related processes without establishing a single underlying mechanism.

Previous reports have described pregnancy-associated hyponatremia in the setting of established SIAD or preeclampsia [12-14]. In this patient, four findings occurred together: a first seizure on postpartum day 8; progressive hyponatremia from 142 mmol/L to a nadir of 112 mmol/L within less than two weeks; pituitary enlargement of approximately 10 x 11 mm on MRI; and uncertain adrenal function because the recorded cortisol result lacked a unit and dynamic testing was not performed. Pituitary enlargement raised secondary adrenal insufficiency and peripartum hypophysitis as alternative explanations for the biochemical findings. The absence of urine osmolality, electrolyte measurements at seizure onset, confirmatory adrenal testing, venographic imaging, and complete contemporaneous blood-pressure and proteinuria measurements limited definitive attribution.

Together, these findings complicated differentiation of SIAD from secondary adrenal insufficiency and other neurological or obstetric causes of postpartum seizure.

## **Case presentation**

### **Patient information and obstetric history**

A 24-year-old woman, gravida 3 para 3, blood group A positive, was admitted on 3 February 2026 in late pregnancy. She delivered a live female infant weighing 2.6 kg by vaginal delivery with episiotomy on 5 February 2026 at 12:27 PM. Apgar scores were 7/10 and 8/10. She had received antenatal care and had no documented history of seizures or major chronic illness. Her obstetric history included two previous preterm vaginal deliveries, the first resulting in stillbirth and the second in a living child.

Moderate microcytic hypochromic anemia was documented during the peripartum period. Hemoglobin was 8.7 g/dL on 3 February, fell to approximately 7.6 g/dL on 6 February, and later improved to 9.6 g/dL on 13 February and 9.4 g/dL on 16 February. She received one packed red-cell transfusion on 4 February and an iron infusion on 9 February. No causal relationship was established between anemia, transfusion, and subsequent hyponatremia, although fluid and solute exposure during treatment remained relevant to its assessment.

### **Neurological event and immediate clinical course**

On 13 February 2026, postpartum day 8, the patient developed one convulsive episode described by witnesses as salivation or frothing, upward eye deviation, tongue biting, and upper-limb movements. The episode was clinically recorded as a focal seizure. Urinary or bowel incontinence was not documented, and a prolonged postictal loss of consciousness was not clearly recorded. An associated confusional state prompted transfer for higher-dependency or intensive monitoring, metabolic evaluation,

neurological review, and neuroimaging. No previous seizure history was documented.

The lowest serum sodium concentration was recorded after, rather than during, the convulsive episode. Although serial investigations demonstrated progressive hyponatremia, sodium and calcium measurements contemporaneous with the seizure were unavailable. The documented sodium nadir of 112 mmol/L occurred on 16 February 2026, three days after the seizure on 13 February 2026. The evolving electrolyte disturbance may have lowered the seizure threshold, although the seizure could not be attributed to a specific measured sodium concentration. The clinical timeline is summarized in Table 1.

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| Date                           | Clinical event / management                                                                                                                                                                                                                   |
|--------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| 3 Feb 2026                     | Admission in late pregnancy. Moderate anemia documented. Serum sodium 142 mmol/L.                                                                                                                                                             |
| 4 Feb 2026                     | One packed red-cell transfusion administered.                                                                                                                                                                                                 |
| 5 Feb 2026                     | Vaginal delivery with episiotomy; live female infant 2.6 kg; Apgar scores 7/10 and 8/10.                                                                                                                                                      |
| 9 Feb 2026                     | Iron infusion administered during postpartum recovery.                                                                                                                                                                                        |
| 13 Feb 2026 (postpartum day 8) | First convulsive episode, clinically characterized as focal with confusional state; transferred for close monitoring and multidisciplinary assessment.                                                                                        |
| 14 Feb 2026                    | CT brain reviewed as showing no acute intracranial abnormality. MRI reported bilateral small subcortical parietal signal abnormalities and pituitary enlargement of approximately 10 x 11 mm.                                                 |
| 14-17 Feb 2026                 | Progressive hypotonic hyponatremia and hypocalcemia evaluated by medicine, neurology, nephrology, endocrinology, obstetrics and other teams; fluid restriction, increased oral salt intake, calcium replacement and antiseizure therapy used. |
| 16 Feb 2026                    | Lowest documented serum sodium 112 mmol/L.                                                                                                                                                                                                    |
| 17 Feb 2026                    | Clinically improved and hemodynamically stable; higher-center endocrinology review recommended because pituitary and adrenal-axis assessment remained incomplete.                                                                             |

## Diagnostic assessment

### Biochemical evaluation of hyponatremia

Serial serum sodium fell from 142 mmol/L on 3 February to 112 mmol/L on 16 February, with several intermediate values reported between 131 and 120 mmol/L. Measured serum osmolality was 256 mOsm/kg, establishing hypotonicity. Urine sodium was 212 mmol/L and renal function remained preserved, with creatinine approximately 0.6-0.9 mg/dL. Clinical teams described the patient as euvolemic and considered SIAD. Associated hypocalcemia was substantial, with a lowest recorded total serum calcium of 6.3 mg/dL and subsequent values around 7.0-8.2 mg/dL during correction. Key laboratory and imaging investigations are summarized in Table 2.

Although the biochemical findings were compatible with SIAD, several essential diagnostic criteria could not be assessed. Urine osmolality was unavailable, preventing demonstration of inappropriate urinary concentration. The timing of urine sodium collection relative to saline administration, oral salt

supplementation, and other interventions was unclear, potentially affecting interpretation of the elevated urine sodium concentration. A morning cortisol value of 5.05 was documented without a unit or assay reference range, and no ACTH stimulation test or equivalent confirmatory assessment was reported. Thyroid results were variable (TSH 3.65 microIU/mL with FT3 2.77 and FT4 1.18 on one assessment; repeat TSH 0.214 with T4 7.75), but these values did not establish severe primary hypothyroidism as the cause of hyponatremia. The pituitary MRI finding further warranted assessment for secondary adrenal insufficiency, which can mimic the biochemical features of SIAD [5,6]. Only some of the Bartter-Schwartz-type diagnostic criteria were satisfied. SIAD therefore remained a provisional diagnosis, while secondary adrenal insufficiency remained a clinically important alternative.

Hypocalcemia may also have contributed to a reduced seizure threshold. Both severe hyponatremia and hypocalcemia are established metabolic precipitants of acute symptomatic seizures [7]. However, the calcium concentration at seizure onset was not available. The available clinical and biochemical findings therefore supported an association with progressive electrolyte disturbances but did not establish a single biochemical cause of the convulsive episode.

### **Neuroimaging and postpartum neurological differential**

Non-contrast CT brain showed no intracranial hemorrhage, hydrocephalus, cerebral edema, or other significant acute parenchymal abnormality. Subsequent MRI reported multifocal small bilateral subcortical parietal T2/FLAIR hyperintense lesions without diffusion restriction or susceptibility blooming. In the postpartum setting, a posterior or parietal T2/FLAIR-predominant pattern raises the possibility of vasogenic edema and PRES, but the formal radiology report did not diagnose PRES and the printed MRI images were insufficient for independent confirmation [15]. Representative MRI images are shown in Figure 1, with additional adjacent axial views in Figure 2.

Cerebral venous sinus thrombosis (CVST) is an important cause of postpartum headache, seizures, focal deficits, and altered sensorium, particularly when vascular imaging has not been completed [3,16]. MR or CT venography results were not documented; consequently, CVST could not be definitively excluded. Complete blood-pressure and proteinuria measurements around the seizure were also unavailable, leaving postpartum eclampsia unresolved. The parietal MRI abnormalities raised PRES as another possibility, although the formal radiology report did not establish that diagnosis. Thus, progressive hypotonic hyponatremia represented a significant metabolic abnormality, while obstetric and cerebrovascular causes remained relevant competing considerations. The diagnostic findings and unresolved alternatives are compared in Table 3.

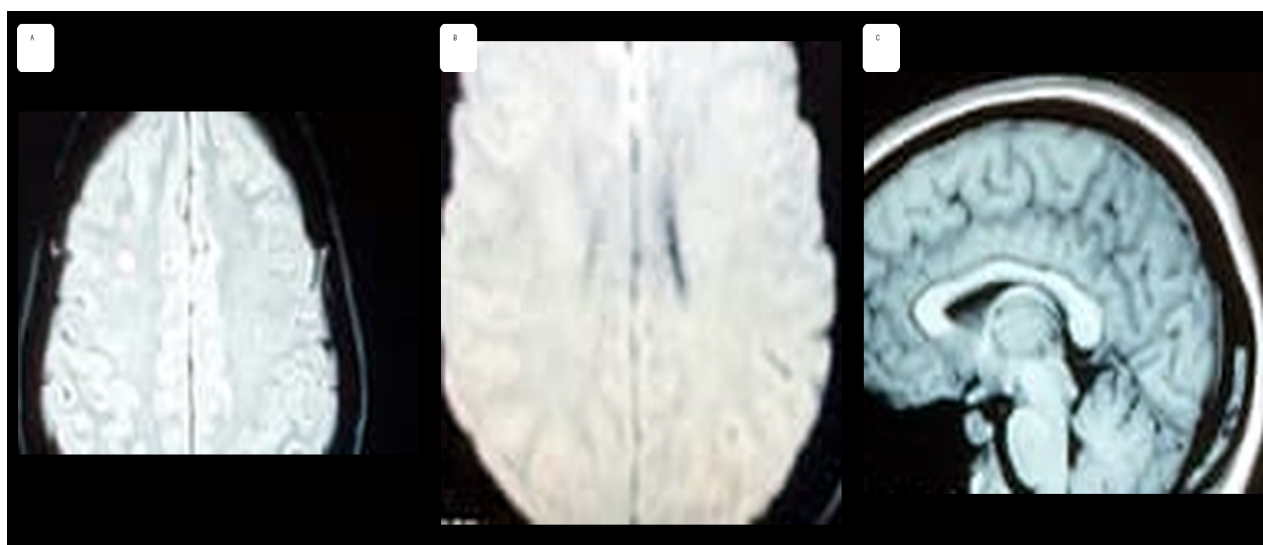


**Figure 1.** Selected de-identified MRI film images: (A) parietal T2/FLAIR view; (B) diffusion-weighted view; (C) sagittal sellar view. The formal report found no parietal diffusion restriction and described pituitary enlargement of approximately 10 x 11 mm.

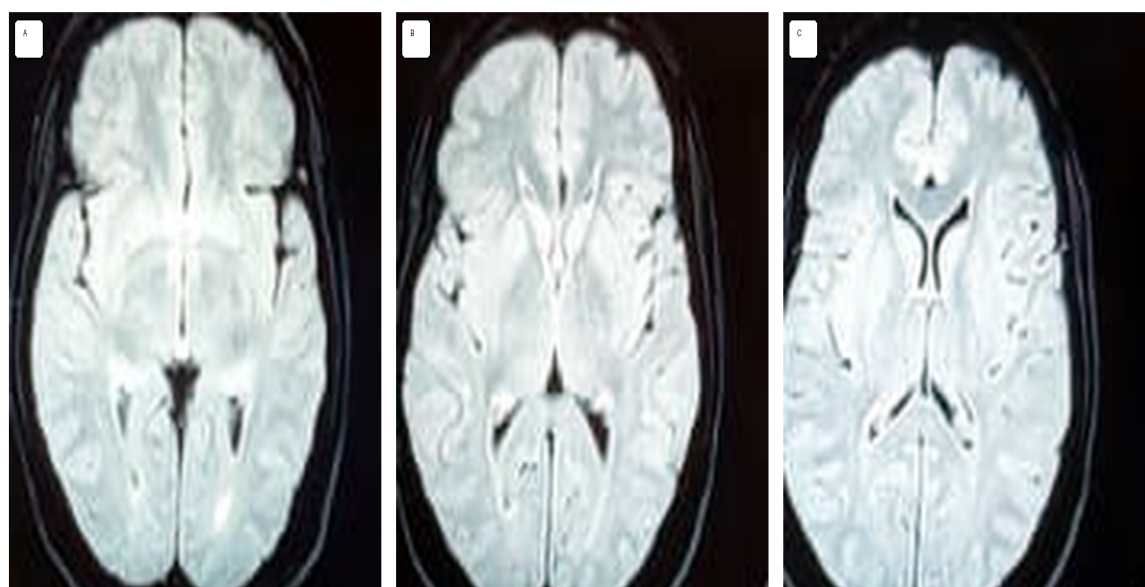


**Figure 2.** De-identified adjacent axial MRI film images illustrating the distribution of the supratentorial signal abnormalities across neighboring levels. Interpretation was based on the formal radiology report.

### Pituitary and endocrine interpretation

The MRI report described pituitary enlargement measuring approximately 10 x 11 mm and recommended dedicated contrast pituitary-protocol imaging. Representative sagittal sellar images are shown in Figure 3. Physiological pituitary enlargement is expected during pregnancy: classic MRI data found the largest glands in the immediate postpartum period, with some healthy postpartum women measuring approximately 10.0-11.8 mm [9]. More recent volumetric work likewise demonstrates increased anterior and total pituitary volume during pregnancy with persistence into the postpartum period [10]. Thus, a

measurement near 10-11 mm shortly after delivery may overlap with physiological change and does not independently establish a pituitary tumor.

Nevertheless, physiological enlargement is not the only possibility. Lymphocytic hypophysitis is disproportionately represented in women during pregnancy or postpartum and can present with symmetrical pituitary enlargement, stalk changes, headache, diabetes insipidus, or partial-to-complete anterior pituitary hormone deficiency [11]. Pituitary apoplexy is much less common but is an important emergency when acute severe headache, ophthalmoplegia, visual loss, reduced consciousness, or acute corticotrophic failure is present. No hemorrhagic sellar features, sudden severe headache, visual loss, or ophthalmoplegia were documented, and fundus examination was normal. Nevertheless, the absence of dedicated contrast pituitary imaging and complete adrenal-axis testing precluded definitive etiologic classification.

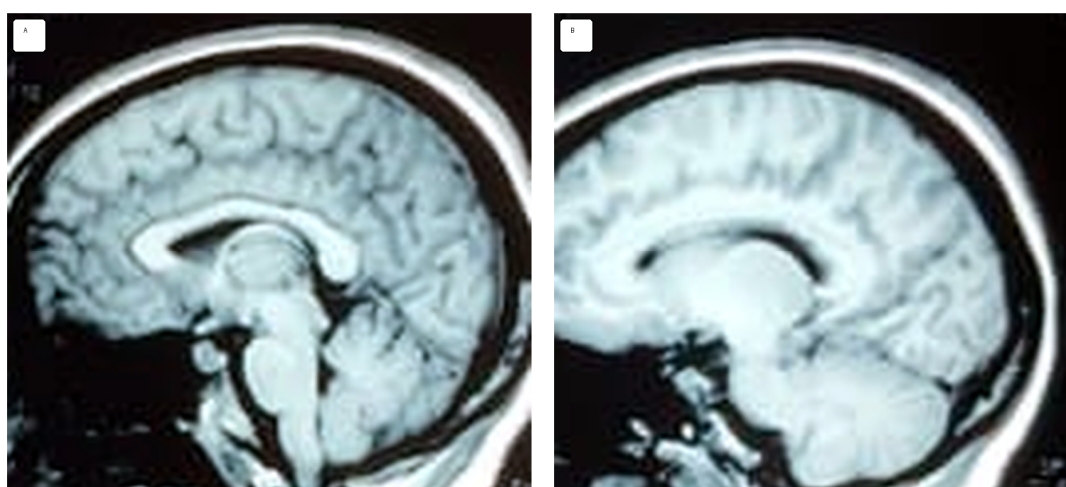


**Figure 3.** De-identified sagittal views of the sellar region from printed MRI films. These images provide anatomical context for the pituitary enlargement of approximately 10 x 11 mm described in the formal report. The printed-film photographs did not permit independent pituitary measurements or exclusion of subtle sellar abnormalities.

**Table 2.** Key investigations relevant to diagnostic interpretation

| Investigation             | Documented finding and interpretation                                                                                                                                       |
|---------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Hemoglobin                | 8.7 g/dL on 3 Feb; approximately 7.6 g/dL on 6 Feb; 9.6 g/dL on 13 Feb; 9.4 g/dL on 16 Feb. Peripheral smear: microcytic hypochromic anemia with neutrophilic leukocytosis. |
| Serum sodium              | 142 mmol/L on 3 Feb, progressively declining to a nadir of 112 mmol/L on 16 Feb. A sodium value contemporaneous with the seizure was unavailable.                           |
| Measured serum osmolality | 256 mOsm/kg, confirming hypotonic hyponatremia.                                                                                                                             |
| Urine sodium              | 212 mmol/L; timing relative to saline or salt therapy was unclear.                                                                                                          |
| Urine osmolality          | Urine osmolality was unavailable; a core diagnostic criterion for SIAD could not be assessed.                                                                               |

| Investigation     | Documented finding and interpretation                                                                                                                 |
|-------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------|
| Serum calcium     | Lowest documented 6.3 mg/dL; later approximately 7.0-8.2 mg/dL during correction.                                                                     |
| Renal function    | Preserved; creatinine approximately 0.6-0.9 mg/dL.                                                                                                    |
| Thyroid profile   | TSH 3.65 microIU/mL with FT3 2.77 and FT4 1.18; repeat TSH 0.214 with T4 7.75. The available results did not establish severe primary hypothyroidism. |
| Morning cortisol  | 5.05; unit not recorded. No confirmatory dynamic adrenal-axis test documented.                                                                        |
| CT brain          | No acute hemorrhage, edema, hydrocephalus, or significant acute parenchymal abnormality.                                                              |
| MRI brain         | Multifocal small bilateral subcortical parietal T2/FLAIR hyperintense lesions without diffusion restriction or susceptibility blooming.               |
| Pituitary imaging | Pituitary reported approximately 10 x 11 mm; dedicated contrast pituitary protocol advised.                                                           |

Table 3. Diagnostic framework: suspected SIAD and competing postpartum causes

| Diagnostic consideration                      | Evidence supporting                                                                                                     | Missing or competing evidence                                                                                                | Interpretation in this case                                                                                                         |
|-----------------------------------------------|-------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------|
| SIAD / syndrome of inappropriate antidiuresis | Hypotonic serum (256 mOsm/kg), high urine sodium (212 mmol/L), clinically described euvoemia, preserved renal function. | Urine osmolality absent; urine-sodium timing relative to treatment unclear; adrenal insufficiency not definitively excluded. | Findings were compatible with, but insufficient to confirm, SIAD.                                                                   |
| Possible acute symptomatic metabolic seizure  | Severe progressive hyponatremia, hypocalcemia, no previous seizure history, and clinical improvement during treatment.  | Contemporaneous serum sodium and calcium values were unavailable; MRI abnormalities were also present.                       | A metabolic contribution was possible, but the lack of peri-ictal electrolyte measurements prevented definitive causal attribution. |
| Postpartum eclampsia                          | Postpartum day 8 is within the recognized delayed postpartum window for new-onset disease.                              | Contemporaneous blood-pressure and proteinuria measurements were incomplete; a hypertensive emergency was not documented.    | Remained a differential diagnosis because the available findings were insufficient for confirmation or exclusion.                   |
| PRES                                          | Bilateral parietal T2/FLAIR abnormalities without diffusion restriction can be compatible with vasogenic                | Formal MRI report did not diagnose PRES; blood-pressure context incomplete.                                                  | PRES remained possible but was not established by imaging.                                                                          |

| Diagnostic consideration         | Evidence supporting                                                                                            | Missing or competing evidence                                                                              | Interpretation in this case                                                                                          |
|----------------------------------|----------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------|
|                                  | edema.                                                                                                         |                                                                                                            |                                                                                                                      |
| Cerebral venous sinus thrombosis | Puerperium and seizure are relevant risk/context factors.                                                      | Dedicated CT/MR venography not documented.                                                                 | Not demonstrated and not definitively excluded.                                                                      |
| Pituitary-adrenal disorder       | Pituitary enlargement plus uncertain morning cortisol makes central adrenal insufficiency clinically relevant. | No dedicated contrast pituitary MRI, ACTH, stimulation test, or complete pituitary panel documented.       | Further endocrine assessment was recommended before attributing hyponatremia solely to SIAD.                         |
| Primary epilepsy                 | New focal-appearing convulsion.                                                                                | No previous seizure history; major metabolic derangement and postpartum context provide provoking factors. | Less favored than a provoked seizure, but long-term classification depends on follow-up and neurological assessment. |

### Therapeutic intervention

Management was multidisciplinary and involved obstetrics, internal medicine, neurology, nephrology, endocrinology, anesthesiology, cardiology, and ophthalmology. The patient underwent close clinical observation and repeated electrolyte testing. Documented measures included calcium gluconate for hypocalcemia, a salt-rich diet and oral salt supplementation, progressive fluid restriction to approximately 800-1000 mL/day during specialist review, sodium valproate following the convulsive episode, and supportive inpatient care. The anemia was treated with one packed red-cell transfusion and an iron infusion. Ophthalmologic review documented a normal fundus without disc edema.

For severe symptomatic hyponatremia accompanied by seizures, current guidance recommends controlled treatment with 3% hypertonic saline and frequent sodium monitoring, with correction sufficient to reverse severe neurological symptoms while minimizing the risk of excessive correction and osmotic demyelination [17]. Treatment notes recorded a decision to

withhold hypertonic saline as sodium concentrations improved. Whether hypertonic saline had been administered beforehand, or the associated dose, timing and sodium-correction rate, could not be established from the available treatment records.

After stabilization, further treatment should be guided by the underlying mechanism. Fluid restriction is a conventional first-line strategy for confirmed SIAD, whereas glucocorticoid replacement is required if adrenal insufficiency is present [5,6,17]. Because adrenal-axis evaluation was incomplete, persistent SIAD could not be assumed without further assessment. Similarly, a single likely provoked seizure does not automatically establish epilepsy, and the duration of sodium valproate should be reviewed by neurology after metabolic normalization and follow-up imaging [8].

### Follow-up and outcome

The patient improved clinically during correction of the electrolyte disturbance and supportive treatment. At discharge, she was conscious, oriented, afebrile, hemodynamically stable, not

dyspneic, and maintaining oxygen saturation on room air. Pallor persisted, but cardiovascular and respiratory examinations were otherwise unremarkable and there was no pedal edema. No recurrent seizure was recorded during inpatient observation.

Because pituitary enlargement and adrenal function remained incompletely characterized, specialist endocrinology review and dedicated pituitary assessment were recommended. Results of post-discharge pituitary MRI, paired serum and urine osmolality measurements, ACTH/cortisol testing, a complete pituitary hormone profile, repeat sodium measurements, EEG, and long-term seizure outcomes were unavailable.

## Discussion

The presentation involved several overlapping potential causes of postpartum seizures and hyponatremia. The patient experienced a first seizure on postpartum day 8 and was subsequently found to have profound progressive hypotonic hyponatremia, hypocalcemia, bilateral parietal MRI signal abnormalities, and pituitary enlargement. Although each finding was clinically relevant, a single underlying disease process was not established.

## Comparison with previously reported cases of postpartum hyponatremia and SIAD

Previous publications have described SIAD in pregnancy and clinically significant hyponatremia accompanying preeclampsia or related hypertensive disorders [12-14]. The present patient experienced a first postpartum seizure accompanied by progressive hypotonic hyponatremia and pituitary enlargement, with incomplete information about adrenal function. The findings did not establish a single underlying cause before discharge.

Three aspects of this presentation warrant consideration. First, the sodium nadir occurred three days after the convulsive episode. A direct relationship between the measured concentration

of 112 mmol/L and the seizure could not be established, consistent with the importance of temporal linkage when classifying acute symptomatic seizures [8]. Second, pituitary enlargement raised the possibility of secondary adrenal insufficiency or peripartum lymphocytic hypophysitis, both of which may be associated with hypotonic euvoletic hyponatremia and may warrant targeted endocrine testing and dedicated pituitary imaging [6,11]. Third, although low serum osmolality, high urine sodium, apparent euvoletic, and preserved renal function supported SIAD, missing urine osmolality and uncertainty about the timing of urine sampling relative to treatment prevented diagnostic confirmation.

Accurate differentiation of these causes depends on the timing of biochemical investigations and correlation with the clinical course. Paired serum and urine osmolality, urine sodium measured before interventions that alter sodium excretion, and serum sodium and calcium values close to seizure onset would assist assessment of a metabolic cause. Morning cortisol with an assay unit and reference range, appropriate adrenal-axis testing, and a complete pituitary hormone profile are important when central adrenal insufficiency is considered. Pituitary-protocol MRI, venographic imaging, serial blood-pressure and proteinuria measurements, and longitudinal neurological and endocrine evaluation may also be indicated according to the clinical presentation. Several of these investigations were unavailable in this patient, limiting a definitive etiologic diagnosis.

## Hyponatremia as a seizure precipitant and the importance of timing

Profound hypotonic hyponatremia is a recognized cause of cerebral edema, encephalopathy, and seizures, and concurrent hypocalcemia can further lower the seizure threshold [7]. Both electrolyte abnormalities could have lowered the seizure threshold in this patient, although neither concentration was measured at seizure onset. However, the lowest sodium concentration of 112

mmol/L was documented on 16 February, three days after the seizure, and a contemporaneous peri-ictal sodium value was unavailable. This distinction is clinically important because classification of an acute symptomatic seizure requires a close temporal relationship with the provoking systemic disturbance [8]. Severe progressive hyponatremia remained a relevant metabolic abnormality, but the nadir measurement did not establish the sodium concentration at seizure onset.

The fall from 142 to 112 mmol/L across less than two weeks also suggests a clinically significant acquired disturbance, although the precise onset rate cannot be reconstructed. Hyponatremia that develops rapidly is generally more likely to cause neurological symptoms because cerebral adaptation is incomplete, whereas chronic hyponatremia may be tolerated at lower concentrations. Because serial sodium measurements close to the seizure were incomplete, the duration of the hyponatremia could not be reliably classified as acute or chronic [5,17].

#### **Diagnostic considerations in suspected SIAD**

The treating teams documented hypotonic euvoletic hyponatremia and considered SIAD. The measured serum osmolality of 256 mOsm/kg, urine sodium of 212 mmol/L, clinical euvolectia, and preserved kidney function are compatible with that diagnosis. However, modern diagnostic frameworks require evidence that urine remains inappropriately concentrated in the presence of hypotonicity and require exclusion of glucocorticoid deficiency, severe hypothyroidism, renal failure, and relevant medications or physiological stimuli [5,6]. Without urine osmolality, inappropriate urinary concentration could not be demonstrated.

Secondary adrenal insufficiency remained an important differential diagnosis because it can mimic SIAD without hyperkalemia. Cortisol deficiency removes tonic suppression of vasopressin and impairs free-water excretion,

producing euvoletic hypotonic hyponatremia. In this patient, the reported morning cortisol of 5.05 cannot be interpreted confidently because the unit and assay reference range were not recorded, and no dynamic testing was documented. The simultaneous finding of pituitary enlargement further increases the clinical importance of excluding central adrenal insufficiency before assigning a definitive SIAD diagnosis. SIAD therefore remained a provisional diagnosis because urine osmolality, definitive adrenal-axis testing, and dedicated pituitary imaging were unavailable.

#### **Postpartum seizure: avoiding automatic attribution to eclampsia or PRES**

Delayed postpartum preeclampsia and eclampsia remain essential considerations because new-onset disease can occur after hospital discharge and after an apparently normal antepartum course [2]. Conversely, not every postpartum seizure is eclamptic. Reviews of peripartum neurological emergencies emphasize CVST, PRES, reversible vasoconstriction, stroke, infection, and metabolic disorders as important mimics [3,4]. Blood-pressure and proteinuria findings around the seizure were incompletely documented; postpartum eclampsia therefore remained in the differential diagnosis.

The parietal T2/FLAIR lesions without diffusion restriction may reflect vasogenic edema, a pattern that can occur in PRES [15]. Interpretation depends on lesion distribution, clinical symptoms, blood pressure, and complete MRI sequences. The formal radiology report did not diagnose PRES, and the imaging abnormalities were therefore considered compatible with a possible vasogenic process rather than diagnostic of PRES. Similarly, CVST was not definitively excluded because dedicated venographic findings were unavailable. Postpartum seizures accompanied by otherwise unexplained headache, focal neurological signs, or persistent symptoms warrant consideration of venous imaging when clinical suspicion remains [16].

**Pituitary enlargement: physiological adaptation versus disease**

Pituitary size changes substantially during pregnancy. Elster and colleagues showed a linear increase in gland height with gestational progression and documented postpartum glands measuring up to approximately 11.8 mm in healthy women [9]. Contemporary volumetric data also demonstrate enlargement of the anterior and total pituitary during pregnancy and residual postpartum differences [10]. Thus, a reported dimension of approximately 10 x 11 mm around the early postpartum period can overlap with physiological lactotroph hyperplasia and does not independently establish a pituitary mass.

The clinical significance changes when pituitary enlargement coexists with possible pituitary hormone dysfunction. Lymphocytic hypophysitis is a recognized peripartum inflammatory disorder and can cause enlargement with ACTH, TSH, gonadotropin, or other hormonal deficiencies [11]. Pituitary apoplexy is rarer but should be suspected with sudden severe headache, visual symptoms, ophthalmoplegia, altered consciousness, or acute adrenal insufficiency; pregnancy and postpartum cases are well documented [18]. Classic clinical and hemorrhagic imaging features of apoplexy were not documented. Dedicated contrast-enhanced pituitary MRI and a complete endocrine assessment could help differentiate physiological enlargement from hypophysitis, adenoma, or other sellar disease.

**Context within pregnancy-associated hyponatremia literature**

Published pregnancy literature confirms that clinically important hyponatremia and SIAD are uncommon but recognized. A review of pregnancies with established SIAD found only a small number of published cases and noted frequent coexistence with preeclampsia in the wider literature [12]. Larger obstetric series show that hyponatremia can accompany preeclampsia and is associated with more severe maternal disease, although the mechanism may be SIAD-

like or related to reduced effective circulating volume and water retention [13]. Case reviews of severe preeclampsia-associated hyponatremia similarly emphasize diagnostic heterogeneity and the need for multidisciplinary management [14]. Although these studies provided relevant clinical context, preeclampsia could not be diagnosed in this patient without the necessary clinical criteria.

**Management implications**

The immediate priority in a convulsing patient with severe symptomatic hypotonic hyponatremia is stabilization and controlled sodium correction. Current treatment standards favor rapid intermittent hypertonic saline for severe neurological symptoms, with frequent biochemical reassessment and careful limitation of total daily correction to reduce osmotic demyelination risk [17]. After stabilization, ongoing treatment depends on identifying the underlying cause of hyponatremia. This is particularly important when SIAD criteria are incomplete or when pituitary disease and adrenal insufficiency remain plausible. Neurological follow-up is also important. Acute symptomatic seizures related to reversible metabolic disturbances have a different recurrence profile from unprovoked seizures, and long-term antiseizure therapy may not be necessary in every patient once the provoking factor has resolved [8]. The MRI signal abnormalities and incomplete vascular evaluation in this case provide additional reasons for specialist follow-up before stopping or continuing antiseizure medication.

**Strengths and limitations**

Strengths include serial biochemical testing, formal CT and MRI assessment, documentation of severe hypotonic hyponatremia and hypocalcemia, and review by multiple specialties. Representative de-identified MRI images were interpreted in conjunction with the formal radiology report. Several factors limited etiologic assessment: sodium and calcium concentrations at seizure onset were unavailable; urine osmolality was not

documented; the timing of urine sodium sampling relative to treatment was uncertain; the cortisol result lacked a unit and confirmatory adrenal testing; contemporaneous blood-pressure and proteinuria measurements were incomplete; dedicated venography and pituitary-protocol contrast MRI were not documented; and EEG, post-discharge neuroendocrine outcomes, hypertonic saline dosing, and sodium-correction rates were unavailable. Consequently, a single cause of the seizure could not be established, and SIAD and other relevant postpartum diagnoses remained incompletely evaluated.

The timing of laboratory sampling and treatment was important because saline administration, salt supplementation, and fluid restriction could influence serum and urinary sodium measurements. Incomplete blood-pressure and proteinuria records also limited assessment for a postpartum hypertensive disorder. Further endocrine and neurological evaluation was recommended to clarify the relationship between pituitary enlargement, possible adrenal dysfunction, and the neurological event.

Severe hypotonic hyponatremia may have contributed to the seizure risk, but SIAD remained provisional, and postpartum eclampsia, PRES, and CVST were not definitively excluded.

## Conclusion

Severe progressive hypotonic hyponatremia accompanied a first postpartum seizure in a patient with hypocalcemia and pituitary enlargement. Low serum osmolality, high urine sodium, apparent euolemia, and preserved renal function supported suspected SIAD, but missing urine osmolality and incomplete adrenal-axis testing prevented confirmation. Pituitary enlargement of approximately 10 x 11 mm may overlap with physiological postpartum changes, although an underlying pituitary-adrenal disorder could not be excluded. Because peri-ictal electrolyte measurements were unavailable, a direct causal relationship between the sodium nadir and the

seizure could not be established. Postpartum eclampsia, PRES, and CVST also remained important differential diagnoses. This presentation emphasizes coordinated obstetric, metabolic, neurological, and endocrine assessment and appropriate specialist follow-up.

## CRedit author statement

**Geetha Guruswamy:** Conceptualization; Data curation; Formal analysis; Methodology; Resources; Writing - original draft preparation.

**Jeyaram Nadarajan Saraswathy:** Data curation; Investigation; Methodology; Supervision; Validation; Visualization.

**Kabiles Jothilingam:** Writing – review & editing.

## Declarations

**Ethics approval:** Ethical approval for this case report was obtained from the Institutional Ethics Committee. The case was reported in accordance with the ethical standards of the institutional research committee and the Declaration of Helsinki.

**Consent for publication:** Written informed consent for publication of the clinical details and anonymized imaging findings was obtained from the patient.

**Availability of data and materials:** All relevant data supporting the findings of this case report are included in the manuscript. Additional information is not publicly available because of patient privacy and confidentiality.

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**Reporting guideline:** The report was structured in accordance with the CARE case-report recommendations [1].

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