TUBERCULOUS ADRENAL ABSCESS ASSOCIATED WITH TUBERCULOSIS OF THE LUNGS AND BRAIN: A CASE REPORT

Phuvitoo SUNGTONG, M.D.[1]

ABSTRACT

Tuberculous adrenal abscess was diagnosed in a 46-year-old man who presented with fever, abdominal pain, and weight loss. Ultrasonography revealed a well-marginated right suprarenal mass with internal hypoechoic areas. By CT scan, it showed thin rim enhancement with multiple septal enhancement and central hypodense areas. Tuberculous adrenal abscess was diagnosed by isolation of tuberculous bacilli from the lesion under CT guidance. He also had evidence of miliary pulmonary tuberculosis from chest radiograph and intracranial tuberculosis from CT scan of the brain.

INTRODUCTION

Tuberculosis is an infectious disease caused by microorganism Mycobacterium tuberculosis. It is on the rise and revisiting both the developed and developing world and is increasing prevalence in both immunocompetent and immunocompromised individuals. It is a chronic, contagious bacterial infection, which can affect several organs of the human body, including the brain, bone, gastrointestinal tract, kidneys, adrenal glands, lymph node, but most commonly it affects the lungs. A case of disseminated tuberculosis which are composed of miliary tuberculosis of the lungs, intracranial tuberculosis, and tuberculous abscess of the adrenal glands is present in the emphasis of radiological findings and differential diagnosis with a review of the current literatures.

CASE REPORT

A 46-year-old man presented with fever, progressive increasing right upper quadrant pain and weight loss 9 kg in approximately 3 months. There was neither cough nor hemoptysis. His past medical history was unremarkable and he had no past history of tuberculosis. There was neither past history of intravenous drug used nor risk factors for human immunodeficiency virus (HIV) infection.

The initial physical examination revealed an obviously unwell patient. He was cachectic, mild pale, and no jaundice. The body temperature was 38 °C, BP 100/70 mmHg, pulse 70 beats/min, and respiratory rate 22 breaths/min. The heart and lungs appear normal. The abdominal examination revealed soft on palpation without point of maximum tenderness. A questionable abdominal mass was palpable on the right side of abdomen. It is deeply located. The rest of physical examination including neurological examination was within normal limits.

Chest radiograph revealed innumerable, 1-3 mm, noncalcified nodules scattering through out both lungs. No cavitary lesion was observed. There was no evidence of mediastinal mass. Minimal right pleural effusion was also noted. (Figure 1A, 1B). Miliary tuberculosis was the most likely diagnosis. However, repeated acid fast smears of the sputum were negative.

[1] Division of Radiology Hatyai Hospital Songkhla Thailand 90110.
E-mail addresses : **phuvitoo@hotmail.com, Phuvitoo@yahoo.com**

Fig. 1 Chest radiograph.

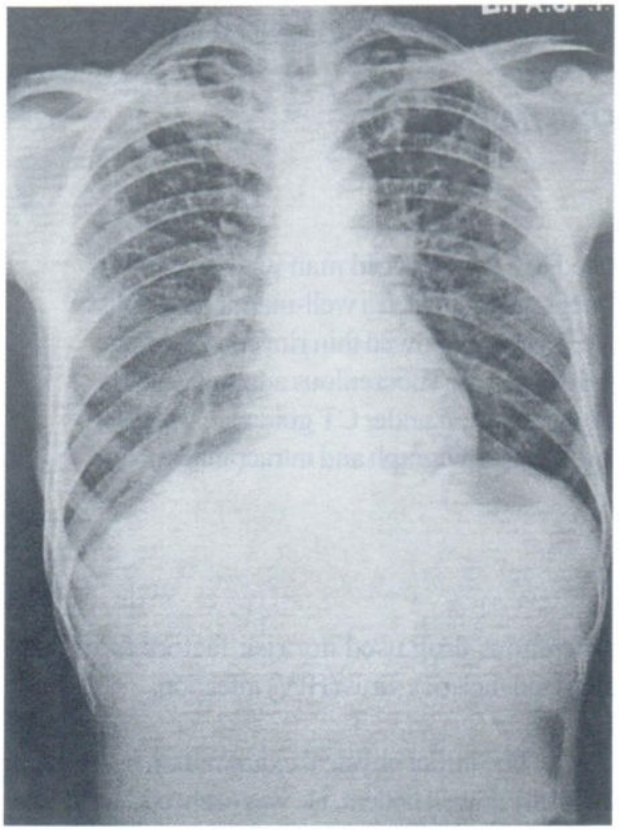

Fig. 1A Posteroanterior chest radiograph revealed diffuse miliary shadows through out both lungs. Small amount of right pleural effusion was evident.

Fig. 1B Close-up right basal lung clearly demonstrated miliary pulmonary infiltrations.

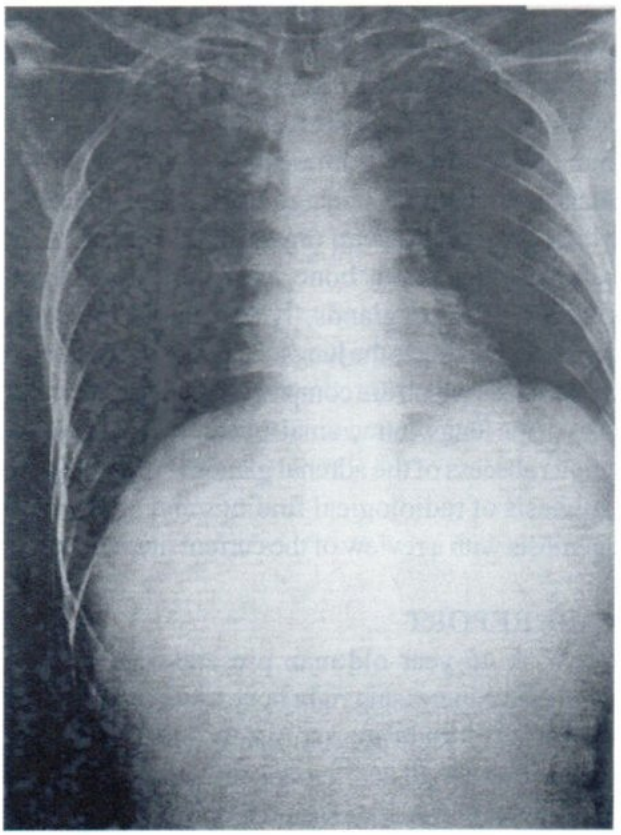

Fig. 1C Follow-up chest radiograph after six weeks of antituberculous treatment showed disappearance of pulmonary infiltrations.

Owning to abdominal pain and a palpable mass, ultrasonography of the abdomen was requested. The abdominal ultrasonography revealed a well -marginated right-sided suprarenal mass with multiple internal hypoechoic areas (Figure 2A, 2B). There was no obvious suprarenal mass on the contralateral side. The liver, gallbladder, pancreas, spleen, and kidneys were normal. There was neither enlarged lymph node nor ascites. Small amount of right pleural effusion was observed.

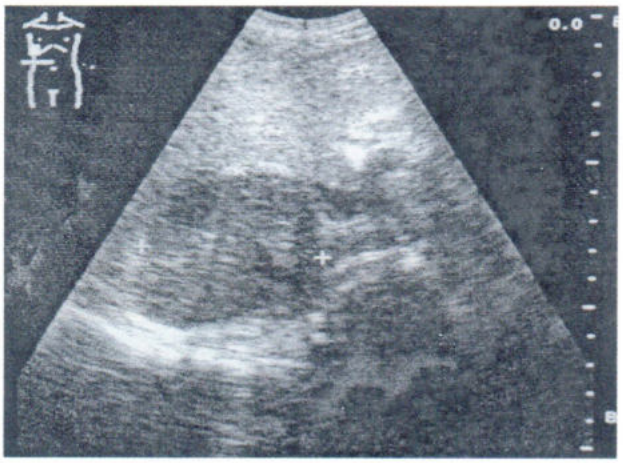

Fig. 2A

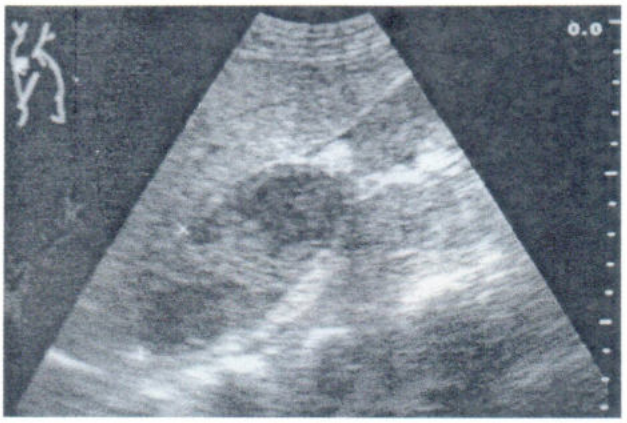

Fig. 2B

Fig. 2 Ultrasonography of the right adrenal gland. Transverse (Figure 2A) and longitudinal (Figure 2B) ultrasonographic images showed a large right suprarenal mass with internal hypoechoic areas.

Computed tomography scan of the abdomen showed a mass originating from the right adrenal gland. On precontrast scan it was inhomogeneous hypodensity and had an attenuation of approximately 27 HU. Neither calcification nor fat density was noted (Figure 3A). Following IV contrast, the mass showed thin rim enhancement with multiple septal enhancement. The internal hypodense areas were not enhanced. Its size was approximately 4x5x6 cm. The left adrenal gland was slightly enlarged and showed rim enhancement with central hypodensity (Figure 3B, 3C). Neither enlarged lymph node nor ascites was noted.

CT-guided aspiration was performed for the correct diagnosis of the right adrenal pathologic condition. It was done by placing the patient in the prone position and using a 20G spinal needle (Figure 3D). Pus was obtained, approximately 20 cc. It was sent for gram stain and AFB stain. Acid fast stain showed organisms as slender red rods. Gram stain showed many WBC and few RBC but no organism. Antituberculous drugs (INH, Rifampicin, PZA, and Ethambutal) were started after the report of adrenal abscess aspiration. The patient was discharged after gradual getting well in 2 weeks.

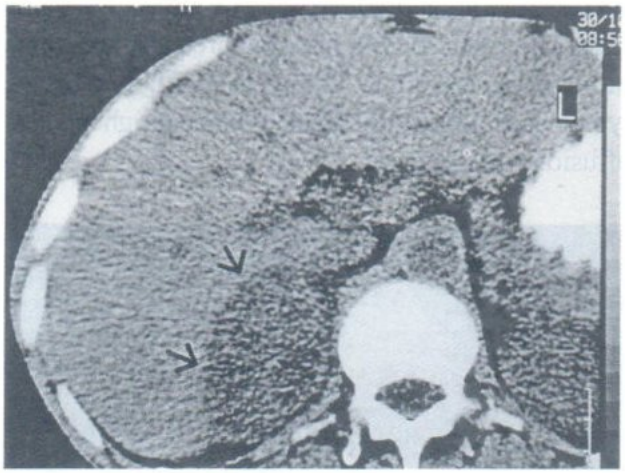

Fig. 3A

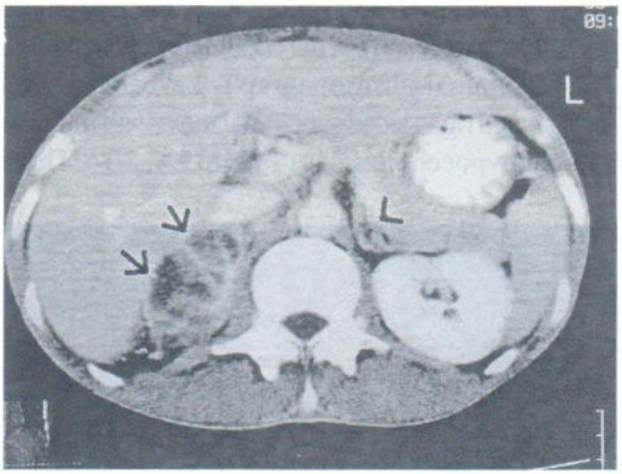

Fig. 3B

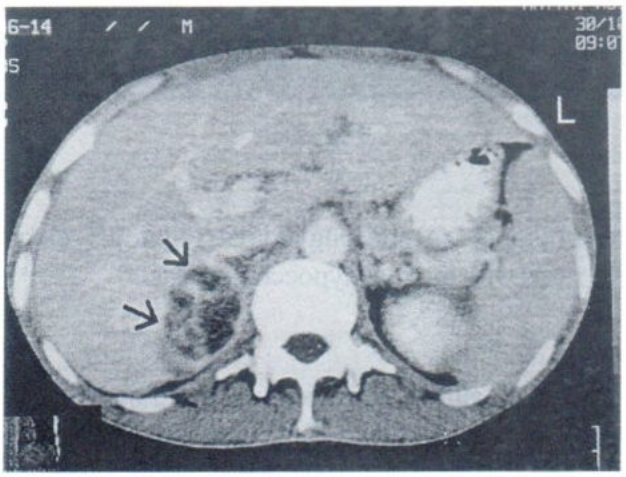

Fig. 3C

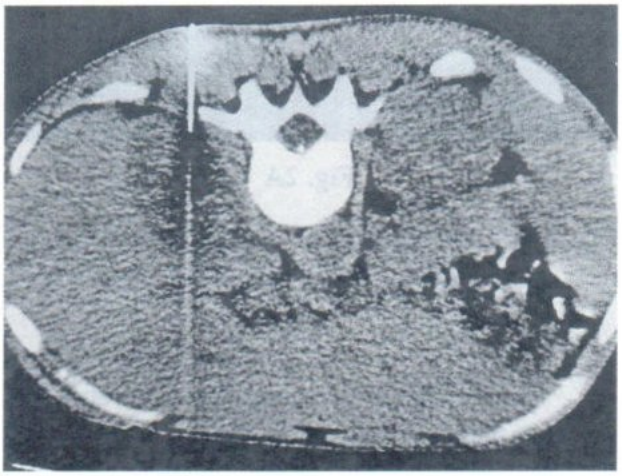

Fig. 3D

Fig. 3 CT scan of the adrenal glands.

Fig. 3A CT scan obtained before I.V. contrast administration showed a large right suprarenal mass with heterogeneous hypodensity (arrows).

Fig. 3B and **3C** Contrast-enhanced CT scan showed rim and septal enhancement of the right adrenal mass with internal hypodense areas (arrows). The left adrenal gland is slightly enlarged with rim enhancement and central hypodensity (arrow head).

Fig. 3D CT-guided aspiration of right adrenal abscess was performed on prone position.

One month later, he was admitted with progressive headache, and drowsiness. The physical examination revealed decreased visual field, papilledema, and impaired cerebellar functions. The body temperature was 36.8 °C, BP 110/70 mmHg, pulse 80 beats/min, and respiratory rate 22 breaths/min.

CT scan of the brain was performed. Multiple, various-sized nodular and ring enhancing lesions with associated brain edema were observed in the cerebrum and cerebellum. They measured less than 1 cm in size. Many miliary tubercles were also noted (Figure 4A, 4B).

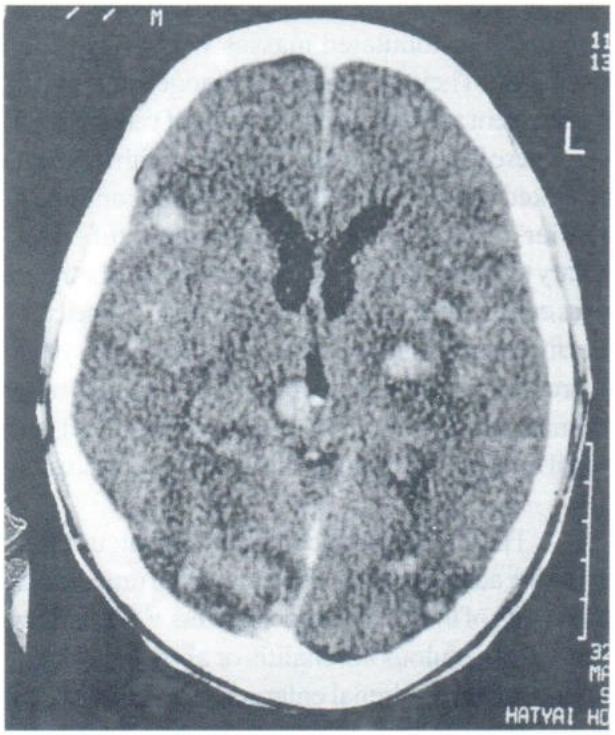

Fig. 4A

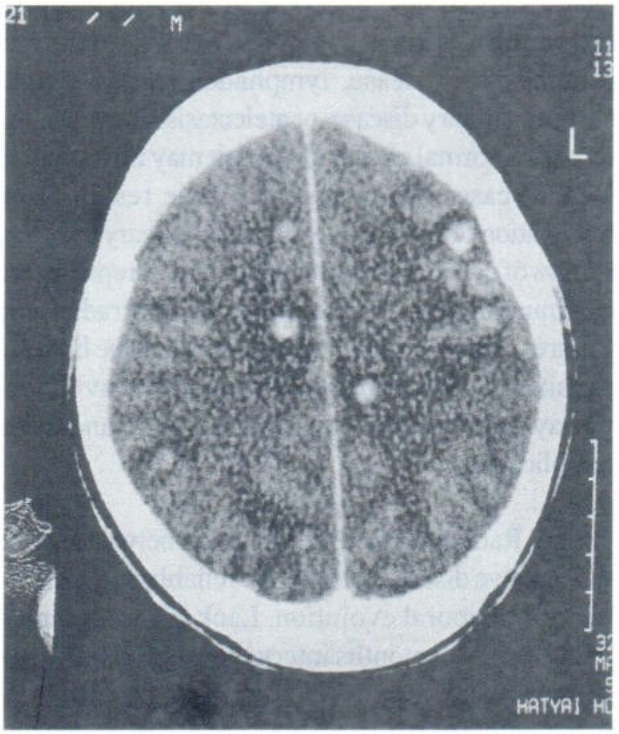

Fig. 4B

Fig. 4 CT scan of the brain.
Fig. 4A and **4B** CT scan of the brain post I.V. contrast showed multiple small nodular and ring enhancing lesions scattered in the brain with associated brain edema.

Chest radiograph of this admission showed disappearance of miliary infiltrations in both lungs. No pleural effusion was noted (Figure 1 C).

The follow up ultrasonography revealed persistence of a large right suprarenal mass. Its size and echogenicity were similar to the previous examination. Right pleural effusion disappeared. No additional abnormal finding of abdominal ultrasonography was observed.

According to persistence of right adrenal abscess after medical treatment, surgical exploration with right adrenalectomy was performed. Large amount of pus inside the bisected adrenal gland was noted. The specimen was submitted for pathological examination and revealed caseous granulomatous inflammation.

After operation, multiple medical problems occurred. The patient got worsen with cardiac arrest and died with disseminated tuberculosis 3 weeks after the operation.

DISCUSSION

Tuberculosis is most commonly limited to the chest but it can affect virtually any organ system in the body and can be devastating if left untreated. Because tuberculosis demonstrates a variety of clinical and radiologic findings and has a known propensity for dissemination from its primary site, it can mimic numerous other disease entities.[1]

Pulmonary tuberculosis is classically divided into primary and postprimary (reactivation) tuberculosis. There is considerable overlap in the radiologic

manifestations between these two entities. Primary tuberculosis typically manifests radiologically as parenchymal disease, lymphadenopathy, pleural effusion, miliary disease, or atelectasis. However, the results of normal chest radiograph may be normal in 15% of cases. Postprimary disease results from reactivation of a previously dormant primary infection in 90% of cases; in a minority of cases, it represents a continuation of the primary disease. The radiologic features of postprimary tuberculosis can be broadly classified as parenchymal disease with cavitation, airway involvement, pleural extension, and other complications.[1,2,3]

Radiographic differentiation between active and inactive disease can only be reliably made on the basis of temporal evolution. Lack of radiographic change in a 4-6 months interval generally indicates inactive disease.[4,5]

Dissemination of tuberculosis may occur during either primary or post primary stages of disease. It results when a focal collection of tubercle bacilli discharges into a blood or lymph vessel, releasing a large number of viable bacilli that embolize to capillary beds in multiple organs.[2,6]

A chest radiographic finding of profuse, tiny, well-defined nodules 1-4 mm in size is termed miliary shadowing. It comes from "mellet seed" (a popular birdseed), and the areas of increased opacity are said to resemble mellet seeds in size and shape.[7] Miliary shadowing is highly suggestive of disseminated tuberculosis in the appropriate clinical setting. However, it may also indicate varicella pneumonia, histoplasmosis, metastases (particularly from thyroid tumor, melanoma, and choriocarcinoma), pneumoconiosis, hemosiderosis, eosinophilic granuloma, or sarcoidosis.[1,7,8]

Central nervous system tuberculosis may take a variety of forms, including meningitis, tuberculoma, abscess, cerebritis, and miliary tuberculosis. Parenchymal disease can occur with or without meningitis and most commonly manifests as either solitary or multiple tuberculomas. At CT, tuberculomas appear as rounded or lobulated masses with low or high attenuation. They demonstrate homogeneous or ring enhancement and have irregular walls of varying thickness. These lesions are often associated with moderate to marked edema, but calcification is uncommon.[1,9] Smaller lesions were termed miliary tubercles. Miliary tubercles appear as numerous round, homogeneous enhancing lesions, less than 2 mm in diameter.[10,11] The differential diagnosis for central nervous system tuberculomas and miliary tubercles includes other granulomatous infections (e.g. cysticercosis), and fungal infections as well as metastatic neoplasms.[1]

The CT appearance of granulomatous infection of the adrenal glands depends on the time, course and activity of the inflammatory process.[12] CT findings of early tuberculous adrenalitis or abscess typically include bilateral adrenal enlargement with a central necrotic area of hypoattenuation and a peripheral enhanced rim but unilateral involvement can occur.[12,13,14,15] Adrenal abscess caused by nontuberculous bacterial infection is uncommon in an adult but occurs more frequently in neonates.[16,17] Neonatal adrenal abscesses can be unilateral or bilateral and are caused by either hematogenous bacterial seeding of normal adrenal glands or seeding of a neonatal adrenal hemorrhage with subsequent abscess formation.[17]

The differential diagnosis of bilateral adrenal enlargement must include other granulomatous infections such as histoplasmosis, cryptococcosis, and blastomycosis. Other diseases that may produce a similar appearance include lymphoma, bilateral adrenal metastases, bilateral hemorrhage, and bilateral primary adrenal tumors.[15]

In the healing stage of adrenal tuberculosis, the adrenal glands become calcified and atrophy. However, its CT appearance is indistinguishable from that of other long-standing granulomatous infections, previous hemorrhage, and idiopathic adrenal calcifications.[12,13,14,15]

Bilateral adrenal involvement occasionally results in adrenal insufficiency or Addison's disease. The disease was first described by Dr. Thomas Addison in 1849. The problem may be due to a disorder of the adrenal glands themselves (primary adrenal insufficiency) or to inadequate secretion of ACTH by the pituitary gland (secondary adrenal insufficiency). Primary adrenal insufficiency is a severe or total deficiency of the hormones made in the adrenal cortex, caused by a destruction of the adrenal cortex. Tuberculosis accounts for about 20% of cases of primary adrenal insufficiency in developed countries. The disease is characterized by weight loss, muscle weakness, fatique, low blood pressure, and sometime darkening of the skin in exposed and nonexposed parts of the body. A definitive diagnosis of Addison's disease requires definitive tests that be carried out. These tests measure the amount of cortisol and aldosterone in blood and urine, and document a lack of the normal increase in the levels of these two hormones after administration of ACTH given by injection. An elevated blood level of ACTH should also be found in Addison's disease.[18,19]

Disseminated tuberculosis of this reported case was composed of miliary pulmonary tuberculosis, intracranial tuberculomas and miliary tubercles, and tuberculous abscess of the bilateral adrenal glands. The diagnosis was made by isolation of tuberculous bacilli from the right adrenal abscess under CT guidance. Miliary pulmonary tuberculosis was strongly evident by miliary shadowing on the intitial chest radiograph which disappeared after six weeks of antituberculous drug treatment. Intracranial tuberculosis were concluded by typical CT findings together with evidence of the disease in previously mentioned organs. Bilateral adrenal involvement in this patient could result in Addison's disease but there was no biochemical investigation for confirming the diagnosis.

CONCLUSION

Tuberculosis of the lungs and brain are common and have been well described radiologically in the literature. Adrenal tuberculosis is not uncommon and is mostly bilateral involvement and may be the cause of adrenal insufficiency. Tuberculous adrenal abscess appears as rim enhancing mass with central hypodensity by CT scan. Old adrenal tuberculosis, the glands may atrophy and calcify.

REFERENCES

1. Harisinghani MG, Mclound TC, Shepard JO, Ko JP, Shroff MM, Mueller PR. Scientific exhibit Tuberculosis from head to toe. Radiographics 2002; 20 :449-470.
2. Leung AN. Pulmonary tuberculosis: The essential. Radiology 1999; 210: 307-322.
3. Kim HY, Song KS, Goo JM, Lee JS, Lee KS, Lim TH. Education exhibit The Thoracic sequelae and complications of tuberculosis. Radiographics 2001; 21 : 839-858.
4. Bass JR, Farer LS, Hopewell PC, Jacobs RF, Snider DE. Diagnostic standards and classification of tubeerculosis. Am Rev Respi dis 1990; 142 : 725-735.
5. Miller WT, MacGregor RR. Tuberculosis: frequency of unusual radiographic findings. AJR 1978; 130 : 867-875.
6. Geppert EF, Leff A. The pathogenesis of pulmonary and miliary tuberculosis. Arch Intern Med 1979; 139 : 1381-1383.
7. Roche CJ, O'Keeffe DP, Duddalwar VA, Torreggiani WC. Selections from the Bufflet of food signs in Radiology. Radiographics 2002; 22 : 1369-1384.
8. Reed JC. Chest radiology Plain films patterns and differential diagnoses. 3rd ed. St Louis, MO: Mosby-Year book. 1991.
9. Engin G, Acunas B, Acunas G, Tunaci M. Scientific exhibit Imaging of extrapulmonary tuberculosis. Radiographics 2000; 20: 471-488.

10. Gee GT, Bazan C, Jinkins R. Miliary tuberculosis involving the brain: MR findings. AJR Am J Roentgenol 1992; 159 : 1075-1076.
11. Jinkins JR, Gupta R, Chang KH, Rodriguez -Carbajal J. MR imaging of central nervous system tuberculosis. Radiol Clin North Am 1995; 33 : 771-786.
12. Kawashima A, Sandler CM, Fishman EK, Charnsangavej C, Yasumori K, Honda H, et al. Spectrum of CT findings in nonmalignant diseases of the adrenal gland. Radiographics 1998; 18 : 393-412.
13. Mayo-Smith WW, Boland GW, Noto RB, Lee M. Satate-of-the-art adrenal imaging. Radiographics 2001; 21 : 995-1012.
14. Wilms GE, Baert AL, Kint EJ, Pringot JH, Gooddeeris PG. Computed tomographic findings in bilateral adrenal tuberculosis. Radiology 1983; 146 : 729-730.
15. Wilson DA, Muchmore HG, Tisdal RG, Fahmy A, Pitha JV. Histoplasmosis of the adrenal glands studies by CT. Radiology 1984; 150 : 779-783.
16. O' Brien WM, Choyke PL, Copeland J, Klappenbach RS, Lynch JH. Computed tomography of adrenal abscess. J Comput Assist Tomogr 1987; 11 : 550-551.
17. Atkinson GO, Kodroff MB, Gay BB, Rickette RR. Adrenal abscess in the neonate. Radiology 1985; 155 : 101-104.
18. Williams GH, Dluhy RC. Disorders of the adrenal cortex. In : Braunwald E, ed. Harrison's principles of internal medicine. 15th ed. New York. MacGraw-Hill Profesional; 2001: 2084-2105.
19. Ten S, New M, Maclaren N. Clinical Review 130: Addison's disease 2001. Journal of Clinical Endocrinology & Metabolism 2001; 86(7): 2909-2922.