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TEMPORARY ADRENAL FAILURE DUE TO TUBERCULOSIS: A RARE PHENOMENON IN RECENT YEARS

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INTRODUCTION

➢Tuberculosis is the most commonly encountered cause of Addison's disease. The exact status of adrenal reserve in tuberculosis still remains controversial, and recovery of adrenal function is unpredictable.

➢There are few reports in the literature suggesting that adrenal insufficiency may improve with anti-tuberculosis therapy during the active phase of the disease. Herein, we presented a case of reversible adrenal failure in a patient with active tuberculosis.

CASE REPORT

➢ A 50-year-old female patient was admitted to our clinic with the complaints of weakness and darkening of the skin. In her history, she had received anti-tuberculosis treatment for the last 8 months. On her physical examination, she had hypotension and hyperpigmentation. Other systemic examinations were in normal ranges. Laboratory findings were presented in Table 1. Thyroid function tests were within normal range, and cortisol and adrenocorticotrophic hormone (ACTH) were 8 µg/dL and 802 pg/mL, respectively. There was no cortisol response to cosyntropin stimulation test, (30th minute cortisol was 10 µg/dL, and 60th minute was 11 µg/dL). Then, we started glucocorticoid therapy. In magnetic resonance imaging, thickness of both adrenal glands were increased. Anti-21 hydroxylase antibody was negative.

➢Anti-tuberculosis treatment were stopped at the 9th month. We repeated the cosyntropin test one month after the therapy was stopped. We detected there was a positive cortisol response to ACTH (cortisol was 25 µg/dL at 30th minute and 29 µg/dL at 60th minute). We stopped the glucocorticoid since we thought adrenal failure improved.

Table 1. Laboratory findings of the case

		normal range
Hemoglobin(gr/dl)	12.9	12-16
Hemotocrit (%)	37.6	36-45
Platelet (mm^3)	189000	150000-450000
Leukocyte (mm^3)	4700	4000-11000
Glucose (mg/dl)	72	74-106
Creatinine (mg/dl)	0.65	0.5-1.2
Sodium (mmol/L)	132	136-145
Potassium (mmol/L)	4.9	3.5-5.1
Aspartate aminotransferase (U/L)	23	0-32
Alanine aminotransferase (U/L)	8	0-33
Cortisol (µg/dl)	8.0	6.2-19.4
Adrenocorticotrophic hormone (pg/ml)	802	0-60

CONCLUCIONS

➢ Adrenal tuberculosis is witnessed in up to 6% of patients with active tuberculosis and is usually bilateral. Adrenal destruction via tuberculosis may cause overt or subclinical adrenal insufficiency. Adrenal cortex has a considerable capacity to regenerate with marked hyperplasia and hypertrophy of cortical cells, noted during the period of active infection.

➢Reversal of adrenal function following anti-tubercular therapy is a controversial issue (1,2). Barnes et al. (2) reassessed adrenal function following the therapy and showed that short synacthin test returned to normal in all patients except one, suggesting adrenal dysfunction to be uncommon and antitubercular drugs that have a favorable effect on adrenal function. While some of the studies showed normalization of adrenal function following therapy in a large number of cases, others indicated a contradicted result.

➢In their study, Prasad et al. (3) evaluated adrenocortical reserve and morphology in tuberculosis and reported that the compromised adrenal reserve and enlargement seemed to reverse with therapy. In a recent study by Laway et al. (4), adrenal cortical function and morphology before and after treatment of pulmonary tuberculosis were investigated, and it was reported that basal and stimulated cortisol is less in patients with pulmonary tuberculosis as compared with healthy controls and increases after antitubercular treatment.

➢We suggest that patients who are diagnosed to have adrenal failure due to tuberculosis should be reevaluated after completion of anti-tuberculosis treatment with dynamic tests.

References

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