Tubercular Addison’ Disease- an Under-Diagnosed Entity

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Sir,

Addison’s disease (AD) or primary adrenal insufficiency is a rare disorder with prevalence estimated 12 cases /100,000 population/ year and contributes to 1.4 million deaths/ year. In developed nations, autoimmune destruction accounts for 70-80% of cases and in developing nations, tuberculosis (TB) remains the most common cause. In the course of active extra-adrenal TB, adrenal insufficiency occurs in 6% of patients.1

We had 6 cases of AD due to TB in 3 years. At the time of presentation of AD due to TB, the extra-adrenal TB is usually evident but clinically silent. Three of our six patients had active extra-adrenal TB and two patients had evidence of old healed tuberculosis and one had active adrenal TB biopsy at presentation. Adrenal TB result from haematogenous/lymphatic routes and commonly involves bilateral glands. >90% of adrenal tissue is destroyed before symptoms manifests with mean interval of 32 ± 14.9 years and mostly in person aged 40-60 years. Our patients presented with symptoms duration ranging from 3 months to 2 years. The slow destruction of adrenal glands manifests clinically as generalised weakness, fatigue, weight loss, anorexia, nausea, vomiting and postural dizziness. Except for one, all five patients presented with these non-specific symptoms, however the diagnosis was considered in the presence of hyperpigmentation, hyponatremia, hyperkalemia, hypoglycaemia and non-responding hypotension.2 The diagnosis of AD needs determination of the level of adrenal dysfunction by short ACTH stimulation test and identification of the specific cause by imaging and tissue diagnosis. Low- dose (1g) ACTH stimulation test perform equally well to that of traditional 250-µg test. In acute state, a randomly timed cortisol level <15µg/dl indicates hypoadrenalism. For tuberculosis, chest and abdominal computed tomography (CT) should be performed, looking particularly apical shadowing and adrenal glands (Figure 1). We selected open adrenalectomy in one patient, who didn’t have evidence of active TB elsewhere. Tissue diagnosis of active TB was established from extra-adrenal involvement in three and two patients had old evidence of TB on imaging. Fine needle aspiration biopsy attempts failed in two patients as the size of adrenal mass was <4cms. The endoscopic ultrasound guided FNA was not available in our institution, which has better sensitivity with fewer adverse events compared with percutaneous route.3 All patients were started on hydrocortisone – preferred glucocorticoid. Only one patient required mineralocorticoid replacement. One patient deteriorated initially when started on ATT with usual dose of hydrocortisone and other collapsed when she was treated with ATT without glucocorticoid. Drugs like rifampicin enhance the glucocorticoid metabolism and patients may need higher doses of steroids. During stress or illness, dose needs to be increased. AD due to TB is mostly irreversible but sometimes recovery is possible after ATT depending upon the residual viable adrenal tissue and adequacy of ATT.

References