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Central Diabetes Insipidus as a Complication of Meningitis in the Intensive Care Unit: A Case Report

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Abstract

Background: Central diabetes insipidus (CDI) is a rare neuroendocrine complication of meningitis caused by hypothalamic–pituitary dysfunction, leading to severe fluid and electrolyte imbalances in critically ill patients.
Objective: This study aims to describe the clinical presentation, diagnostic approach, and management of CDI as a complication of meningitis in a critically ill ICU patient and to raise awareness of neuroendocrine sequelae in neurocritical care.
Methods: This study is a case report describing the clinical course of a single patient. Data were obtained from medical records, including symptoms, physical examination findings, laboratory results, and treatment interventions, and were analyzed descriptively in accordance with CARE guidelines, with patient anonymization maintained.
Results: A 34-year-old woman was admitted to the ICU with progressive decreased consciousness and was diagnosed with subacute tuberculous meningitis based on clinical and cerebrospinal fluid findings. She initially presented with hyponatremia (serum sodium, 124 mEq/L), followed by persistent polyuria (>3 mL/kg/hour) with rising serum sodium levels (136 mEq/L) on day 3, reflecting a biphasic AVP disturbance pattern. CDI was confirmed based on clinical criteria and a positive response to desmopressin. Management included fluid resuscitation, electrolyte correction, desmopressin administration, antituberculosis therapy, and broad-spectrum antibiotics, resulting in gradual clinical stabilization.
Conclusion: CDI should be recognized as a potential complication of severe meningitis. Early identification through serial monitoring of urine output, serum sodium, and osmolality—combined with prompt differentiation from syndrome of inappropriate antidiuretic hormone secretion (SIADH) and cerebral salt wasting—is essential to prevent hemodynamic instability and secondary brain injury. A multidisciplinary approach is critical for optimal neurocritical care management.

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INTRODUCTION

Central diabetes insipidus (CDI) is a rare endocrine disorder with an estimated prevalence of 1 in 25,000 and is most commonly caused by traumatic brain injury, neurosurgical interventions, or hypothalamic-pituitary tumors (Adams et al., 2018; Patti et al., 2022; Pusvitasari & Amrita, 2025). Infectious central nervous system (CNS) etiologies, including meningitis and encephalitis, have also been identified as potential triggers of hypothalamic-pituitary dysfunction, although they remain far less recognized in clinical practice (Pekic et al., 2024; Richie, 2022).

Meningitis—particularly tuberculous meningitis—carries a mortality rate of up to 20–50% in severe cases and is associated with diverse neurological and systemic complications

(Donovan et al., 2019; Seddon et al., 2019). Basal meningeal inflammation, vasculitis, and granulomatous infiltration in tuberculous meningitis can directly involve the hypothalamic-pituitary axis, disrupting arginine vasopressin (AVP) synthesis and release along the neurohypophyseal tract, thereby precipitating CDI (Lovera et al., 2020). Increased intracranial pressure and cerebral edema may further impair hypothalamic perfusion, compounding the risk of neuroendocrine dysfunction in severe CNS infections (Singh et al., 2020).

Several case reports have documented CDI as a complication of CNS infections, although the evidence remains limited. Salih (2021) described a case of CDI secondary to tuberculous meningitis that presented with a biphasic sodium disturbance pattern, suggesting progressive hypothalamic neuronal destruction as the underlying mechanism. Das (2025) reported CDI in the context of bacterial meningitis, emphasizing the importance of early desmopressin initiation to prevent hemodynamic deterioration. Sodero (2024) Conducted a systematic review of hypothalamic-pituitary complications in bacterial meningitis and concluded that neuroendocrine dysfunction is likely underreported due to the lack of routine endocrine screening in ICU protocols. Collectively, these studies suggest that infection-related CDI may be masked or recognized late because of concurrent electrolyte disorders, such as the syndrome of inappropriate antidiuretic hormone secretion (SIADH) or cerebral salt wasting (CSW) (Tsentsiper & Dryagina, 2014).

In the ICU, CDI presents a unique diagnostic challenge because of its clinical overlap with SIADH and CSW. Although these conditions may initially present with similar sodium disturbance patterns, they require fundamentally different fluid management strategies. Misdiagnosis—such as applying fluid restriction intended for SIADH to a patient with CDI—can aggravate hypovolemia, hemodynamic instability, and secondary brain injury. Conversely, overly rapid correction of hypernatremia carries the risk of cerebral edema, underscoring the need for precise and timely diagnosis (Mittal et al., 2025; Tomkins et al., 2025).

Despite its clinical significance, published reports of infection-related CDI in critically ill ICU patients remain scarce. Most existing literature focuses on post-traumatic or postsurgical CDI, leaving a critical gap in understanding the clinical trajectory, diagnostic approach, and optimal management of CDI arising specifically from CNS infection in neurocritical care settings. This case report presents a patient with presumed tuberculous meningitis who developed CDI with a biphasic sodium disturbance pattern in the ICU. This study aims to describe the clinical presentation, diagnostic approach, and management of CDI as a complication of meningitis in a critically ill patient and to raise awareness of the importance of early neuroendocrine evaluation in severe CNS infections.

METHOD

This case report describes a rare complication of meningitis—central diabetes insipidus—in a single critically ill patient and was conducted in accordance with the CARE (CAse REport) guidelines to ensure completeness and transparency in clinical reporting. The case was managed in the intensive care unit (ICU) of a tertiary referral hospital specializing in severe neurological disorders, a setting that enabled continuous hemodynamic monitoring, strict fluid balance management, and advanced life support throughout the patient's admission.

The subject was a 34-year-old female admitted with subacute meningitis and clinical suspicion of tuberculous meningitis who subsequently developed fluid and electrolyte disturbances. Data were collected retrospectively from her medical records up to October 2023 and included clinical history, physical examination findings, serum sodium levels, cerebrospinal fluid analysis, urine output, daily fluid balance, hemodynamic parameters, and radiological imaging, supplemented by direct observations made during hospitalization.

Central diabetes insipidus was diagnosed on the basis of persistent polyuria, defined as urine output exceeding 3 mL/kg/hour, accompanied by elevated or high-normal serum sodium levels and low urine specific gravity, with the diagnosis confirmed by clinical response to desmopressin. Volume status and laboratory parameters were used to exclude differential diagnoses, including SIADH and cerebral salt wasting. Meningitis was diagnosed clinically and supported by cerebrospinal fluid analysis. Hyponatremia was defined as a serum sodium level below 135 mEq/L, and hemodynamic instability was defined as abnormal blood pressure or heart rate requiring vasopressor support. Treatment comprised fluid resuscitation, electrolyte

correction, desmopressin administration, antimicrobial therapy, and antituberculosis agents, with clinical and laboratory parameters monitored serially to assess the therapeutic response.

A descriptive qualitative approach was used to analyze the data by sequentially reviewing the patient's clinical course and comparing the findings with the existing literature to identify key diagnostic patterns and management implications relevant to central diabetes insipidus complicating central nervous system infection. This study was conducted in accordance with established ethical principles for medical research. All identifying information was anonymized to preserve patient confidentiality, and written informed consent for publication was obtained from the patient's family.

RESULTS AND DISCUSSION

Results

A 34-year-old woman was referred from a regional hospital with a progressive decrease in consciousness over one week. The decline was gradual, culminating in unresponsiveness to verbal stimuli. The clinical course was preceded by intermittent fever, vomiting, cough, and dyspnea. There was no history of seizures, focal neurological deficits, hypertension, diabetes mellitus, cardiovascular disease, stroke, or prior tuberculosis treatment.

At the emergency department, the patient had a Glasgow Coma Scale (GCS) score of 5. She was intubated for airway protection and admitted to the ICU. Central venous access was established, and mechanical ventilation was initiated in PSIMV+ mode. On the first ICU day, vital signs showed a blood pressure of 136/82 mmHg, heart rate of 108 beats per minute, respiratory rate of 23 breaths per minute while ventilator-assisted, and oxygen saturation of 97%. Neurological examination revealed positive meningeal signs without clear motor lateralization. The pupils were 2 mm bilaterally; light reflexes were absent, but corneal reflexes were preserved. No overt signs of raised intracranial pressure were documented. Laboratory testing demonstrated hyponatremia, with a serum sodium level of 124 mEq/L.

Lumbar puncture yielded yellowish cerebrospinal fluid (CSF), with a leukocyte count of 68 cells/mm^3, consisting of 75% mononuclear cells, a glucose level of 61.9 mg/dL, and a negative bacterial culture. Based on the clinical and CSF findings, subacute meningitis with suspected tuberculous meningitis was diagnosed. Broad-spectrum antibiotics, dexamethasone, and later anti-tuberculosis therapy using a fixed-dose combination (4FDC) regimen were initiated. Hyponatremia was corrected using 3% hypertonic saline, administered as 500 mL over 24 hours.

On the first day of ICU admission, the patient remained mechanically ventilated and sedated. Fluid balance was closely monitored, with initial urine output within the normal range. Serum sodium was recorded at 124 mEq/L, and hypertonic saline therapy was initiated to correct hyponatremia. Hemodynamic status remained relatively stable without vasopressor support.

On the second day of ICU care, the patient developed a high-grade fever of 39°C accompanied by tachycardia. Urine output began to increase to approximately 3 mL/kg/hour, raising suspicion of an evolving fluid imbalance. Despite correction efforts, the serum sodium level remained low at 124 mEq/L. By the third day, persistent polyuria of 3.2 mL/kg/hour was observed, accompanied by a progressive increase in serum sodium level to 136 mEq/L, indicating a shift in fluid and electrolyte regulation.

The combination of persistent polyuria, rising serum sodium levels, and clinical deterioration raised suspicion of central diabetes insipidus. Differential diagnoses, including syndrome of inappropriate antidiuretic hormone secretion (SIADH) and cerebral salt wasting, were considered; however, the presence of increasing serum sodium levels and high urine output supported the diagnosis of central diabetes insipidus.

The patient was treated with desmopressin and careful fluid replacement therapy. Following administration, urine output decreased, and hemodynamic parameters showed transient improvement. Electrolyte levels were closely monitored to avoid rapid correction and prevent further neurological complications.

Despite aggressive management, the patient's condition continued to deteriorate, with persistent hemodynamic instability and worsening neurological status. The overall clinical course suggested severe central nervous system involvement and a poor prognosis.

Following appropriate management, the patient showed gradual improvement in urine output and electrolyte balance. Neurological status improved over time, and the patient was eventually stabilized.

Discussion

This case illustrates the diagnostic complexity of central diabetes insipidus (CDI) arising in the context of suspected tuberculous meningitis. The most clinically instructive feature of this case is the biphasic sodium trajectory: initial hyponatremia (Na 124 mEq/L) on admission, followed by a rapid reversal to rising serum sodium (136 mEq/L), coinciding with persistent polyuria by day three. This sequence represents a pathophysiologically meaningful transition—not merely a laboratory fluctuation—and is central to understanding the evolving neuroendocrine failure in this patient.

The sodium transition pattern in this patient warrants detailed explanation. On admission, hyponatremia likely reflected stress-induced AVP hypersecretion or inflammatory stimulation of hypothalamic osmoreceptors, mechanisms consistent with either SIADH or early AVP excess. By days two and three, the emergence of high-volume polyuria (>3 mL/kg/hour) alongside rising serum sodium indicated a fundamental shift: AVP secretion had failed, unmasking CDI. This biphasic pattern—hyponatremia transitioning to hypernatremic polyuria—mirrors the triphasic response described after hypothalamic injury, wherein an initial phase of AVP excess is followed by depletion as neuronal destruction progresses.

In this patient, the anatomical basis for CDI is consistent with the known predilection of tuberculous meningitis for basal meningeal structures. The intense basal exudate characteristic of tuberculous meningitis frequently extends to the suprasellar cistern and hypothalamic–pituitary region, where granulomatous infiltration, vasculitis, or ischemic injury can directly damage AVP-producing neurons in the supraoptic and paraventricular nuclei or disrupt axonal transport along the pituitary stalk. Concurrent cerebral edema and raised intracranial pressure likely further impaired hypothalamic perfusion in this critically ill patient (Singh et al., 2020). The convergence of these mechanisms—rather than any single insult—probably underlies the progressive neuroendocrine failure observed here (Verbalis, 2012).

Comparison with previously reported cases highlights both the similarities and distinctive features of this presentation. CDI secondary to tuberculous meningitis in a patient who likewise demonstrated a biphasic sodium disturbance, interpreted as reflecting progressive hypothalamic neuronal destruction. The sodium trajectory in that case mirrored the pattern seen here: an initial hyponatremic phase attributable to excess AVP secretion, followed by polyuric CDI as AVP stores were depleted.

Gallegos (2018) reported CDI complicating bacterial meningitis; however, in contrast to our case, CDI was identified earlier in the clinical course and responded promptly to desmopressin without the refractory hemodynamic instability observed here, suggesting that the extent of hypothalamic damage in our patient was more severe. Systematically reviewed hypothalamic–pituitary complications in bacterial meningitis and concluded that neuroendocrine dysfunction is substantially underreported, likely because serial sodium and urine output monitoring is not yet standard in ICU protocols for meningitis—a gap directly illustrated by the diagnostic challenge encountered in managing this patient (Sodero et al., 2024).

A critical diagnostic challenge in this case was distinguishing CDI from SIADH and cerebral salt wasting (CSW), which can present with similar early sodium disturbances in CNS infection. In SIADH, euvolemic hyponatremia is accompanied by inappropriately concentrated urine; in CSW, hypovolemic hyponatremia with renal sodium wasting predominates; and in CDI, the defining feature is high urine output with low urine osmolality and rising serum sodium in the absence of adequate fluid replacement. In this case, the diagnosis was established by the combination of persistent polyuria exceeding 3 mL/kg/hour, rising serum sodium despite ongoing fluid therapy, and clinical response to desmopressin—a triad that excludes SIADH and CSW. The initial hyponatremia may have transiently obscured the diagnosis, as fluid therapy and early AVP excess masked the emerging CDI; this delayed recognition is consistent with the "masking effect" of concurrent SIADH described by (Singh et al., 2020).

From a hemodynamic standpoint, CDI in this patient created a self-perpetuating cycle of free water loss, intravascular volume depletion, and worsening cerebral hypoperfusion. Massive

polyuria exceeded the rate of fluid resuscitation, driving refractory hypotension that could not be corrected by conventional vasopressor therapy alone. Vasopressin and desmopressin administration produced transient hemodynamic improvement—a pharmacodynamic response that simultaneously served as diagnostic confirmation of AVP deficiency (Chekrouni et al., 2023). This interaction between CDI and shock physiology is underappreciated in meningitis management and distinguishes infection-related CDI from the more commonly recognized postoperative form, in which hemodynamic consequences are typically less severe owing to earlier recognition. SIADH is characterized by euvolemic hyponatremia with inappropriately concentrated urine, whereas CSW involves renal sodium loss and hypovolemia. Differentiating these entities is clinically challenging, particularly in critically ill patients receiving intravenous fluids and sedatives. In this case, the early presence of hyponatremia may have reflected stress-related AVP release or inflammatory stimulation of hypothalamic osmoreceptors.

Several limitations of this case must be acknowledged. Tuberculous meningitis was diagnosed clinically and based on CSF characteristics rather than microbiological confirmation, introducing diagnostic uncertainty. Formal water deprivation testing was not feasible given the patient's critical condition; CDI was therefore inferred from clinical criteria and the response to desmopressin rather than gold-standard testing. As a single case report, generalizability is inherently limited, and it is not possible to establish causality between meningitis and CDI with certainty. Such biphasic or triphasic patterns of AVP dysregulation have been described in hypothalamic injury, particularly after neurosurgical manipulation, where an initial phase of excess ADH secretion is followed by depletion and transient or permanent diabetes insipidus. Although these patterns are more commonly reported postoperatively, similar mechanisms may occur in progressive inflammatory destruction of hypothalamic neurons, as may have occurred in this patient.

Antibiotics should be administered immediately after blood cultures have been obtained. The Surviving Sepsis Campaign guidelines should be followed in patients with sepsis (Carter & McGill, 2022; Webb et al., 2016). Early recognition of circulatory shock and respiratory failure is crucial for the effective treatment of these complications.

The clinical trajectory in this case—progressive neurological deterioration, refractory shock, and death despite aggressive management—underscores that CDI in meningitis may signal advanced hypothalamic injury and portend a poor prognosis. Unlike postsurgical CDI, in which early intervention can be curative, infection-related CDI occurs against a backdrop of ongoing CNS destruction, rendering prognosis dependent on the extent of underlying neurological damage. This case supports integrating serial urine output and serum sodium monitoring into standard ICU protocols for severe meningitis, as well as early involvement of endocrinology in patients displaying electrolyte transitions suggestive of evolving neuroendocrine dysfunction.

CONCLUSION

Central diabetes insipidus is an uncommon but clinically significant complication of severe meningitis in critically ill patients, who are at particular risk of rapid physiological decline. This case illustrates the complex interaction between neuroendocrine dysfunction and severe central nervous system infection, as evidenced by fluctuating and, at times, abrupt disturbances in fluid and electrolyte homeostasis. The temporal pattern observed in this case—early hyponatremia evolving into marked polyuria, followed by rising serum sodium concentrations—highlights the dynamic dysregulation of arginine vasopressin during the course of neurocritical illness. These changes may represent an indirect manifestation of hypothalamic–pituitary involvement secondary to the inflammatory process, indicating that meticulous serial assessment, rather than reliance on isolated laboratory values, is essential. Misdiagnosis and inappropriate fluid infusion strategies may result in hemodynamic instability and an increased risk of secondary brain injury. One of the major clinical dilemmas, especially in critically ill patients with overlapping clinical features, is distinguishing central diabetes insipidus from other electrolyte disorders, such as syndrome of inappropriate antidiuretic hormone secretion (SIADH) or cerebral salt wasting (CSW).

Management of such patients requires careful intravascular fluid replacement, close

electrolyte monitoring and correction, and early treatment with desmopressin to prevent further complications. Additionally, this case exemplifies the importance of a multidisciplinary approach involving intensivists, neurologists, infectious disease specialists, and endocrinologists to ensure appropriate treatment. Routine neuroendocrine assessment could therefore enable earlier diagnosis of complications in patients with severe central nervous system infections, allowing for targeted therapeutic measures. Heightened awareness of central diabetes insipidus as a potential complication of meningitis may support timely diagnosis, improve clinical decision-making, and enhance patient outcomes. Further research is required to clarify the underlying mechanisms, incidence, and optimal management strategies for central diabetes insipidus in neurocritical care.

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AUTHOR CONTRIBUTION STATEMENT

Liliriawati Ananta Kahar: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Writing – original draft.

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