Intraoperative Central Diabetes Insipidus during Aneurysmal Clipping Surgery: An Unusual Phenomenon

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Abstract

Central diabetes insipidus (DI) is a known complication associated with pituitary surgeries occurring in postoperative period. However, development of DI following aneurysmal subarachnoid hemorrhage (SAH) is rarely reported. We describe here a case of intraoperative DI in a patient undergoing aneurysmal clipping surgery that posed a challenge for both diagnosis and management. Intraoperative development of central DI was attributed to the evolving ischemic injury to the hypothalamus at the time of rebleeding that was not apparent in the preoperative scan. DI resolved postoperatively after 18 hours of medical management. A careful observation of preoperative scans and vigilant monitoring may help in early diagnosis and management of such complication in the perioperative period.

Keywords
- central diabetes insipidus
- intraoperative
- subarachnoid hemorrhage
- intracranial aneurysm

Introduction

Central diabetes insipidus (DI) is one of the known complications associated with pituitary surgeries mostly occurring in the postoperative period. Even though it has infrequently been linked to aneurysmal clipping surgery, its prevalence is still primarily in postoperative period. There is no description of its intraoperative development in such surgeries as yet. We describe here a case of intraoperative DI in a patient undergoing aneurysmal clipping surgery that posed a challenge for both diagnosis and management.

Case Report

A 55-year-old female, diagnosed with subarachnoid hemorrhage (SAH) with modified Hunt and Hess grade 3 due to ruptured left middle cerebral artery (MCA) aneurysm 2 days back, was posted for aneurysmal clipping. Preoperatively, the patient developed sudden neurological deterioration and an urgent noncontrast computed tomography (CT) scan was done that showed a rebleeding with massive intracranial hemorrhage and left MCA territory infarct (►**Fig. 1A**). The patient was immediately taken up for craniotomy and aneurysmal clipping in the theater where standard neurosurgical monitoring along with arterial blood pressure and central venous pressure (CVP) was instituted. A standard anesthetic management protocol was followed with general anesthesia induction with 1 mg midazolam, 100 µg fentanyl, 250 mg thiopentone, and 6 mg vecuronium. After induction, 60 g (1 gm/kg) of 20% mannitol was given over 30 minutes for brain relaxation. Anesthesia was maintained with propofol infusion 75 to 100 µg/kg/min, vecuronium infusion (0.1 mg/kg/h), and fentanyl boluses (1µg/kg/h). Goal-directed intravenous fluid administration was done with maintenance of blood gas parameters and normoglycemia. Almost 2 hours after the surgical incision, a high urine output (700–1,000 mL) was observed for about 10 minutes, which further increased to 100 to 150 mL per minute. Arterial blood gas analysis

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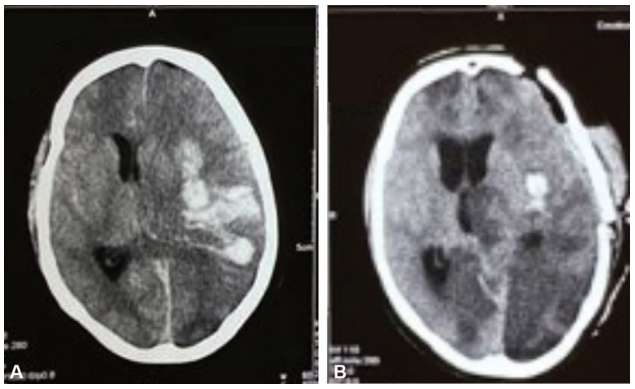

Fig. 1 **(A)** Preoperative axial noncontrast computed tomography (NCCT) head after rebleeding showing left temporoparietal hematoma with surrounding edema, midline shift, compression of third ventricle and no appreciable hypodensity in thalamic region. (**B**) Postoperative axial NCCT head showing left temporal hematoma with displacement of third ventricle with hypodensity in thalamic region with ventriculomegaly.

revealed raised serum sodium (~165 mEq/L) with increased serum osmolality (303 mOsm/L) as compared to preoperative and post-induction baseline serum sodium value of 142 and 138 mEq/L, respectively. Urine-specific gravity was found to be 1.005 (hypo-osmolar urine). The hemodynamic measurements were mean arterial pressure (MAP) of 80 to 85 mmHg, CVP of 1 cm H2O, and pulse pressure variation (PPV) of 18 to 22. Since, all these findings were consistent with DI—intravenous vasopressin infusion started at 5 mU/kg/h and titrated within 1 to 10 mU/kg/h and maintenance fluid shifted to 0.45% normal saline. After 45 minutes urine output reduced to approximately 200 to 250 mL/h and serum sodium decreased to 155 mEq/L and subsequently, vasopressin dose was titrated to maintain serum sodium around 155 mEq/L till the end of surgery. Postoperatively, patient shifted to neurosurgical intensive care unit on mechanical ventilation with vasopressin infusion. A CT scan done during this time revealed residual temporal hematoma with infarct in MCA territory and cerebellum and hypodensity in left thalamic region (►**Fig. 1B**). The vasopressin infusion was tapered off within 18 hours following resolution of polyuria and hypernatremia. Patient was electively ventilated for 48 hours. As the patient improved neurologically with Glasgow coma scale of E4VtM5, weaning from mechanical ventilation was initiated. However, the patient developed pulmonary complications and unfortunately succumbed on 5th postoperative day.

Discussion

The sudden onset of intraoperative polyuria in aneurysmal clipping surgery may pose a diagnostic dilemma for the anesthesiologist. The common causes of intraoperative polyuria are overzealous fluid administration, intraoperative use of mannitol, uncontrolled hyperglycemia, anesthetic agent-induced DI with certain differentiating features (►**Table 1**). Osmotic diuresis due to mannitol leads to increased sodium excretion with high urinary osmolality and specific gravity (> 1.005) and decreased serum sodium levels.[1] The patient had no history of diabetes and maintained normoglycemia throughout the perioperative period. Also, common anesthetic agents that can precipitate DI like dexmedetomidine, sevoflurane, and ketamine were not used during anesthetic management.[2] The bedside urine-specific gravity of this patient was found to be less than 1.005 with severe hypernatremia and increased serum osmolarity favoring central DI. Following this, we chose to give vasopressin infusion as it has more rapid onset and shorter action (t1/2 10–15 min) contrary to routinely used desmopressin.[3] Also, 0.45% normal saline was initiated and after 45 minutes, urine output was reduced and serum sodium measurements showed decreasing trend further indicating responsiveness to treatment and supporting central DI as the cause of polyuria.

Transient DI is an uncommon complication of SAH that is more often reported with ruptured anterior communicating artery aneurysm and mostly in postoperative period.[4–8] DI after aneurysmal SAH may be caused by damage to the supraoptic and paraventricular nuclei in the anterior hypothalamus. These nuclei receive their blood supply from

Table 1 Common causes of polyuria with their differentiating features

	Hemodynamics	Serum osmolarity	Serum Na+	Urine osmolarity	Urine specific gravity	Urinary spot Na+	Serum glucose	Precipitating drug
Fluid overload	Hypertension, CVP↑,↓ PPV	N/ ↓	N/ ↓	N/ ↓	N/↓	N/↓	N	Absent
Intraoperative mannitol	Hypotension, ↓CVP, ↑ PPV	↑	N/ ↓	↑	↑	N/↑	N/↑	Absent
Uncontrolled hyperglycemia	Hypotension, ↓CVP, ↑ PPV	↑	↓/↑	↑	↑	↓	↑	Absent
Drug-induced DI	Hypotension, ↓CVP, ↑ PPV	↑	↑	↓	↓	↓	N	Present
Central DI	Hypotension, ↓CVP, ↑ PPV	↑	↑	↓	↓	↓	N	Absent

Abbreviations: CVP, central venous pressure; DI, diabetes insipidus; PPV, pulse pressure variation.

small penetrating branches arising from the anterior cerebral and anterior communicating artery.[9] Intraoperative development of central DI in this patient was attributed to the evolving ischemic injury to the hypothalamus at the time of rebleeding that was not apparent in preoperative scan. However, in postoperative CT scan (6 hours post-procedure) infarct in thalamic/hypothalamic region was evident and was the probable cause of intraoperative manifestation of central DI in this patient with SAH. The likely mechanism of occurrence of transient central DI in spite of ischemic injury to hypothalamic region could be a partial injury to hypothalamus resulting in infarct that led to initial transient DI with subsequent stabilization. The hypothalamic nuclei are present bilaterally and, in this patient, it is evident that there is more hypodensity on the left side than on the right side suggestive of incomplete or partial insult that probably led to transient DI. Alternatively, it could be penumbra zone that recovered following treatment.

Conclusion

Development of DI during aneurysmal surgery was unexpected and unanticipated. Initially, common causes of polyuria were considered and ruled out. The cause of intraoperative DI in this case was found to be pre-existing ischemic injury of hypothalamic region that subsequently evolved to infarct which was not evident in the preoperative scan. We suggest carefully observing preoperative scan with cautiously monitoring urine output and electrolytes in the perioperative period may enable anticipation, prompt diagnosis, and management of intraoperative DI.

Conflict of Interest
None declared.

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