Original Article

CT Findings in Mycobacterial and Fungal Infection of the Adrenal Glands

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Abstract

Introduction: To determine the CT findings of mycobacterial and fungal infection of the adrenal gland.

Methods: CT findings of patients with mycobacterial and fungal infection at adrenal glands were reviewed retrospectively by two reviewers, independently. Several CT parameters were recorded. Quantitative parameters were reported as mean, range, and standard deviation. Qualitative parameters were reported as percentage. The Kappa statistic was used to assess interobserver agreement.

Results: Seventeen patients, 8 with adrenal tuberculosis, 7 with adrenal histoplasmosis, 1 with adrenal cryptococcosis, and 1 with adrenal non-tuberculous mycobacterial infection, were included. Nine patients (52.9%) had primary adrenal insufficiency which are found only in adrenal tuberculosis and adrenal histoplasmosis groups. The CT findings that found in adrenal tuberculosis and adrenal histoplasmosis groups are bilateral involvement (100%), enlarged size of adrenal gland/mass forming lesion (100%), multiloculated abscess or necrosis (75% and 57.1%), and perilesional fat stranding (75% and 85.7%). The interobserver agreements are good to excellent with Kappa of 0.81 - 1.00.

Conclusions: Many CT findings are found in mycobacterial and fungal infection of adrenal glands, such as bilateral involvement, enlarged size of adrenal gland/mass forming lesion, multiloculated abscess/necrosis, and perilesional fat stranding. It is difficult to diagnose the mycobacterial and fungal infection by CT imaging. The clinical correlation, laboratory, and pathologic findings are needed to diagnose the etiology of primary adrenal insufficiency.

Keywords: Adrenal tuberculosis, Adrenal histoplasmosis, CT

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Introduction

Adrenal glands are the organs that are located in retroperitoneum, lying at bilateral suprarenal area. The adrenal gland is an active site of a variety of hormones syntheses, such as mineralocorticoids, glucocorticoids, and sex hormones. Patients with abnormalities of adrenal gland may present with symptoms of hormone excess or, less likely, hormone deficiency. Some patients with adrenal abnormalities are asymptomatic.[1]

Infection of adrenal gland is one of the most common causes of adrenal gland abnormalities. Chronic infection which causes granulomatous inflammation can destroy adrenal gland resulting in primary adrenal insufficiency, or Addison's disease.[1, 2] The clinical onset of Addison's disease is gradual and may be difficult to recognize. The clinical manifestations of adrenal insufficiency are characterized by weakness, malaise, nausea, fatigue, anorexia, and abdominal pain, together with orthostatic hypotension, constipation, weight losing, salt craving, and hyperpigmented skin. Sometimes, the patient has adrenal crisis resulting in severe hypotensive crisis (shock) or severe electrolyte imbalance, which are life-threatening.[3]

Mycobacterium tuberculosis and histoplasmosis infection are the most common pathogens that cause primary adrenal insufficiency.[2, 4-6] There are some reports showed that other pathogens such as Cryptococcus,[7] Aspergillus,[8] Coccidioidomycosis, and Blastomycosis[9, 10] can cause primary adrenal insufficiency. Cytomegalovirus (CMV) is also known as the common pathogen that causes the disease in children. Bacterial infection is a less common cause of adrenal gland infection and rarely chronic infection to destroy adrenal gland.

CT scan plays an important role in evaluation of etiology for patient with primary adrenal insufficiency. The CT images may guide the diagnosis of the adrenal abnormalities including infection or tumors. Some previous studies reported the radiographic findings of mycobacterial tuberculosis and histoplasmosis such as enlarged size of adrenal glands, calcifications, and abnormal enhancement pattern.[11]

The objectives of this study is to determine the findings of mycobacterial and fungal infection of the adrenal gland in Siriraj Hospital.

Methods

Patients

This retrospective study was approved by the institutional review board and the informed consent was not required. The study included the patients age more than 15 years old from 2 databases.

The first; searching on hospital databases, by searching code on ICD-10 system, with diagnosis mycobacterium or tuberculosis infection of adrenal gland, histoplasmosis infection of adrenal gland and cryptococcosis infection of adrenal gland and also other pathogens that reported in the literatures such as nocardiosis and aspergillosis. The patients who had MDCT scan of adrenal glands and had the imaging saved to PACS from January 2007 to June 2018 at Siriraj Hospital were included.

The second; searching from the CT reports for words "Adrenal insufficiency", "Addison's disease", "Adrenal mass", "Adrenal tuberculosis", "Adrenal TB", "Tuberculosis of adrenal gland", "Adrenal histoplasmosis", "Histoplasmosis adrenalitis", or "Granulomatous disease of adrenal gland" on reports from the picture archiving and communication system (PACS) from January 2007 to June 2018.

These patients from two aforementioned databases who had been diagnosed mycobacterium, tuberculosis, and fungal infection of adrenal glands by one of these criteria were included.

A. Diagnosed by tissue diagnosis or positive culture from FNA/Biopsy at adrenal gland

B. Diagnosed by tissue diagnosis or positive culture from other sites

C. Treatment diagnosis (No pathological proven but had been diagnosed infection by the clinician)

The lists of 77 patients by searching from ICD-10 database and 679 patients by keyword searching in CT reports from PACS are obtained. Finally, as correlated with the pathology or final diagnosis, 17 patients were included for data collecting.

The demographic data of the patients, including sex, age of the patients, underlying disease, host status, and chief complaint by electronic medical record review.

Imaging and imaging interpretation

The CT examinations were processed by 64-MDCT scanners in variable protocols due to several indications and parts of expected diseases. All of the included examinations had both non-contrast and post-contrast enhanced phases.

The CT findings were independently reviewed by one radiologist, with subspecialty in genitourinary imaging (20 years of experience), and one resident of radiology department at PACS. Discrepancies in qualitative parameters were adjusted by consensus and in quantitative parameters were averaged. The lesions at each side of adrenal gland that had different characteristics were recorded separately.

CT findings were assessed and recorded, including size of lesion (maximum diameter in axial view), size of adrenal gland (atrophy/normal/enlarged size or mass forming), location (unilateral/bilateral), heterogeneity (homogeneous and heterogeneous), calcification, intralesional hemorrhage, necrosis or multiloculated abscess, border (smooth/irregular), enhancement pattern (peripheral rim enhancement/inhomogeneous enhancement), perilesional fat stranding, and other findings that indicated intraabdominal inflammation e.g. lymphadenitis, ascites.

Follow up results were also recorded if the patients were followed up by clinicians and had medical records. Moreover, the lesions that had been followed up after treatment by CT scan on PACS were reviewed and classified as improve/stable lesion/not improve.

Statistical Analysis

Quantitative parameters, such as age of the patients and size of the lesions, were reported as mean and standard deviation. Qualitative parameters, such as sex, underlying disease, host status, chief complaint, the presence of primary adrenal insufficiency, and other CT parameters were reported as percentage. The Kappa statistic was used to assess interobserver agreement of two readers. The degree of interobserver agreement, indicated by kappa values, was interpreted as follows: 0 - 0.20, poor agreement; 0.21 - 0.40, fair agreement; 0.41 - 0.60, moderate agreement; 0.61 - 0.80, good agreement; and 0.81 - 1.00, excellent agreement.[12]

Results

Patient characteristics

Seventeen patients were included in this study, 8 patients (47.1%) with adrenal tuberculosis, 7 patients (41.2%) with adrenal histoplasmosis, 1 patient (5.9%) with nontuberculous mycobacterium (NTM) infection of adrenal gland and 1 patient (5.9%) with adrenal cryptococcosis (Table 1).

In adrenal tuberculosis, 2 patients (25%) were diagnosed by method A, 4 patients (50%) by method B and 2 patients (25%) by method C. Five patients (71.4%) were diagnosed adrenal histoplasmosis by method A, and 2 patients (28.6%) by method B. One adrenal NTM was distinguished by method B. One adrenal cryptococcosis was included by method C.

Eleven were men (64.7%) and six were women (35.3%). Mean (± S.D.) age was 58.23 ± 13.6 years, range from 40 to 81 years. The frequencies of underlying disease were as follows: diabetic mellitus 29.4%, hypertension 29.4%, chronic kidney disease 5%, and HIV 29.4%. The chief complaints are prolonged fever 25.3%, fatigue 23.5%, weight loss 23.5%, abdominal pain 11.8%, and cervical lymph node enlargement 5.9%.

Nine patients (52.9%) which were four patients of adrenal tuberculosis and five patients of adrenal histoplasmosis had primary adrenal insufficiency. Six patients (35.3%) were immunocompromised hosts which were Human Immunodeficiency Virus (HIV) infections in five patients and interferon-gamma (IFN- γ) autoantibodies in one patient.

Table 1 Demographic data of the patients

	Mean ± S.D. or Number (%) (n = 17)
Male	11 (64.7)
Age (year)	58.23 ± 13.60
Underlying disease	
Diabetic mellitus	5 (29.4)
Hypertension	5 (29.4)
Chronic kidney disease	1 (5.0)
HIV	5 (29.4)
Chief complaints	
Prolong fever	6 (25.3)
Fatigue	4 (23.5)
Weight loss	4 (23.5)
Abdominal pain	2 (11.8)
Cervical lymph node enlargement	1 (5.9)
Primary adrenal insufficiency, yes	9 (52.9)
Immunocompromised host*	6 (35.3)
Disease	
Adrenal Tuberculosis	8 (47.1)
Adrenal Histoplasmosis	7 (41.2)
Nontuberculous Mycobacterial infection of adrenal gland	1 (5.9)
Adrenal Cryptococcosis	1 (5.9)

*Immunocompromised host: HIV infections in 5 patients and interferon-gamma (IFN- γ) autoantibodies in 1 patient

Imaging and imaging interpretation

All CT characteristics were depicted in Table 2. All of adrenal tuberculosis and histoplas mosis (100%) had bilateral involvement (location) and enlarged adrenal size. (Figures 1 and 2) No presence of hemorrhage in both diseases. The mean size (± S.D.) of lesion of adrenal tuberculosis was 3.48 ± 2.01 cm and 4.87 ± 1.26 cm for adrenal histoplasmosis.

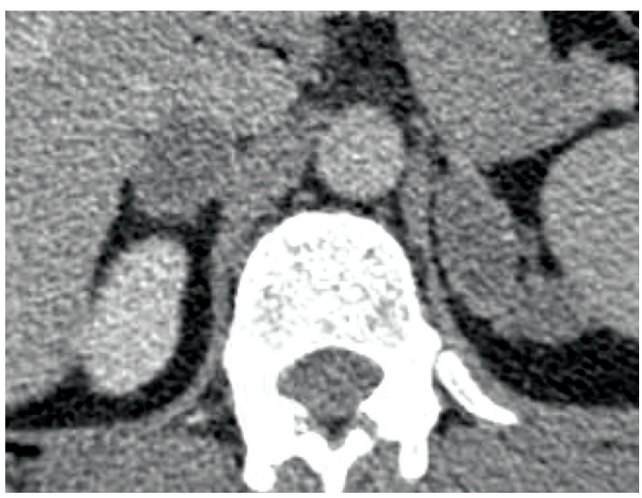

Figure 1 Bilateral adrenal lesions.

Contrast enhanced CT shows bilateral adrenal enlargement at bilateral adrenal glands and pathologic proven adrenal tuberculosis.

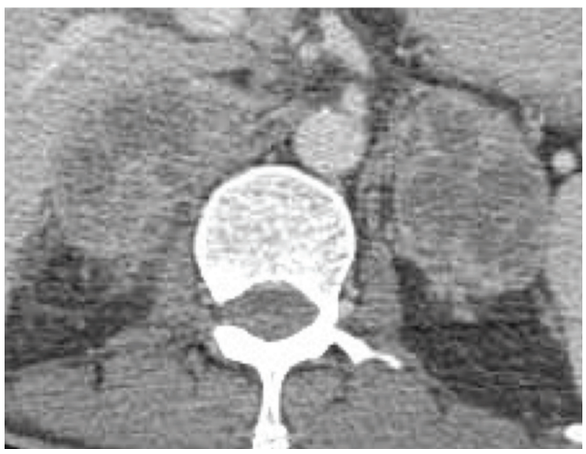

Figure 2 Multiloculated abscess formation with perilesional fat stranding.

Contrast enhanced CT of adrenal histoplasmosis shows bilateral multiloculated abscesses and perilesional fat stranding.

In adrenal tuberculosis patients, 37.5% of patients had intralesional calcifications (Figure 3) which were amorphous calcification 66.7% and mixed coarse and amorphous calcification 33.3%. Three fourth (75%) of patient with adrenal tuberculosis had multiloculated abscess or necrosis and had perilesional fat stranding. 62.5% had smooth border lesion. 50% of patients had peripheral enhancement pattern and 50% of patients had inhomogeneous enhancement pattern. The associated findings that found in patients with adrenal tuberculosis were lymphadenitis (50%), splenic abscess (25%), and pleural effusion (12.5%).

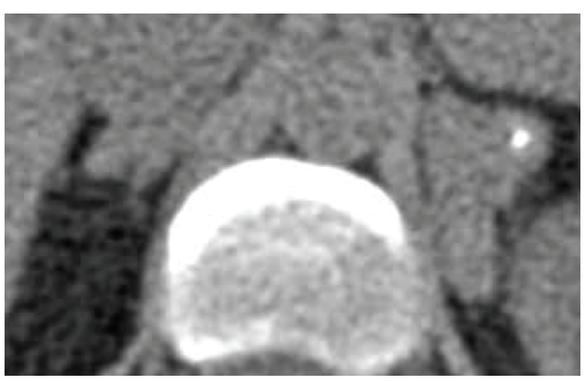

Figure 3 Enlargement of left adrenal gland with intralesional calcification.

Non-contrast enhanced CT scan of adrenal tuberculosis in patient with disseminated tuberculosis infection shows enlargement of left adrenal gland with presence of coarse calcification.

While in adrenal histoplasmosis patients, two patients (28.6%) had intralesional calcifications which one (50%) had coarse calcification and another (50%) had amorphous calcification. The rate of multiloculated abscess or necrosis in this group was about 57.1% (Figures 2 and 4). 71.4% had smooth border of lesion. About 85.7% had perilesional fat stranding (Figure 4). Most of adrenal histoplasmosis showed peripheral rim enhancement (85.7%) and 14.7% had both peripheral rim and inhomogeneous enhancement. The associated findings that found in patients with adrenal histoplasmosis were ascites (42.9%), pleural effusion (42.9%), hepatosplenomegaly (42.9%), peritoneal thickening (42.9%), lymphadenitis (28.6%), and pulmonary nodules (14.3%) (Table 3).

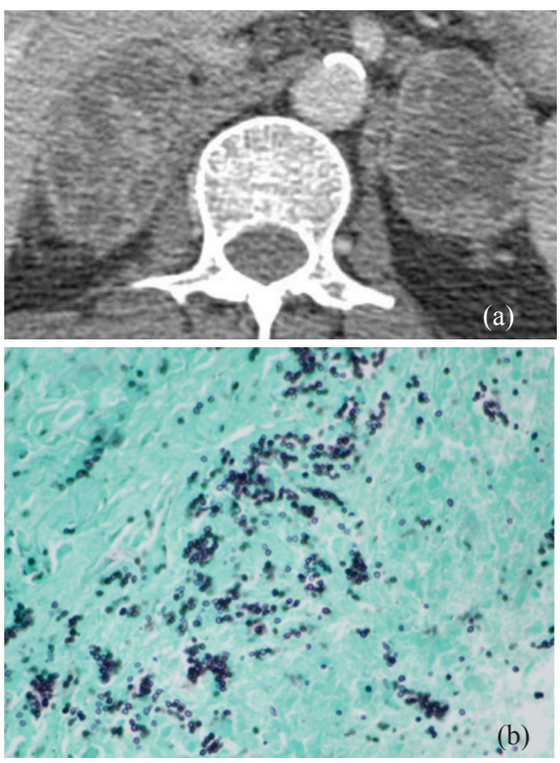

Figure 4 (a) Contrast enhanced CT scan shows bilateral adrenal enlargement with multiloculated abscesses and perilesional fat stranding in patient with pathological proven adrenal histoplasmosis. (b) The Gomori methenamine silver (GMS) stain of adrenal tissue from CT-guided core needle biopsy of the same patient shows positive budding yeasts which are consistent with histoplasmosis morphology.

Adrenal NTM, found in a patient with interferon-gamma (IFN- γ) autoantibodies, had unilateral lesion and presented with enlarged adrenal size. The density of lesion was homogeneous and had internal amorphous calcification. It had irregular border and had inhomogeneous enhancement. The associated findings that found were lymphadenitis, pleural effusion and splenic abscess.

The adrenal cryptococcosis also presented unilaterally. It showed enlarged size of adrenal gland. The lesion was smooth border, homogeneous density without internal calcification and had peripheral rim enhancement. No associated finding that indicated intraabdominal inflammation was found in this patient.

The interobserver agreement of CT parameter of location, adrenal size, density of mass, the presence of calcification, the presence of hemorrhage, the presence of multiloculated abscess or necrosis, the border of lesion, the presence of perilesional fat stranding, and the enhancement pattern were good to excellent agreement with Kappa of 0.81 - 1.00 in range. (location k of 0.76, adrenal size k of 1.0, density of mass k of 0.89, the presence of calcification k of 0.95, the presence of hemorrhage k of 1.0, the presence of multiloculated abscess or necrosis k of 0.95, the border of lesion k of 0.95, the presence of perilesional fat stranding k of 0.94, and the enhancement pattern k of 0.81)

Ten patients (59%) had CT imaging follow up in PACS. Nine patients (90%) had decreased size of adrenal lesion. One patient had unchanged size. Fourteen patients (76.5%) were followed up by clinician and all of them had clinical improvement. Two patients were dead. One patient was loss to follow up.

Table 2 CT Findings of adrenal tuberculosis and adrenal histoplasmosis

CT Findings of	Adrenal TB (n = 8) Mean ± S.D. or Number (%)	Adrenal Histoplasmosis (n = 7) Mean ± S.D. or Number (%)
Bilateral location	8 (100)	7 (100)
Enlarged size of adrenal glands	8 (100)	7(100)
Size of lesions (cm)	3.48 ± 2.01	4.87 ± 1.26
Homogeneous density of mass	4 (50)	7 (100)
Presence of calcification	3 (37.5)	2 (28.6)
Shape of calcification		
Coarse	0 (0)	1 (50)
Amorphous	2 (66.7)	1 (50)
Mixed coarse and amorphous	1 (33.3)	0 (0)
Presence of hemorrhage	0 (0)	0 (0)
Multiloculated abscess or necrosis	6 (75)	4 (57.1)
Smooth Border	5 (62.5)	5 (71.4)
Perilesional fat stranding	6 (75)	6 (85.7)
Enhancement pattern		
Peripheral rim enhancement	4 (50)	6 (85.7)
Inhomogeneous enhancement	4 (50)	0 (0)
Both peripheral rim and homogenous enhancement	0 (0)	1 (14.3)

Table 3 Associated CT findings in adrenal tuberculosis and adrenal histoplasmosis

Associated findings of	Adrenal TB (n = 8) Number (%)	Adrenal Histoplasmosis (n = 7) Number (%)
Lymphadenitis	4 (50)	2 (28.6)
Ascites	0 (0)	3 (42.9)
Pleural effusion	1 (12.5)	3 (42.9)
Hepatosplenomegaly	0 (0)	3 (42.9)
Peritoneal thickening	0 (0)	3 (42.9)
Splenic abscess	2 (25)	0 (0)
Pulmonary nodules	0 (0)	1 (14.3)

Discussion

This research objective is to determine the CT findings of mycobacterium and fungal infection of adrenal glands which can cause adrenal destruction and lead to primary adrenal insufficiency (Addison's disease). Unfortunately, there is only one patient in each cryptococcosis and nontuberculous mycobacterium infection group. The discussion is mainly about adrenal tuberculosis and histoplasmosis.

The important findings of adrenal tuberculosis and histoplasmosis in this study are bilateral involvement, enlarged size of adrenal gland/mass forming lesion, multiloculated abscess or necrosis and perilesional fat stranding.

The study found that 100% of patients with adrenal tuberculosis and adrenal histoplasmosis had bilateral involvement which was also reported in other previous studies.[3, 13-16] These findings are probably due to the mechanism of infection of these pathogens. Adrenal infection most frequently occurs in the setting of disseminated infection.[13] Mycobacterium hematogenously disseminates to the adrenal glands and causes adrenal dysfunction by inducing degeneration of cells within adrenal cortex.[13] Caseous necrosis and tuberculous granuloma can be found in the early stage of adrenal gland infection.[16] While in histoplasmosis, some previous studies reported that up to half of patient with disseminated *Histoplasmosis capsulatum* had been found the infection of adrenal glands.[13] The mechanism of adrenal gland destructions in *Histoplasmosis capsulatum* infection was thought to occur via directed infection followed by extra and intracapsular vasculitis that led to extensive infarction and caseation of adrenal gland.[14] *Cryptococcus neoformans* is typically infected immunodeficiency patients, particularly in advanced acquired immune deficiency syndrome (AIDS). In spite of the propensity of this fungus to cause disseminated infection, the adrenal dysfunction is uncommon. However, it can occur by caseating granuloma. Infection either can present as isolate lesion with no obvious adrenal dysfunction or can result in disseminated cryptococcosis and cause adrenal failure.[13, 15] One cryptococcus in this study had unilateral lesion.

Consistent with other previous studies, all of the patients in the study had enlarged size of adrenal gland or mass forming lesion.[3, 13-16] These findings may be since all of the pathogens in this study can cause caseous granuloma in adrenal gland.[13] Moreover, as mentioned above, these pathogens can cause vasculitis resulting in necrosis. The study found that 75% of patients with adrenal tuberculosis and 57.1% of adrenal histoplasmosis had multiloculated abscess or necrosis in adrenal glands. Perilesional fat stranding could be due to inflammation.

Some previous studies reported about the calcification in adrenal glands in adrenal tuberculosis patients that appeared in inactive infection, late stage or chronic infection and in post-treatment status.[3, 13, 17] Some case reports of adrenal histoplasmosis also mentioned about calcification in adrenal glands. In this study, the presence of calcification in lesions are detected in both adrenal tuberculosis and adrenal histoplasmosis, about 37.5% and 28.6%, respectively. Both coarse calcification and amorphous calcification appearances were presented in both pathogen group. In adrenal tuberculosis patients, two patients, with amorphous calcification and mixed coarse and amorphous calcification

were found before treatment by anti-tuberculosis drugs. One patient with amorphous calcification was previously treated by anti-tuberculosis drugs due to pulmonary tuberculosis for 1 week. One patient, in adrenal histoplasmosis group, was detected amorphous calcification in pre-treatment imaging. Another patient with coarse calcification was found in imaging which was done 1 year after treatment by anti-fungal drug due to histoplasmosis lymphadenitis. It can be inferred that the presence of calcification can be found in both pre-treatment and post-treatment group and it is not the specific finding to identify the pathogen.

Four patients (50%) of adrenal tuberculosis and five patients (71%) of adrenal histoplasmosis had primary adrenal insufficiency. Adrenal insufficiency will present in patient with more than 90% adrenal destruction.[16] Even though one reported that treatment with anti-tuberculosis drugs does not improve or recover adrenal function,[13] there is evidence that early antifungal therapy can lead to at least partial recovery of adrenal function. Early detection of adrenal infection by CT scan would have some benefit for these patients and prevent them from lifelong corticosteroid supplements.

Ten of seventeen patients had imaging follow up after treatment. 90% of them had decreased size of adrenal lesion. Fourteen patients had clinical follow-up and all of them had clinical improvement.

However, the CT finding of the adrenal gland enlargement or mass forming can be neoplasm.[1] The additional common CT finding of multiloculated low density or abscess may indicate infection process but not specific finding for each pathogen. The tissue diagnosis or culture is needed for definite pathogens or diagnosis.

The study reveals tuberculosis and histoplasmosis are the common organism to infected adrenal glands. The limitation of the study is a small sample size of other fungal infections.

There are many CT findings that found mostly in mycobacterial and fungal infection of adrenal glands, such as bilateral involvement, enlarged size of adrenal gland/mass forming lesion, multiloculated abscess or necrosis and perilesional fat stranding. It is difficult to give the definite pathogen by only CT imaging. The clinical correlation, laboratory and pathologic findings are needed. Nevertheless, the CT imaging may have benefits in differential diagnosis of adrenal infection, helping clinician to decide about early treatment and may save patients from lifelong corticosteroid supplements.

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