CASE REPORT

A Diagnostic Odyssey: Post-Surgical Polyuria and Hyponatremia Unmasking Multiple Myeloma-Associated SIADH-A Case Report

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ABSTRACT

Hyponatremia and polyuria are common postoperative complications, often posing significant diagnostic challenges. This case report details a complex scenario of a 56-year-old male with hypertension and diabetes who developed polyuria post-femur cyst evacuation, initially misinterpreted as diabetes insipidus and treated with desmopressin. Subsequent severe symptomatic hyponatremia necessitated a comprehensive re-evaluation, revealing a shift towards syndrome of inappropriate antidiuretic hormone secretion (SIADH). Further investigation, particularly through fractional excretion studies, led to the diagnosis of SIADH secondary to underlying multiple myeloma, a rare etiology. The patient's condition stabilized with targeted treatment for SIADH and subsequent chemotherapy for multiple myeloma. This case highlights diagnostic challenges in electrolyte imbalances, the use of fractional excretion studies to differentiate hyponatremia etiologies, and the importance of considering rare paraneoplastic causes, such as multiple myeloma, in SIADH.

Keywords: *Desmopressin, fractional excretion, multiple myeloma, syndrome of inappropriate antidiuretic hormone secretion, diabetes insipidus, polyuria, hyponatremia.*

INTRODUCTION

Hyponatremia is a frequently encountered electrolyte abnormality in hospital admissions, particularly in the postoperative period [1]. Moreover, post-surgical polyuria, when unexplained by oral or parenteral hydration, often leads to an initial presumption of diabetes insipidus (DI), sometimes resulting in premature treatment with desmopressin. Our case illustrates a diagnostically challenging scenario where a patient initially presented with features mimicking DI but subsequently developed severe hyponatremia, leading to a complex diagnostic journey culminating in the identification of the syndrome of inappropriate antidiuretic hormone secretion (SIADH) secondary to multiple myeloma. The etiology of hyponatremia was determined based on the specific circumstances of this case, using fractional excretion studies [2-4]. This unusual association, along with the diagnostic process, offers valuable clinical insights.

CASE REPORT

A 56-year-old gentleman with a significant history of hypertension for 5 years and diabetes for 2 years was referred from a peripheral hospital with a complaint of abnormal sodium readings. He was admitted there electively for the evacuation of a bone cyst in the right femur. He underwent cyst evacuation and bone grafting with fixation 3 days ago, on November 12. His baseline serum electrolytes, urine analysis, and echocardiography were unremarkable. On the first postoperative day, he was noted to have an eight-liter urine output. This output increased to 12 liters the next day. Upon evaluation, his serum sodium was 140 mEq/L, with a serum osmolality of 273 mOsm/kg and urine osmolality of 128 mOsm/kg. At that time, he was considered a case of diabetes insipidus and managed accordingly. Initially, he was started on indomethacin and hydrochlorothiazide, with little improvement. Later, desmopressin nasal spray was initiated, leading to an improvement in his urine output to 2 to 2.5 L. However, his serum sodium then started to drop, as detailed in Table **1**.

Table 1: Trends in serum sodium levels (Immediate postoperative period).

Date / Time	Serum Sodium (mEq/L)
November 12, 6 am	140
November 13, 12 pm	138
November 14, 6 am	132
November 14, 12 pm	129
November 14, 6 pm	124
November 15, 6 am	127
November 15, 12 pm	120
November 15, 6 pm	119

On November 15, he developed a tonic-clonic seizure, which was managed with 3% saline boluses. He was subsequently referred to a tertiary care setting. A CT brain plain was performed before transfer and was unremarkable.

When he was received in our hospital, he complained of pain at the surgery site. On examination, he was stable,

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especially volume-wise, euvolemic. On laboratory evaluation, his sodium was now 115 mEq/L, and his creatinine was 1.4 mg/dL, up from 0.6 mg/dL preoperatively. Other detailed lab workup is mentioned in Table **2**.

Table 2: Initial laboratory findings upon tertiary care admission.

Test	Patient Value	Normal Range
Calcium	8.35 mg/dL	8.5-10.5 mg/dL
Phosphorous	5.09 mg/dL	2.5-4.5 mg/dL
Magnesium	1.76 mg/dL	1.7-2.2 mg/dL
albumin	3.51 g/dL	3.5-5.0 g/dL
hemoglobin	9.3 g/dL	13.5-17.5 g/dL
hematocrit	27%	41%-53%
WBC	25.6×10^9 /L	$4.0\text{-}11.0\times10^9$ /L
PlatPlatelets	336×10^9 /L	$150\text{-}400\times10^9$ /L
Urine analysis	pH5, specific gravity 1.025, Albumin negative, RBC numerous, Leucocytes 4, WBC Esterase negative	-

The initial impression was tonic-clonic seizures secondary to drug-induced acute hyponatremia (desmopressin), complicated with AKI secondary to Septic ATN. Consequently, urine Sodium and urine osmolality were sent. He was started on 3% saline at 25 mL/hr, and desmopressin was stopped. Additionally, he was commenced on intravenous antibiotics. His further management is summarized in Table **3** and Fig. (**1**).

Table 3: Subsequent management and clinical course.

Date	Investigation	Value	Input (mL)	Output (mL)	Management
November 17	Sodium	126-132 mEq/L	1350	2300	3% saline at 20 mL/hr + monitoring
-	Urine Sodium	16 mEq/L	-	-	-
-	Urine Osmolality	338 mOsm/kg	-	-	-
-	WBC	16×10^9/L	-	-	-
-	Creatinine	1.12 mg/dL	-	-	-
November 18	Sodium	135 mEq/L	1800	3300	Hypertonic saline stopped + water restriction
-	Creatinine	0.86 mg/dL	-	-	-
November 19	Sodium	133 mEq/L	1700	3300	-

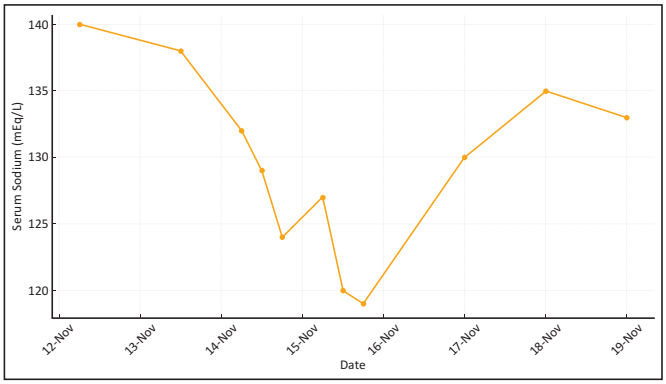

Fig. (1): Trend of sodium over time.

At this time, he was stable and being considered for discharge. But now he again complained of polyuria. Subsequently, we considered polyuria with SIADH *vs.* salt wasting (renal/cerebral) given his recent history. Now, his urine sodium was 142 mEq/L, with urine osmolality 496 mOsm/kg (serum sodium 133 mEq/L). The main difference between these two differentials depends on the volume assessment, but due to his recentfemur surgery, it was inconclusive. Hence, based on an extensive literature review [2-4], we calculated the fractional excretion of uric acid and phosphate at 16.94 and 20.68, respectively. Initially, he was started on a high-salt diet (10 g daily) and fluid restriction, since he had previously responded to hypertonic saline. Moreover, after a few days, due to lack of responsiveness and uncontrolled blood pressures, he was started on demeclocycline 300 mg bid and fludrocortisone 0.1 mg daily. Now, the sodium level (135 mEq/L) has stabilized, and the patient has been discharged. In the follow-up clinics, his serum sodium remained within the normal range, and the repeat fractional excretion of uric acid was 8.9% [2-4]. Moreover, Persistent SIADH without an identifiable cause, normocytic anemia, elevated serum proteins, and the presence of urinary kappa light chains raised suspicion for a plasma cell disorder, prompting bone marrow biopsy. Bone marrow biopsy demonstrated 38% clonal plasma cell infiltration, confirming the diagnosis of multiple myeloma.

Urine kappa light chains and an M-spike on serum electrophoresis supported the diagnosis (biopsy image not included due to institutional restriction). Subsequently, a diagnosis of SIADH secondary to multiple myeloma was made. It was also supported by the improved fractional excretion of urea [2, 3]. His demeclocycline and fludrocortisone were tapered off. Later, the patient was referred to the hem-oncologist. He was started on a VRd chemotherapy regimen (bortezomib, lenalidomide, dexamethasone) and received localized radiotherapy as per hematology recommendations.

DISCUSSION

This case highlights several important clinical points. Firstly, it illustrates a significant diagnostic challenge. Hyponatremia is common after surgery and is often misattributed to stress, fluid shifts, or drug effects such as opioids or desmopressin [1]. Our patient's initial presentation mimicked diabetes insipidus (DI) due to polyuria. Still, the subsequent development of profound hyponatremia following desmopressin administration necessitated a re-evaluation of the diagnosis, ultimately pointing towards SIADH. This transition in clinical picture is diagnostically complex and a crucial learning point.

Secondly, the utility of fractional excretion studies is underscored. This case reinforces their diagnostic value,

particularly in differentiating causes of hyponatremia [3, 4]. The calculated fractional excretion of uric acid and phosphate helped to differentiate between cerebral salt wasting, renal salt wasting, and SIADH [2, 3]. Specifically, an elevated FEUrea initially, which later normalized, is characteristic of SIADH. In addition, our use of repeat fractional excretion post-treatment helped confirm resolution and guide de-escalation of the therapy [2-4].

Thirdly, plasma cell dyscrasia, specifically multiple myeloma, is a rare cause of SIADH and an important consideration [5, 6]. SIADH is typically linked to small-cell lung carcinoma, central nervous system lesions, or various medications [7, 8]. Plasma cell dyscrasias, including monoclonal gammopathy of undetermined significance (MGUS) and multiple myeloma (MM), are an overlooked etiology. Mechanisms may include: Ectopic ADH production, IL-6 or other cytokine-mediated ADH release, or Tubular damage from light chains impairing water handling [8].

Additionally, the complication of desmopressin-induced hyponatremia underscores the risks of premature therapeutic trials before establishing a firm diagnosis [9]. The initial treatment for suspected DI with desmopressin inadvertently worsened the patient's hyponatremia, leading to a seizure [5]. This emphasizes the need for a thorough diagnostic workup, especially in dynamic clinical scenarios involving electrolyte disturbances.

Finally, in our case, the simultaneous use of demeclocycline and fludrocortisone is not standard practice for SIADH. Given the unique and evolving clinical circumstances, it was deemed justified to manage the complex and fluctuating electrolyte imbalances before the definitive etiology was established.

CONCLUSION

This case report illustrates a rare and complex presentation of SIADH secondary to multiple myeloma, initially masquerading as diabetes insipidus. It emphasizes the diagnostic challenge in differentiating causes of polyuria and hyponatremia, highlighting the critical role of fractional excretion studies in guiding diagnosis and management. Clinicians should be aware of multiple myeloma as a rare but significant underlying cause of SIADH and exercise caution with empirical treatments that can worsen electrolyte imbalances before a firm diagnosis is established.

CONSENT FOR PUBLICATION

Informed consent was taken from the patient.

CONFLICT OF INTEREST

The author declares no conflict of interest.

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Declared none.

GENERATIVE AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

During the preparation of this work, the author limitedly used ChatGPT (GPT-4, OpenAI) to get language suggestions and do minor proofreading in some parts of the manuscript. After using this tool/service, the author reviewed and edited the content as needed and takes full responsibility for the content of the published article.

REFERENCES

1. Sanada M, Tominaga H, Kawamura I, Tokumoto H, Ogura T, Taniguchi N, *et al.* Incidence and risk factors for hyponatremia in postoperative spinal surgery patients. Spine Surg Relat Res 2023; 8(3): 267-71.
DOI: https://doi.org/10.22603/ssrr.2023-0158

2. Maesaka JK, Imbriano L, Mattana J, Gallagher D, Bade N, Sharif S, *et al.* Differentiating SIADH from cerebral/renal salt wasting: failure of the volume approach and need for a new approach to hyponatremia. J Clin Med 2014; 3(4): 1373-85.
DOI: https://doi.org/10.3390/jcm3041373

3. Maesaka JK, Imbriano LJ, Miyawaki N. Determining fractional urate excretion rates in hyponatremic conditions and improved methods to distinguish cerebral/renal salt wasting from the syndrome of inappropriate secretion of antidiuretic hormone. Front Med (Lausanne) 2018; 5: 319.
DOI: https://doi.org/10.3389/fmed.2018.00319

4. Bassi V, Fattoruso O. The role of fractional excretion of uric acid in the differential diagnosis of hypotonic hyponatraemia in patients with diuretic therapy. Cureus 2020; 12(4): e7762.
DOI: https://doi.org/10.7759/cureus.7762

5. Tang R, Zhang JJ, Zhong YP. A report on the syndrome of inappropriate antidiuretic hormone secretion associated with multiple myeloma. Chin Med J (Engl) 2020; 133(12): 1493-4.
DOI: https://doi.org/10.1097/CM9.0000000000000837

6. Abraham A, Shafi F, Iqbal M, Kollipara R, Rouf E. Syndrome of inappropriate antidiuretic hormone due to multiple myeloma. Mo Med 2011; 108(5): 377-9.

7. Björck E, Samuelsson J. Syndrome of inappropriate secretion of antidiuretic hormone (SIADH) after treatment with cyclophosphamide, alpha-interferon and betamethasone in a patient with multiple myeloma. Eur J Haematol 1996; 56(5): 323-5.
DOI: https://doi.org/10.1111/j.1600-0609.1996.tb00724.x

8. Dixon BN, Daley RJ, Buie LW, Hsu M, Park JH, Brentjens RJ, *et al.* Correlation of IL-6 secretion and hyponatremia during CD19 CAR-T therapy. Clin Nephrol 2020; 93(1): 42-6.
DOI: https://doi.org/10.5414/cn109872

9. Achinger SG, Arieff AI, Kalantar-Zadeh K, Ayus JC. Desmopressin acetate (DDAVP)-associated hyponatremia and brain damage: a case series. Nephrol Dial Transplant 2014; 29: 2310-15.
DOI: https://doi.org/10.1093/ndt/gfu263