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Central Diabetes Insipidus in a Patient with Acute Myeloid Leukemia: A Case Report

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ABSTRACT

Central diabetes insipidus (CDI) is an uncommon but potentially life-threatening complication in patients with hematologic malignancies. We report the case of a 30-year-old man who presented with upper respiratory symptoms, followed by progressive fatigue, polydipsia, and polyuria. Laboratory evaluation revealed pancytopenia, and bone marrow biopsy confirmed the diagnosis of AML-M2. Despite initial chemotherapy, the patient experienced persistent polyuria and hypernatremia (148–156 mmol/L).

Endocrine evaluation revealed a urine osmolality of 159 mOsm/kg, a serum osmolality of 298 mOsm/kg, and a desmopressin challenge confirmed CDI (>50% increase in urine osmolality). MRI demonstrated the loss of the posterior pituitary hyperintensity and deviation of the pituitary stalk.

The patient was treated with intranasal desmopressin, resulting in symptomatic improvement. Despite therapy, the leukemia proved refractory, and the patient eventually succumbed to disease progression. This case underscores the importance of early recognition of CDI in patients with AML, as timely diagnosis and management can mitigate severe complications.

Keywords: acute myeloid leukemia, central diabetes insipidus, polyuria

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1. Introduction

Acute myeloid leukemia (AML) is a clonal hematopoietic stem cell malignancy characterized by the uncontrolled proliferation and accumulation of immature myeloid precursors in the bone marrow.

It is a biologically heterogeneous disease with diverse genetic, molecular, and clinical subtypes, including AML with recurrent genetic abnormalities, AML with myelodysplasia-related changes, therapy-related AML, AML, not otherwise specified, and myeloid sarcoma (1).

These classifications provide critical guidance for prognosis and therapeutic decision-making, including conventional chemotherapy, targeted agents, and hematopoietic stem cell transplantation (2).

One of the rare but clinically significant complications of AML is diabetes insipidus (DI), a disorder characterized by impaired water balance due to inadequate secretion or action of antidiuretic hormone arginine vasopressin (AVP) (3, 4).

DI is classified into two main types: central diabetes insipidus (CDI), caused by deficient AVP production due to hypothalamic or pituitary dysfunction, and nephrogenic diabetes insipidus, resulting from renal resistance to AVP (5). In AML, CDI may result from leukemic infiltration, ischemia, hemorrhage, or autoimmune mechanisms targeting the hypothalamic–pituitary axis (6).

Recognizing DI in AML patients is crucial, as delayed diagnosis can lead to severe hypernatremia, dehydration, and hemodynamic instability (7).

This report describes a rare case of AML complicated by CDI, highlighting the clinical, radiologic, and laboratory findings, as well as the associated diagnostic and therapeutic challenges.

2. Case Presentation

A 30-year-old man was admitted to our hospital with a one-month history of upper respiratory tract symptoms, including sore throat, productive cough, and intermittent hemoptysis. Initial outpatient management included intramuscular penicillin G, dexamethasone, and a cough suppressant, followed by a five-day course of oral levofloxacin, which provided transient relief.

Notably, the patient presented with a five-year history of self-referred phlebotomy sessions performed at irregular intervals (approximately every 3 to 4 months), during which 300–500 mL of blood was withdrawn per session. He attributed this practice to perceived "detoxification" benefits, though no formal medical indication was identified. During the current illness, he underwent an additional 300 mL phlebotomy session at a local clinic, after which he developed progressive fatigue, anorexia, and unintentional weight loss (8 kg over 4 weeks). Concurrently, he reported new-onset xerostomia, polydipsia (6–7 L/day), and polyuria (5–6 L/day), prompting further evaluation. Initial laboratory findings revealed anemia and thrombocytopenia: white blood cells (WBC)**:** count, 4.4 × 10^9/L, hemoglobin (Hb)**:** level, 9 g/dL, platelets count, 28 × 10^9/L, serum sodium level, 140 mmol/L, serum potassium level, 4 mmol/L, creatinine level, 1.26 mg/dL, blood urea nitrogen**:** level, 15 mg/dL.

Given the presence of bicytopenia, he was referred to a hematologist and admitted for further evaluation. On admission, the patient appeared cachectic (BMI: 17.8 kg/m²) and pale.

Vital signs included a blood pressure of 120/80 mmHg, a regular heart rate of 110 beats/min, a body temperature of 37.5°C, and an oxygen saturation of 94% on room air. Physical examination revealed petechiae on the palate and ecchymoses on the extremities. Neurological and abdominal examinations were unremarkable.

Repeat blood tests showed pancytopenia: WBC: 2.8 × 10^9/L (neutrophils: 17.9%), Hb: 9.8 g/dL, MCV: 107 fL, platelet count: 30 × 10^9/L, PT: 13 seconds, INR: 1.17, PTT: 24 seconds, creatinine: 1.16 mg/dL, phosphorus: 4.9 mg/dL, sodium:138 mmol/L, and potassium:4.13 mmol/L. Given these findings, further endocrine workup revealed impaired glucose metabolism: HbA1c: 6.1%, fasting blood glucose: 148 mg/dL.

Bone marrow aspiration and biopsy were performed. Flow cytometry of the marrow aspirate revealed a predominant population of immature myeloid cells (45% blasts) with the following immunophenotype:

- B-cell markers: CD19 (2%), CD20 (2%), CD22 (2%)
- T-cell markers: CD2 (19%), CD3 (18%), CD7 (16%)
- Myeloid markers: CD13 (29%), CD15 (5%), CD33 (45%), CD14 (negative)
- Non-lineage markers: CD10 (negative), CD11b (4%), HLA-DR (14%)
- Stem/progenitor markers: CD34 (4%), CD38 (55%), CD117 (41%)
- Other markers: CD45 (71%), CD64 (19%), Glycophorin A (negative), CD41 (negative)

The findings indicated a predominance of immature myeloid cells with CD33, CD13, and CD117 positivity, supporting a diagnosis of AML-M2, and chemotherapy initiation was recommended. However, the patient declined treatment and left the hospital against medical advice. One week later, the patient was readmitted with epistaxis, fever (39.9°C), and presyncope. Chest CT scan revealed bilateral pleural effusions and diffuse alveolar opacities with a halo sign, suggestive of pneumonia. Empiric antibiotic and antifungal therapy were initiated. Given persistent tachycardia and dyspnea, pulmonary embolism was suspected. However, pulmonary CTA ruled out thromboembolism and showed only diffuse ground-glass opacities (GGO). Echocardiography was normal (EF: 55%), and abdominal ultrasound showed no hepatosplenomegaly. Induction chemotherapy with a 7+3 regimen (idarubicin 12 mg/ m² daily for 3 days and cytarabine 100 mg/ m² by continuous infusion for 7 days) was administered alongside granulocyte colony-stimulating factor (G-CSF; 300 mcg/day).

Persistent polyuria and polydipsia prompted further workup. Urinalysis revealed low specific gravity (1.003), sterile pyuria, and normal glucose. Despite glycemic control, symptoms persisted. Serum sodium fluctuated between 138–145 mmol/L, and potassium dropped to <3.5 mmol/L during chemotherapy, requiring IV potassium replacement. The patient was eventually discharged in improved condition. Following two cycles of chemotherapy, repeat marrow biopsy showed persistent AML (40% blasts). Salvage therapy with high-dose cytarabine (HiDAC; 3 g/m² every 12 hours on days 1–6) was initiated. The patient continued to report polydipsia (>6 L/day) and polyuria despite anxiolytic treatment for presumed psychogenic polydipsia. Subsequent labs revealed serum sodium levels of 148–156 mmol/L, serum osmolality of 298 mOsm/kg, urine osmolality of 159 mOsm/kg, and urinary sodium of 125 mmol/L. A desmopressin challenge led to a >50% increase in urine osmolality (to 650 mOsm/kg), confirming CDI. MRI findings were consistent with CDI, showing a relative decrease in size of the posterior pituitary hyperintensity with rightward deviation of the pituitary stalk. Intranasal desmopressin (0.05 mL, 20 U twice daily) was started, resulting in symptomatic improvement. Despite therapy, the patient failed to achieve remission and was referred for FLAG-NK cell therapy. He died two weeks' post-discharge due to progressive disease.

3. Discussion

Diabetes insipidus (DI) is rare but clinically significant in patients with hematologic malignancies, including acute myeloid leukemia (AML). CDI may precede, coincide with, or follow the diagnosis of AML. In some cases, it may be the initial presenting feature, emphasizing the need for clinicians to maintain a high index of suspicion in patients presenting with unexplained polyuria and hypernatremia (8). In our case, symptoms of CDI developed early in the disease course and persisted despite chemotherapy, suggesting persistent hypothalamic–pituitary dysfunction. Due to the absence of electrolyte disturbances and an elevated HbA1c level, these symptoms were initially attributed to hyperglycemia.

However, over time, approximately 2.5 months after definitive AML diagnosis the patient was ultimately diagnosed with central DI. In comparing our case with published reports, karyotypic analyses in previous studies have suggested a potential association between DI and chromosomal abnormalities, particularly involving chromosomes 3 and 7 (6, 9, 10). In our case, due to the patient's lack of cooperation and subsequent mortality, genetic testing and karyotypic evaluation could not be performed. However, the refractory nature of his AML, persistent CDI despite intensive chemotherapy, and poor overall outcome closely resemble the poor-risk profiles reported in cases with monosomy 7 and inv (3) (q21q26) (8). Management of CDI includes AVP replacement, typically with intranasal or oral desmopressin. In AML-associated CDI, the clinical course may correlate with leukemic control; however, some cases show persistent CDI despite hematologic remission, while others experience resolution after successful treatment (7). Our patient's CDI persisted despite induction and salvage chemotherapy. Regarding neuroimaging findings, while some studies have reported normal brain CT and MRI findings in patients with AML-associated DI (9), others have documented loss of high signal intensity in the neurohypophysis on T1-weighted MRI scans (8). This latter finding was consistent with the MRI results in our case. While direct leukemic infiltration was not histologically confirmed, the radiologic abnormalities and clinical response to desmopressin supported the diagnosis of CDI secondary to AML.

4. Conclusion

This case highlights the complex interplay between hematologic malignancies and endocrine dysfunction. CDI in AML is rare but poses significant diagnostic and therapeutic challenges.

Early endocrinological evaluation, including the desmopressin challenge test and neuroimaging, is essential to prevent life-threatening electrolyte imbalances. Further research is needed to elucidate the underlying mechanisms linking AML, cytogenetic abnormalities, and pituitary dysfunction to improve early detection and management strategies.

5. Declarations

5.1 Acknowledgments

We would like to thank the patient and his family for their cooperation in preparation of this case report.

5.2 Ethical Considerations

Written informed consent was obtained from the patient for the publication of this case report and any accompanying images. This study was approved by the Research Ethics Committee of Tabriz University of Medical Sciences under the approval code IR.TBZMED.REC.1404.017.

5.3 Authors' Contributions

All authors contributed to the study conception, data collection, manuscript drafting, and final approval of the final version.

5.4 Conflict of Interest

The authors declare no conflicts of interest regarding the publication of this manuscript.

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5.6 Using Artificial Intelligence Tools (AI Tools)

The authors declare that no artificial intelligence (AI) tools were used in the writing or analysis of this study.

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