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Addison's disease due to tuberculous adrenalitis

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Abstract
Addison's disease is defined as an inability of the adrenal gland to produce steroids in sufficient quantities (primary adrenal insufficiency). Tuberculosis is still the main cause of Addison's disease in developing countries. Most of the patients with adrenal tuberculosis are asymptomatic, and in those patients with symptoms, the clinical picture is nonspecific. The diagnostic approach includes measurement of morning serum cortisol levels (decreased), adrenocorticotropic hormone (elevated), characteristic findings on imaging studies, and sometimes adrenal biopsy. Regarding treatment, in addition to steroid replacement, hypovolemia and hyponatremia should be treated with adequate fluid replacement. Specific therapy against Mycobacterium tuberculosis should also be initiated. In this article, a clinical case of Addison's disease due to tuberculous adrenalitis is reported.

Keywords: Addison's disease, adrenal insufficiency, tuberculosis

Introduction
Addison's disease (AD) is defined as an inability of the adrenal gland to produce steroids in sufficient quantities (primary adrenal insufficiency) [1]. In this article, a clinical case of AD due to tuberculous adrenalitis is reported.

Case report
A 69-year-old woman with a history of diabetes and hypertension was referred for weakness, decreased appetite, abdominal pain, nausea, 8-kg weight loss, and skin hyperpigmentation of 6 months duration. She also had recurrent hypoglycemia and low blood pressure, and recently discontinued her regular medications (metformin and losartan). Physical examination revealed blood pressure of 80/60 mmHg and generalized hyperpigmentation of the skin, nails (Figure 1), and tongue (Figure 2), with no other abnormalities.
Initial laboratory studies showed hyponatremia (106 mEq/l, normal: 135-145 mEq/l) and glucose of 79 mg/dl. Thyroid profile (no abnormalities), morning serum cortisol (2.36 µg/dl, normal: 3.7-19.4 µg/dl), and morning adrenocorticotropic hormone (821 pg/ml, normal: 4-48 pg/ml) were requested, which suggested primary adrenal insufficiency as the cause of the symptoms and hyponatremia (due to the significantly low morning serum cortisol level, a dynamic short cosyntropin stimulation test was not necessary). Adrenal antibodies were negative (which ruled out autoimmune origin) and the Mantoux test was positive.
Abdominal contrast-enhanced tomography showed a simple cyst in the lower pole of the right kidney and enlargement of both adrenal glands (Figure 3), which suggested a probable tuberculous adrenalitis. There was no evidence of pulmonary tuberculosis on chest radiography, but urine stains for mycobacteria were positive (indicating renal tuberculosis). A diagnosis of adrenal insufficiency due to tuberculous adrenalitis was established. Serology for human immunodeficiency virus was negative. Treatment with prednisone, fludrocortisone, and antituberculous drugs was started, significantly improving of the clinical picture and biochemical alterations in the following months.

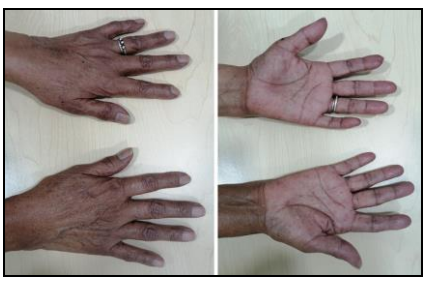

Fig 1: Hyperpigmentation of skin and nails

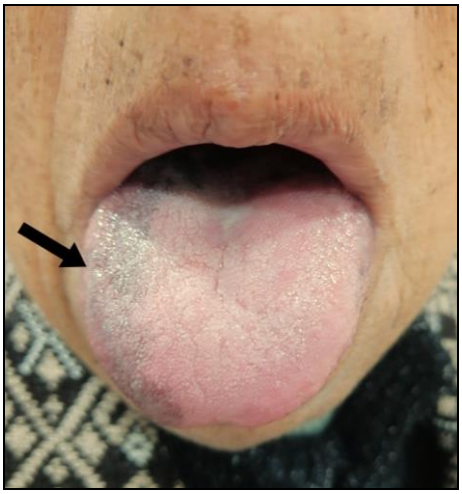

Fig 2: Hyperpigmentation of the tongue (black arrow)

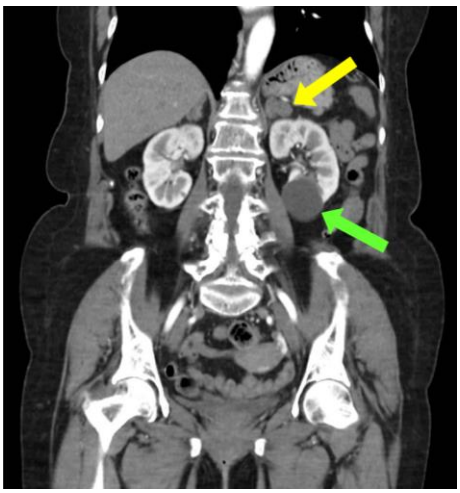

Fig 3: Bilateral adrenal enlargement (yellow arrow) and simple kidney cyst (green arrow), on CT

Discussion

Thomas Addison described the disease that bears his name in 1855 when he demonstrated bilateral adrenalitis due to tuberculosis (TB) in 6 patients [1]. Primary adrenal insufficiency requires the destruction of at least 90% of the gland to be manifest [2]. Currently, with the wide use of anti-TB drugs, the incidence of adrenal TB is reduced. In developed countries, 75-80% of AD cases are caused by autoimmune adrenalitis, whereas TB and other causes (histoplasmosis, blastomycosis, lymphoma, metastatic adrenal deposits, or bilateral adrenal hemorrhage) cause the remaining 7-20% of cases. Otherwise, TB is still the main cause of AD in developing countries [2, 3].

Genitourinary, pulmonary, or other extrapulmonary TB (or even more rarely primary due to reactivation of the disease) are the most common cause of adrenal TB [1]. The main risk factors for extrapulmonary TB are HIV infection, diabetes, chronic kidney disease, cirrhosis, female sex, advanced age, alcoholism, and smoking [4]. Most of the patients with adrenal TB are asymptomatic, and in those patients with symptoms, the clinical picture is nonspecific. Signs and symptoms of adrenal TB include anorexia (75-100%), fever (72-94%), weakness (72-100%), fatigue (70-100%), hyperpigmentation (65-94%), gastrointestinal symptoms such as nausea, vomiting, abdominal pain, constipation, and diarrhea (58-92%), hypotension (50-90%), salt cravings (5-16%), giddiness (4-12%), vitiligo (10-20%) and muscle or joint pain (5-10%) [1].

Morning serum cortisol values >20 mcg/dl (>550 nmol/l) exclude the diagnosis of AD. Levels between 5-19 mcg/dl (138-550) require confirmation with a dynamic short cosyntropin stimulation test. AD can be confirmed with significantly low morning plasma cortisol levels of <5 µg/dl (<138 nmol/l) without a confirmatory test. In addition, the diagnosis is suggested by measurement adrenocorticotropic hormone (ACTH) levels with values twice the upper limit. Among other laboratory manifestations are anemia, lymphocytosis, and eosinophilia (11-15%), hypoglycemia (18%), hypercalcemia (21%), hyponatremia (90%), and osteopenia or osteoporosis (28%) [4].

Typical features of adrenal TB on CT scans are enlarged glands, low central attenuation, and calcifications with peripheral enhancement [2, 3]. Bilateral adrenal involvement is present in 80% of cases [5]. Histopathological findings of adrenal TB include tuberculous granuloma, caseous necrosis, fibrosis, and calcification [2]. However, diagnosis of tuberculous adrenalitis may not require adrenal biopsy if the following findings are observed: bilateral enlarged adrenal mass on the CT scan and the demonstration of active extra-adrenal TB, especially in areas with a high burden of tuberculosis (as in our case) [5].

The cornerstone of AD treatment is glucocorticoid (prednisolone, hydrocortisone, or dexamethasone) and mineralocorticoid (fludrocortisone) replacement therapy. Hypovolemia and hyponatremia should be treated with adequate fluid replacement [1, 4]. Specific treatment for TB adrenalitis is the same duration as for pulmonary TB with 4 drug therapy in the intensive phase, for 8 weeks (rifampin, isoniazid, pyrazinamide and, ethambutol) followed by 2 drug therapy in the continuation phase, for 18 weeks (rifampin and isoniazid) [6]. HIV co-infection should be ruled out in patients with risk factors [4]. The prognosis of adrenal function in patients treated with anti-TB drugs is unclear. Some studies show evidence of rescue of adrenal function, although an absence of adrenal recovery has also been seen 2-5 years after therapy [6].

Conclusion

This case illustrates Addison's disease due to tuberculous adrenalitis in a 69-year-old woman with diabetes and hypertension. Symptoms included weakness, hyperpigmentation, and hypotension, with diagnostic findings of low cortisol, high ACTH, and bilateral adrenal enlargement. Treatment combined glucocorticoid and mineralocorticoid replacement with antituberculous therapy, leading to significant improvement. This highlights the need to consider adrenal tuberculosis in Addison's disease, especially in high-burden regions, and underscores the importance of comprehensive treatment strategies.

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Consent: No identifying markers are included as part of medical images. No consent is required.

References

1. Gupta S, Ansari AM, Gupta AK, Chaudhary P, Bansal LK. Current approach for diagnosis and treatment of adrenal tuberculosis-our experience and review of

literature. Surg. J (NY); c2022. p. 8-7.
DOI: https://doi.org/10.1055/s-0042-1743523.
2. Arambewela M, Ross R, Pirzada O, Balasubramanian SP. Tuberculosis as a differential for bilateral adrenal masses in the UK. BMJ Case Rep., 2019, 12.
DOI: 10.1136/bcr-2018-228532.
3. Soedarsoa MA, Nugrohob KH, Dewi M. A case report: Addison disease caused by adrenal tuberculosis. Urol. Case Rep. 2018;20:12-14.
DOI: https://doi.org/10.1016/j.eucr.2018.05.015.
4. Eslava DF, Cortés HT, Fuentes-Trespalacios R. Tuberculous adrenalitis: In regard to a recent update. Repert. Med. Cir. 2022;31(1):33-41.
DOI: 10.31260/RepertMedCir.01217372.1133.
5. Tran NQ, Phan CC, Doan TTP, Tran TV. Bilateral adrenal masses due to tuberculosis: how to diagnose without extra-adrenal tuberculosis. Endocrinol. Diabetes Metab. Case Rep. 2021;1:21-0093.
DOI: https://doi.org/10.1530/EDM-21-0093.
6. Achira AA, Abushanab D, Elbadawi H, Dhar S, Taha W. Primary adrenal insufficiency due to tuberculous adrenalitis in a patient without active pulmonary tuberculosis. J Hum Endocrinol. 2019;4:014.
DOI: 10.24966/HE-9640/100014.

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