Case Report

Central diabetes insipidus secondary to neurocysticercosis in a pediatric patient: A rare clinical intersection

Abhinav Anil[1], Surabhi Das[2], Anil Kumar[3]

From [1]Consultant, Department of Emergency Medicine and Diabetology, Yashlok Hospital, Dhanbad, Jharkhand, India, [2]Senior Registrar, Department of Cardiothoracic and Vascular Surgery, Intensive Therapy Unit, The Mission Hospital, Durgapur, West Bengal, India, [3]Consultant, Department of General and Laparoscopic Surgery, Yashlok Hospital, Dhanbad, Jharkhand, India

ABSTRACT

Neurocysticercosis (NCC), caused by the larval stage of *Taenia solium*, is a leading cause of acquired epilepsy in endemic regions. While neurological symptoms predominate, endocrine manifestations such as central diabetes insipidus (DI) are exceedingly rare. We report a unique case of a 12-year-old male who presented to the emergency department with a 5-day history of profound polyuria and polydipsia. Evaluation confirmed central DI via a desmopressin challenge. Neuroimaging revealed a solitary ring-enhancing lesion in the left high parietal lobe, without direct hypothalamic-pituitary involvement. The patient responded rapidly to a regimen of albendazole, corticosteroids, and desmopressin. This case highlights the potential for NCC to cause distal inflammatory disruption of the pituitary stalk, necessitating clinical suspicion for atypical metabolic presentations in endemic areas.

Key words: Diabetes, Emergency department, Neurocysticercosis

Neurocysticercosis (NCC) remains one of the most significant public health challenges in developing nations. In the Indian subcontinent, the prevalence is estimated between 1% and 4% in specific endemic clusters [1]. Conventionally, the clinical spectrum of NCC is categorized by the location and stage of the cysts. While focal seizures are the hallmark of parenchymal NCC, extra-neural and systemic manifestations are often overlooked. Endocrine dysfunction in NCC is rare, documented in <1% of cases, and traditionally associated with mechanical effects of cysts in the basal cisterns or direct pituitary compression [2].

This case demonstrates that systemic inflammatory responses to the colloidal vesicular stage of the parasite can disrupt the neuroendocrine axis indirectly [3,4]. Identifying this in the emergency department (ED) is critical to avoid mismanaging what is essentially a reversible metabolic emergency.

CASE PRESENTATION

A 12-year-old male presented to the ED of Yashlok Hospital with a 5-day history of relentless thirst and urinary frequency, consuming 7–8 L of water daily, associated with muscle weakness and lethargy.

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Physical examination showed signs of moderate dehydration (dry mucosa, delayed capillary refill). Vital signs were as follows: Heart rate 102 bpm, blood pressure 102/68 mmHg, temperature 98.4°F. Neurological examination was normal. In the ED setting, the diagnostic approach for this 12-year-old patient followed a logical, high-priority sequence designed to first stabilize the metabolic crisis and then identify the underlying neurological trigger. The process can be broken down into four distinct clinical phases.

Phase 1: Clinical Recognition and Metabolic Profiling

The initial step was to differentiate between simple dehydration and a complex polyuric-polydipsic syndrome. Physical assessment showed findings of dry mucosa and delayed capillary refill, confirming significant volume depletion. Blood glucose and glycated hemoglobin (HbA1C) were immediately tested. Since these were normal (94 mg/dL and 5.2%), osmotic diuresis from diabetes mellitus (DM) was excluded. The combination of hypernatremia (149 mEq/L) and hyperosmolality (308 mOsm/kg), alongside a very low urine specific gravity (1.002) pointed specifically toward diabetes insipidus (DI). Table 1 shows the laboratory investigations of the patient. The normal HbA1C and random blood glucose results were critical in excluding

Correspondence to: Dr. Abhinav Anil, Yashlok Hospital, Baliapur Road, Govindour, Dhanbad – 828 109, Jharkhand, India. E-mail: drabhinav.anil@gmail.com

DM, the most common cause of polyuria in pediatric patients, as a differential diagnosis. The stable leukocyte count suggested the absence of acute bacterial sepsis. Liver function test results established a healthy baseline for hepatic function, ensuring the patient could safely tolerate the subsequent 28-day course of albendazole, which carries a risk of drug-induced hepatotoxicity. Baseline serum and urine biochemistry suggested the presence of hypernatremia and high serum osmolality, alongside an inappropriately dilute urine (low osmolality), which is the biochemical hallmark of DI. It indicates the kidneys' inability to conserve free water despite systemic dehydration.

Phase 2: Differentiation of DI Etiology

Once DI was suspected, the clinicians needed to determine if the issue was in the brain (Central) or the kidneys (Nephrogenic). This was achieved through the water deprivation test. In this test, the patient was denied fluids for 8 h. His serum sodium rose further to 154 mEq/L, and his urine failed to concentrate, proving his body could not produce or utilize antidiuretic hormone (ADH) (Vasopressin) endogenously. The "Desmopressin Challenge": Exogenous ADH (Desmopressin) was administered. The results showed that his urine osmolality jumped by 68% (from 118 to 252 mOsm/kg). This rapid response proved the kidneys were receptive to ADH, confirming that the "factory" (the pituitary/hypothalamus) was the site of failure (Table 2).

Phase 3: Neuro-anatomical Investigation

With a diagnosis of central DI, the team shifted from the "what" to the "why." Contrast-enhanced computed tomography (CECT) was performed to look for structural damage to the hypothalamic-pituitary axis, such as tumors (Craniopharyngioma) or infections (Tuberculosis). The imaging showed a normal sellar region but revealed a solitary ring-enhancing lesion in the left parietal lobe. The presence of a "scolex" (the head of the parasite) within the lesion confirmed the diagnosis of NCC (Figs. 1 and 2).

Phase 4: Therapeutic Challenge (The Final Proof)

The final "diagnostic" step in an ED is often the response to treatment. High-dose dexamethasone was started. The rapid normalization of serum sodium (from 154 to 140 mEq/L) within 48 h following steroid administration supported the hypothesis that the DI was caused by inflammatory stunning rather than permanent tissue destruction. By following this sequence, the medical team was able to bridge the gap between a simple symptom (thirst) and a complex parasitic infection, ensuring the patient received targeted antiparasitic and hormonal therapy (Table 3).

DISCUSSION

The clinical presentation of polyuria and polydipsia

Table 1: Laboratory investigations of the patient

Parameter	Investigation	Result	Reference range	Status
Complete hemogram and metabolic screening (Day 1)	Hemoglobin	13.2 g/dL	12.0–15.5 g/dL	Normal
	Total leukocyte count	9,800/mm^3	4,000–11,000/mm^3	Normal
	Random blood glucose	94 mg/dL	70–140 mg/dL	Normal
	HbA1C	5.2%	<5.7%	Normal
Liver function tests- pre-treatment baseline	SGOT (AST)	32 U/L	<40 U/L	Normal
	SGPT (ALT)	28 U/L	<41 U/L	Normal
	Total bilirubin	0.8 mg/dL	0.2–1.2 mg/dL	Normal
Baseline serum and urine biochemistry (Day 1, 10:30 am	Serum sodium (Na+)	149 mEq/L	135–145 mEq/L	High
	Serum osmolality	308 mOsm/kg	275–295 mOsm/kg	High
	Urine osmolality	105 mOsm/kg	300–900 mOsm/kg	Low

HbA1C: Glycated hemoglobin, SGOT: Serum glutamic oxaloacetic transaminase, AST: Aspartate aminotransferase, SGPT: Serum glutamic pyruvic transaminase, ALT: Alanine aminotransferase

Table 2: Water deprivation and desmopressin challenge results (Day 1)

Phase of test	Time	Serum sodium (mEq/L)	Serum osmolality (mOsm/kg)	Urine osmolality (mOsm/kg)
Baseline (Admit)	10:30 am	149	308	105
End of deprivation	06:30 pm	154	318	118
Post-desmopressin	08:30 pm	145	295	252

Interpretation: The failure of the urine to concentrate during deprivation ruled out primary polydipsia. The dramatic>50% rise in urine osmolality (from 118 to 252 mOsm/kg) following desmopressin confirmed a central deficiency of antidiuretic hormone, rather than a renal resistance (nephrogenic diabetes insipidus)

Table 3: Serial monitoring and treatment response (Days 2–3)

Day	Status	Serum sodium (mEq/L)	Serum osmolality (mOsm/kg)	Urine specific gravity
Day 2	Post-steroid start	144	292	1.012
Day 3	Pre-discharge	140	288	1.018

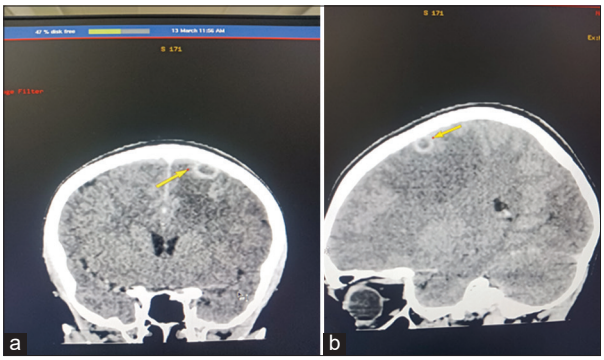

Figure 1: (a) Contrast-enhanced computed tomography (CECT) Brain - axial and coronal views demonstrate a solitary, ring-enhancing lesion located in the left high parietal lobe (yellow arrows). There is evidence of significant perilesional hypodensity, consistent with vasogenic edema associated with neurocysticercosis (Source: A.P. Diagnostic and Imaging Centre, Dhanbad, Jharkhand); The yellow arrow points to a small, well-defined, circular, hyperdense lesion located within the brain parenchyma, close to the inner table of the skull. (b) CECT brain montage comprehensive view of the cranial sections showing the anatomical distance between the parietal lesion and the pituitary region (Source: A.P. Diagnostic and Imaging Centre, Dhanbad, Jharkhand)

in a pediatric patient mandates a broad and systematic differential diagnosis. In this case, several primary conditions were meticulously excluded, such as DM: Effectively ruled out via normal glucose and HbA1C (Table 1). Primary polydipsia: Excluded by the patient's inability to concentrate urine during water deprivation (Table 2). Nephrogenic DI: Ruled out by the positive response to exogenous desmopressin [5,6]. Tuberculoma: In endemic regions, central nervous system tuberculosis is the primary mimic. The radiological presence of a scolex and the rapid response to albendazole favored NCC [7,8]. Craniopharyngioma: Ruled out by the normal appearance of the sella on CECT Brain [9,10].

The most intriguing aspect of this case is the anatomical mismatch between the lesion (parietal lobe) and the clinical manifestation (Central DI). We hypothesize that this occurred through a mechanism of distal neuro-inflammatory crosstalk. During the colloidal vesicular stage of NCC, the blood-brain barrier is compromised, leading to the local release of

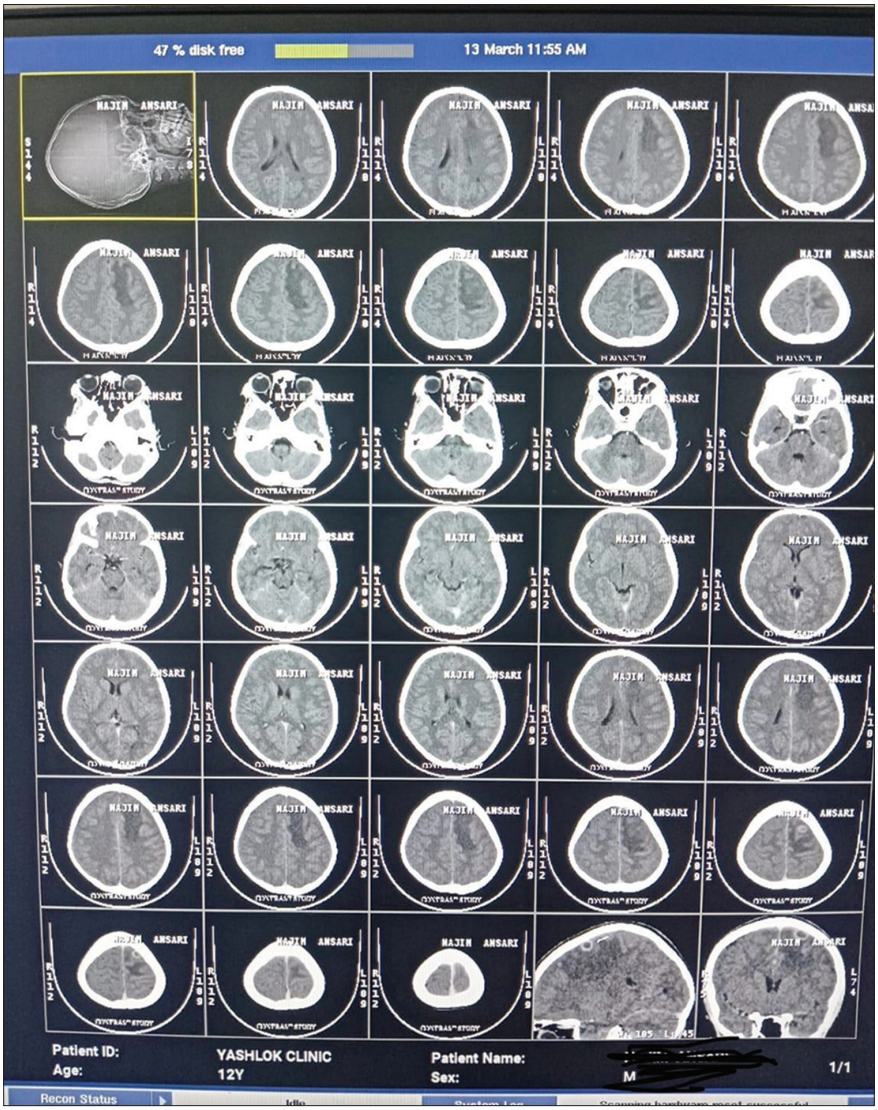

Figure 2: Contrast-enhanced computed tomography brain report confirms the presence of a 10 mm × 9mm cortical ring-enhancing lesion in the left high parietal lobe. The impression specifically notes the absence of calcification and the presence of significant vasogenic edema, identifying the lesion as highly suggestive of a granulomatous infection, specifically Neurocysticercosis in the colloidal vesicular stage. This documentation provides the definitive radiological basis for the patient's diagnosis and underscores the absence of anatomical abnormalities in the basal cisterns or ventricles, further supporting the theory of a distal inflammatory trigger for the central diabetes insipidus. (Source: A.P. Diagnostic and Imaging Centre, Dhanbad, Jharkhand)

pro-inflammatory cytokines such as tumor necrosis factor-α, interleukin (IL)-1, and IL-6 [11-13]. These molecules can enter the cerebrospinal fluid and reach the hypothalamus and pituitary stalk. The neurohypophysis, located outside the traditional blood-brain barrier, is uniquely vulnerable to circulating inflammatory mediators [14]. This "stunning" of the magnocellular neurons leads to a transient suppression of arginine vasopressin release. This theory is supported by the patient's rapid recovery following corticosteroid therapy, suggesting that the dysfunction was inflammatory rather than structural [4,9].

Pediatric NCC is often viewed through the lens of neurology, yet this case demonstrates its ability to mimic a metabolic emergency. The "protean" nature of NCC refers to its various forms; however, Central DI remains rare. Emergency physicians must maintain clinical suspicion in endemic regions. If a child presents with polyuria, even without seizures, intracranial imaging should be considered if the metabolic profile suggests a central origin [10,15].

CONCLUSION

This case highlights the potential for NCC to cause distal inflammatory disruption of the pituitary stalk, necessitating clinical suspicion for atypical metabolic presentations in endemic areas.

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AUTHORS' CONTRIBUTIONS

Dr. Abhinav Anil: Conceptualized the study, managed the clinical treatment of the patient, and drafted the primary manuscript. Dr. Surabhi Das: Conducted the literature review, assisted in manuscript editing, and formatted technical data.

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