

Mind Your Step

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Abstract:

Miliary tuberculosis can mimic many clinical disorders, and Addison's disease may be a late complication of tuberculous infection. The coexistence of miliary tuberculosis and Addison's disease has occasionally been described. We report a case in which subacute adrenal failure was the manifestation of miliary tuberculosis in a case of diabetes.

Case Report

A 58-year-old male from Chinsurah presented to a primary care physician with history of low-grade fever for last 3 weeks. Fever was associated with occasional productive cough. By occupation, he was a farmer. He was a diabetic but non-hypertensive and was on treatment with Glipizide and Metformin combination. No previous history of operation or major medical illness were noted. He had stopped smoking cigars 5 years before this.

The primary care physician prescribed him oral co-amoxiclav and suggested to undergo blood investigation.

As the patient remained febrile after taking medicines for 7 days, he was referred to our clinic. He was re-examined and underwent blood investigation. On repeated enquiry the patient also complained of weight loss of 3 kg within last 3 to 4 months and occasional dizziness. His physical examination was unremarkable except for mild pallor and palpable spleen. Pulse of 96/min (regular) and blood pressure

of 108/70 mm of Hg were observed. His blood investigation is as follows:

Hb 10.8 gm/dl, TLC 6800, N58L36M2E4B0, ESR 50, CRP 16. LFT WNL, FBS 176, PPBS 291, HbA1C 8.8%, Na 134 meq/lit, K 3.4 meq/lit.

USG abdomen and chest X-ray (Fig. 1) PA were taken. USG detected mild hepatosplenomegaly.



Figure 1: showing the xray chest

It was diagnosed as a case of miliary tuberculosis. Oral anti-diabetic was stopped and insulin was started. Anti-tuberculosis drug (ATD) was started according to DOTS protocol from our chest clinic.

The patient was kept on observation when ATD was started. He suffered from one episode of hypoglycaemia. An endocrinologist was consulted and insulin dose was optimised. Much to our dismay, the patient suffered from another two episodes of hypoglycaemia on following 3 days. Patient complained of profound generalised weakness and we found gradual deterioration of general well-being.

The patient was re-examined and revealed slightly lower systolic blood pressure of 100 mm of Hg with a diastolic of 70 mm of Hg. Blood parameters were repeated, and the results are as follows:

Hb 9.8 mg/dl, TLC 3400/cmm, ESR 40 mm 1st hour, LFT (WNL), Na 128 meq/lit, K 5.8meq/lit. Considering this hyponatremia with slight hyperkalemia started Serum cortisol was started with the following dose:

S. cortisol– 2.3 mcg/dl (Normal 5–23 mcg/dl).

Then a contrast enhanced CT scan of the abdomen was taken, focussing the adrenals (Fig. 2).

Bilateral adrenal hyperplasia (right> left) was noted with few micro-calcification which was suggestive of adrenal tuberculosis.

The patient was then started on steroids with oral hydrocortisone, and gradual improvement was noticed.

Discussion

This case illustrates problems related to the diagnosis and management of both adrenal failure and miliary tuberculosis. The features observed at the time of the patient's admission to hospital-weakness, hypotension, hyperkalaemia - were characteristic enough to warrant immediate steroid therapy, which resulted in clinical and biochemical improvement. The absence of hyperpigmentation is

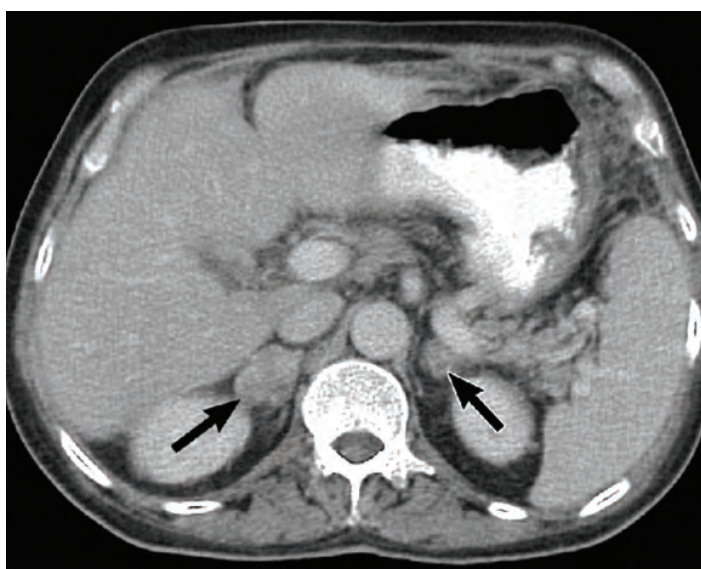


Figure 2 showing CT scan of the abdomen

in keeping with the recent onset of the insufficiency¹. A dynamic stimulation test would have confirmed the functional abnormality. The relation between tuberculosis and adrenal failure is complex. In classic Addison's disease the tuberculosis appears inactive and the adrenal cortical tissue is replaced with granulomas, often partially calcified, which results in a characteristic clinical picture. Reactivation of tuberculosis, either in a localised form (e.g. in an area of the lung) or in a disseminated miliary form^{2,3}, may occur in patients with adrenal failure. The symptoms and signs will be those of active tuberculosis as well as chronic adrenal insufficiency. Miliary dissemination may arise from a reactivated lesion in the adrenals. Miliary tuberculosis may also involve the adrenals incidentally, producing random granulomas that do not cause functional impairment, or massively, resulting in acute or subacute adrenal failure⁴.

References

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