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CASE REPORT

The role of Chest HRCT in diagnosis active tuberculosis & lung destruction

Adi Soekardi[1,2]*, Aziza Ghanie Icksan[1,2], Audrina Ernes[1]

Abstract

Tuberculosis is a public health problem caused by *Mycobacterium tuberculosis*. In 2021 there will be 10.6 million cases in the world, and Indonesia ranks 2nd with 700,000 cases. In 2022, there will be 17,303 cases in North Sumatra Province and 2,430 cases in Medan City. In current practice, evaluation and diagnosis of active tuberculosis relies on bacteriological examination and *Chest Radiographs*. However, *Chest Radiographs* have limited specificity and high intraobserver and interobserver variability. HRCT is also not widely used as a routine option for patients suspected of suffering from tuberculosis, even though HRCT has high accuracy in detecting tuberculosis. We present a case of a patient with active pulmonary tuberculosis and lung destruction e.c. advanced pulmonary tuberculosis (MDR-Tuberculosis) (declared cured in 2018 after 2 years of treatment). This patient was evaluated with chest radiograph and chest HRCT. In this case, the initial findings on the *Chest Radiograph*, showed the impression of inactive tuberculosis but on further examination with chest HRCT, there was a *tree in bud* image which indicated active tuberculosis. This shows that there are limitations in diagnosing tuberculosis activity using *Chest Radiographs*. Due to the significant role of HRCT in the diagnosis of tuberculosis activity, it is necessary to consider the use of HRCT in the evaluation of patients with tuberculosis.

Keywords: tuberculosis, lung destruction, Chest Radiography, HRCT

Introduction

Tuberculosis is an infectious disease, caused by bacterial infection (*Mycobacterium tuberculosis)*.[1] Tuberculosis is a public health problem and the leading cause of death worldwide from a single infectious agent before the COVID-19 pandemic (mortality rate of 50%).[2,3] A quarter of the global population is estimated to be infected with Tuberculosis. By 2021, worldwide, 10.6 million people fell ill with Tuberculosis.[4] Indonesia reaches 2nd place in the world with 700,000 TB cases by 2022.[3] North Sumatra province contribute 17,303 cases and Medan with the most TB cases in North Sumatra (2,430 cases in 2022).[2,4,5] Tuberculosis is one of the top 10 diseases in Royal Prima Hospital both as outpatient and inpatient care. In 2021, there were 277 inpatient cases with tuberculosis and 1529 outpatient visits.[6]

Tuberculosis can be categorized into six classes based on clinical and radiological findings. Bacteriological testing and chest radiographs are currently used in clinical practice for tuberculosis evaluation and diagnosis of active tuberculosis. Chest radiographs, however, have limited specificity and eventhough HRCT has higher sensitivity and specificity, it has not become the routine choice of modality in patient with suspected tuberculosis.[7,8]

Case Report

A 31-year-old female presented to the emergency department with complaints of dyspnea and cough (1 month). She had a history of advanced pulmonary tuberculosis (MDR Tuberculosis) 7 years ago

Affiliation
[1]Department of Radiology, Universitas Prima Indonesia
[2]Consultant Cardiothoracic Radiologist, Royal Prima Hospital

Correspondence
dokteradisoekardi@gmail.com

which was declared cured in 2018 after 2 years of treatment. She was diagnosed with tuberculosis in 2014 and began MDR treatment in 2016. She had a history of recurrent, uncontrolled asthma exacerbations and limitation in doing sports activities due to complaints of tightness related to uncontrolled asthma since childhood and was not receiving optimal therapy to control her asthma. She has residual chronic lung disease which has a significant influence on her quality of life (physical activity restrictions, frequent hospital visits and hospitalizations), as well as health expenditure.

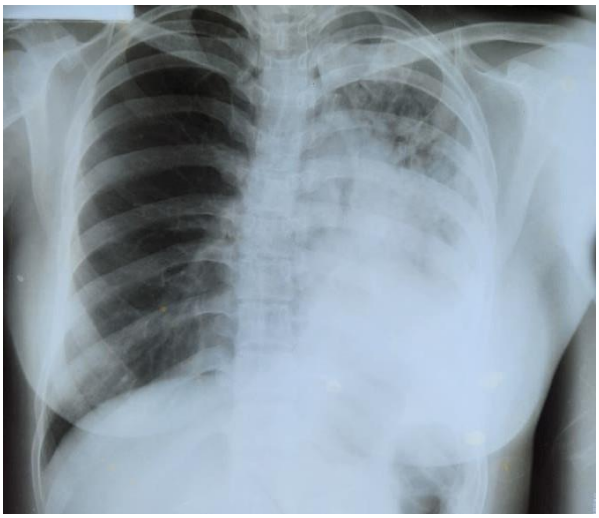


Figure 1. Chest Radiograph (2014).
Active tuberculosis.

There are patchy opacity in the apex - upper zone of the left lung accompanied by a shift of the mediastinal compartment to the left thoracic cavity and blunting of the left costophrenic angle suggesting active tuberculosis with suspected left pleural effusion.

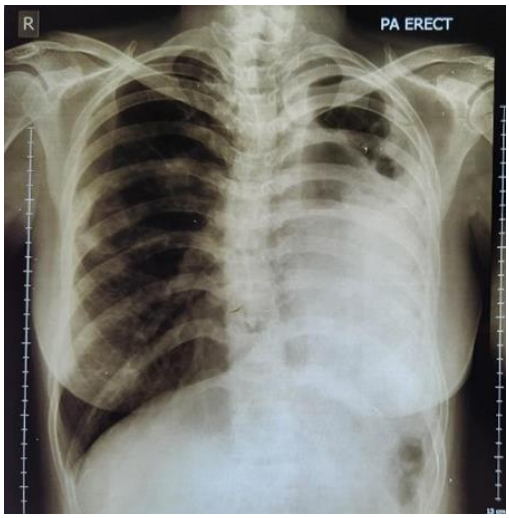


Figure 2. Chest Radiograph (2016).
Active tuberculosis and destroyed left lung.

Two years after treatment showed patchy opacity in the upper and middle zones of the right lung, suggesting active tuberculosis, and destroyed the left lung. The patient started MDR Tuberculosis therapy.

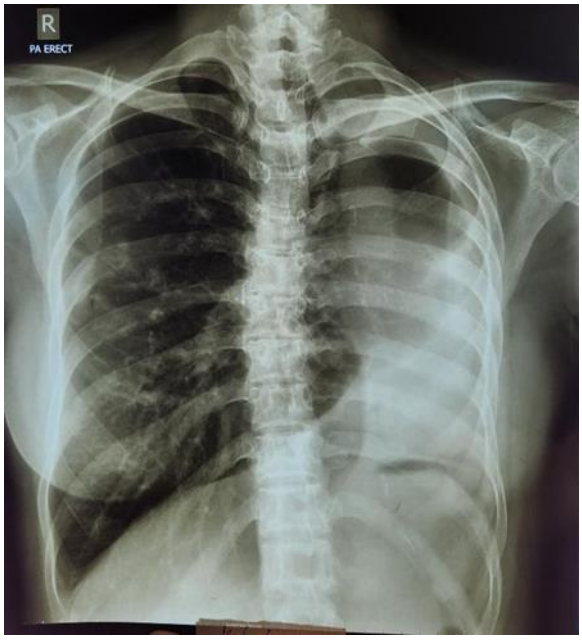


Figure 3. Chest Radiograph (2017).
Active tuberculosis with destroyed left lung.

One year after MDR Tuberculosis treatment, there is patchy opacity in the upper-lower zone of the right lung and destroyed left lung suggesting active tuberculosis with destroyed left lung.

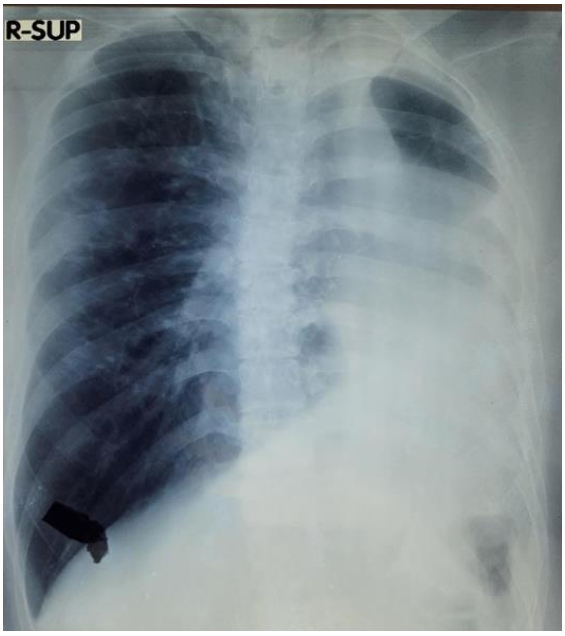


Figure 4. Chest Radiograph (2018).
Previous tuberculosis disease (inactive) with destroyed left lung.

After 2 years of MDR Tuberculosis Treatment. No patchy opacity and other signs of active tuberculosis. There are multiple nodules, and calcification in upper and middle zone of the right lung.

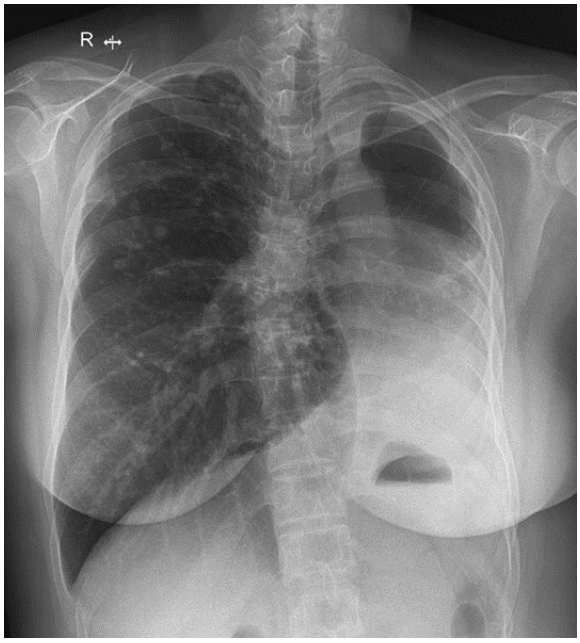


Figure 5. Chest Radiograph (2023).
Inactive tuberculosis with destroyed left lung.

There are multiple nodules, and calcification in upper and middle zone of the right lung, shifting of mediastinal compartment to the left thoracic cavity, inhomogeneous opacity in the left thoracic cavity, and compensatory hyperinflation of the right lung with herniation to the left, which suggesting previous tuberculosis disease (inactive) with destroyed left lung.

Physical examination showed BP (140/90 mmHg), RR (28 times/min), HR (110 bpm), Tempt. (36 ºC), SpO2 (70%), GCS (15), Inspection: asymmetric left thoracic cavity, rhonchi +/-, wheezing +/-. Laboratory investigations showed Hb (14.9 g/dl), WBC (9 x10^3/ µL), Hematocrit (43.1 %), ESR (14 mm/1 hour), neutrophils (58%). Chest radiograph shows multiple nodules, and calcification in upper and middle zone of the right lung, shifting of mediastinal compartment to the left thoracic cavity, inhomogeneous opacity in the left thoracic cavity, and compensatory hyperinflation of the right lung with herniation to the left. The Non-Contrast Chest HRCT scan demonstrates left lung destruction with shifting of mediastinal structure to the left, ipsilateral ribs crowding, contralateral hyperinflated lung as well as lung herniation to the left side. In addition to the tuberculosis sequelae findings, there is a sign of active tuberculosis in the form of tree in bud appearance.

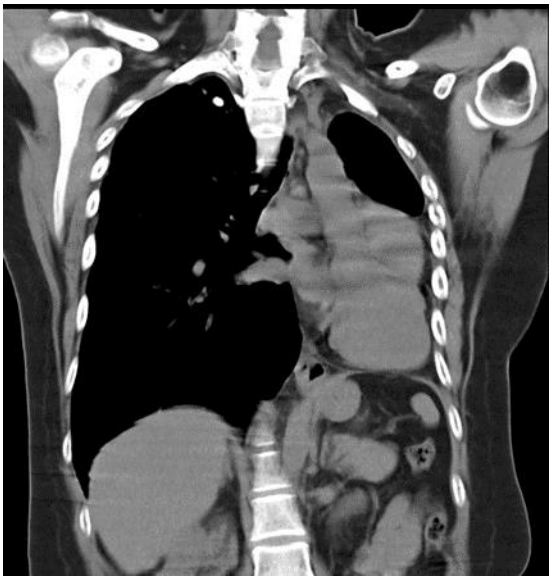
Discussion

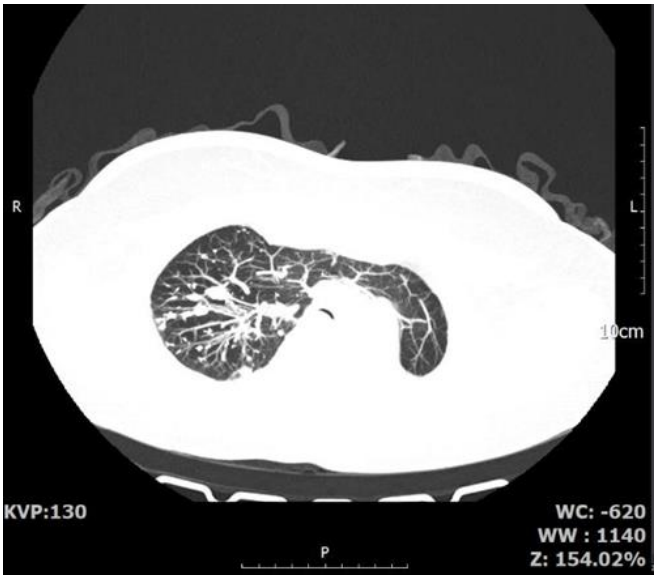
Tuberculosis is an airborne infectious disease caused by Mycobacterium tuberculosis.[9] Infection with tubercle bacteria that is genetically resistant to isoniazid and rifampicin is known as MDR-Tuberculosis.[10] The pathophysiology cycle of active tuberculosis begins and ends with aerosolization, which happens when a person with active tuberculosis forcefully expires (e.g coughing). Aerosolization, phagocytosis by macrophage, phagolysosome blockage and replication by M. Tuberculosis, TH1 response, granuloma formation, clinical symptoms, and transmission are the seven processes that make up the pathophysiology of active tuberculosis.[11] Patient with active pulmonary tuberculosis may be asymptomatic or having symptoms such as nighttime fever, sweating, weight loss, fatigue, and loss of appetite. Other additional clinical manifestations in patients with highly contagious pulmonary tuberculosis are persistent cough with or without bloody sputum, breathlessness and chest pain.[1,9]

Based on clinical and radiological findings, tuberculosis can be classifed into class 0 (no exposure and no infection of tuberculosis), class 1(exposure to tuberculosis; no infection), class 2 (latent tuberculosis), class 3 (active tuberculosis), class 4 (inactive tuberculosis/ previous tuberculosis disease) and class 5 (suspected tuberculosis; diagnosis pending).[7] To diagnose tuberculosis, several tests need to be performed, including laboratory tests and imaging. Detection of latent tuberculosis can be done through interferon-gamma release assay or tuberculin skin test. Active tuberculosis detection can be done through NAATs (Nucleic Acid Amplification Tests) including DNA extraction and polymerase chain reaction (PCR) amplification; Xpert MTB/RIF assay (Molecular diagnostic test Xpert mycobacterium tuberculosis/rifampicin resistance), and histopathological examination (staining using acid- fast method and culture) as well as medical imaging.[1,7]

Imaging plays a crucial role in initial evaluation, diagnosis and treatment of active tuberculosis.[7] In current practice, diagnosis of active TB relies on bacteriological examination and chest radiograph. [8] The same practice is also conducted in Indonesia, where patients with suspected TB are subjected to bacteriological examination (microscopic examination (AFB), TB molecular rapid test and culture). A chest radiograph will be conducted in case of the bacteriological test is negative.[12]

Chest radiographs have been used for almost a century in diagnosing Tuberculosis and play a major role in the management of patient with Tuberculosis infection. [8,13] Typically, a single PA view is obtained, sometimes with adjunctive views (lordotic view or dual-energy radiography).[7] It can show endobronchial and hematogenous spread of infection as well as airspace and airway sequelae of Tuberculosis.





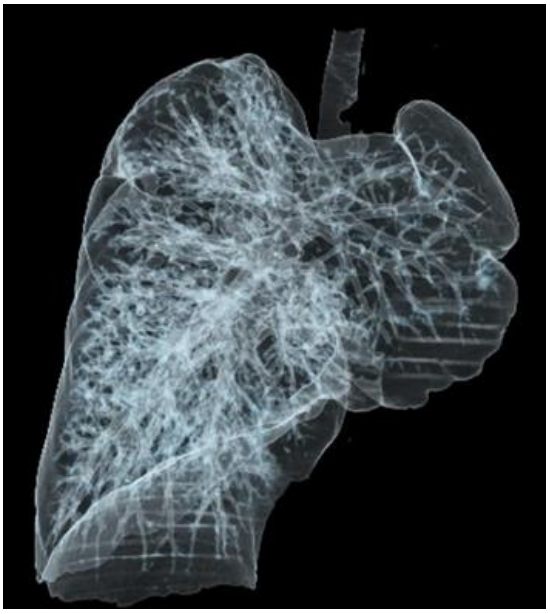


Figure 6. Chest HRCT (2023).
Active Tuberculosis with destroyed left lung.

Non-Contrast Chest HRCT demonstrates left lung destruction with shifting of mediastinal structure to the left, ipsilateral ribs crowding, contralateral hyperinflated lung as well as lung herniation to the left side. In addition to the tuberculosis sequelae findings, there is a sign of active tuberculosis in the form of tree in bud appearance.

Endobronchial spread in Active Tuberculosis can be seen as bronchial wall thickening, branching opacity, endobronchial plug and poorly defined centrilobular nodules (5-10 mm). Hematogenous spread to the pulmonary parenchyma on chest radiograph gives the appearance solid bilateral micronodules (miliary pattern) and may have basal predominance (more common among immunocompromised patients).[13] However, chest radiographs have limited specificity, high intraobserver and interobserver variability.[8,13] Chest radiographs may give a normal imaging in 15% of tuberculosis patients, and misdiagnosis in 30% cases of primary tuberculosis.[13]

HRCT play an important role in patients with suspected mycobacterial infection.[13] HRCT has a high Sensitivity (89.09%) and Specificity (79.25%) for diagnosing tuberculosis.[14] Compared to chest radiograph, HRCT is more sensitive in the detection, characterisation and distribution of subtle findings in parenchymal disease.[15] It can show abnormalities in almost 50% of patients with normal chest radiograph and persistent symptoms to empiric antibiotic therapy.[8] HRCT also helpful in differentiating between active and inactive tuberculosis and assessing medical (antituberculous) treatment efficacy.[13] It is also more sensitive in detecting endobronchial spread (a finding that indicate a presesence of activity), detecting reactivation within areas of dense fibrocalcific disease and more accurately define lymph node disease as well as can reveal miliary and pleural disease that not visible on chest radiograph. [13] Imaging findings in active tuberculosis including patchy unilateral/ bilateral consolidation, tree in bud, cavitation (variable morphology of cavity wall (thin/thick), hilar-mediastinal lymphadenopathy with ring enhacement and central necrosis, pleural effusion, miliary disease and bronchopleural fistula, empyema necessitans.[13] The most important findings to distinguish active from inactive tuberculosis are branching linear opacity, airspace consolidation, centrilobular nodules and tree-in- bud appearance. [13]

Post tuberculous lung disease or sequelae is a result of direct damage caused by mycobacterium tuberculosis in the lower respiratory tract and host immune response. These processes lead to lung

parenchyma and vasculature damage, airway distortion, decreased elasticity, muscular components of bronchial walls destruction, structural pathology, and distorted anatomical images.[16] Airway and Airspace sequelae including bronchiectasis, cavities, broncholithiasis, tracheobronchial stenosis and extensive lung destruction. Others major sequelae of tuberculosis involving pleura (chylothorax, bronchopleural fistula, fibrothorax), mediastinum (fibrosing medistinitis), large vessels (pseudoaneurysm) and bone (spondylitis and osteomyelitis) are variably depicted in chest radiograph and need other imaging methods such as CT and MRI.[13]

The appearance of strains resistant to pharmacological treatment may increase the possibility of complications and sequelae.[17] Lung destruction is an ireversible sequela that result from Pulmonary Tuberculosis. Destroyed lung is defined as combination of pleural and parenchymal lung destruction with bronchiectasis, cavitation, loss of lung volume and ipsilateral mediastinal herniation. [17,18] It is characterized with left-sided predominance and anterior/posterior (retrocardiac) herniation of the contralateral lung.[17] Based on radiological patttern destroyed lung can be classified into without (25%) and with cystic bronchiectasis (85%).[17] The structure of the left pulmonary bronchus, which is longer and smaller in diameter and crosses a narrow anatomical area, the aortopulmonary window, is what causes left-predominance lung damage.[18]

Lung function impairment may persist in up to 50% of tuberculosis patients who have microbiologically cured.[19] Destroyed lung can remain asymptomatic for ten years or so after the initial illness, but then they can cause a number of issues, including progressing dyspnea that results in irreversible respiratory insufficiency and recurrent pulmonary infectious episodes.[18] Some of complications of destroyed lung including empyema, hemorrhage, secondary fungal infections and sepsis.[8]

We present a case of a patient with active pulmonary tuberculosis and lung destruction e.c. advanced pulmonary tuberculosis (MDR-Tuberculosis) (declared cured in 2018 after 2 years of treatment). This patient was evaluated with chest radiograph and chest HRCT. Chest radiograph findings in 2014 sugesting an active tuberculosis with destroyed left lung, and after 2 years of tuberculosis treatment (2016), the chest radiograph still sugesting an active tuberculosis with destroyed left lung, and this patient start MDR Tuberculosis therapy. In 2017, one year after MDR Tuberculosis treatment there are still patchy opacity in upper and lower zone of the right lung which suggesting active tuberculosis. In Chest radiograph 2018, there are multiple calcified nodules in upper and middle zone of the right lung which is suggesting inactive tuberculosis. In 2023, this patient came to emergency department with main complaint of dyspnea and was examined by chest radiograph and chest HRCT. The findings on the chest radiograph (2023), depicted an impression of inactive tuberculosis but on further examination with chest HRCT, there was a tree-in bud appearance that suggested active tuberculosis. This shows that there are limitations in the diagnosis of tuberculosis activity using chest radiograph. HRCT examination is needed in evaluating active tuberculosis because HRCT has much better sensitivity and specificity than chest radiograph.

Conclusion

Due to the significant role of HRCT in the diagnosis of tuberculosis activity, it is necessary to consider the use of HRCT in the evaluation of patients with tuberculosis.

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