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Metabolic Encephalopathy Due to Severe Hypernatraemia and Uraemia as the Presenting Manifestation of Dengue Fever in a Psychiatrically Vulnerable Patient: A Case Report

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Abstract: ***Background: Dengue fever, caused by dengue virus serotypes 1-4 (DENV 1-4) and transmitted by Aedes aegypti, is endemic across tropical and subtropical regions including the Indian subcontinent. While the haemorrhagic and plasma leakage phenotypes are well characterised, neurological complications remain under-recognised. The concurrent occurrence of severe hypernatraemia and uraemia as dual metabolic drivers of encephalopathy in dengue has been exceptionally rarely described in the literature. Case Presentation: We report a 37-year-old divorced female with a one-month history of depressive illness and markedly diminished oral intake who presented with sudden-onset altered sensorium. Investigations revealed severe hypernatraemia (serum sodium 170 mEq/L), uraemia (blood urea 246 mg/dL; serum creatinine 4.2 mg/dL), haemoconcentration (haematocrit 49.9%), leukocytosis (18,750 cells/mm³), transaminitis, and positive dengue IgM serology. The patient was anuric on day 1. Stroke protocol neuroimaging was normal. The calculated free water deficit was 6.4 litres. Gradual free water replacement via nasogastric tube with concomitant intravenous 5% dextrose, targeting a correction rate not exceeding 10-12 mEq/L per 24 hours, resulted in complete neurological recovery by day 5, with normalisation of serum sodium (143 mEq/L), renal parameters, and haematological indices by day 6. Conclusion: This case underscores that dengue fever may present atypically as metabolic encephalopathy driven by the dual insult of severe hypernatraemia and uraemia, particularly in individuals with pre-existing psychiatric illness and social vulnerability. Early metabolic evaluation and judicious correction of these derangements are life-saving. Clinicians in endemic regions should include dengue in the differential diagnosis of unexplained metabolic encephalopathy.***

Keywords: Dengue encephalopathy; Hypernatraemia; Uraemia; Altered sensorium; Metabolic encephalopathy; Acute kidney injury; Dengue fever; Psychiatric vulnerability; Free water deficit

1. Introduction

Dengue fever is one of the most rapidly disseminating arboviral infections globally, with an estimated 390 million infections occurring annually, of which approximately 96 million manifest clinically. **[9]** The disease is caused by four antigenically distinct serotypes of dengue virus (DENV 1-4), belonging to the family *Flaviviridae*, and is transmitted predominantly by the female *Aedes aegypti* mosquito. In India, dengue is hyperendemic, with epidemic peaks coinciding with the monsoon season. The clinical spectrum is broad, ranging from an undifferentiated febrile illness to severe dengue characterised by plasma leakage, haemorrhage, and organ dysfunction, as defined by the World Health Organization (WHO) 2009 revised classification. **[10]** Neurological complications of dengue, encompassing direct neuroinvasion (dengue encephalitis), immune-mediated mechanisms (acute disseminated encephalomyelitis, Guillain-Barré syndrome), and metabolic encephalopathy, are reported in 0.5-6.2% of hospitalised dengue cases. **[6]** However, severe hypernatraemia as the primary metabolic derangement driving encephalopathy in dengue has been documented only in isolated reports. Hypernatraemia is defined as a serum sodium concentration exceeding 145 mEq/L; values above 160 mEq/L constitute a medical emergency associated with significant neurological morbidity and mortality, particularly when onset is acute. **[2, 7]**

Psychiatric illness, particularly depressive disorder with anhedonia and reduced oral intake, impairs the physiological thirst response and predisposes to chronic dehydration. In the context of a febrile illness such as dengue, this pre-existing vulnerability may precipitate or dramatically accelerate the development of severe hypernatraemia.

We report a diagnostically challenging case of severe hypernatraemic encephalopathy as the presenting manifestation of dengue fever in a young woman with untreated depressive illness, and discuss the pathophysiological interplay, diagnostic approach, and therapeutic considerations relevant to this rare but instructive clinical scenario. To our knowledge, this constellation-dengue-associated severe hypernatraemia complicated by acute kidney injury in a psychiatrically vulnerable patient- has not been previously reported in indexed literature.

2. Case Report

Patient Demographics and Presenting Complaint

A 37-year-old female, a housewife, divorced and living alone without a social support network, presented to the Department of Internal Medicine with acute-onset altered sensorium of less than 24 hours' duration. Her family reported that she had been evaluated at a psychiatric outpatient clinic for a one-month history of low mood, anhedonia, social withdrawal, and markedly diminished oral intake; however, no

pharmacological therapy had been initiated prior to this admission.

Clinical Examination

On general examination, the patient was obtunded with a depressed level of consciousness. The mucous membranes and tongue were dry, and skin turgor was reduced, consistent with significant dehydration. There were no signs of meningeal irritation (no neck stiffness, negative Kernig's and Brudzinski's signs). No petechiae, purpura, or rash were evident. Fundoscopy was unremarkable.

Vital parameters revealed tachycardia (heart rate 110 beats per minute), hypotension (blood pressure 100/70 mmHg), tachypnoea (respiratory rate 24 breaths per minute), and peripheral oxygen saturation of 98% on room air. The patient was afebrile at the time of assessment, though a febrile prodrome was reported by the family. Systemic examination of the cardiovascular, respiratory, and abdominal systems was unremarkable. Neurological examination, insofar as it could be performed in the context of altered sensorium, did not reveal focal deficits.

Investigations

Laboratory investigations obtained at admission are detailed in Table 1. Salient findings included severe hypernatraemia (serum sodium 170 mEq/L), hyperchloraemia (chloride 130 mEq/L), acute kidney injury with markedly elevated serum creatinine (4.2 mg/dL) and blood urea (246 mg/dL), haemoconcentration (haematocrit 49%), leukocytosis (total leucocyte count 18,750 cells/mm³), microcytosis (mean corpuscular volume 76 fL), and transaminitis (alanine aminotransferase 128 U/L; aspartate aminotransferase 55 U/L) with mildly elevated serum bilirubin. Serum potassium was within normal limits at 3.6 mEq/L. Dengue NS1 antigen and IgM enzyme-linked immunosorbent assay (ELISA) were positive, consistent with acute primary dengue infection. Stroke protocol computed tomography (CT) and magnetic resonance imaging (MRI) of the brain were unremarkable, excluding haemorrhagic or ischaemic stroke, cerebral venous sinus thrombosis, and structural lesions. Urinalysis was not possible on day 1 as the patient was anuric. Cerebrospinal fluid (CSF) examination and dengue polymerase chain reaction (PCR) were not performed, as the encephalopathy resolved fully with correction of hypernatraemia, obviating the clinical need.

Table 1: Laboratory Investigations at Admission

Parameter	Value at Admission
COMPLETE BLOOD COUNT	
Haemoglobin (g/dL)	14.9
Total Leucocyte Count (/mm³)	18,750 ↑
Platelet Count (/mm³)	1,95,000
Haematocrit (%)	49 ↑
Mean Corpuscular Volume (fL)	76 ↓
RENAL FUNCTION TESTS	
Blood Urea (mg/dL)	246 ↑
Serum Creatinine (mg/dL)	4.2 ↑
Serum Sodium — Na^+ (mEq/L)	170 ↑↑
Serum Potassium — K^+ (mEq/L)	3.6 (Normal)
Serum Chloride — Cl^- (mEq/L)	130 ↑
LIVER FUNCTION TESTS	
Total Bilirubin (mg/dL)	1.8 ↑
Direct Bilirubin (mg/dL)	1.1 ↑
AST / SGOT (U/L)	55 ↑
ALT / SGPT (U/L)	128 ↑
Alkaline Phosphatase (U/L)	104
SEROLOGY AND IMAGING	
Dengue NS1 Antigen	Positive
Dengue IgM (ELISA)	Positive
Stroke Protocol CT / MRI Brain	No acute intracranial pathology
Urine Output (Day 1)	Anuric

Diagnosis

The following diagnoses were established: (1) **Severe hypernatraemic encephalopathy** (serum sodium 170 mEq/L) secondary to prolonged poor oral intake in the context of depressive illness, compounded by dengue-related fever, insensible losses, and impaired osmoregulation; (2) **Dengue fever** (WHO 2009 classification: dengue with warning signs- altered sensorium, organ impairment); (3) **Dengue-associated acute kidney injury** (DAKI), likely pre-renal with a possible intrinsic component; and (4) **Dengue hepatitis**.

The free water deficit was calculated using the standard formula as described in Harrison's Principles of Internal Medicine: **[2]**

Free Water Deficit (litres) = 0.5 × lean body weight (kg) × [(serum Na / 140) − 1]

Assuming an estimated lean body weight of 50 kg: Free Water Deficit = 0.5 × 50 × [(170/140) − 1] = 25 × 0.214 ≈ **6.4 litres**.

Management

A nasogastric (Ryle's) tube was inserted for enteral access. Free water was administered orally via the nasogastric tube with concurrent intravenous 5% dextrose in water (D5W). Sodium correction was titrated to achieve a reduction of no more than **10-12 mEq/L per 24 hours**, in accordance with current guidelines for chronic hypernatraemia (duration >48 hours), to prevent the sequelae of cerebral oedema from overly rapid correction. **[2,7]** Serial serum electrolytes and renal function tests were performed every 12-24 hours. Intravenous antipyretics and supportive hydration were provided. No specific antiviral therapy was administered.

Given the absence of haemorrhagic manifestations and normal platelet counts, corticosteroids and platelet transfusions were withheld. The patient was monitored in a high-dependency unit during the acute phase.

Clinical Progress and Outcome

The clinical course demonstrated a clear and sequential neurological improvement directly paralleling the correction of serum sodium, as documented in Table 2. By day 5, the patient had achieved complete neurological recovery with full orientation to time, place, and person. Renal function showed progressive improvement, and urine output was restored from day 2 onwards. The patient was subsequently referred to the psychiatry department for definitive management of her depressive illness and for social support coordination prior to discharge.

Table 2: Serial Haematological, Renal, and Electrolyte Parameters During Hospitalisation

Day	Na+ (mEq/L)	Urea (mg/dL)	Creat (mg/dL)	Hb (g/dL)	TLC (/mm3)	Plts (/mm3)	HCT (%)	Clinical Status
Day 1 (Admission)	**170**	**246**	**4.2**	14.9	**18,750**	1,95,000	49.9	Obtunded; anuric; initiated free water replacement via NGT and IV D5W
Day 2	**164**	**167**	**2**	12.1	**12,670**	1,50,000	41.5	Partial improvement; urine output restored; irritability persisting
Day 3	**159**	**109**	**1.5**	**10.9**	10,170	**1,20,000**	**35.5**	Obeying commands; irritability resolved; not yet verbalising
Day 4	**151**	38	0.8	**9.8**	8,360	**1,00,000**	**32.5**	Intermittent, partially relevant verbal responses elicited
Day 5	**149**	24	0.7	**10.6**	10,580	**1,10,000**	**33.8**	Fully conscious; oriented to time, place, and person; complete recovery
Day 6	143	15	0.5	**10.5**	7,960	1,58,000	**32.8**	Stable; ambulant; serum sodium and renal parameters normalised

Values in red = outside normal reference range; values in green = within normal limits. Na+ = serum sodium; Creat = creatinine; Hb = haemoglobin; TLC = total leucocyte count; Plts = platelets; HCT = haematocrit; NGT = nasogastric tube; D5W = 5% dextrose in water.

3. Discussion

This case presents an instructive convergence of dengue fever, dual metabolic encephalopathy (hypernatraemia and uraemia), and untreated psychiatric illness. The serial monitoring data in Table 2 provide compelling objective evidence for the mechanistic link between metabolic correction and neurological recovery, and reveal that initial haemoconcentration substantially amplified the apparent severity of several haematological parameters at admission.

Dual Metabolic Mechanism: Hypernatraemia and Uraemia

The encephalopathy was driven by two concurrent and synergistic metabolic derangements. Hypernatraemia results from a net deficit of water relative to sodium, most commonly due to inadequate intake, increased insensible losses, or impaired renal water conservation. **[2]** Hypovolaemic hypernatraemia was precipitated by a one-month history of poor oral intake secondary to depressive illness, substantially increased insensible losses from dengue-associated hyperthermia, **[1,3]** and dengue virus-mediated hypothalamic dysfunction with impaired osmoregulation, attenuating the physiological thirst response and appropriate antidiuretic hormone (ADH) secretion. **[3]** The resulting 6.4-litre free water deficit corresponds to approximately 13% of total body water loss- sufficient to produce severe neurological depression. Concurrently, uraemia arising from DAKI produced a second, independent encephalopathic insult through neurotoxic accumulation of nitrogenous waste products, causing synaptic dysfunction and cerebral oedema. **[5]** The admission blood urea of 246 mg/dL with anuria on day 1 represents a degree of uraemia amply sufficient to contribute to the obtunded state. Importantly, severe hypernatraemia itself promotes intrarenal vasoconstriction and reduces glomerular filtration rate, creating a bidirectional relationship that exacerbates both derangements simultaneously.

Serial Parameter Trends: Objective Evidence of Recovery

The serial monitoring data provide several important clinical insights. First, the progressive decline in haematocrit from 49.9% at admission to 32.8% by day 6 confirms significant haemoconcentration at baseline. This implies that the admission leukocytosis (18,750 cells/mm³) and haemoglobin (14.9 g/dL) were at least partially artifactual, reflecting haemoconcentration rather than true leucocyte proliferation or polycythaemia. Post-rehydration leucocyte normalisation (7,960 cells/mm³ by day 6) and fall in haemoglobin to 10.5 g/dL support this interpretation.

Second, the renal recovery trajectory- urea from 246 mg/dL to 15 mg/dL and creatinine from 4.2 to 0.5 mg/dL over six days- is consistent with predominantly pre-renal AKI responding to volume repletion. Third, the platelet nadir at day 4 (1,00,000/mm³) with spontaneous recovery to 1,58,000/mm³ by day 6 is characteristic of the dengue thrombocytopenic dip, further corroborating the aetiology. Fourth, the stepwise improvement in consciousness from day 3 through day 5 precisely mirrors the rate of sodium correction, providing strong mechanistic evidence that hypernatraemia was the dominant encephalopathic driver, with uraemia contributing synergistically.

Dengue-Associated Acute Kidney Injury (DAKI)

AKI complicates 0.9-5.7% of hospitalised dengue cases. **[5,8]** The pathogenesis of DAKI is multifactorial, including hypovolaemia, direct cytopathic viral tubular injury, immune complex deposition, and rhabdomyolysis. **[5]** In this patient, the prompt recovery of renal function with rehydration and the absence of myoglobinuria favour a predominantly pre-renal mechanism with possible mild tubular injury.

Psychiatric Illness and Social Vulnerability

The patient's untreated depressive illness occupies a central pathogenic role. As described in Harrison's, the neurovegetative features of depression- including hypophagia, adipsia, psychomotor retardation, and social withdrawal- critically impaired her capacity to maintain adequate fluid intake over the preceding month. **[4]** Living alone and being recently divorced eliminated compensatory social mechanisms that might otherwise have prompted earlier medical attention. This case exemplifies how social determinants of health interact directly with acute medical illness to amplify severity and delay presentation.

Therapeutic Considerations

As detailed in Harrison's, sodium correction should not exceed 10-12 mEq/L per 24 hours in chronic hypernatraemia to avoid cerebral oedema. **[2]** The total correction from 170 mEq/L to 143 mEq/L over six days (27 mEq/L over 144 hours) represents an average rate of approximately 4.5 mEq/L per 24 hours — well within the safe threshold- resulting in excellent neurological recovery without rebound complications. Simultaneous renal recovery with rehydration

resolved the uraemic component, thereby eliminating both metabolic drivers of encephalopathy in a coordinated fashion.

4. Conclusion

We present a rare and instructive case of severe metabolic encephalopathy attributable to the synergistic dual insult of hypernatraemia and uraemia as the predominant manifestation of dengue fever in a psychiatrically vulnerable patient. The comprehensive serial monitoring data provide objective, day-by-day documentation of parallel metabolic correction and neurological recovery. Three key observations merit emphasis: first, dengue fever can present atypically as severe metabolic encephalopathy without classical haemorrhagic features; second, hypernatraemia and uraemia may act synergistically as encephalopathic insults and must both be identified and corrected; and third, psychiatric comorbidity and social isolation are clinically significant risk factors that amplify metabolic derangements in acute infectious illness. Serum electrolytes and renal function tests must be obtained as a priority in any dengue patient with altered sensorium.

Key Learning Points

1) Dengue fever may present atypically as severe metabolic encephalopathy without haemorrhagic manifestations — serum electrolytes and renal function tests must be obtained urgently in all dengue patients with altered sensorium.
2) Hypernatraemia and uraemia can act as synergistic encephalopathic insults; identifying and correcting both simultaneously is essential to complete neurological recovery.
3) Initial haemoconcentration may artefactually elevate haemoglobin and leucocyte counts; post-rehydration haematological reassessment is mandatory for accurate interpretation.
4) Psychiatric illness- particularly depressive disorder with hypophagia and adipsia — and social isolation are independent risk factors that amplify metabolic derangements during acute febrile illness.
5) Gradual correction of hypernatraemia (≤10-12 mEq/L per 24 hours) with serial monitoring of all metabolic parameters guides safe and effective therapy.

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