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Urine Output Control in Central Diabetes Insipidus with Severe Hypernatremia After Traumatic Brain Injury: A Case Report

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ABSTRACT

Introduction: Central diabetes insipidus (CDI) is a secondary injury complication of traumatic brain injury (TBI). Neurohypophysis injury causes posterior pituitary insufficiency to secrete arginine vasopressin (AVP) in hyperosmolality condition, lead to hypovolemic hypernatremia thus increasing mortality and morbidity. Prevalence of hypernatremia in TBI patient is more than 35% with possible causes of dehydration and hypovolemia and mortality rate 86.8%.

Case Presentation: We reported a case of a 20-year-old man with CDI and severe hypernatremia post TBI. He underwent ventriculoperitoneal shunt surgery and admitted to intensive care unit post operative. Patient showed polyuria with urine output 3.2 ml/kg/hour with a sodium level of 190 mmol/L. Hypernatremia correction with KA-EN 3B intravenously and oral fluid intake was given to replace free water deficit. Oral desmopressin was given to compensate AVP deficiency to reducing ongoing fluid loss. A good response was achieved on the second day of treatment, indicated by a decrease in urine output to 1.4 mL/kg/hour and decrease in sodium levels with target 10-12 mEq/L/day. No side effects of desmopressin found in this patient. Our case shows that close monitoring and appropriate therapy result in good outcomes in CDI patients with severe hypernatremia post TBI.

Conclusion: Patients with CDI and severe hypernatremia after TBI responded well after receiving desmopressin therapy to treat AVP deficiency, as well as fluid replacement with KA-EN 3B and oral intake according to free water deficit.

Keywords: traumatic brain injury, central diabetes insipidus, severe hypernatremia, polyuria

INTRODUCTION

Traumatic brain injury (TBI) is a leading cause of death and lifelong disability, accounting for 9% of deaths worldwide. Central diabetes insipidus is one of the complications after TBI with 15-28% incidence. Neurohypophysis injury causes impaired secretion of arginine vasopressin (AVP) which triggers polyuria, polydipsia, and dehydration.

Gempeller et al. stated that CDI was found in 14.82% of TBI patients, with 32.18% of patients experiencing hypernatremia. Study by Wu et al. mentioned the prevalence of hypernatremia in patients with TBI is more than 35% with the possible cause is

insufficient water intake or excessive water loss resulting in dehydration and hypovolemia with a mortality rate in severe hypernatremia (>160 mEq/L) reaching 86.8%.[1-3]

The goal of diabetes insipidus therapy is to correct the water deficit and replace the AVP deficit.[4] In this case, we report a patient with central diabetes insipidus and severe hypernatremia after TBI who responded well to intravenous KA-EN 3B fluid administration, oral fluid intake, and oral desmopressin.

CASE PRESENTATION

A 20-year-old man with decreased consciousness and polyuria that appeared 2 weeks after the accident. Pre-surgical physical examination revealed GCS E3V3M5, BP 128/87 mmHg, pulse 50 beats per minute, respiratory rate 16 beats per minute, temperature 36.6°C, and oxygen saturation 98% room air. Pupils are isochoric, pupillary reflexes are positive. Urine output was 3.7 mL/kg/hour. Extremity motor strength 55555/33333/55555/33333. Head MRI showed communicating hydrocephalus with surrounding trans ependymal edema (Figure 1). The patient underwent VP shunt surgery and was admitted to the intensive care unit postoperatively.

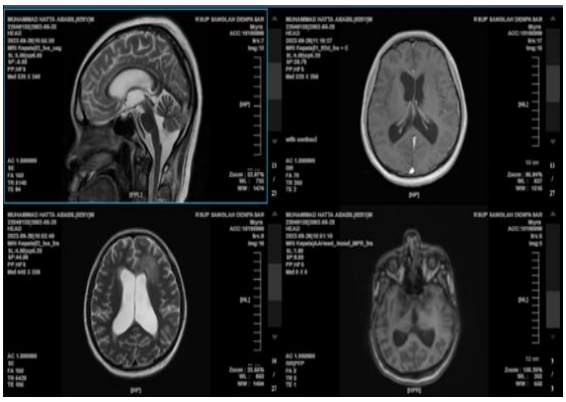


Figure 1. MRI Head Axial

Postoperative examination the patient obtained urine output 3.2 mL/kg/hour, plasma osmolality 397 mOsm/kg, and serum osmolality 611 mOsm/kg. The patient had a free water deficit of 10.3 liters. KA-EN 3B 148 mL/hour was administered intravenously and per oral fluids 50 mL/hour. Desmopressin 0.2 mg every 8 hours orally was given to treat AVP deficiency. The patient was monitored and found to have decreased urine output and electrolytes. On the fifth day of treatment, the patient was transferred to the intermediate ward and therapy was continued.

DISCUSSION

Traumatic brain injury (TBI) is a significant public clinical problem associated with high mortality and acute and chronic morbidity. Central diabetes insipidus (CDI) is one of the complications of TBI. Damage to 80-90% of hypothalamic vasopressin neurons can impair the secretory function of arginine vasopressin (AVP) from the posterior pituitary gland triggering dehydration and hypernatremia.[4]

Diabetes insipidus is one of the main causes of polyuria polydipsia syndrome and is characterized by high hypotonic urine production, more than 50 mL/kg/24 hours, accompanied by polydipsia of more than 3 L/day.[5] Hannon's prospective study, DI was so common as a pre-fatal event that 80% of TBI patients were at risk for central DI.[6]

Our patient had consciousness and cognitive impairment that affected thirst. Water deprivation test was not accurate in our patient, so we used Seckl and Dunger criteria for the diagnosis of diabetes insipidus. Measurement of plasma AVP or plasma copeptin was not recommended, as the risk of DI has been shown to be closely related to the severity of trauma (as measured by the Glasgow Coma Scale) and the presence of cerebral edema on brain radiology.[6] Our patient presented with symptoms of polyuria with urine production up to 5.7 liters/24 hours. Laboratory examination showed severe hypernatremia with serum sodium of 190 mmol/L, hypotonic urine with urine specific gravity is 1002, and serum osmolality of 375 mOsm/kg confirming the diagnosis of CDI after TBI.

The principles of diabetes insipidus management are replacing free water deficit

with appropriate fluid intake and replacing AVP deficiency with synthetic analog therapy.[7] Desmopressin is administered orally which is the most effective route because plasma concentrations can be achieved within 40-55 minutes.[8] A tablet dose of 0.2 mcg is given every 8 hours via NG tube.

Hypernatremia is corrected slowly because cerebral tissue is hypersensitive to osmotic changes and can cause worsening of cerebral edema and demyelination. Correction is determined according to the free water deficit with a correction rate of no more than 0.5 mmol/hour or 10-12 mmol/24 hours. Selection of intravenous fluids with different sodium content will affect the speed of sodium reduction. Administration of fluids per oral or NGT is recommended to be carried out immediately considering that is the safest route to prevent too rapid a decrease in sodium and measurement of serum sodium every 4 hours during fluid resuscitation with repetition every 12 hours until clinically and biochemically stable.[8]

The patient was admitted to the ICU for 5 days, admitted to the intermediate ward for 7 days, and admitted to inpatient ward for 6 days. Before the patient was discharged, a repeat laboratory examination was performed and desmopressin therapy was continued at a dose of 0.1 mg every 8 hours orally. The patient was discharged on the 18th day and planned for control 5 days later.

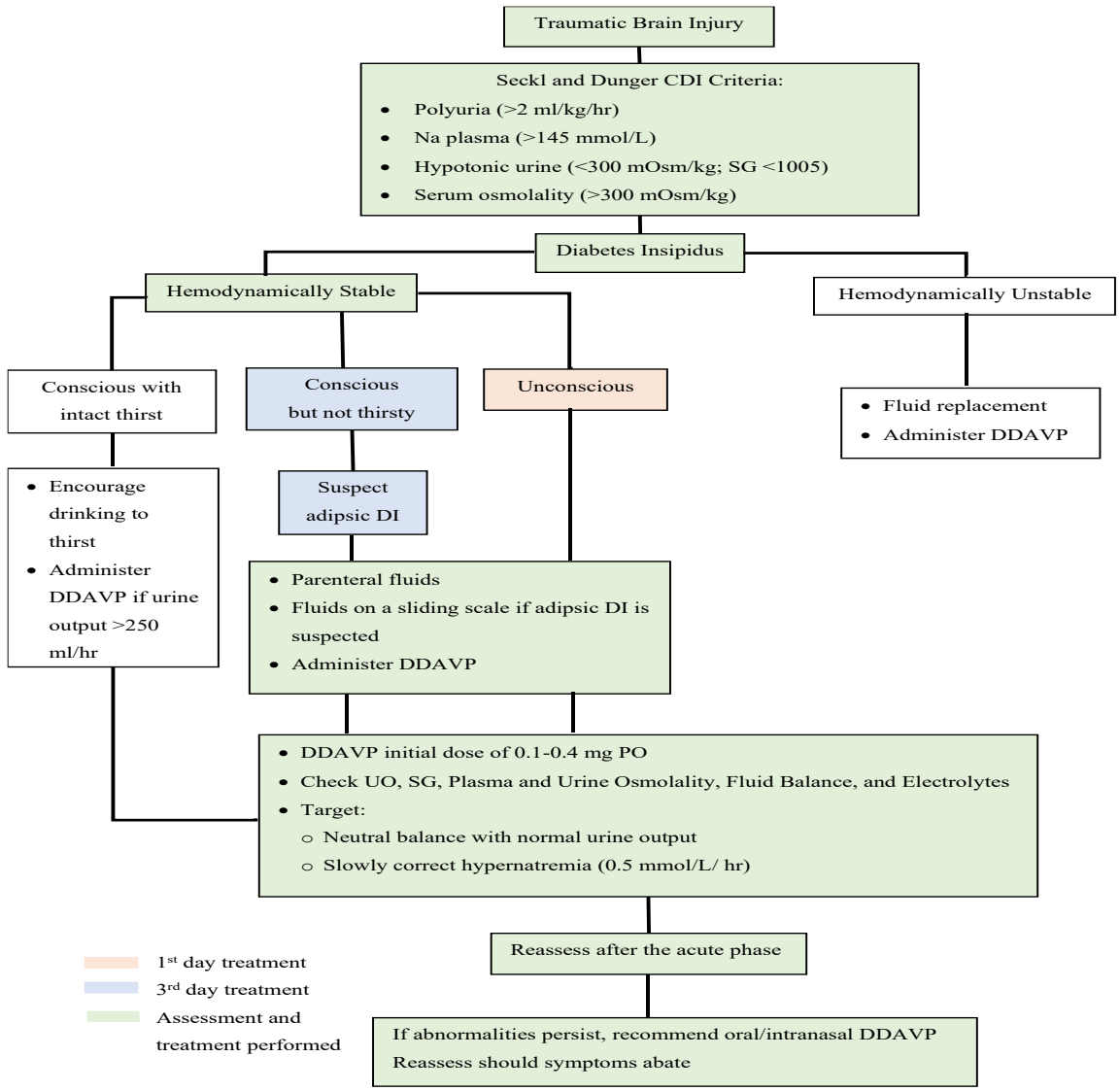
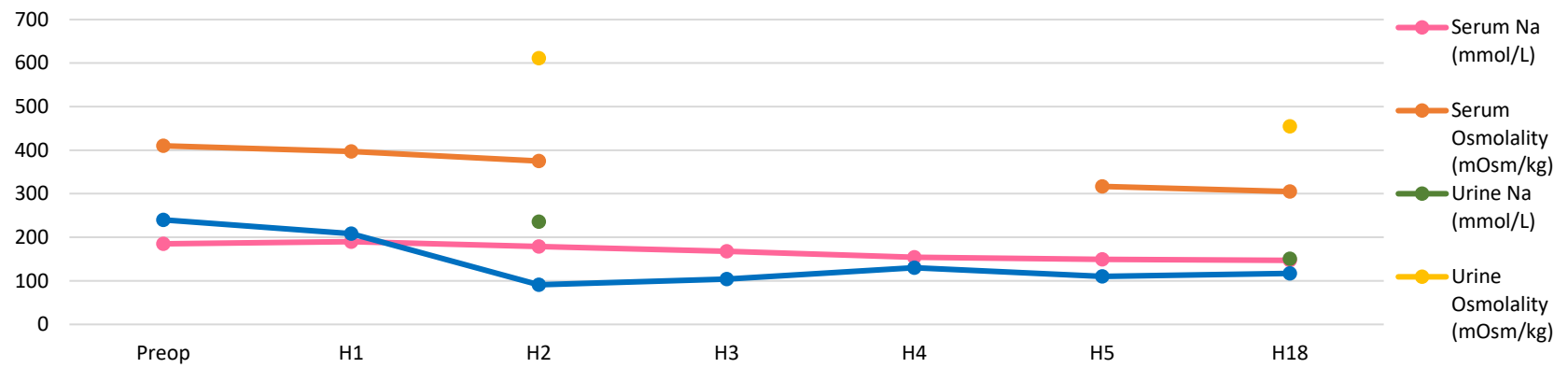


Figure 2. Diagnosis and Management Algorithm of CDI after TBI.[14]



	Intermediate	**ICU**					**Inpatient Ward**
Respiration	FM	Ventilator		FM	NC		Spontaneous breathing
SpO2	100	99	99	98	99	99	99
FiO2	40	40	40	50	28	28	21
Hypotension							
Vasopressor		Norepinephrine; Dobutamine					
Antibiotic		Ceftriaxone					
Corticosteroid	Dexamethasone						
Diuretic	Acetazolamide						
Analgetic	Paracetamol	Fentanyl; Paracetamol			Paracetamol		
Sedation		Dexmedetomidine					
Ulcer Protection		Omeprazole					
Nutrition	Peptibren						Porridge
IVFD	RF	Kaen 3B				RF	
		148 ml/jam	97 ml/jam	62 ml/jam	60 ml/jam		
Clear water	6x100 ml per NGT	50 ml/hr per NGT			50 ml/hr per oral		
DDAVP		Desmopressin					
		0.2 mg every 8 hr per NGT		0.2 mg every 12 hr per NGT	0.2 mg every 12 hr per oral		0.1 mg every 8 hr per oral

Graphics 1. Monitoring and Therapy during Treatment.

Tabel 1. Demographics, underlying disease, clinical presentation, management, and prognosis of patients with CDI reported in the literature.

Year/Reference	Gender/Age	Underlying Disease	Clinical Presentation	Therapy	DDAVP Dosage	Prognosis
2023; Pratana et al.	Male, 20 yo	Traumatic Brain Injury	Decreased consciousness, polyuria, hypernatremia	IVFD Kaen 3B	Desmopressin orally 0.2 mg every 8 hr → 0.2 mg every 12 hr → 0.1 mg every 8 hr	Discharge on the 18th day of treatment
2021; Jameel et al.[9]	Female, 7 yo	Craniopharyngioma	Polyuria, hypernatremia, irritability	Dexamethason IV 0.2 mg/kg/day every 6 hr Hydrocortison e1.25 mg every 6 hr	Vasopressin SC 4 IU/day every 6 hr → 6 IU/day Desmopressin orally 0.1 mg/day every 12 hr	Discharge on the 20th day of treatment
2020; Yang et al.[10]	Female, 59 yo	Guillain-Barré syndrome, Empty Sella Syndrome, hypopituitarism	Polyuria	IVIG 20 gr (5 days) Methylprednisolone IV 1000 mg (5 days) → 500 mg (5 days)	DDAVP high dose (single dose)	Improved condition after 6 months
2019; Smedegaard et al.[11]	Female, 41 yo	Pituitary apoplexy	Polyuria, hypernatremia, irritability Polyuria, polydipsia, persistent hypernatremia	-	Desmopressin 0.1 mg/day	-
2018; Zain et al.[12]	Female, 40 yo	St. IV breast cancer with bone and leptomeningeal metastases	Decreased consciousness, polyuria, polydipsia, hypernatremia, hypercalcemia	IVFD Dextrose 5% Pamidronate IV 90 mg (single dose) Calcitonin IV 200 IU every 12 hr (4 days)	Desmopressin IV 2 mcg (single dose)	Passed away
2010; Itshayek et al.[13]	Male, 19 yo	Traumatic Brain Injury	Polydipsia, polyuria, hypernatremia, decreased consciousness, irritability	Parenteral fluid	intranasal DDAVP	-

We conducted a literature search to support our case, including on PubMed and ProQuest for cases reported as post-TBI CDI cases with hypernatremia from 2010 to 2021, and a comparison was made with our case. We found similarities in the administration of desmopressin as the therapy of choice for CDI with hypernatremia given through various routes, although the underlying disease is different. Not all cases described the management for hypernatremia, some cases included parenteral fluid administration and 5% dextrose, and treatment for other complications such as hypercalcemia. The case of Itshayek et al. in 2010 has similarities with our case, namely TBI as the underlying disease, but there are differences in the route of administration of DDAVP and the patient's outcome is not included. Until 2023, there were no more cases with TBI as the underlying disease, but all cases were diagnosed with CDI and almost all of them had hypernatremia with different degrees of severity. All references reported in the literature have been summarized in Table 1.

CONCLUSION

Patients with CDI and severe hypernatremia after TBI responded well after receiving desmopressin therapy to treat AVP deficiency, as well as fluid replacement with KA-EN 3B and oral intake according to free water deficit. This case shows that appropriate monitoring and therapy can result in good outcomes in patients with CDI and severe hypernatremia after TBI.

Declaration by Authors

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Conflict of Interest: No conflicts of interest declared.

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