

Case Report

Adrenal Histoplasmosis: A Rare Cause of Primary Adrenal Insufficiency in an Immunocompetent Host

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Abstract

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Background: Adrenal histoplasmosis is an uncommon yet important cause of primary adrenal insufficiency, often misdiagnosed due to its nonspecific presentation. This case highlights the diagnostic and management challenges of adrenal histoplasmosis in an immunocompetent patient.

Case Presentation: A 42-year-old immunocompetent male presented with progressive fatigue, weight loss and postural dizziness for six months. Physical examination revealed pallor, postural hypotension and a BMI of 19 kg/m². Systemic examination was unremarkable except for hypotension (132/86 mmHg with a postural drop of 32 mmHg). Laboratory investigations showed anemia (Hb 8.6 g%), hyponatremia (Na 127 mmol/L), hyperkalemia and an elevated ESR (58 mm/h). Endocrine evaluation revealed a low morning cortisol level (2.3 µg/dL) with elevated ACTH (148 pg/mL), suggestive of primary adrenal insufficiency.

Imaging revealed bilateral adrenal enlargement with peripheral enhancement on Contrast-Enhanced Computed Tomography (CECT) of the abdomen. Ultrasound-guided Fine-Needle Aspiration Cytology (FNAC) of the adrenal glands demonstrated intracellular 2-3 micron spores within histiocytes and giant cells, confirming *Histoplasma capsulatum* infection. Culture further supported the diagnosis. The patient was treated with intravenous liposomal amphotericin B, followed by oral itraconazole, along with hydrocortisone replacement therapy. He showed significant clinical improvement with normalization of adrenal function on follow-up.

Conclusion: Adrenal histoplasmosis should be considered in the differential diagnosis of primary adrenal insufficiency, even in immunocompetent patients, especially in endemic regions. Early diagnosis with FNAC and culture, along with prompt antifungal therapy, is crucial for preventing adrenal crisis and improving patient outcomes.

Keywords: Adrenal Histoplasmosis; Primary Adrenal Insufficiency; *Histoplasma capsulatum*; Fungal Adrenalitis; Immunocompetent Host

Introduction

Adrenal histoplasmosis is an uncommon but significant cause of primary adrenal insufficiency, primarily seen in endemic areas such as Southeast Asia and the Americas. The disease is often underdiagnosed due to its nonspecific clinical presentation and insidious progression. Most reported cases occur in immunocompromised individuals, but an increasing number of cases are being identified in immunocompetent hosts. Early recognition and appropriate antifungal treatment are essential to prevent life-threatening adrenal crisis [1].

Case Presentation

A 42-year-old male, a farmer from eastern India, presented with complaints of fatigue, unintentional weight loss (7 kg over six months) and recurrent postural dizziness. He denied fever, chronic cough, night sweats or gastrointestinal symptoms. There was no history of tuberculosis exposure, HIV risk factors or immunosuppressive therapy [2,3].

Examination Findings: The patient appeared cachectic with pallor but no lymphadenopathy, cyanosis, icterus or pedal edema. Blood pressure was 132/86 mmHg, with a postural drop of 32 mmHg. Systemic examination was unremarkable, with normal cardiorespiratory and neurological assessments.

Laboratory Investigations

Parameter	Result	Normal Range
Hemoglobin	8.6 g%	13.0-17.0 g%
ESR	58 mm/h	<20 mm/h
Serum Creatinine	1.1 mg/dL	0.6-1.2 mg/dL
Sodium	127 mmol/L	135-145 mmol/L
Potassium	4.4 mmol/L	3.5-5.0 mmol/L
HbA1c	7.6%	<5.7%
TSH	3.4 IU/mL	0.5-5.0 IU/mL
Free T4	1.22 ng/dL	0.8-2.0 ng/dL
8 AM Cortisol	2.3 µg/dL	5-25 µg/dL
8 AM ACTH	148 pg/mL	<46 pg/mL
HIV	Negative	-
Urine RE/ME/CS	NAD	-
Chest X-ray	NAD	-
USG Abdomen	Bilateral adrenomegaly	-
CECT Abdomen	Bilateral peripherally enhancing hypodense adrenals	-
UGIE	NAD	-
NCV	Mild mixed polyneuropathy of LL	-
FNAC Adrenals	2-3 micron spores within histiocytes and giant cells	-
Culture	Growth of <i>Histoplasma capsulatum</i>	-

Diagnosis and Treatment

A diagnosis of adrenal histoplasmosis leading to primary adrenal insufficiency was made. The patient was started on intravenous liposomal amphotericin B (3 mg/kg/day) for two weeks, followed by oral itraconazole (200 mg twice daily for 12 months). Hydrocortisone replacement therapy was initiated to manage adrenal insufficiency. Over the following months, the patient showed significant clinical improvement with weight gain and normalization of blood pressure.

Discussion

Histoplasmosis is a fungal infection caused by *Histoplasma capsulatum*, commonly affecting the lungs but with potential systemic dissemination, including the adrenal glands. Adrenal involvement occurs via hematogenous spread, particularly in chronic disseminated histoplasmosis. While most cases occur in immunocompromised hosts, our case highlights its occurrence in an immunocompetent patient, emphasizing the importance of early recognition. Bilateral adrenal enlargement with peripheral enhancement on imaging is a characteristic feature. FNAC with fungal culture remains the gold standard for diagnosis. Delayed diagnosis can result in adrenal crisis, emphasizing the need for prompt antifungal therapy.

Conclusion

Adrenal histoplasmosis is an important differential diagnosis of primary adrenal insufficiency, even in immunocompetent individuals. Clinicians should maintain a high index of suspicion, particularly in endemic regions. Early diagnosis using FNAC and culture, coupled with antifungal therapy and corticosteroid replacement, is essential for optimal patient outcomes.

Conflict of Interest

The authors declare that they have no conflict of interest.

Consent to Participate

Not applicable.

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Data Availability

Not applicable.

Author's Contribution

The authors contributed equally.

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