

Adrenal mass mimicking the incidentaloma in a patient with newly diagnosed adrenal failure due to tuberculosis

Murat Mehmet RİFAİOĞLU¹, Ersin Şükrü ERDEN², Emine Nur RİFAİOĞLU³, İhsan ÜSTÜN⁴, Cumali GÖKÇE⁴

¹ Mustafa Kemal Üniversitesi Tıp Fakültesi, Üroloji Anabilim Dalı, Hatay,

² Mustafa Kemal Üniversitesi Tıp Fakültesi, Göğüs Hastalıkları Anabilim Dalı, Hatay,

³ Mustafa Kemal Üniversitesi Tıp Fakültesi, Dermatoloji Anabilim Dalı, Hatay,

⁴ Mustafa Kemal Üniversitesi Tıp Fakültesi, Endokrinoloji ve Metabolizma Hastalıkları Bilim Dalı, Hatay.

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A 53 year-old male patient was admitted to the outpatient clinic due to the findings regarding prostatism. For the investigation, a urinary ultrasonography (USG) was also performed. USG displayed a right spherical, 32.6 mm in diameter adrenal mass with calcification. The lesion was diagnosed as adrenal incidentaloma (AI) and consulted to the endocrinology section. In the patient's detailed history, there was also orthostatic hypotension, weight lost, and fatigue for three months. The patient -ex smoker for six years- had 64-pack year history of smoking. Also, he had had one-month drug therapy due to tuberculosis (TB) pleurisy 12 years ago in the past history.

On physical examination, facial and gingival hyperpigmentation and decreased respiratory sounds at lower zone of right hemithorax was found (Figure 1a,b). Laboratory findings revealed that there are hyponatremia: 134 (136-146) mmol/L, hyperkalemia: 5.3 (3.5-5.1) mmol/L. On hormonal assessment, there were low basal cortisol level: 2.59 (6.7-22.6) µg/dL and high ACTH level: 96 (7.2-63.3) pg/mL. Therefore, ACTH

stimulation test was performed, and peak cortisol response was 4.08 µg/dL. After the history, physical examination, laboratory and dynamic tests, the case was diagnosed as adrenal failure (AF). Computerized tomography (CT) showed a calcified mass 2.5 x 2.5 cm in size and peripheral enhancement of right adrenal gland (AG) in which had calcified areas, and relative small left AG with millimetric calcifications. Moreover, CT scan of the chest revealed pleura thickening at basis of the right pulmonary regarding previous pulmonary TB (Figure 1c,d,e).

Here, we presented a case with AF due to TB in a patient with adrenal calcification who was admitted to the clinic in terms of AI. AF is commonly due to autoimmune adrenalitis in Western countries, but it should be kept in mind that adrenal TB is still an important etiology in developing countries.

Calcifications are seen in 59% of adrenal TB cases but in only 8% of adrenal tumors. Adrenal TB generally presents with a low attenuation center and peripheral enhancement on CT, a finding seen in 47% of patients

Yazışma Adresi (Address for Correspondence):

Dr. Murat Mehmet RİFAİOĞLU, Mustafa Kemal Üniversitesi Tıp Fakültesi, Üroloji Anabilim Dalı,
HATAY - TÜRKİYE

e-mail: muratrfai@yahoo.com

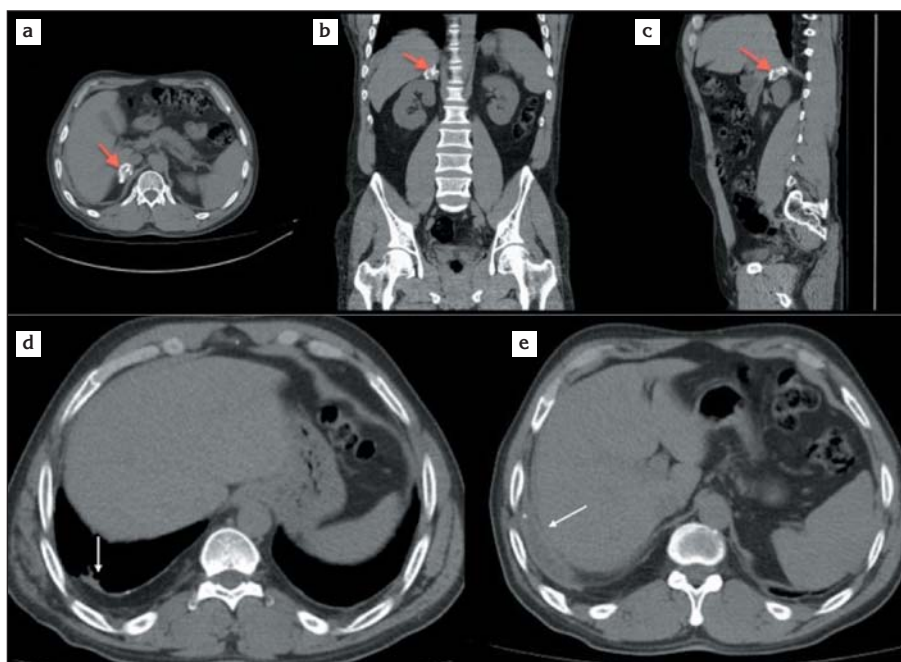


Figure 1. Axial (a), coronal (b) and sagittal (c) computerized tomography revealed right adrenal calcification and distortion. Computerized tomography revealed pleura thickening at basis of the right pulmonary, arrow (d,e).



Figure 2. Periorbital hyperpigmentation, arrow (a), gingival hyperpigmentation (b).

with TB but in only 9% of primary adrenal tumors (1,2). CT findings can differentiate TB from a primary tumor of the AGs with high sensitivity and an acceptable specificity when combined with the endocrinological examination (3).

In acute pulmonary TB, the activation of the hypothalamo-pituitary-adrenal (HPA) axis results in more cortisol secretion from the AGs, which become enlarged. From our group, Gulmez et al. demonstrated that adrenal enlargement is reduced after appropriate TB therapy and CT demonstrated no calcification either befo-

re or after the therapy. In contrast to this report from the same center, anti-TB therapy was not satisfactory in aspect of the development of calcification and distortion after the previous TB (3,2). In another study, Kelestimur et al. claimed that atrophied AGs with fibrosis, calcifications and adrenal enlargement regarding CT findings are common in patients with chronic pulmonary TB (4,2). Additionally, calcifications and soft tissue masses are seen in the adrenal TB (1,2). Also, in the other preliminary report by Gokce and Kelestimur et al., it is pointed that the cases with past TB have cal-

cifications and tortuosity in the AGs (5). Therefore, adrenal findings related to chronic pulmonary TB, adrenal TB, and the past pulmonary TB may be overlapped as the present case. Also, this calcification may confuse with adrenal mass (AI) as in the current case.

TB may affect many of the endocrine glands including the hypothalamus, pituitary, thyroid and adrenals. The most commonly involved endocrine organ in TB is the AG as in our case. TB may directly or indirectly affect AGs. TB Addison's disease is still an important cause of primary AF particularly in the developing countries. HPA axis is also involved in TB and recent findings revealed that HPA axis is activated rather than underactivated in active pulmonary TB. Activated HPA axis in TB causes increased cortisol secretion which results in a shift in the Th1/Th2 balance towards Th2. T cell dysfunction due to high cortisol and low DHEAS levels may be responsible for immunologically-mediated tissue damage in TB (2).

Although acute AF due to adrenal TB is rare, it should not be missed because of the possibility of adrenal crisis, which is a potentially life threatening disorder during physiological distress. In acute pulmonary TB infection, the adrenals are under stress in active pulmonary TB and secrete more cortisol as a result of increased ACTH secretion by the pituitary gland (3). In a study by Kelestimur et al. initially showed that the mean basal cortisol level and 60-minute cortisol response to Synacthen were significantly higher in acute pulmonary TB than in chronic pulmonary TB and healthy subjects (4). Additionally, in patients with acute pulmonary TB, they also showed that cortisol reserve is normal.

Moreover, in the preliminary report, the authors evaluated AGs in terms of functionally (1 µg and 250 µg ACTH tests) and morphologically (adrenal CT) in patients with previous pulmonary TB. Prospectively, they followed up 26 cases with past TB for two years, and it was found that some patients had overt and subclinical AF. Also, morphologically, it is claimed that AGs have tortuosity and calcifications due to previous TB. In ad-

dition, they concluded that 1 µg ACTH test (low dose test) is important for the diagnosis of subclinical AF due TB (5). In the current case, the adrenal mass (AI) due to calcification and AF may be because of previous pulmonary and adrenal TB. As a result, it is may be overlapped in case of functionally and morphologically.

In conclusion, adrenal findings regarding chronic pulmonary TB, adrenal TB, and the past pulmonary TB may be overlapped as the present case. Sometimes, this calcification may confuse with adrenal mass as in the current case. Also, for the differential diagnosis of the AF, it should be kept in mind that TB may affect all the cortex and medulla of the AG, but autoimmune adrenalitis is only seen at the cortex of AG.

CONFLICT of INTEREST

None declared.

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