

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

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Running title: Postpartum seizure with severe hyponatremia

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DOI: 10.5281/zenodo.23435882

[Received 09-09-2026,
Accepted 15-10-2026,
Published 17-10-2026]

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Abstract

Background: New-onset seizure in the postpartum period is a neurological emergency in which eclampsia must be considered promptly, but vascular, structural, infectious, endocrine, and metabolic causes may produce an indistinguishable presentation. Severe hypotonic hyponatremia is a reversible seizure precipitant, whereas syndrome of inappropriate antidiuresis (SIAD) remains a diagnosis of exclusion. To our knowledge, the co-occurrence of a first-ever postpartum seizure, severe progressive hypotonic hyponatremia, MRI-documented pituitary enlargement, and an adrenal axis that could not be adequately characterized has rarely been reported together, and this combination, rather than a confirmed endocrine diagnosis, is what makes the present case instructive.

Case presentation: A 24-year-old gravida 3 para 3 woman developed a first focal convulsive seizure on postpartum day 8 after vaginal delivery. Serial investigations documented progressive hyponatremia from 142 mmol/L before delivery to a nadir of 112 mmol/L on postpartum day 11, measured serum osmolality 256 mOsm/kg, urine sodium 212 mmol/L, hypocalcemia to 6.3 mg/dL, and preserved renal function. The exact serum sodium at the moment of seizure was not recoverable from the available record. CT brain showed no acute intracranial abnormality. MRI demonstrated multifocal small bilateral subcortical parietal T2/FLAIR hyperintense lesions without diffusion restriction or susceptibility blooming and reported pituitary enlargement of approximately 10 x 11 mm. Clinical teams considered symptomatic hypotonic euvolemic hyponatremia and SIAD; however, urine osmolality was not documented, the timing of urine sodium relative to therapy was unclear, and a morning cortisol of 5.05 lacked a recorded unit or confirmatory adrenal-axis

Cite this article: Jeyaram Nadarajan Saraswathy, Geetha Guruswamy and Kabilesh Jothilingam, 2026. Postpartum Focal Seizure associated With Severe Progressive Hypotonic Hyponatremia and Pituitary Enlargement: A Care Case Report. Advanced Natural Sciences: Life and Health Sciences, 3(2), 168-182

testing. Management included close monitoring, electrolyte correction, calcium supplementation, fluid restriction, increased oral salt intake, sodium valproate, and multidisciplinary review. The patient improved clinically and was referred for dedicated endocrine and pituitary assessment. The nadir sodium concentration was documented three days after the convulsive episode, and no time-stamped peri-ictal sodium, calcium, or urine osmolality value was retrievable; the seizure is

therefore reported as associated with, and not attributed to, a serum sodium of 112 mmol/L.

Conclusion: Severe progressive hypotonic hyponatremia was a clinically important and potentially reversible contributor to this postpartum seizure presentation, but definitive attribution to SIAD cannot be made from the available data. The coexistence of pituitary enlargement and incompletely characterized adrenal function strengthens the need for structured endocrine follow-up. Postpartum seizures should be evaluated with parallel obstetric, metabolic, cerebrovascular, and endocrine pathways rather than being attributed to a single diagnosis prematurely. SIAD is consequently retained as a provisional, criteria-incomplete diagnosis, and postpartum eclampsia, posterior reversible encephalopathy syndrome (PRES), and cerebral venous sinus thrombosis (CVST) could not be completely excluded on the basis of the available record.

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 temporal biochemical documentation is important before assigning 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 instructive feature of this presentation is the simultaneous presence of a first postpartum seizure, severe progressive hypotonic hyponatremia, pituitary enlargement, and incomplete adrenal-axis data; such a constellation requires provisional rather than definitive diagnostic labelling and

structured record retrieval before causal claims are made.

Introduction

Case reports are most clinically useful when diagnostic uncertainty, competing explanations, and limitations of the available evidence are made explicit; this report is structured in accordance with the CARE reporting recommendations [1]. New-onset seizure during the puerperium is a time-critical presentation. Postpartum preeclampsia or eclampsia may arise de novo after delivery, including after an apparently uncomplicated pregnancy, and the period from 48 hours to 6 weeks postpartum remains clinically relevant [2]. Nevertheless, pregnancy and the puerperium also increase the diagnostic complexity of seizure because cerebral venous thrombosis, posterior reversible encephalopathy syndrome (PRES), reversible cerebral vasoconstriction syndrome, ischemic or hemorrhagic stroke, infection, and non-obstetric neurological disease can overlap with 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; the distinction matters because it influences prognosis and the need for long-term antiseizure treatment [8]. The postpartum endocrine context adds another layer. 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]. This case therefore required interpretation of severe hyponatremia, seizure, MRI signal abnormalities, and pituitary enlargement as potentially related but not necessarily caused by a single mechanism.

Published reports of pregnancy-associated hyponatremia and SIAD have predominantly described antenatal or intrapartum presentations, frequently in women with preeclampsia, hyperemesis, or an already established diagnosis of SIAD, and have usually reported either a confirmed endocrine cause or an isolated electrolyte disturbance without concurrent sellar imaging findings [16-18]. The present report differs in four respects that are seldom documented together in a single patient: the neurological event was a first-ever seizure occurring late in the puerperium, on postpartum day 8; the hyponatremia was progressive and

severe, falling from a normal antenatal concentration of 142 mmol/L to a nadir of 112 mmol/L within less than two weeks; MRI reported pituitary enlargement of approximately 10 x 11 mm, which raised secondary adrenal insufficiency and peripartum hypophysitis as competing explanations for the same biochemical picture; and the adrenal axis could not be characterized because the single cortisol value lacked a recorded unit and no dynamic testing was performed. The contribution of this case is therefore not another confirmed instance of postpartum SIAD, but a transparently documented example of how the absence of a small number of investigations, namely urine osmolality, peri-ictal electrolytes, dynamic adrenal testing, venographic imaging, and a contemporaneous blood-pressure and proteinuria series, can prevent secure attribution even when the biochemical pattern initially appears characteristic.

This case is notable because it highlights the diagnostic challenge of severe progressive postpartum hypotonic hyponatremia occurring alongside a first seizure and pituitary enlargement, while demonstrating the difficulty of distinguishing SIAD from secondary adrenal insufficiency and other postpartum neurological causes when key biochemical and imaging data are incomplete.

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. Her pregnancy was documented as booked, and no previous seizure disorder or major chronic medical illness was recorded in the available clinical notes. 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. The record does not establish a direct causal relationship between anemia, transfusion, and the subsequent sodium disturbance, but these interventions are relevant when reconstructing postpartum fluid and solute exposure.

Neurological event and immediate clinical course

On 13 February 2026, postpartum day 8, the patient developed one convulsive episode described by attendants as drooling or frothing of saliva, upward rolling of the eyes, tongue bite, and upper-limb movements. The treating notes characterized the event 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.

Importantly, the lowest recorded sodium value occurred after the seizure rather than at the same time. The available dataset documents progressive hyponatremia across the admission but does not preserve a clearly time-stamped sodium value within the immediate peri-ictal window. Consequently, a metabolic acute symptomatic seizure is clinically plausible but cannot be proven solely from the nadir sodium concentration. Specifically, the lowest recorded serum sodium of 112 mmol/L was obtained on 16 February 2026, three days after the convulsive episode of 13 February 2026, and no sodium, calcium, or urine osmolality result carrying a time stamp within the peri-ictal window could be recovered from the archived record. Throughout this report the seizure is therefore described as having occurred during a period of progressive hypotonic hyponatremia, and is not attributed to any single measured sodium concentration.

Table 1. Clinical timeline

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 focal convulsive seizure 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.

Date	Clinical event / management
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 a documented nadir of 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.

Although the biochemical pattern is compatible with SIAD, the diagnostic dataset is incomplete by standard criteria. Urine osmolality was not documented, so inappropriate urinary concentration cannot be demonstrated. The record does not clearly indicate whether urine sodium was collected before or after saline, salt supplementation, or other interventions, which is relevant because therapy can alter urinary sodium excretion. A morning cortisol result of 5.05 was recorded without a unit, and no ACTH stimulation test or equivalent confirmatory assessment was documented. 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 the available values do not establish severe primary hypothyroidism as the explanation for the hyponatremia. Because secondary adrenal insufficiency can closely mimic SIAD biochemically, the pituitary MRI finding makes this exclusion especially important [5,6]. Taken together, these gaps mean that the Bartter-Schwartz-type criteria for SIAD were only partly satisfied. SIAD is accordingly reported here as a provisional working diagnosis rather than a confirmed one, and secondary adrenal insufficiency remains an unexcluded alternative that would produce an indistinguishable euvolemic hypotonic pattern. The calcium abnormality provides a second plausible contributor to seizure threshold. Both severe hyponatremia and hypocalcemia are established metabolic precipitants of acute symptomatic seizures [7]. However, as with sodium, the exact calcium level at the time of the seizure is not clearly time-stamped in the available record. This limitation supports the more cautious wording that the seizure was associated with progressive dyselectrolytemia rather than definitively caused by a single measured value.

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 available printed-film photographs do not permit independent neuroradiological adjudication [12].

Cerebral venous sinus thrombosis is another important postpartum cause of headache, seizure, focal deficits, and altered sensorium, and dedicated venographic imaging is generally required when clinical suspicion remains [3,13]. The manuscript record did not document MR venography or CT venography. Therefore, CVST cannot be declared excluded on the basis of the available material. Similarly, a complete

contemporaneous blood-pressure and proteinuria series around the seizure was not available in the supplied record, so postpartum eclampsia cannot be ruled in or ruled out solely from this dataset. The most defensible interpretation is that metabolic disturbance was prominent while obstetric and vascular causes required parallel consideration. To state this explicitly: in the absence of a complete peripartum blood-pressure and proteinuria record, dedicated MR or CT venography, and a full multisequence MRI review, postpartum eclampsia, PRES, and CVST could not be completely excluded in this patient, and none of these diagnoses should be considered formally ruled out on the strength of the data presented.

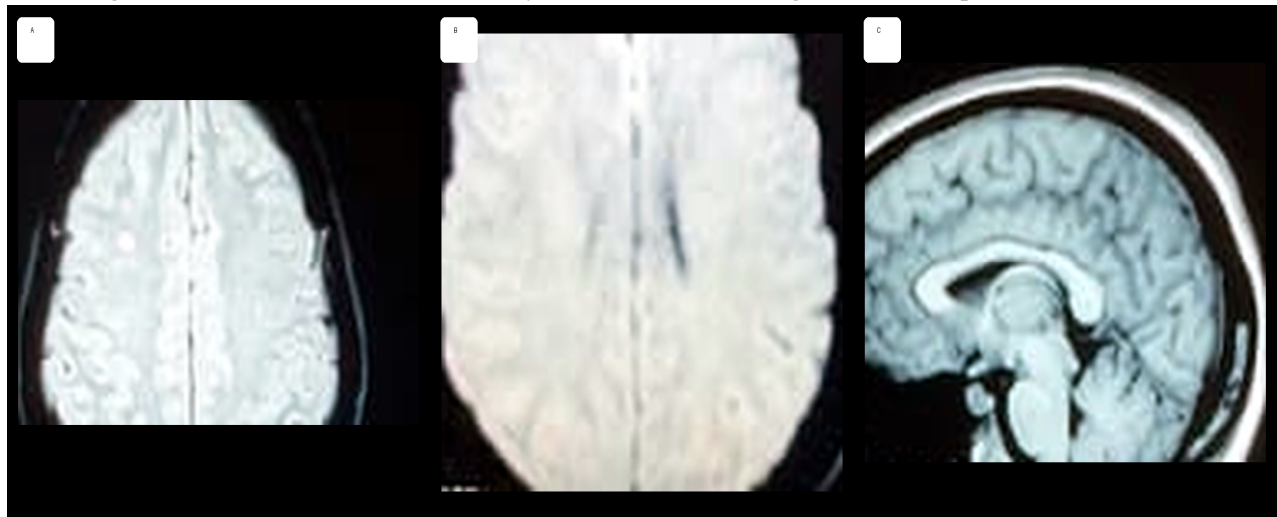


Figure 1. Selected de-identified MRI film images from the index study. (A) Representative axial T2/FLAIR-dominant parietal slice. (B) Representative diffusion-weighted image from the same examination; the formal report stated that the small bilateral parietal lesions did not show diffusion restriction. (C) Mid-sagittal image through the sellar region. The formal report described pituitary enlargement of approximately 10 x 11 mm. These photographs are illustrative; sequence interpretation and measurements should be based on the original DICOM data and formal neuroradiology report.

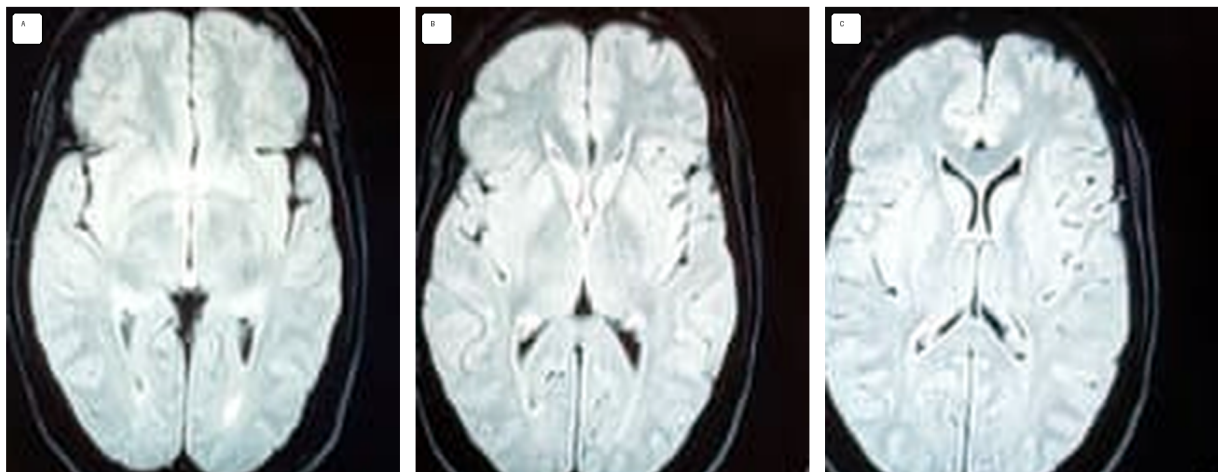


Figure 2. Additional de-identified adjacent axial MRI film slices from the same examination, included to show the distribution of the supratentorial signal pattern across neighboring levels. No new radiological diagnosis is inferred from these photographs; the formal report remains the reference interpretation.

Pituitary and endocrine interpretation

The MRI report described pituitary enlargement measuring approximately 10 x 11 mm and recommended dedicated contrast pituitary-protocol imaging. 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 should not automatically be labeled a 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. The available record did not describe hemorrhagic sellar features, sudden severe headache, visual loss, or ophthalmoplegia, and fundus examination was normal; nevertheless, absence of a dedicated contrast pituitary study and incomplete adrenal-axis testing prevents definitive etiologic classification.

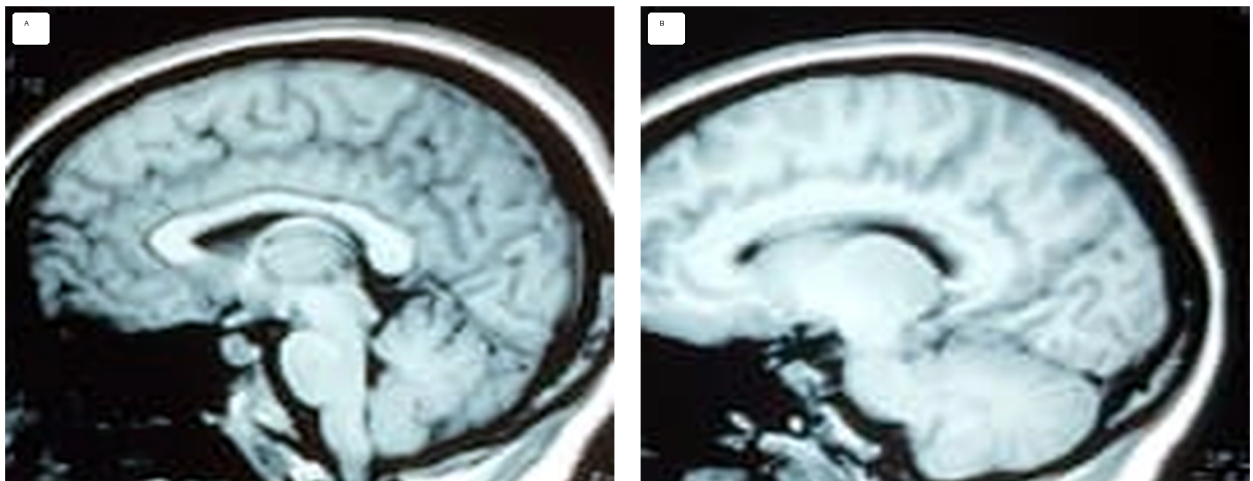


Figure 3. De-identified sagittal sellar-region views selected from the printed MRI film. These adjacent images provide anatomical context for the pituitary finding reported as approximately 10 x 11 mm. Because they are photographs of printed films rather than source DICOM images, they are not suitable for independent pituitary measurement or for excluding hypophysitis, microadenoma, or subtle hemorrhagic change.

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. Exact peri-ictal sodium was not clearly time-stamped.

Investigation	Documented finding and interpretation
Measured serum osmolality	256 mOsm/kg, confirming hypotonic hyponatremia.
Urine sodium	212 mmol/L; timing relative to saline/salt therapy not documented.
Urine osmolality	Not documented; therefore a core SIAD criterion could not be verified.
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. Severe primary hypothyroidism not established.
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 euolemia, preserved renal function.	Urine osmolality absent; urine-sodium timing relative to treatment unclear; adrenal insufficiency not definitively excluded.	Compatible but not proven; best described as suspected or provisional SIAD.
Acute symptomatic metabolic seizure	Progressive profound hyponatremia, hypocalcemia, no previous seizure history, clinical improvement with correction.	Exact serum sodium/calcium within the peri-ictal window not preserved; MRI abnormalities were also present.	Metabolic contribution is likely, but strict temporal causal attribution is limited.
Postpartum eclampsia	Postpartum day 8 is within the recognized delayed postpartum window for new-onset disease.	Complete contemporaneous blood-pressure/proteinuria dataset not available; no clear hypertensive emergency documented in supplied manuscript.	Must remain in the initial differential; cannot be adjudicated from available data alone.
PRES	Bilateral parietal T2/FLAIR abnormalities without diffusion restriction can be	Formal MRI report did not diagnose PRES; blood-pressure context incomplete.	Possible imaging differential, not established.

Diagnostic consideration	Evidence supporting	Missing or competing evidence	Interpretation in this case
	compatible with vasogenic 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.	Requires endocrine follow-up 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 for seizure control or short-term prophylaxis, 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 seizure, contemporary guidance prioritizes controlled treatment with 3% hypertonic saline and frequent sodium measurement, with an early limited rise sufficient to reverse severe neurological symptoms while avoiding excessive correction and osmotic demyelination [14]. The source notes refer to a later decision to withhold hypertonic saline once sodium began improving, but the exact concentration, bolus or infusion regimen, timing,

and rate of sodium correction were not recoverable from the available manuscript record. These details should be added if they can be verified from medication charts because they are clinically important for a high-quality case report.

Once immediate neurological risk has resolved, therapy should address the underlying mechanism. Fluid restriction is a conventional first-line strategy for confirmed SIAD, whereas glucocorticoid replacement is required if adrenal insufficiency is present [5,6,14]. In this case, the incomplete adrenal-axis evaluation means that long-term management should not assume persistent SIAD without reassessment. 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. By 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 documented in the available inpatient record.

Because pituitary enlargement had not been fully characterized and the adrenal-axis assessment remained incomplete, higher-center endocrinology review and dedicated pituitary evaluation were recommended. The manuscript record does not provide post-discharge pituitary MRI, repeat serum and urine osmolality, ACTH/cortisol testing, prolactin or complete pituitary hormone assessment, repeat sodium trajectory, EEG, or long-term seizure outcome. These follow-up data would materially strengthen the final publication if available.

Discussion

This case is clinically informative because it demonstrates how several plausible postpartum diagnoses can coexist and how premature diagnostic closure can overstate causality. 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. Each finding is clinically relevant, but the available evidence does not prove that all arose from one disease process.

Novelty and relation to previously reported postpartum hyponatremia and SIAD cases

Placing this case against the existing literature clarifies what it does and does not add. A review of pregnancy in women with known SIAD identified only a small published experience and focused on antenatal management of an already established diagnosis [16]. Obstetric series and case reports of clinically important hyponatremia have largely arisen in the context of preeclampsia, where the sodium disturbance accompanies a defined hypertensive disorder and is interpreted within it [17,18]. In those reports the diagnostic question is usually the severity or management of a recognized entity. In the present patient the

diagnostic question was different in kind: the presenting event was a first seizure in the late puerperium, the hyponatremia was progressive and reached a profound nadir, and the aetiological label itself remained unresolved at discharge.

Three features give the case its distinct instructional value. First, the temporal dissociation between the seizure and the nadir sodium concentration illustrates a reasoning error that is easy to make retrospectively, namely reading the most abnormal laboratory value in an admission as the cause of an earlier clinical event; ILAE criteria for acute symptomatic seizure require close temporal linkage to the provoking insult, which this dataset cannot demonstrate [8]. Second, the coexistence of pituitary enlargement converted a routine hyponatremia work-up into a differential that included secondary adrenal insufficiency and peripartum lymphocytic hypophysitis, both of which can generate euvolemic hypotonic hyponatremia with a high urine sodium and neither of which can be excluded without dynamic adrenal testing and pituitary-protocol contrast imaging [11,6]. Third, the case demonstrates that a biochemical pattern widely regarded as typical of SIAD, that is hypotonicity with a urine sodium of 212 mmol/L in a clinically euvolemic patient with preserved renal function, is not diagnostic when urine osmolality is missing and the timing of the urine sample relative to saline and oral salt therapy is uncertain.

The reporting value of the case therefore lies in the explicit mapping of uncertainty rather than in the assertion of a diagnosis. It also defines a minimum dataset that should be secured whenever severe postpartum hyponatremia coincides with a seizure: paired serum and urine osmolality with urine sodium obtained before therapeutic intervention, time-stamped peri-ictal sodium and calcium, an 8-9 a.m. cortisol reported with its assay unit and reference range together with plasma ACTH and a short corticotropin stimulation test, a complete anterior pituitary hormone profile, dedicated pituitary-protocol contrast MRI, MR or CT

venography, a full blood-pressure and proteinuria series, a reconciled medication and intravenous fluid chart, and documented post-discharge endocrine and neurological follow-up. Cases in which this dataset is complete can settle attribution; cases such as the present one show why attribution should be withheld when it is not.

Hyponatremia as a seizure precipitant and the importance of timing

Profound hypotonic hyponatremia is a recognized cause of cerebral edema, encephalopathy, and seizure, and concurrent hypocalcemia can further lower the seizure threshold [7]. This makes dyselectrolytemia a strong candidate contributor. However, the nadir sodium of 112 mmol/L was documented on 16 February, three days after the seizure. The exact sodium within the immediate peri-ictal interval is not available. This distinction is more than semantic: ILAE definitions of acute symptomatic seizure emphasize a close temporal association with the provoking systemic insult [8]. Accordingly, the article is strongest when it describes severe progressive hyponatremia as an important associated and potentially causal metabolic disturbance, rather than implying that the seizure occurred at a documented sodium of 112 mmol/L.

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. Since serial values around the seizure are incomplete, the safest approach is to emphasize symptom severity and serial monitoring rather than to classify the entire episode as unequivocally acute or chronic [5,14].

Why SIAD remains provisional

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 euvoletism, 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]. The absence of urine osmolality is therefore a major limitation rather than a minor missing laboratory value.

Adrenal insufficiency deserves particular emphasis because secondary adrenal failure may 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. For these reasons SIAD is designated a provisional diagnosis throughout this report. A definitive label should be applied only after urine osmolality measured during hypotonicity, adrenal-axis confirmation by dynamic testing, and dedicated pituitary imaging have been obtained and reviewed together.

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]. In the present record, the available blood-pressure and

proteinuria documentation around the event is incomplete, so the case should not be framed as having definitively excluded eclampsia.

The parietal T2/FLAIR lesions without diffusion restriction could be compatible with vasogenic edema, a pattern encountered in PRES [12]. Yet imaging diagnosis requires correlation with distribution, blood pressure, symptoms, and complete MRI sequences. The formal report did not diagnose PRES; therefore, describing the lesions as "compatible with a reversible vasogenic process including PRES in the differential" is more defensible than labeling the case as PRES. Likewise, the absence of documented venography means that CVST was not definitively excluded. A postpartum seizure with unexplained headache, focal findings, or persistent symptoms should prompt dedicated venous imaging when clinical suspicion remains [13].

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 by itself prove 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 [15]. The current record lacks the classic clinical and hemorrhagic imaging features of apoplexy, but a dedicated contrast-enhanced pituitary MRI and complete endocrine profile are necessary to distinguish 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 [16]. 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 [17]. Case reviews of severe preeclampsia-associated hyponatremia similarly emphasize diagnostic heterogeneity and the need for multidisciplinary management [18]. These reports are relevant as differential context, but they should not be used to retroactively diagnose preeclampsia 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 [14]. After stabilization, the cause of hyponatremia should be revisited rather than assumed. 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. The imaging figures have been restricted to de-identified representative slices and interpreted according to the formal report rather than by retrospective over-reading of photographed films.

The principal limitations are clinically important: the exact serum sodium and calcium at the time of seizure are not clearly documented; urine osmolality is absent; the timing of urine sodium relative to therapy is uncertain; the cortisol result lacks a unit and confirmatory adrenal testing; a complete contemporaneous blood-pressure/proteinuria series is not available; dedicated MR venography and pituitary-protocol contrast MRI are not documented; EEG and post-discharge neuroendocrine outcomes are unavailable; and hypertonic-saline dosing and sodium-correction rates cannot be reconstructed. These gaps preclude definitive attribution of the event to SIAD alone and should remain explicit in the submitted manuscript.

Recognizing that several of these gaps are potentially recoverable rather than inherent, a structured retrieval of the original source documentation has been undertaken and is directed at four categories of record. The first is the laboratory information system output for the whole admission, printed with date and time stamps, so that every sodium, calcium, osmolality, thyroid, and cortisol result, including the assay unit and reference range for the cortisol value, can

be positioned accurately relative to the seizure and to each therapeutic intervention. The second is the complete inpatient prescription and intravenous fluid chart, including oxytocin, hypotonic fluids, diuretics, antiemetics, analgesics, hypertonic saline volumes and infusion times, and antiseizure medication, since drug and fluid exposure is directly relevant both to the cause of the hyponatremia and to the reconstruction of correction rates. The third is the nursing observation and antenatal record, providing the serial blood-pressure readings and all urine protein results from admission to discharge, which are required before postpartum preeclampsia or eclampsia can be meaningfully addressed. The fourth is the post-discharge file, comprising any repeat sodium and paired osmolality measurements, dynamic adrenal and anterior pituitary testing, pituitary-protocol contrast MRI, venographic imaging, EEG, and clinical review at higher centres.

Any of these documents recovered before publication will be reported in full, with the diagnostic interpretation and treatment chronology revised accordingly; items that remain unavailable are declared above as explicit limitations. Until that verification is complete, the conservative formulation used throughout this manuscript is retained: severe progressive hypotonic hyponatremia was a clinically important and potentially reversible contributor to the seizure, suspected SIAD is provisional, and eclampsia, PRES, and CVST remain incompletely excluded alternatives.

Conclusion

A postpartum seizure associated with progressive profound hypotonic hyponatremia requires urgent correction of reversible metabolic abnormalities while eclampsia, PRES, CVST, and other neurological causes are evaluated in parallel. In this patient, low serum osmolality, high urine sodium, clinical euvolemia, and preserved renal function supported suspected SIAD, but the

diagnosis was incomplete because urine osmolality and definitive adrenal-axis evaluation were unavailable. Pituitary enlargement of approximately 10 x 11 mm may overlap with physiological postpartum change, yet the uncertain cortisol result makes dedicated pituitary and endocrine follow-up clinically necessary. The central lesson is not that one diagnosis explains every abnormality, but that rigorous postpartum seizure assessment should integrate obstetric, metabolic, neurological, and endocrine evidence and state diagnostic uncertainty explicitly. The novelty of this report lies in that combination of a first postpartum seizure, severe progressive hypotonic hyponatremia, and pituitary enlargement assessed with an incomplete biochemical and imaging dataset, and in the resulting discipline of reporting SIAD as provisional while eclampsia, PRES, and CVST remain formally unexcluded.

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.

Kabilesh Jothilingam: Writing – review & editing.

Declarations

Ethics approval: Ethical approval for this case report was obtained from the relevant Institutional Ethics Committee. The report was prepared in accordance with institutional ethical standards and the principles of the Declaration of Helsinki.

Consent for publication: Written informed consent was obtained from the patient for publication of her clinical details and anonymized radiological images. Patient confidentiality and anonymity have been maintained throughout the report.

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

Competing interests: The authors declare that they have no competing interests.

Funding: No specific funding was received for this work.

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

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