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Case Report

Primary Adrenal Tuberculosis Masquerading as Adrenal Adenoma: A Rare Cause of Addison's Disease

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A B S T R A C T

Background: Primary adrenal tuberculosis is a rare clinical entity and an uncommon cause of Addison's disease. While adrenal tuberculosis is usually secondary to pulmonary or genitourinary tuberculosis, isolated primary adrenal involvement without evidence of disease elsewhere is exceptionally rare.

Case presentation: We report the case of a 46-year-old male who presented with generalized weakness and disorientation. Laboratory investigations revealed markedly elevated adrenocorticotropic hormone (ACTH) levels and profoundly low morning cortisol, consistent with primary adrenal insufficiency. Contrast-enhanced CT abdomen demonstrated a right adrenal mass suggestive of a non-functional adrenal adenoma. The patient underwent right adrenalectomy; however, histopathology revealed caseous necrosis and necrotizing granulomatous lymphadenitis consistent with tuberculosis. No evidence of extra-adrenal tuberculosis was identified, confirming the diagnosis of primary adrenal tuberculosis. The patient was managed with lifelong hormone replacement therapy (hydrocortisone and fludrocortisone) and a 12-month course of antituberculous therapy.

Conclusion: This case highlights the diagnostic challenges of adrenal tuberculosis presenting as an adrenal mass. It underscores the importance of considering infectious etiologies, particularly tuberculosis, in the differential diagnosis of adrenal lesions in endemic regions.

Keywords: Primary adrenal tuberculosis; Addison's disease; Adrenal adenoma; Adrenal insufficiency; Case report

Introduction

Most typically, pulmonary and genitourinary TBs are the primary cause of adrenal TB, which is nearly invariably a subsequent condition to TB elsewhere[1].

Although any of the endocrine glands-the pituitary, thyroid, adrenal, hypothalamus, and pancreas-can be impacted by tuberculosis, the adrenal gland is the organ most frequently affected[2]. Only a small number of cases with primary adrenal TB are documented in the literature, making it a rare clinical entity. Only one instance of primary adrenal TB was found in a systematic evaluation conducted by Edlin in 1978, which examined 370 patients with extrapulmonary TB over a ten-year period[3]. Tuberculous Addison's disease has a comparatively late beginning because the majority of adrenal TB cases are discovered 10 to 15 years after the initial infection[4].

The primary adrenal insufficiency that results from the adrenal gland's inability to generate adequate steroid hormones is known as Davison's disease. Thomas Addison originally described Addison's illness in 1855. He showed how TB causes the bilateral adrenal glands to be destroyed in six cases. Although TB has become less frequent in the West since then, it continues to be the leading cause of adrenal insufficiency in poorer nations[5]. Fever, lethargy, anorexia, and stomach pain are examples of the nonspecific clinical signs and symptoms of isolated adrenal involvement in tuberculosis. Only after at least 90% of the adrenal gland has been destroyed do symptoms of adrenal insufficiency manifest[6].

Case Presentation

A 46-year-old male was brought to the emergency department of Lady Reading Hospital Peshawar with complaints of generalized weakness for several days, associated with mild disorientation at the time of presentation. There was no history of fever, cough, weight loss, night sweats, or gastrointestinal complaints. The patient did not report any prior similar episodes, nor did he have a known history of chronic medical conditions such as diabetes mellitus, hypertension, or tuberculosis. Family and social history were also unremarkable.

On examination, the patient appeared lethargic but arousable. His vital signs showed a blood pressure of 100/60 mmHg, random blood sugar of 65 mg/dL, oxygen saturation within normal range, and normal body temperature. No hyperpigmentation of the skin or mucous membranes was noted, and systemic examination did not reveal any significant abnormalities.

Initial management included intravenous fluid resuscitation, and hypertonic saline was administered in view of his low blood sugar and generalized weakness. Following stabilization, baseline investigations were sent. Complete blood count, renal and liver function tests, electrolytes, and inflammatory markers were all within normal limits. However, endocrine evaluation showed striking abnormalities: plasma adrenocorticotropic hormone (ACTH) was markedly elevated at 937.1 pg/mL, while early morning serum cortisol was profoundly reduced at 0.30 µg/mL **(Table 1)**. These findings were strongly suggestive of primary adrenal insufficiency (Addison's disease).

Table 1:

tests	results	Normal range	Interpretation
ACTH	937.1pg/mL	7.2-63.3pg/mL	Very raised ACTH
Cortisol	0,30 ug/dL	2.9-17.3	Very low cortisol
RBS	65 mg/dL	120-200mg/dL	Very low level

The other laboratory investigations also revealed several abnormalities consistent with adrenal insufficiency. The patient had hypoglycemia, with a random blood glucose level of 65 mg/dL, which can occur due to decreased gluconeogenesis in the setting of cortisol deficiency. Electrolyte analysis demonstrated hyponatremia (serum sodium 128 mmol/L) and mild hyperkalemia (serum potassium 5.6 mmol/L), findings that are commonly associated with mineralocorticoid deficiency in primary adrenal insufficiency. Additionally, the serum bicarbonate level was reduced to 19 mmol/L, indicating a mild metabolic acidosis. In contrast, other hematological parameters such as hemoglobin (13.2 g/dL) and total leukocyte count (7.8 $\times 10^9$/L) were within normal limits, suggesting the absence of significant infection or hematologic abnormalities **(Table 2)**.

Table 2:

tests	results	Normal range	Interpretation
Serum Sodium (Na^+)	128 mmol/L	135–145 mmol/L	Hyponatremia
Serum Potassium (K^+)	5.6 mmol/L	3.5–5.0 mmol/L	Mild hyperkalemia
Serum Bicarbonate	19 mmol/L	22–28 mmol/L	Mild metabolic acidosis
Hemoglobin	13.2 g/dL	13–17 g/dL	Normal
Total Leukocyte Count	7.8 $\times 10^9$/L	4–11 $\times 10^9$/L	Normal

To further evaluate the etiology, a contrast-enhanced CT scan of the abdomen with adrenal protocol was performed. Imaging revealed a well-defined, low-density, lipid-poor adrenal mass on the right side, measuring approximately 3 × 2 cm, and situated adjacent to the inferior vena cava **(Figure 1)**.

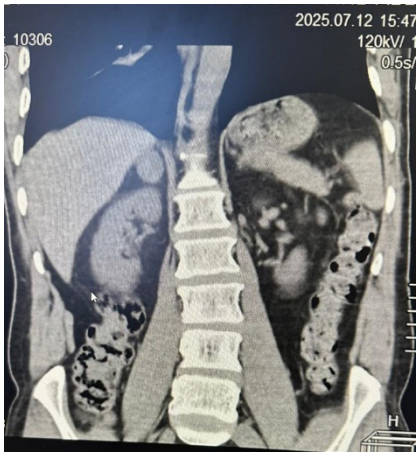

Figure 1:

The lesion was initially suspected to represent a non-functional adrenal adenoma, and in correlation with the hormonal profile, Addison's disease secondary to a non-functional right adrenal adenoma was considered the most likely diagnosis.

The patient was scheduled for right adrenalectomy. Surgery was uneventful, and the excised specimen was submitted for histopathological evaluation. To our surprise, histopathology demonstrated complete effacement of nodal architecture by caseous necrosis, along with necrotizing granulomatous lymphadenitis. These findings were consistent with tuberculosis **(Table 3)**.

Table 3:

Test	Result	Interpretation
Chest x ray	Normal	No pulmonary TB
Sputum AFB smear	Normal	No active pulmonary TB
Mantoux test	positive	Prior TB exposure
Interferon Gamma Release Assay	positive	Supports TB infection
ESR	Mildly elevated	Suggestive of inflammation
CT Chest	No lessions	No extra-adrenal TB

Following histopathological confirmation of tuberculosis, a comprehensive tuberculosis workup was performed to identify any primary focus or disseminated disease. However, no evidence of pulmonary or extra-adrenal tuberculosis was found on imaging or microbiological testing. These findings supported the diagnosis of isolated primary adrenal tuberculosis.

As there was no evidence of pulmonary or extrapulmonary tuberculosis elsewhere on clinical assessment, chest X-ray, or laboratory workup, a final diagnosis of primary adrenal tuberculosis was established. The patient was treated with lifelong adrenal hormone replacement therapy using hydrocortisone and fludrocortisone, along with a 12-month course of antituberculous medications consisting of isoniazid, rifampin, ethambutol, and pyrazinamide.

Thus, a patient initially suspected to have Addison's disease due to a non-functional adrenal adenoma was ultimately found to have primary adrenal tuberculosis, a rare presentation in the absence of any other tuberculous focus.

Discussion

Primary adrenal tuberculosis is a rare but recognized cause of Addison's disease, particularly in regions where tuberculosis is prevalent. Isolated adrenal involvement without evidence of disease elsewhere is uncommon and can pose a diagnostic challenge. In this case, the patient was initially suspected to have a non-functional adrenal adenoma based on imaging and hormonal findings, but histopathology revealed adrenal tuberculosis. This emphasizes the need to keep infectious causes in the differential diagnosis of adrenal masses.

The early diagnosis of primary adrenal insufficiency is typically hampered by nonspecific signs and symptoms, which might mimic other illnesses such gastrointestinal issues and psychological disorders. These are the typical signs of tuberculosis in the adrenal glands. According to Bleicken et al., 68% of cases were misdiagnosed before adrenal tuberculosis was confirmed. Primary adrenal insufficiency was frequently misdiagnosed as gastrointestinal diseases, as was the case in our instance. Occasionally, individuals wait until they are admitted to the hospital with a severe adrenal crisis before seeking medical help for the nebulous symptoms of weakness, fatigue, or anorexia[7].

Low morning plasma cortisol concentrations, particularly values below 83 nmol/L (or 3 µg/dL), in combination with markedly elevated plasma adrenocorticotropic hormone (ACTH) levels, are considered classic diagnostic indicators of primary adrenal insufficiency. The measurement of cortisol in the early morning is particularly important, as this is the time when endogenous cortisol secretion should normally peak. Failure of cortisol to reach an adequate level, coupled with a compensatory rise in ACTH, strongly suggests impaired adrenal function and forms the biochemical basis for diagnosing Addison's disease[8].

Adrenal biopsy may not be necessary to diagnose tuberculous adrenalitis causing primary adrenal insufficiency if the following symptoms are noted: Particularly in regions with a high TB incidence, a bilateral enlarged adrenal mass on the CT scan is accompanied by signs of active extra-adrenal tuberculous illnesses[9].

In our case, an adrenal biopsy was necessary to make a diagnosis because our patient showed no signs of extra-adrenal tuberculous illnesses.

Adrenal gland dysfunction has been documented in patients with active extra-adrenal TB. According to Laway, et al., granulomatous adrenalitis in patients with active pulmonary tuberculosis may be the source of the enlargement of the adrenal glands. Compared to healthy controls, these patients' basal and stimulated plasma cortisol levels were lower. Following TB therapy, cortisol levels and adrenal hypertrophy returned to normal[10].

Conclusion

Primary adrenal tuberculosis is an uncommon but important cause of Addison's disease, especially in tuberculosis-endemic regions. Its presentation can mimic adrenal neoplasms and is often diagnosed only after histopathological confirmation. Early recognition is crucial, as delayed diagnosis may result in adrenal crisis and life-threatening complications. This case emphasizes the need for clinicians to maintain a high index of suspicion for adrenal tuberculosis in patients presenting with adrenal insufficiency and adrenal masses, even in the absence of pulmonary or extra-adrenal tuberculosis. A combination of hormonal evaluation, imaging, and histopathology remains essential for accurate diagnosis and appropriate management.

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