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Case Report

Central Diabetes Insipidus as An Unusual Sequelae of Non-Dominant Hemisphere Ischemic Stroke

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Abstract

Introduction: Central diabetes insipidus (CDI) is an uncommon complication of ischemic stroke and usually occurs when the hypothalamic–pituitary axis is affected. Its development following a middle cerebral artery (MCA) infarct is rare but clinically significant, as delayed recognition can lead to dehydration and hypernatremia.

Objective: To present a rare case of transient CDI following a right MCA infarction and highlight the importance of early diagnosis in post-stroke patients presenting with polyuria.

Material and Methods: This case was evaluated through retrospective review of hospital records. Clinical data, including neurological findings, urine output, serum sodium, serum and urine osmolality, and fluid balance, were collected. Imaging studies (CT and MRI) were reviewed to assess the infarct and exclude hypothalamic–pituitary injury. Treatment with desmopressin and the patient's clinical response were documented. Patient confidentiality was maintained according to institutional guidelines.

Results: Following a right MCA infarct, the patient developed significant polyuria and rising serum sodium. Laboratory studies confirmed CDI. Desmopressin therapy led to rapid normalization of urine output and serum sodium. The CDI was transient, resolving after short-term treatment without recurrence.

Conclusion: CDI can occur as a rare complication of MCA infarction. Clinicians should consider CDI in stroke patients with unexplained polyuria, as prompt diagnosis and treatment can prevent metabolic complications and support recovery. The occurrence of CDI following a stroke, along with its complete resolution during neurological recovery, points to a vascular mechanism as the underlying cause of this condition.

Take home message: Central diabetes insipidus is a rare but important complication of middle cerebral artery infarction. Early recognition of post-stroke polyuria enables timely diagnosis and desmopressin treatment, preventing hypernatremia and dehydration. Transient CDI following ischemic stroke suggests a reversible vascular mechanism.

Keywords: *Polyuria, Stroke, Diabetic Insipidus*

Introduction

Central diabetes insipidus (CDI) is an uncommon consequence of ischemic stroke, seen in less than 1% of cases, usually caused by damage to the hypothalamic-pituitary axis, predominantly associated with suprasellar or posterior circulation strokes.

The occurrence of CDI after a middle cerebral artery (MCA) stroke is very rare but carries significant clinical implications. If not recognized promptly, it can lead to severe dehydration and hypernatremia. However, with early detection and treatment using desmopressin, the outlook is generally positive and often reversible. [1-7]

Case Presentation

A 75-year-old male with a history of diabetes mellitus and hypertension presented with complaints of diarrhea, vomiting, and one episode of generalized tonic-clonic seizure. The patient had a prior history of stroke in 2021, leading to left hemiparesis, and had experienced recurrent deep vein thrombosis and pulmonary embolism, rendering him bedbound since that time. On examination, the patient was in a stuporous state, with pupils of normal size responsive to light. He had left hemiplegia with 0/5 power and an extensor plantar reflex on the left side. An MRI of the brain revealed a right MCA infarct affecting the frontoparietal and temporal region [Figure 1].

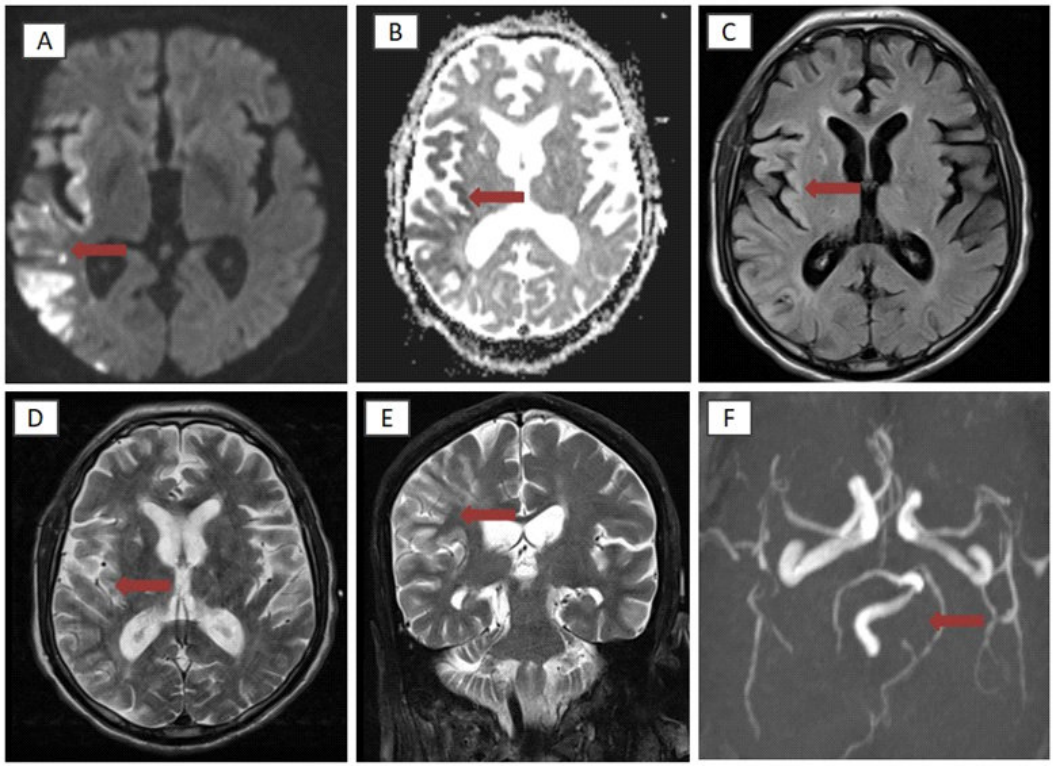

Figure 1. MRI brain axial section showed diffusion restriction in right temporo parietal region (A) with ADC reversal (B) as marked with red arrow and Fluid-Attenuated Inversion Recovery / T2 weighted imaging showing corresponding hyperintensity in temporo parietal cortex (C, D) and T2 weighted coronal section showing hyper intensity in temporo parietal cortex (E) with no involvement of thalamus or hypothalamus. MR angiogram showed non-visualization of the left vertebral artery (F).

The patient was treated with anti-platelets, statins, anti-epileptics, antibiotics, and other symptomatic treatments. On the fourth day, the patient experienced increased urine output (over 3000 ml in a 24-hour period), with serum sodium levels rising from 135 to 141 and then to 145 mmol/l on consecutive days, alongside a serum osmolality of 304 mosm/kg. The patient had hypotension, tachycardia, and other indicators of dehydration, with urine osmolality was normal [Figure 2]. A diagnosis of diabetes insipidus was made, and desmopressin nasal spray, at a dosage of 10 mcg twice daily, was administered. Over the following two days, the patient's urine output decreased to 2000 ml, and developed hyponatremia of 134. Desmopression was stopped and hyponatremia was corrected. Patient showed improvement in the Glasgow Coma Scale (GCS). The authors obtained consent from the patient’s relatives for publication.

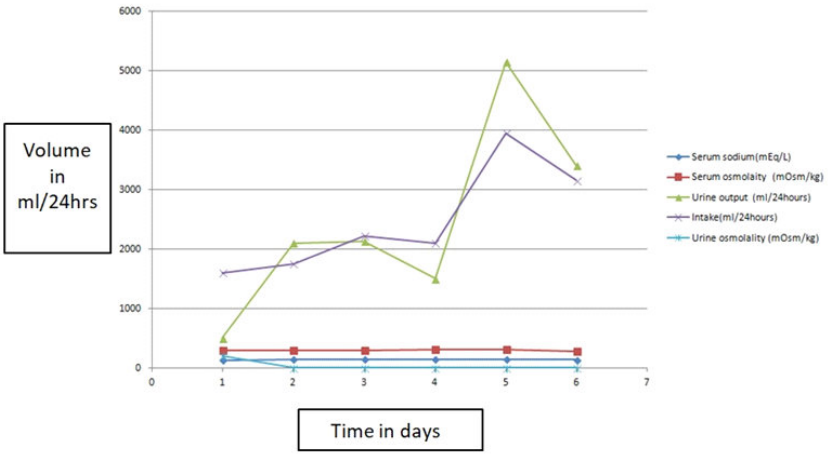

Figure 2. Showing series of laboratory parameters in relation to urine output during hospital stay. [A line diagram with the X-axis representing time in days and the Y-axis representing intake and output measured in ml per 24 hours. Two separate lines display trends over time: one for fluid intake and another for fluid output. Serum sodium (Meq/L), Serum osmolality (mOsm/kg), Urine output (ml/24hours), Intake (ml/24hours), and Urine osmolality (mOsm/kg)]

Discussion

Central diabetes insipidus is an uncommon complication associated with brain infarction. [1] The development of CDI in this patient appears to stem from ischemia-related dysfunction of hypothalamic–neurohypophyseal circuits [Figure 3]. Ischemia of the posterior pituitary caused by the inferior hypophyseal arteries has been suggested as a potential reason for idiopathic CDI. [2] Our patient did not undergo a water deprivation test due to his altered clinical state, but the presence of hypernatremia accompanied by elevated serum osmolality, increased urine output, and reduced urinary osmolality established the diagnosis of CDI. The patient's response to desmopressin and the normalization of urine output further confirmed this diagnosis.

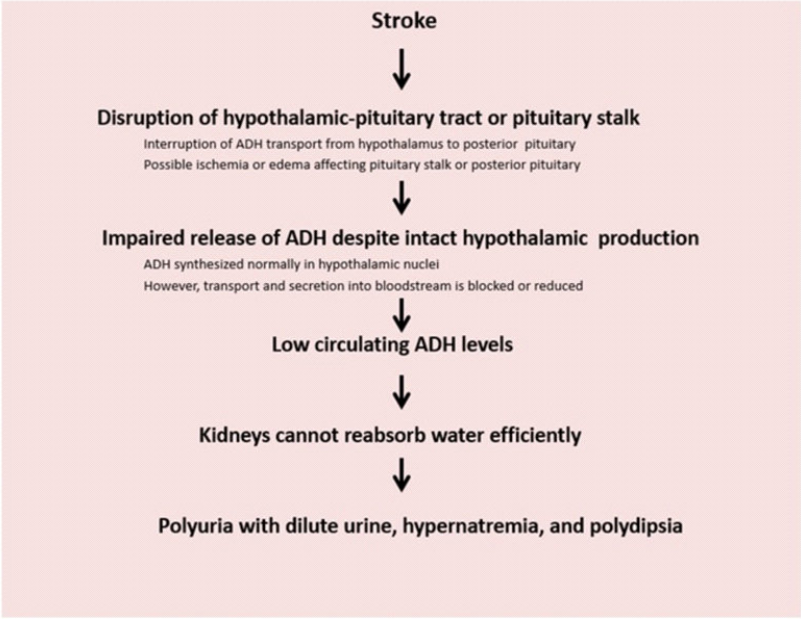

Figure 3. Pathophysiology of central diabetes Insipidus (CDI) following stroke.

CDI appears to be rare in the context of ischemic infarction, potentially resulting from heightened osmoreceptor activity due to hypothalamic and posterior pituitary hypoperfusion.[3] Transient CDI has been also reported in cardiopulmonary bypass,[4] in neurological intensive care patients after discontinuation of vasopressin therapy,[5] withdrawal of vasopressin in septic shock [6] and after vasopressin infusion.[7] Imaging showed a normal pituitary, eliminating concerns of pituitary pathology. The delayed onset of polyuria may be attributed to stored arginine vasopressin (AVP) within the posterior pituitary. Despite various potential underlying mechanisms being proposed, the evidence of transient CDI in conjunction with ischemic brain infarction suggests a vascular cause.

Conclusion

Central diabetes insipidus, although uncommon, can arise as a complication following middle cerebral artery infarction and should be thoroughly evaluated in stroke patients who exhibit polyuria without an obvious cause. Prompt identification and appropriate treatment are essential to avoid significant metabolic disruptions and facilitate overall recovery. The temporary nature of CDI, which resolves entirely in line with neurological recovery, provides strong evidence for a reversible vascular cause as the underlying pathophysiological mechanism.

Conflict of Interest

The authors declare no conflict of interest.

Acknowledgement

None

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