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Case Report

Central diabetes insipidus following minor head trauma in a child with normal neuroimaging

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ABSTRACT

Central diabetes insipidus (CDI) is an uncommon endocrine consequence of head injury in children and is typically associated with moderate to severe trauma. Development of CDI after trivial head injury, particularly in the presence of normal neuroimaging, is rare and may be easily overlooked. We report a 4-year-old child who presented with polyuria and polydipsia following a minor head injury, with laboratory findings consistent with central diabetes insipidus and normal magnetic resonance imaging of the brain. Prompt initiation of desmopressin resulted in rapid clinical improvement. This case emphasizes the need for clinical vigilance for CDI in children presenting with unexplained polyuria even after apparently insignificant head trauma.

Keywords: Central diabetes insipidus, Minor head injury, Polyuria, Children, Normal magnetic resonance imaging

INTRODUCTION

Diabetes insipidus is characterized by impaired urinary concentrating ability, leading to excessive excretion of dilute urine and compensatory polydipsia.[1] Central diabetes insipidus results from deficient synthesis or secretion of arginine vasopressin due to damage involving the hypothalamic–posterior pituitary axis.[2] In pediatric practice, trauma-related central diabetes insipidus (CDI) is usually seen following severe head injury, neurosurgical interventions, or intracranial hemorrhage.[3,4]

Occurrence of CDI after minor head trauma is distinctly uncommon and may remain underdiagnosed, especially when neuroimaging is normal.[4,5] Reporting such cases is important to increase awareness among clinicians and to highlight that even mild trauma can disrupt vasopressin regulation.

CASE REPORT

A 4-year-old previously healthy child was brought with complaints of excessive thirst and passage of large volumes of urine for 10 days. The symptoms were progressive and interfered with daily activities and sleep. There was a history of minor head injury 3 months earlier due to a fall from standing height. The child cried immediately after the fall and had no loss of consciousness, vomiting, seizures, or abnormal behavior. No medical attention was sought at that time.

On examination, the child was alert, active, and hemodynamically stable. There were no signs of dehydration and child drinks water eagerly 4-5 l/day. Growth parameters were appropriate for age. Systemic and neurological examinations were within normal limits (Table 1).

Investigations

The child had persistently increased urine output exceeding 5 ml/kg/hour. Urinalysis revealed dilute urine with a low specific gravity. Serum investigations showed elevated serum sodium and increased serum osmolality, with inappropriately low urine osmolality. Blood glucose levels were normal, excluding osmotic diuresis.

Magnetic resonance imaging (MRI) of the brain with pituitary protocol did not reveal any structural abnormality. The posterior pituitary bright spot was not visualized.

Based on clinical presentation and biochemical findings, a diagnosis of central diabetes insipidus was established.

Table 1: Timeline of clinical events.

Day / period	Event
~3 months prior to admission	History of minor head trauma (fall from standing height); no loss of consciousness or immediate complications
10 days prior to admission	Onset of polyuria and polydipsia; progressive increase in urine output
Day of admission	Admitted with excessive thirst and urination; vitals stable; systemic examination normal
Hospital stays	Fluid balance monitoring showed polyuria; urine specific gravity persistently low; water deprivation test suggestive of diabetes insipidus
During evaluation	MRI brain with pituitary protocol normal; posterior pituitary bright spot not visualized
Diagnostic intervention	Desmopressin nasal spray challenge administered
Post-desmopressin	Marked reduction in urine output; increase in urine specific gravity confirming central diabetes insipidus
Discharge	Clinically stable; discharged on desmopressin nasal spray (10mcg intranasal once daily) with follow-up advice with monitoring of serum sodium and urine output.

Treatment and follow-up

Oral desmopressin was initiated at a low dose and titrated according to clinical response. A significant reduction in urine output and improvement in polydipsia was observed within 48 hours. Serum sodium levels normalized subsequently. The child remains on regular follow-up and continues to do well on maintenance therapy.

DISCUSSION

Diabetes insipidus is a disorder of water balance characterized by inability to concentrate urine, resulting in excretion of large volume of dilute urine and excessive thirst. Central Diabetes Insipidus occurs due to deficient synthesis or release of vasopressin. Post-traumatic central diabetes insipidus is attributed to injury to the hypothalamic–neurohypophyseal system, resulting in impaired vasopressin release.[4] While commonly reported after severe traumatic brain injury, its occurrence following minor trauma is rare.[5] Microscopic axonal injury or transient vascular compromise, undetectable on conventional imaging, may explain CDI in such cases.[6]

The absence of the posterior pituitary bright spot-on magnetic resonance imaging (MRI), although not specific, supports the diagnosis of CDI.[2,7] Normal neuroimaging does not exclude hypothalamic–pituitary dysfunction, and persistent polyuria and polydipsia following head injury warrant biochemical evaluation for diabetes insipidus irrespective of injury severity.[3,4]

A clear biochemical response to desmopressin, with normalization of serum sodium and concentration of urine, confirms central diabetes insipidus and helps differentiate it from nephrogenic causes (Table 2).[2] Early diagnosis and treatment with desmopressin are associated with excellent outcomes and prevent complications such as dehydration, hypernatremia, and growth impairment.[1]

Table 2: Serum electrolyte and osmolality response to desmopressin.

Parameter	Before desmopressin	After desmopressin
Urine output (ml/kg/hour)	>5	1-2
Urine sodium (mmol/l)	8	42
Serum sodium (mEq/l)	Elevated (154)	Normal (140)
Serum osmolality (mOsm/kg)	Increase (304)	Normal (284)
Urine osmolality (mOsm/kg)	Low (168)	Increased (560)
Urine specific gravity	<1.005	≥1.010

Post-traumatic central diabetes insipidus is attributed to injury to the hypothalamic–neurohypophyseal system, resulting in impaired vasopressin release. While commonly reported after severe traumatic brain injury, its occurrence following minor trauma is rare. Microscopic axonal injury or transient vascular compromise, undetectable on conventional imaging, may explain CDI in such cases.

The absence of the posterior pituitary bright spot-on MRI, although not specific, supports the diagnosis of CDI. This case highlights that normal neuroimaging does not exclude hypothalamic–pituitary dysfunction. Persistent polyuria

and polydipsia following head injury, regardless of severity, warrant evaluation for diabetes insipidus.

Early diagnosis and appropriate treatment with desmopressin are associated with excellent outcomes and prevent complications such as dehydration, hypernatremia, and growth impairment.

CONCLUSION

Central diabetes insipidus can rarely develop after minor head trauma in children, even in the absence of radiological abnormalities. Pediatricians should maintain a high index of suspicion for CDI in children presenting with unexplained polyuria and polydipsia after head injury. Early recognition and treatment are crucial for optimal outcomes.

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