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Case Report

Primary Adrenal Insufficiency Presenting with Primary Hypogonadism

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ABSTRACT
Addison's disease is a rare endocrine disorder affecting adrenal function, leading to reduced cortisol and aldosterone production (Husebye et al., 2014). Primary hypogonadism in men results from testicular dysfunction, causing low testosterone levels and elevated gonadotropins (Wu et al., 2008). The simultaneous occurrence of both conditions is extremely rare. This case study presents a 56-year-old male with symptoms of generalized weakness, salt cravings, gynecomastia, and sexual dysfunction. The patient had a history of incomplete tuberculosis treatment and underwent left-sided orchiectomy for a testicular abscess. Laboratory investigations confirmed primary adrenal insufficiency and primary hypogonadism. Imaging revealed adrenal calcifications, suggesting a tubercular etiology. The patient was treated with hydrocortisone, fludrocortisone, and testosterone replacement therapy, resulting in significant clinical improvement. This case highlights the importance of recognizing endocrine dysfunction in patients with a history of tuberculosis and emphasizes the role of comprehensive hormone replacement therapy in management.

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INTRODUCTION

Addison's disease, or primary adrenal insufficiency, is a rare disorder caused by the destruction of the adrenal glands, most commonly due to autoimmune adrenalitis, infections like tuberculosis, or adrenal damage (Betterle et al., 2002). It leads to reduced production of cortisol and aldosterone, resulting in symptoms such as fatigue, weight loss, hyperpigmentation, dizziness, and salt cravings. The condition affects 40–60 people per million worldwide (Brosnan & Gowing, 1996).

Primary hypogonadism in men occurs due to testicular dysfunction, leading to reduced testosterone levels and elevated gonadotropins (LH and FSH). Symptoms include reduced libido, infertility, erectile dysfunction, and gynecomastia (Wu et al., 2008). Causes include genetic conditions, infections, trauma, or autoimmune diseases.

The co-occurrence of Addison's disease with primary hypogonadism is exceptionally rare. This case report describes a 56-year-old male presenting with both conditions, likely due to incompletely treated tuberculosis. The objective is to document the diagnostic challenges and management strategies for this rare combination.

CASE REPORT: Co-occurrence of Addison's Disease and Primary Hypogonadism in a 56-Year-Old Male

Patient History: A 56-year-old male presented with generalized weakness, easy fatigability, salt cravings, sexual dysfunction, gynecomastia, dizziness on standing, and multiple falls over the past five years. He had no known comorbidities, no history of substance use, and no significant family history.

Medical History:

- History of pulmonary tuberculosis seven years ago, with incomplete treatment (drug defaulter).
- Left-sided orchiectomy for a testicular abscess.

Examination Findings:
Vital signs:

- Temperature: Normal
- Pulse: 88 beats/min

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- Blood pressure: 132/82 mmHg (lying down), 112/72 mmHg (standing up)
- SpO_2: 97% (room air)
- Skin hyperpigmentation on gums, tongue, and palm creases.
- Absence of secondary sexual characteristics and presence of gynecomastia.

Investigations:

Blood Chemistry

- Hyponatremia: 126 mEq/L (normal: 135–145 mmol/L)
- Hyperkalemia: 5.3 mEq/L (normal: 3.5–5 mmol/L)
- Random blood glucose: 116 mg/dL
- Complete blood count: Normal

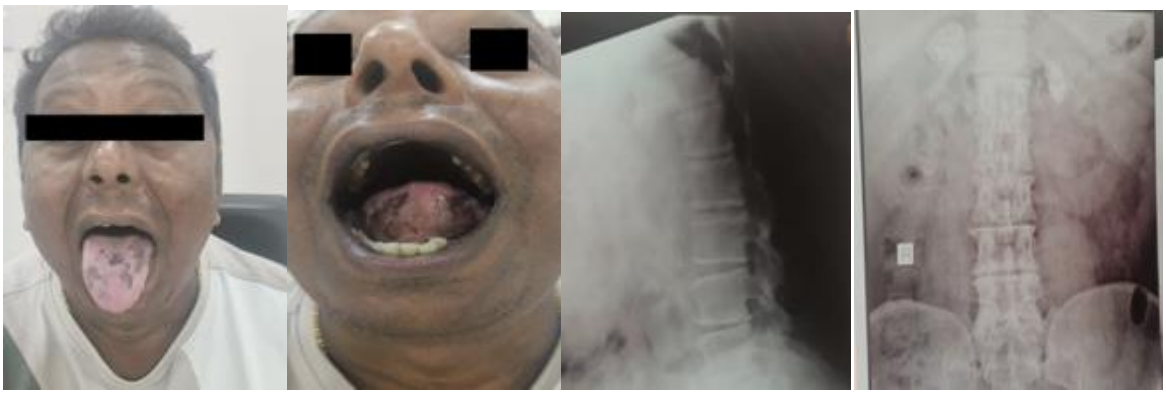
Adrenal Function Tests

- Morning cortisol: 3.4 mcg/dL (normal: 5–23 mcg/dL)
- ACTH: 490 pg/dL (normal: 9–52 pg/dL)
- Direct renin concentration: 485.40 µU/mL (normal: 4.4–46.1 µU/mL)

Labs	Patient's results	Reference Range
S. Sodium	126	135-145 mmol/l
S. Potassium	5.3	3.5-5 mmol/l
Cortisol	3.4	5-23 mcg/dl
ACTH	490	9-52 pg/dl
Direct Renin Concetration	485.40	4.4-46.1 MicroU/mL
LH	26.67	1.5-9.3
Testosterone Total	1.30	2.27-10.3

Imaging

Abdominal X-ray showed bilateral adrenal gland calcifications, consistent with primary adrenal insufficiency (Erichsen et al., 2009).



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Hormonal Evaluation

- Luteinizing hormone (LH): 26.67 IU/L (normal: 1.5–9.3 IU/L)
- Testosterone: 1.30 ng/mL (normal: 2.27–10.3 ng/mL)
- Follicle-stimulating hormone (FSH): Normal
- Findings confirmed primary hypogonadism.

Other Investigations

- ECG, chest X-ray, and abdominal ultrasound: Normal.
- Brain MRI ruled out Nelson syndrome.

Diagnosis: The patient was diagnosed with primary adrenal insufficiency and primary hypogonadism, likely secondary to incompletely treated tuberculosis.

Treatment and Outcome:

The patient was treated with:

- Hydrocortisone: 10 mg in the morning, 5 mg in the evening.
- Fludrocortisone: 100 mcg daily.
- Testosterone replacement**:** 250 mg intramuscular injections.

Following treatment, the patient showed significant improvement in energy levels, salt cravings, dizziness, and sexual function, allowing him to resume normal daily activities.

DISCUSSION

The coexistence of Addison's disease and primary hypogonadism is exceptionally rare. Addison's disease is commonly caused by autoimmune destruction, but in regions with a high prevalence of tuberculosis, adrenal tuberculosis remains a significant cause (Vaidya et al., 2009). Adrenal calcifications in this case strongly suggest tubercular adrenalitis as the underlying etiology.

Testicular dysfunction leading to primary hypogonadism can be due to infections, trauma, or autoimmune processes. This patient's history of left-sided orchiectomy for a testicular abscess indicates a past infectious or inflammatory event, which may have contributed to testicular dysfunction. Elevated LH with low testosterone levels confirmed the diagnosis of primary hypogonadism.

Endocrine dysfunction in tuberculosis is often overlooked. The destruction of the adrenal glands due to granulomatous inflammation can lead to adrenal insufficiency, and testicular involvement can result in hypogonadism. The gradual onset of symptoms over five years in this case made early diagnosis challenging.

CONCLUSION

This case highlights a rare combination of Addison's disease and primary hypogonadism, likely secondary to incompletely treated tuberculosis. The patient presented with symptoms of adrenal insufficiency and androgen deficiency, confirmed through laboratory findings and imaging. Treatment with glucocorticoids, mineralocorticoids, and testosterone replacement led to significant clinical improvement.

This case emphasizes the importance of recognizing endocrine complications in patients with a history of tuberculosis and the role of comprehensive hormone replacement therapy in improving quality of life. Early recognition and appropriate treatment are crucial for managing such rare endocrine disorders.

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