Annals of
Clinical Case Studies and Medical Images

Neurology ACCSMI-1002 Case Study

Postoperative Diabetes Insipidus after Spinal Cord Tumor Resection: Report of Two Cases and Literature Review

Dan Lei* and Guihuai Wang

Department of Neurosurgery, Tsinghua University Affiliated Beijing Tsinghua Changgung Hospital, Beijing, China

Corresponding author:** ***Dan Lei, *Department of Neurosurgery, Tsinghua University Affiliated Beijing Tsinghua Changgung Hospital, Beijing, China;* ***Email:*** *ld94616@163.com*

Received Date : *02 March, 2026*
Accepted Date : *03 April,, 2026*
Published Date : *07 April, 2026*

Citation: *Dan Lei, Guihuai Wang. Postoperative Diabetes Insipidus after Spinal Cord Tumor Resection: Report of Two Cases and Literature Review. Ann Clin Case Stud Med Images. 2026; 1(1): 1002.*

Abstract

Background: Diabetes insipidus (DI) after spinal surgery is extremely rare and may be missed because osmotic diuresis or cerebral salt wasting (CSWS) can mimic the presentation. Clear diagnostic framing and time-linked biochemical testing are therefore essential.

Case Description: Two patients developed DI following spinal cord tumor resection. A 40-year-old woman with a recurrent thoracolumbar intradural tumor manifested polyuria up to 5,000 mL/day with low urine specific gravity and improved promptly with oral desmopressin. A 13-year-old boy who underwent long-segment intramedullary tumor resection developed hypotonic polyuria on postoperative day 3. Outside the 0.5–1-hour window after mannitol infusion (osmotic diuresis), persistent polyuria was accompanied by urine osmolality <60 mOsm/kg, urine specific gravity 1.001–1.003, low plasma osmolality (~260 mOsm/kg), hyponatremia, hypokalemia and hypouricemia; a desmopressin challenge increased urine osmolality to ~500 mOsm/kg and reduced urine output, confirming central AVP deficiency. Both patients recovered fully after temporary DDAVP therapy and electrolyte management.

Conclusion: Postoperative DI can occur after spinal cord tumor resection and may be under-recognized. Differentiating hypotonic polyuria due to AVP deficiency from osmotic diuresis and CSWS requires timed biochemical testing and, when feasible, desmopressin/copeptin-guided confirmation. Early recognition prevents serious electrolyte disturbances.

Keywords: Spinal cord tumor; Diabetes insipidus; Postoperative complication; Neurosurgery

Introduction

Diabetes insipidus (DI) is defined by hypotonic polyuria due to inadequate secretion or action of arginine vasopressin (AVP). While postoperative DI is classically associated with suprasellar or pituitary procedures, spinal surgery–related DI is scarcely reported and easily confounded by osmotic diuresis (e.g., mannitol) or salt-wasting states. We present two cases of AVP-responsive postoperative DI after spinal cord tumor resection and synthesize pathophysiological hypotheses relevant to complex spinal procedures, aiming to offer a practical diagnostic framework for postoperative polyuria in neurosurgical practice.

Case Description

Case 1

A 40-year-old woman underwent resection of a recurrent thoracolumbar intradural tumor (pathology: myxopapillary ependymoma). On postoperative day 2, she developed polyuria up to 5000 mL/day with a low urine specific gravity (1.009). She was treated empirically with oral desmopressin, which rapidly normalized urine output. At that time, plasma and urine osmolality were not measured.

Her symptoms resolved by postoperative day 13, and she remained asymptomatic during follow-up.

Case 2

A 13-year-old boy underwent resection of a large intramedullary spinal cord tumor extending from the medulla to T12. The procedure lasted 15 hours, with 600 mL blood loss. On postoperative day 3, he developed marked polyuria (>4000–5000 mL/day). During mannitol infusion, a transient increase in urine output occurred within 1 hour, but persistent polyuria continued beyond this period. Laboratory testing revealed urine specific gravity of 1.001–1.003 and urine osmolality < 60 mOsm/kg, with concurrent plasma osmolality of 260 mOsm/kg, hyponatremia, hypokalemia, and hypouricemia. Desmopressin administration increased urine osmolality to 500 mOsm/kg and decreased urine volume, confirming central DI. The patient required oral DDAVP and electrolyte supplementation for 35 days, achieving full recovery.

Discussion

Diagnostic Framework for Postoperative Polyuria

Polyuria is commonly defined as >3 L/day in adults (or >2 L/m²/day in children). A practical pathway in the early postoperative setting includes: (1) confirm hypotonic polyuria (urine osmolality <300 mOsm/kg or specific gravity ≤1.005); (2) review time-locked exposures—particularly mannitol, loop diuretics and high-volume fluids; (3) screen plasma osmolality and sodium, together with urine electrolytes; (4) differentiate AVP deficiency from primary polydipsia and AVP resistance; and (5) where feasible, use desmopressin response and/or copeptin-based diagnostics to confirm mechanism. In our second case, very low urine osmolality with brisk desmopressin response was diagnostic for central DI despite concurrent hyponatremia.

Distinguishing AVP Deficiency from Osmotic Diuresis and CSWS

Osmotic diuresis (e.g., mannitol) typically causes a temporally restricted surge in urine output that parallels drug administration and resolves as the osmotic load is cleared; urine osmolality is not profoundly low. In Case 2, persistent polyuria outside the mannitol window with extremely hypotonic urine argued against pure osmotic diuresis.

CSWS features extracellular volume depletion, low serum sodium and uric acid, and high urinary sodium losses; however, urine is usually not profoundly hypotonic and patients often display thirst/salt craving and overt dehydration. Our adolescent patient had low serum sodium and uric acid yet strikingly low urine osmolality and a robust desmopressin response—findings more consistent with AVP deficiency than CSWS.

Why Might Spinal Surgery Precipitate AVP Deficiency?

Multiple, non-mutually exclusive mechanisms may operate in complex spinal procedures:

Neuroaxis traction: extensive intramedullary tumor resection and dural manipulation may transiently perturb long-tract inputs influencing hypothalamic–pituitary AVP release (an analogy to traction-related DI described after deformity correction).

Neuroinflammation within the subarachnoid space: blood and inflammatory mediators after extensive spinal manipulation could secondarily dysregulate hypothalamic–pituitary signaling in a self-limited fashion.

Autonomic dysregulation: disruption of spinal sympathetic pathways involved in renal handling of water and electrolytes may modify the renal set-point for free-water reabsorption and interact with AVP signaling.

Peri-Anesthetic Factors: prolonged anesthesia and certain agents (e.g., volatile anesthetics, α2-agonists) have been linked to perioperative DI; however, such drug-induced diuresis tends to be short-lived and resolves upon drug cessation, which did not fully explain our course.

Endocrine Stress Responses: elevated cortisol observed in Case 2 may reflect stress hypercortisolemia; while glucocorticoids promote sodium retention and water reabsorption, the overall biochemical profile remained dominated by hypotonic polyuria responsive to AVP replacement.

Practical Bedside Points for Neurosurgeons

Time the Labs: pair urine/plasma osmolality with the timing of mannitol to avoid misclassification.

Look for Extreme Hypotonicity: urine osmolality <100 mOsm/kg strongly favors AVP deficiency in this context.

Use a desmopressin challenge early when compatible with hemodynamics; a ≥50% rise in urine osmolality with falling urine output supports central DI.

Treat and re-evaluate: temporary DDAVP with careful sodium and fluid management is typically effective; reassess for spontaneous resolution as neuroinflammation subsides.

Document volume status carefully to avoid over- or under-correction of sodium in children and low-weight patients.

Limitations

Our first case lacked formal osmolality assessment; copeptin testing was unavailable. Nonetheless, convergent clinical features and DDAVP responsiveness, together with negative suprasellar imaging, support central DI. Larger series with standardized endocrine testing are needed to define incidence and mechanisms after complex spinal tumor surgery.

Conclusion

Diabetes insipidus can occur after spinal cord tumor resection and should be considered when polyuria develops postoperatively. Differentiating osmotic diuresis from AVP deficiency through biochemical testing and desmopressin challenge is crucial for accurate diagnosis and management.

Acknowledgment

The authors thank the medical and nursing staff involved in the care of these patients for their invaluable assistance.

References

1. Rosenbaum BP, Steinmetz MP. Central diabetes insipidus after staged spinal surgery. Global Spine J. 2013; 3(4): 257–260.
2. Kong Kam Wa T, McCarthy KF. Intra-operative diabetes insipidus associated with cervical spine traction during staged scoliosis surgery. Anaesth Rep. 2020; 8(2): 123–126.
3. Kuzeyli K, Cakir E, Baykal S, Karaarslan G. Diabetes insipidus secondary to penetrating spinal cord trauma: case report and

literature review. Spine (Phila Pa 1976). 2001; 26(21): E510–E511.

4. Mutter CM, Smith T, Menze O, Zakharia M, Nguyen H. Diabetes Insipidus: Pathogenesis, Diagnosis, and Clinical Management. Cureus. 2021; 13(2): e13523.

5. Yee AH, Burns JD, Wijdicks EF. Cerebral salt wasting: pathophysiology, diagnosis, and treatment. Neurosurg Clin N Am. 2010; 21(2): 339–352.

6. Van Decar LM, Reynolds EG, Sharpe EE, Harbell MW, Kosiorek HE, Kraus MB. Perioperative Diabetes Insipidus Caused by Anesthetic Medications: A Review of the Literature. Anesth Analg. 2022; 134(1): 82–89.