

SHORT REPORT

Acute Adrenal Insufficiency in a 30-Year-Old Woman with Active Pulmonary Tuberculosis

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Acute adrenal insufficiency is a complicated disease that can be overlooked easily because of its non-specific symptoms. The main cause of this disease worldwide was tuberculosis in the past, and it is still a major cause in developing areas (1). We report a case of adrenal insufficiency in an active pulmonary tuberculosis patient.

Case Report

A 30-year-old woman was admitted to hospital because of dizziness, nausea, vomiting, anorexia and weight loss. Nine months before, she was sent to a state tuberculosis clinic with fever, night sweats, cough and weight loss. In that clinic, active pulmonary tuberculosis was diagnosed by X-ray, ARB in sputum and sputum culture. She was treated with a 4 drug combination therapy. After she was discharged from hospital, her symptoms, especially nausea, vomiting and loss of appetite, persisted. She was examined in many clinics and these complaints were attributed to pulmonary tuberculosis and the side-effects of the anti-tuberculosis drugs. She was given saline infusions many times to correct her hyponatremia but her problems were not resolved. During her physical examination her blood pressure was 90/50 mmHg. Because of severe orthostatic hypotension, she could not stand up. She weighed 42 kg. She had generalized hyperpigmentation and darkening with prominent gingival and buccal mucosa. The thyroid gland was diffusely enlarged. The

results of the laboratory examination were as follows: WBC, 6700/mm³ (neutrophil 61%, lymphocyte 23%, monocyte 11%); hemoglobin, 11.4 g/dl; hematocrit, 33.5%; platelets, 276,000/mm³; ESR, 110 mm/h; fasting blood glucose, 79 mg/dl; BUN, 14 mg/dl; uric acid, 9.6 mg/dl; albumin, 3.7 mg/dl; sodium, 129 mmol/l; potassium, 4.7 mmol/l; chloride, 98 mmol/l; TSH, 0.0514 mIU/ml; free triiodothyronine (FT₃), 3.83 pg/ml; free thyroxine (FT₄), 1.4 ng/dl; ACTH, 50 pg/ml; cortisol, 1.7 µg/dl; antithyroglobulin Ab, (-); antimicrosomal Ab, (-); tuberculin skin test, 22 mm; and sodium in 24 h urine, 7.95 g. An X-ray of the chest revealed secondary non-homogeneous consolidations in both apical areas due to a previous specific infection. A CT scan of the chest and abdomen demonstrated consolidations in the left apicoposterior, lower lobe superior, right upper lobe superior and posterior segments. There was a heterogeneous, contrasted 4 x 3 cm solid mass lesion with a necrotic center in the left adrenal gland with probable punctuated calcifications (Figure).

As we suspected a malign lesion in the differential diagnosis, a fine needle aspiration biopsy of the adrenal gland was performed. The pathological result was non-diagnostic. In order to establish a diagnosis, an explorative laparotomy and surgical biopsy were performed. The pathological results revealed necrotic granulomatous inflammation as in tuberculoma. There was diffuse, homogeneous minimal hyperplasia in thyroid ultrasonography and scintigraphy. Therapy with



Figure. CT scan of the abdomen, showing a unilateral adrenal mass that was found to be tuberculous in origin after laparotomy and pathological examination.

prednisolone and propylthiouracil was initiated well before laparotomy, and resulted in a rapid improvement in the patient's condition.

With improvements in anti-tuberculosis therapy and the decreased incidence of tuberculosis all around the world, adrenal insufficiency due to tuberculosis has become a rare condition. Before the 1950s, the major cause of Addison's disease was tuberculosis, but nowadays the major cause is autoimmune destruction of adrenals, accounting for 80-90% of cases. Tuberculosis accounts for as little as 10% of cases (1). In the course of active pulmonary tuberculosis and extrapulmonary tuberculosis, adrenal insufficiency due to acute infection can be seen. In disseminated tuberculosis, this situation can be seen more frequently (2). In the literature, a prevalence of hypoaldosteronism due to acute pulmonary tuberculosis is reported in between 0% and 55% of cases (3). On the other hand, awareness of adrenal insufficiency is important and lifesaving in a tuberculosis patient, because uncured hypoaldosteronism may lead to sudden unexplained deaths (4).

In Addison's disease, most of the symptoms are non-specific. If the adrenal gland is involved in active pulmonary tuberculosis, these symptoms may be

overlooked and be put down to the side-effects of anti-tuberculosis drugs. In all tuberculosis patients with hypotension, hyponatremia resistant to saline infusion or hyperpotassemia, adrenal involvement should be considered. Early and effective anti-tuberculosis treatment may result in an improvement in adrenal function (5).

CT scanning is a proven non-invasive technique for the differential diagnosis of tuberculous adrenalopathy (6). In active tuberculosis infection, the adrenal glands are usually enlarged. Additionally, in adrenal insufficiency due to fungal infections, metastatic malignancies, lymphoma and AIDS, the adrenals can be enlarged (7). Enlargement can be unilateral and massive, like in our case. It can mimic malignant neoplasm and surgical explorative laparotomy may be needed. The diagnostic value of a fine needle aspiration biopsy is not well established.

The adrenals become smaller with fibrosis in chronic tuberculosis. Calcification can be seen radiographically in 50% of cases (8). If there is adrenal atrophy, anti-tuberculosis therapy may not be required (9).

Grave's disease may be seen in 20-25% of patients with adrenal insufficiency due to autoimmune adrenal destruction. Our patient had thyrotoxicosis but anti-thyroglobulin and anti-microsomal antibodies were absent. Therefore, she had 2 coincidental non-autoimmune endocrinopathies similar to a previous case report by Casten et al. (10).

In conclusion, the symptoms of adrenal insufficiency are non-specific, and there may be difficulties in diagnosing this problem in tuberculosis patients. Adrenal insufficiency must be considered in all tuberculosis patients who have persistent symptoms like nausea, vomiting, hypotension and hyponatremia resistant to saline infusions, and further evaluations may be required for diagnosing adrenal dysfunction.

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