

Pyopneumothorax as an Advanced Complication of Tuberculosis

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ABSTRACT

Tuberculosis is a common infectious disease in our community. The management of this disease become more difficult and also the prognosis when there were an advanced complication and a high prevalence of multidrugs resistance. We report a 40-year-old female patient with pyopneumothorax with history of discontinuing tuberculosis drugs before.

Key words: Pyopneumothorax, Tuberculosis

INTRODUCTION

Tuberculosis is an infectious disease caused by *Mycobacterium tuberculosis* (*M.tuberculosis*). Clinical features include a generally prolonged latency period between initial infection and overt disease, prominent pulmonary disease (although other organ can be involved), and a granulomatous response associated with intense tissue inflammation and damage.¹⁻⁴

Symptoms of respiratory tuberculosis are cough, sputum, blood spitting, chest wall pain, breathlessness, localized wheeze, and frequent cold. General symptoms are loss of weight, fever and sweating, tiredness, and loss of appetite. On physical examination there might be rales in the upper part of one or both lungs. Later there may be dullness to percussion or even bronchial breathing in the upper part of both lungs. Diagnosis can be made by finding acid fast bacilli in a direct smear of the sputum and performing chest x-ray.¹⁻⁴

Almost all patients with newly diagnosed tuberculosis can be cured if they are properly treated. The antituberculosis drugs should be given as combination of several drugs with suitable dose, at least for 6 months. The first line (essential) drugs are rifampicin (R), isoniazid

(H), streptomycin (S), ethambutol (E), pyrazinamide (Z). Inadequate treatment would cause drug resistance to the antituberculosis drugs.¹⁻⁷

PYOPNEUMOTHORAX

The signs and symptoms of extrapulmonary tuberculosis depend on the organ involved. In pleural effusion, clinically, it feel pain while breathing (pleuritic pain), fever, slight irritating cough, breathlessness on exertion, dullness on percussion over the lower part of the chest, no sound of air entry. The pleura may be affected in three different ways :⁸⁻¹⁰

1. Effusion which develops within a few months of primary infection in children and young adults
2. Effusion which develops as a result of lung disease in older adults. Rarely this may be go on to a purulent effusion (empyema).
3. Rupture of a tuberculous cavity and escape of air into the pleural space. This allow air to escape into the space between the lung and the chest wall. The tuberculosis from the ruptured cavity produce a purulent effusion (empyema). The air and the pus together are called pyopneumothorax.

There are two important things in initial management of pleural effusion, the administration of suitable or prompt antibiotic and insertion of water sealed drainage (WSD) when necessary. Management of pleural effusion include chest tube and image direct catheter, intrapleural antibiotics, intrapleural fibrinolytics, thoracoscopy, decortication, or open drainage. WSD is expected to drain out as much as possible so the lung can inflate with the aid of negative pressure of the WSD. The benefit of using intrapleural antibiotics is still on clinical trial however several studies show positive outcomes. The goal of administration of fibrinolytics is to prevent fibrinolysis, to increase fluid drainage, and prevent possible surgical intervention. Thoracoscopy should be considered in patients with loculated effusion irresponsive to intra-

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pleural thrombolytics. If the lungs can not inflate due to thick fibrin covering the visceral pleura, decortication is the best choice of treatment¹⁰

CASE REPORT

Mrs. Y, 40-year-old, came to the emergency room of Cipto Mangunkusumo General Central National Hospital because of chest pain since three days before admission.

Seven years before admission she took antituberculosis drugs two times after she got cough and hemoptysis. Unfortunately, she stopped the medication by her self because she felt better. One year later she got the same symptom again, and she went to primary health care. After she had performed chest x-ray and sputum analysis, she got antituberculosis drugs (combipack). The injection drug was avoided because of sign of allergy was shown in the skin test. Six month later, she still had dyspnea and stopped the medicine by her self. There were histories of night sweating and weight loss.

Three days before admission she got chest pain in the right side especially when she was taking a deep breath. The pain was not radiated, associated with activities, and she felt better if she slept with two or three pillows. There were no difference between slept with left or right side on the bed. She also had a productive cough with purulent sputum. Her voiding and defecation were normal.

She has been married and has seven children. There were no history of asthma, hypertension, diabetes melitus, and cardiovascular disease before either her family. She had allergy to streptomycin.

On admission, the temperature was 37,5°C, the pulse was 100 times/minutes, and the respiration was 30 times/minutes. The blood pressure was 100/60 mmHg. The body weight was 29,5 kg and the height was 150 cm. The patient was mentally alert. The conjunctiva was not pale and the sclera was not icteric. Jugular venous pressure was 5-2 cmH₂O. The heart sound was normal. From the inspection the right side of the chest was left from her left side on the breathing. In the right side, the fremitus was decreased and the percussion sound was hipersonor until the 5th right rib and dull next. The breath sound was absent. The auscultation of the left lung was vesicular and there was rales especially in the apex. The abdomen was supple, no shifting dullness, and no tenderness. Liver and spleen were not palpable. The extremities were warm and there were not edema.

Laboratory result showed hypoxia. There were right

hydropneumothorax with atelectasis component and left lung tuberculosis in radiography finding.

The diagnosis of the patient were right hydropneumothorax and lung tuberculosis. The patient underwent tube drainage procedure by surgery department. They performed tube insertion into 6th intercostal in mid axillary line. There were initial bubble, undulation but no initial bleeding, no initial fluid, and no expiratory bubble.

The next eight hour, the patient was moved to (5th floor of IRNA B). In the examination the patient was still dyspnea. From the tube there yellowish greenish fluid came out to the bottle. There were crepitation on the right chest.

On the ward the patient's problems was concluded as right pyopneumothorax, subcutaneous emphysema of the right anterior chest, lung tuberculosis, and poor nutrition status. The treatment were O₂, 4-6 L/m, ceftriaxone 2 gram once daily, rifampicine 300 mg once daily, isoniazid 300 mg once daily, pyrazinamide 250 mg three times daily, ethambutol 250 mg three times daily, ofloxacin 400 mg twice daily, paracetamol 500 mg three times daily, bromhexine-HCl 15 ml three times daily, and high calory rich protein diet. The chest physiotherapy was also planned.

On the 6th admission day the pain was decrease but the patient still had dyspnea. There were still hydropneumothorax on second radiographic findings. The pleural fluid analysis showed exudate. From the three sputum examinations of acid bacilli, there was only 1 positif result. The fluid production was about 100 cc daily. On the 9th hospital day the result of pleural fluid culture was *Pseudomonas aeruginosa* that resistant to almost all antibiotics except ceftazidime. The microorganism from sputum culture was *Pseudomonas sp.* There were data about ceftriaxone resistancy. Due to clinical assesment the ceftriaxone was continued.

The radiogram on 13th day didn't show any improvement of the lung expansion. The next plan was bronchoscopy to look for something that possibly obstruct the air track that could disturb lung expansion. Unfortunately the patient had a sudden bronchospasm and died on 17th day of admission.

DISCUSSION

This case was a classic history of lung tuberculosis. The first infection that incomplete treatment continued with caverne on the apex of the lung. The next treatment did not completed either. The last treatment (4 years before) least for 6 month but she stopped by herself because it worsened. The tuberculosis manifestation in

the lung became worst. And the patient came with pyopneumotorax as late complication of the tuberculosis. There might be an acid bacilli resistant in this patient because of several interrupted or discontinued tuberculosis treatment. However there was no acid bacilli in the culture so there was no result of bacil resistance to anti-tuberculosis drugs.

There were old chest radiogram, when she took 1st, 2nd, and 3rd antituberculosis treatment. The first showed light cloudy on the apex area. On the second radiogram, the cloudy became worst. The third radiogram showed cavity on the right apex area.

The diagnoses of pyopneumothorax was based on history chest pain, dyspnea. On physical examination, we got the right chest was left on the respiration, low fremitus, and hipersonor-dull on percussion. The chest radiogram showed air fluid level, and pleural fluid analysis referred to the exudative fluid. The sputum microorganism culture results matched to pleural fluid lets us thinking there was infection and connection between pleural space and bronchial air track.

Lung tuberculosis based on history of hemoptysis, body weight lose, night sweating, had a antituberculosis drug before. We can classify this tuberculosis as type 2. The drugs that should be given are RHZES but the patient had allergy to streptomycin and also there was possibility the tuberculosis already resistant to the drug that already given before. Especially when she had the third treatment that could not cure her illness.

Table 1. Antituberculosis Drug Base on Body Weight

Antituberculosis Drugs	Dose	Maximal Dose	Dose in This Patient (BW 29,5 - 30 kg)
Rifampicine	10 mg/kg	600 mg	300 mg/kg
Isoniazid	5 mg/kg	300 mg	150 mg/kg
Pirazinamide	15 - 30 mg/kg	2 g	450 - 750 mg
Elhambutol	15 - 30 mg/kg	2,5 g	450 - 750 mg
Streptomycin	15 mg/kg	1 g	450 mg

Poor status nutrition based on body weight and height. The body mass index was 13,33 kg/m² (normally 18-5-22,9 kg/m²). The plan was to provide high calory and rich protein diet. The body weight target was 45 kg.

On the 6th day we already discussed this patient with thorax surgery staff. The problem was the chest radiogram was not showed improvement although the patient felt better. From the of discussion we concluded there were a bronchopleural fistel caused by infection that disturb lung expansion. The nonspecific and specific antibiotic should be given at least 3-4 weeks. If the expansion

of the lung was not showed, the thoracis surgery would performed pneumectomy. Unfortunately the patient was dying before.

There were several interesting point of this patient's disease. First, how long the pyopneumothorax has occurred in this patient. Second, which one occurred first. Third, what was the connection between disease and interrupted or discontinued treatment of antituberculosis drugs. We thought that the cause poorly improvement of lung expansion to bronchopleural fistel but there was no expiratoar bubble from the chest drain. From the history, the patient had been felt dyspneu although she already on antituberculosis drug for sixth months of the last treatment that her took. The dyspneu became worst until her coming to this hospital. The patient should have a caverne or bullae that broke up and there were 'silent' pneumothorax that might be bigger. The result were the lung collapsed and poor aeration on the pleural space.

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