Clinical Image

Bilateral Adrenal Histoplasmosis

Porntharukchareon et al. Adrenal Histoplasmosis

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An 85-year-old man presented with a 4-kilogram weight loss and anorexia for 3 months. He had no history of tuberculosis or other major illness. His physical examination showed no fever or hyperpigmentation. His chest x-ray was normal. A computed tomography (CT) scan of the abdomen revealed bilateral adrenal masses: a 2.0x2.9 cm mass of right adrenal gland and a 3.7x4.3 cm mass of left adrenal gland (Figure 1). The serologic testing for HIV was negative. His morning serum cortisol was 5.4 μg/dL following with abnormal response to 250-μg ACTH stimulation test (peak serum cortisol of 17 μg/dL). He was diagnosed with primary adrenal insufficiency (AI), which was confirmed by an elevated ACTH level. A CT-guided fine needle aspiration (FNA) cytology of the adrenal lesion revealed the features of Histoplasma spp. (Figure 2). He was treated with liposomal amphotericin B (180 mg daily) for 2 weeks followed by oral itraconazole (400 mg daily) for 1 year. A follow-up CT scan after 6 months of treatment Follow up CT scan showed a slightly decreased in size of the adrenal masses. However, his adrenal function had not recovered.

Histoplasmosis is a disease caused by the dimorphic fungus Histoplasma capsulatum, which is endemic in the certain regions of the world including Southeast Asia (1). Inhalation of its spores from soils contaminated with bat and bird droppings is the main route of infection. Although most infected patients are asymptomatic, localized or disseminated forms can occur in immunocompromised individuals or elderly people.

The adrenal gland is one of the organ systems commonly involved in disseminated histoplasmosis. Bilateral adrenal involvement is common. However, the presence of AI has been reported only in 20-50% of the cases (2). The CT findings of adrenal histoplasmosis may vary depending on the stage of the disease. Typical findings include bilateral adrenal masses with preservation of normal outline, peripheral enhancement and central hypodensity (3). Similar features are also described in other systemic infections (tuberculosis or other fungal infections), metastatic cancers, adrenal hemorrhage, lymphoma and pheochromocytoma. Calcifications can be seen in the late stage. A percutaneous biopsy or FNA cytology of the mass is needed to confirm the diagnosis. Histopathological examination of H. capsulatum shows an intracellular small spherical or oval yeast forms surrounded by a clear ring of space. Additional diagnostic tests include tissue culture, antigen detection and serology. The recommended treatment for the disseminated form, particularly in critically ill hospitalized patients is amphotericin B for 1-2 weeks followed by a 12-month course of an itraconazole. In milder cases, a course of itraconazole for one year is proposed but its blood levels should be monitored. Steroid replacement is required in patients with AI, although reversal of adrenal dysfunction has been reported. Recurrence of the disease has been reported in about 10-15% after cessation of
treatment, and therefore long-term follow-up is necessary (3).

References

FIG. 1: CT upper abdomen venous phase showing 2.0x2.9 cm mass of right adrenal gland and 3.7x4.3 cm mass of left adrenal gland. Both masses (white arrow) showed rim enhancement with septation compatible with thin-walled abscess.
FIG. 2: Calcofluor-White staining showing round shape yeast-like organisms measuring 2-4 μm with narrow-based budding, morphologically compatible with Histoplasma spp. (×1000)