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Tuberculous Addison's Disease Presenting as Primary Adrenal Insufficiency: A Case Report From a Tuberculosis-Endemic Region

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Abstract

Primary adrenal insufficiency is a rare but life-threatening disorder, most often autoimmune in developed countries but still frequently caused by tuberculosis in endemic regions. We report a 42-year-old man with previously treated spinal tuberculosis who presented with progressive hyperpigmentation, fatigue, and recurrent presyncope. Evaluation revealed diffuse pigmentation, postural hypotension, low cortisol, markedly elevated ACTH, hyponatremia, hyperkalemia, and high plasma renin activity. CT showed bilateral adrenal enlargement with central necrosis, and biopsy confirmed caseating granulomas diagnostic of adrenal tuberculosis. The patient improved with anti-tuberculosis therapy and steroid replacement, highlighting the importance of considering adrenal tuberculosis in the differential diagnosis of Addison's disease in tuberculosis-endemic regions.

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Introduction

Primary adrenal insufficiency (PAI), also known as Addison's disease, is an endocrine disorder characterized by the insufficient production of glucocorticoids, mineralocorticoids, and adrenal androgens due to the destruction of the adrenal cortex. (1) The clinical presentation is often insidious, with nonspecific symptoms such as chronic fatigue, weight loss, and gastrointestinal disturbances, which can delay diagnosis. More specific signs, including hyperpigmentation of the skin and mucosa and postural hypotension, often appear later in the disease course. (2) The etiology of PAI varies geographically. In the Western world, autoimmune adrenalitis accounts for over 80% of cases. (3) However, in developing and TB-endemic countries like India,

tuberculosis remains a significant cause. (4) Tuberculous involvement of the adrenal glands is now uncommon globally but remains clinically relevant in regions where tuberculosis is endemic. Tuberculous adrenalitis results from the hematogenous spread of *Mycobacterium tuberculosis* to the adrenal glands. Although clinical signs of adrenal insufficiency typically manifest only after more than 90% of the adrenal cortex has been destroyed, the diagnosis requires a high index of suspicion. (5)

We report the case of a 42-year-old male who presented with classic features of Addison's disease and was subsequently diagnosed with adrenal tuberculosis, underscoring the need for continued vigilance for this infectious etiology.

Case Presentation

A 42-year-old male presented with a two-year history of progressive generalized darkening of the skin, most prominent on his face, hands, palmar creases, and buccal mucosa. The hyperpigmentation was insidious in onset and gradually worsened. He also complained of persistent fatigue, generalized weakness, and unintentional weight loss of 5 kg over the past year.

Additionally, he described three episodes of presyncope characterized by lightheadedness and near-fainting on prolonged standing. His past medical history was significant for spinal tuberculosis (Pott's spine) ten years earlier, for which he had completed a full course of anti-tuberculosis therapy. There was no history of autoimmune disease, diabetes, or other chronic illness.

On examination, the patient was alert and oriented but appeared fatigued. His supine blood pressure was 120/70 mmHg with a pulse rate of 88 beats/min, which fell to 100/60 mmHg and 104 beats/min, respectively, after standing for three minutes, consistent with postural hypotension. There was diffuse bronze-like hyperpigmentation over sun-exposed areas, palmar creases, knuckles, and the oral mucosa. The remainder of the systemic examination was unremarkable.

Initial biochemical investigations revealed hyponatremia (129 mEq/L) and hyperkalemia (5.7 mEq/L), with normal renal and liver function tests. Random blood glucose was 78 mg/dL. Morning (8 AM) serum cortisol was markedly reduced at 2.8 µg/dL (reference range: 5–25 µg/dL), while plasma ACTH was significantly elevated at 850 pg/mL (reference: <46 pg/mL). Plasma renin activity was also raised (28 ng/mL/hr; normal supine: 0.5–1.6 ng/mL/hr). An ACTH stimulation test using 250 µg of cosyntropin produced an inadequate cortisol response, with a peak level of 3.5 µg/dL at 60 minutes, confirming primary adrenal insufficiency.

Contrast-enhanced CT of the abdomen showed bilateral adrenal enlargement with peripheral rim enhancement and central hypodense necrotic areas, without calcification, findings highly suggestive of adrenal tuberculosis. To confirm the diagnosis, a CT-guided fine-needle aspiration biopsy of the right adrenal gland was performed, which revealed epithelioid cell granulomas with central caseous necrosis and Langhans-type giant cells. Ziehl-Neelsen staining demonstrated acid-fast bacilli, confirming the diagnosis of tuberculous adrenalitis.

The patient was initiated on hormone replacement therapy with oral hydrocortisone (15 mg in the morning and 5 mg in the evening) and fludrocortisone (0.1 mg once daily). He was counseled regarding stress dosing of glucocorticoids during illness and the need for lifelong hormone replacement. Concurrently, anti-tuberculosis therapy was started with isoniazid 300 mg, rifampicin 600 mg, pyrazinamide 1500 mg, and ethambutol 1200 mg daily for two months, followed by a continuation phase with isoniazid and rifampicin for seven additional months, for a total duration of nine months.

At a three-month follow-up, the patient showed significant clinical improvement, with resolution of fatigue and postural symptoms. Serum sodium and potassium levels normalized, and the diffuse hyperpigmentation had begun to fade. He tolerated anti-tuberculosis therapy well without adverse effects. Lifelong adrenal hormone replacement was continued, and regular follow-up with endocrinology and infectious disease services was advised.

Discussion

Adrenal tuberculosis develops from the hematogenous spread of *M. tuberculosis* from a primary site, most commonly the lungs. (6) The adrenal glands are highly vascular organs, making them susceptible to such dissemination. However, adrenal function is typically preserved until over 90% of the cortical tissue is destroyed, leading to a long latent period between initial infection and clinical presentation. (5) Our patient's history of spinal TB a decade prior is consistent with this pathophysiology of reactivation or slow progression of a latent adrenal focus. Tuberculosis historically accounted for the majority of Addison's disease cases before the recognition of autoimmune adrenalitis.

The clinical presentation, as seen in our patient, is classic for Addison's disease. Hyperpigmentation, a hallmark sign, results from the elevated levels of ACTH and other pro-opiomelanocortin-derived peptides stimulating melanocytes. (2) Postural hypotension is a direct consequence of mineralocorticoid deficiency leading to volume depletion.

CT imaging plays a crucial role in the etiological diagnosis. In active adrenal tuberculosis, CT typically shows bilateral, symmetrical enlargement of the adrenals with peripheral enhancement and central necrosis. (7) In later, chronic stages, the glands may become atrophic and calcified.

Figure.1 Generalized cutaneous and mucosal hyperpigmentation

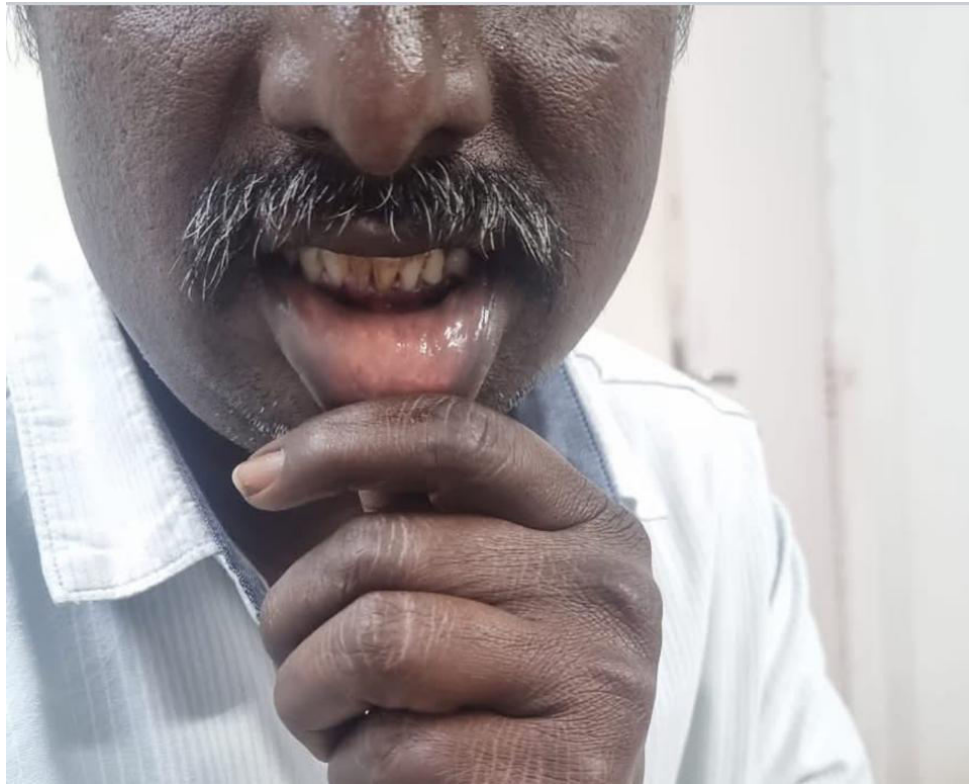


Figure.2 CT-guided biopsy of the right adrenal gland

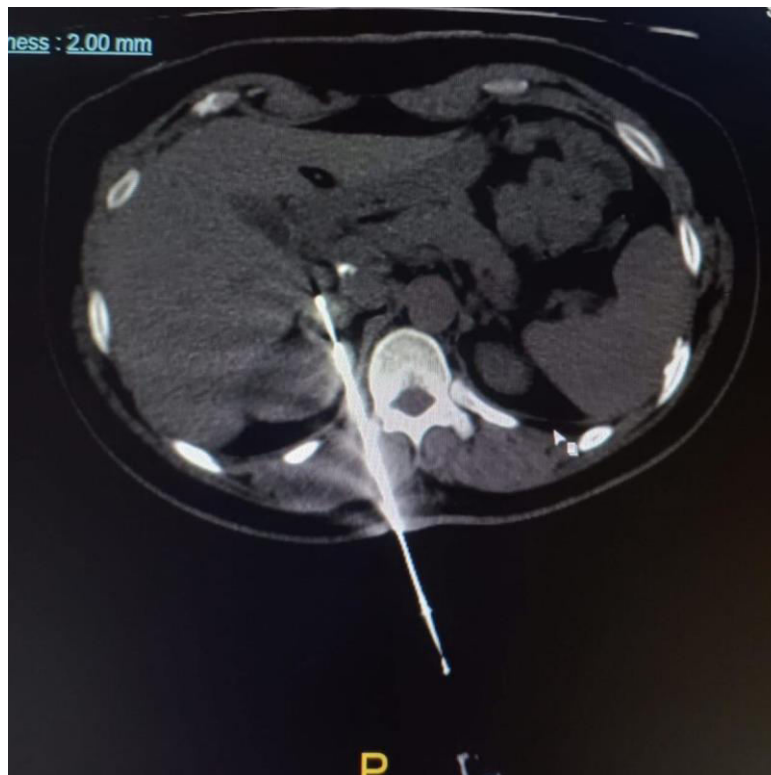


Figure.3 Contrast-enhanced CT showing bilateral adrenal enlargement with central necrosis

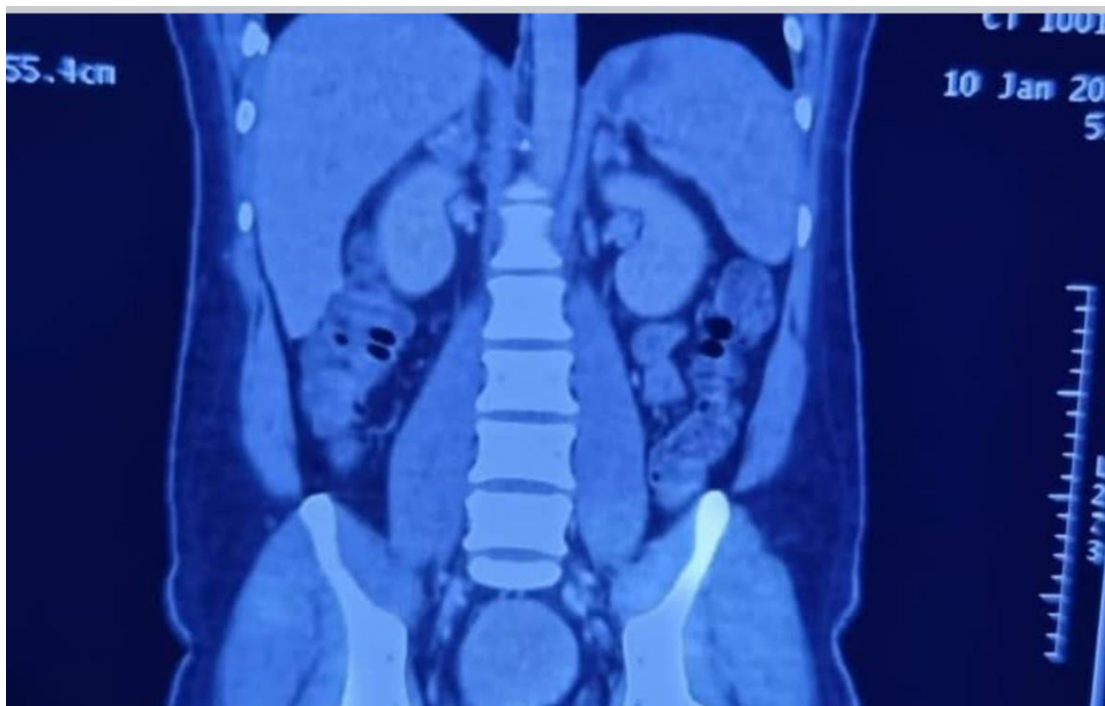
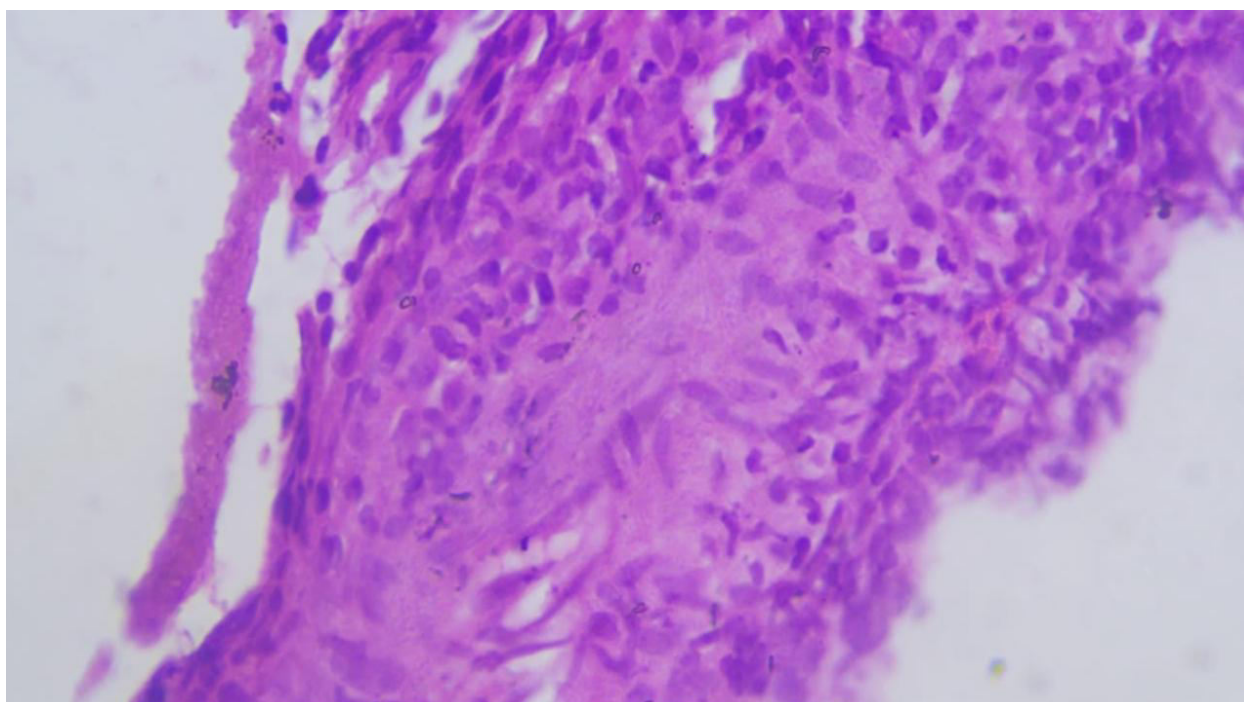


Figure.4 Histopathology showing epithelioid granuloma with caseous necrosis



Our patient's imaging findings were classic for the active stage of the disease. While imaging is highly suggestive, histopathological or microbiological confirmation is the gold standard for diagnosis.

The management of tuberculous Addison's disease is twofold: lifelong hormone replacement for the irreversible adrenal insufficiency and a full course of anti-tuberculosis therapy to eradicate the infection. (8) It

is important to note that initiating ATT can sometimes accelerate the metabolism of hydrocortisone (due to enzyme induction by rifampicin), potentially necessitating an increase in the glucocorticoid dose. (4)

Adrenal function recovery is rare, even with successful treatment of the underlying tuberculosis.

In conclusion, this case highlights that tuberculosis remains a vital and treatable cause of primary adrenal insufficiency in endemic settings.

A high index of suspicion should be maintained in patients presenting with suggestive symptoms, particularly if there is a personal history of tuberculosis.

Prompt diagnosis and initiation of both hormone replacement and anti-tuberculosis therapy are essential to prevent life-threatening adrenal crises and achieve favorable outcomes.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Conflicts of Interest

The authors have declared that no competing interests exist.

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