

LETTER TO EDITOR (VIEWERS CHOICE)

HEMATOLOGICAL MANIFESTATIONS IN ADDISON'S DISEASE

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KEYWORDS

Addison's disease, Hematological manifestations, Pediatrics Endocrinology, Pediatrics Hematology.

ARTICLE HISTORY

Received 13 November 2024

Accepted 24 February 2025

Clinical findings in Addison's disease can be diverse and are rarely associated with underlying hematological abnormalities.

We present a previously healthy 14-year-old female transferred to a tertiary-level hospital after receiving intravenous fluid therapy during a two-day hospitalization. She had a one-month history of epigastric pain, anorexia and 6 Kg weight loss (12%). Nausea and occasional vomiting became persistent in the week before admission. In physical examination, moderate to severe signs of dehydration were evident. Blood analysis showed non-regenerative normocytic normochromic anemia (Hb 9,4 g/dL; RDW 13%; Reticulocytes 31,200/ μ L; normal peripheral blood smear), hyponatremia (128 mmol/L) and hypochloremia (101 mmol/L). Hydroelectrolyte corrections were performed, leading to improvement. During her hospital stay, thoracic imaging (X-ray and tomography) was performed due to dyspnea episodes, revealing a small to medium-sized right pleural effusion, three subpleural micronodules and multiple lymph nodes. The largest lymph node measured 12 mm in diameter with central hypodensity, suggestive of necrosis. Infectious and immune causes were excluded and elevated ACE levels (197,9 U/L) were detected. Due to clinical symptoms, the persistent non-regenerative anemia and the development of mild neutropenia (1420/ μ L), a bone marrow examination was conducted, indicating a hypocellular bone marrow (50% of cellularity) and excluding hematologic oncological disease. A lymph node aspiration biopsy was also performed, which did not reveal evidence of malignancy in cytology and immunophenotyping. After 17 days restricted of physical activity, she was asymptomatic, with a resolution of the hyponatremia, neutropenia (4200/ μ L) and improved anemia (Hb 11,3 g/dL; reticulocytes 141,700/ μ L), she was discharged with suspicion of sarcoidosis. Due to a recurrence of symptoms, after minimal physical effort, she returned to the emergency department three days after

discharge. Blood analysis detected metabolic acidosis (pH 7,25; HCO₃ 11,7 mmol/L), hyponatremia (127 mmol/L), hypochloremia (99 mmol/L), hyperkalemia (5,1 mmol/L) and hypoglycemia (35 mg/dL) and worsening of the anemia (Hb 10,2 g/dL). Given the clinical and laboratory manifestations, the possibility of an adrenal crisis was considered. Confirmation was obtained through hormonal assays: Aldosterone 7 pg/mL (50–250 pg/mL), Cortisol 0,3 μ g/dL (6–23 μ g/dL in the morning), Renin 310 pg/mL (5–20 pg/mL) and ACTH 2630 pg/mL (10–60 pg/mL) and the presence of anti-adrenal antibodies (1/32), leading to the diagnosis of Addison's disease. Around 3 months after starting treatment for Addison's disease with glucocorticoid and mineralocorticoid supplementation (Hydrocortisone and Fludrocortisone), she was asymptomatic, with normalization of ACE levels, anemia (Hb 13,1 g/dL), pleural effusion and lymphadenopathy, maintaining the presence of the 3 subpleural micronodules.

Addison's disease is a primary adrenal disorder characterized by insufficient production of cortisol and aldosterone and increased levels of ACTH_{1,2}. Despite being rare, hematological manifestations linked to glucocorticoid deficiency can occur. These nonspecific hematological changes include normocytic normochromic anemia, neutropenia, lymphocytosis and eosinophilia.^{1,2} In this case, two of the most common hematological abnormalities associated with Addison's disease were identified. This case highlights the importance of a holistic approach to patients to avoid unnecessary investigation and to ensure timely and appropriate treatment, thereby preventing potentially life-threatening complications.

Compliance with Ethical Standards

Funding: None

Conflict of Interest: None

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