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CASE REPORT

COMPLICATED *KLEBSIELLA PNEUMONIA* INFECTION CAUSING LUNG ABSCESS: CASE REPORT

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ABSTRACT

Background: Lung abscess is a severe pulmonary infection characterized by localized suppuration and necrosis of lung parenchyma, often associated with high morbidity and mortality. Klebsiella pneumoniae is a frequent causative pathogen, especially in immunocompromised patients.

Objectives: To present a case of lung abscess caused by Extended-Spectrum Beta-Lactamase (ESBL) producing Klebsiella pneumoniae in a patient with dual immunosuppression due to HIV infection and uncontrolled diabetes mellitus, and to highlight the clinical challenges in its management.

Results:A 58-year-old male with HIV infection on regular antiretroviral therapy and uncontrolled type 2 diabetes mellitus presented with shortness of breath, productive cough, and fever. Radiological examination revealed a large cavitary lesion with an air-fluid level in the left hemithorax, accompanied by empyema and pneumothorax. Chest tube drainage yielded 1500 mL of purulent fluid. Microbiological culture identified ESBL Klebsiella pneumoniae. The patient was initially treated with intravenous levofloxacin and metronidazole, later switched to moxifloxacin based on antibiotic sensitivity, leading to significant clinical improvement.

Conclusion: This case emphasizes the complexity of managing lung abscess in immunocompromised patients with uncontrolled comorbidities. Risk factors such as large cavity formation, empyema, multidrug-resistant pathogens, and poor metabolic control contribute to poor prognosis. Effective management requires adequate drainage, culture-guided antibiotic therapy, and control of underlying conditions. Prolonged antibiotic therapy with close radiological and clinical follow-up is recommended to optimize outcomes and prevent recurrence.

Keywords**:** Klebsiella pneumoniae, lung abscess, empyema, HIV, type 2 diabetes mellitus, ESBL, chest tube drainage

INTRODUCTION

Central suppuration and necrosis of the lung parenchyma tissue can cause damage and lung abscess resulting small or large and single or multiple cavities. Epidemiologically, it causes morbidity and mortality rate around 15-20% cases. Better antibiotic treatment and advance surgical intervention lessen the complication of lung abscesses except in high-risk patients such as prone to aspiration in cerebrovascular disease patient, alcoholism, and in immunocompromised population.[1] If treated carefully, uncomplicated lung abscess should have better prognosis with cure rate of 90-95% and low mortality rate 0,01%[3] .

Pure empyema without lung abscess has different consideration for surgical intervention. In lung abscess, such intervention is only reserved for complicated cases or unresponsive to antibiotic treatment. Complicated lung abscess could also result as empyema, increasing the risk of dissemination focal infection to adjacent lung parenchymal lobe, hemoptysis, and fibrosis, lowering lung function after infection[3] .

Most cases of lung abscess involve polymicrobial infection (aerobe and anaerobe microorganisms). Anaerobic bacteria are predominated by gram negative: *Bacteroides fragilis, Fusobacterium capsulatum* and *necrophorum;* and gram positive: *Peptostreptococcus* and *microaerophilic streptococci*. Isolate aerobe bacterias mostly found: *methicillin resistant Staphylococcus aureus (MRSA), Streptococcus pyogenes, S. pneumonia, Klebsiella pneumonia, Pseudomonas aeruginosa, Haemophilus influnza* (type B), *Acinetobacter spp, Escherichia coli,* and *Legionella spp*[8]. *Klebsiella pneumonia* is one of the most prevalent isolates found in lung abscess especially in Taiwan. *K. pneumonia* is actually a facultative gram-negative rod with large polysaccharide capsule an Enterobacteriaceae class transmitted to upper respiratory tract through inhalation (droplets). Mortality caused by lung abscess ranging from 25-50%. It's mostly found as nosocomial bacterial infection but also could be found in community acquired population (cause of pneumonia 6,3% cases in Indonesia)[2].

Risk factors associated with complicated and rapidly progressing lung abscess includes immunocompromised patients and multidrug-resistant bacteria[13]. HIV patients with type 2 DM have greater susceptibility to infection due to immune defects from hyperglycemia and chronic HIV viral replication. These

conditions require cautious and careful management. Uncontrolled risk factors could worsen immune defect causing complicated infection. Here we reported a case in a male with ESBL *Klebsiella pneumonia* infection complicated by lung abscess in HIV patient with type 2 DM.

CASE

Anamnesis

A 58-year-old male living in Surabaya was admitted to emergency room in Dr. Soetomo General Academic Hospital with chief complaint shortness of breath in the last 3 days. The complaint was preceded by cough with yellow-non-bloody phlegm and has not recovered in the last 3 weeks. There was no nausea and vomiting. High fever was denied but he frequently reported nighttime feverish with cold sweat since the last 2 weeks. The patient lost 5 kgs of weight in a month. He felt dyspnea especially during daily activities such as walking around the house less than 10 meters. It lessened with sleeping on the left side with two pillows. There were no swelling of both legs and no urinary problem. It was reported that the patient had taken antibiotics given by general practitioner but the complaint did not improve after 5 days.

Past medical history revealed that he was diagnosed having HIV infection with tenofovir-lamivudine-efavirenz combination since 2016. He was first diagnosed having lung tuberculosis infection which he then recovered after completion of 6-month treatment with anti-tuberculosis drug in the same year. He took the ARV regularly afterwards. He was also diagnosed as non-insulin-dependent diabetes mellitus (type 2 DM) receiving glibenclamide 5 mg/a day in the last 6 month before admission. The patient had no history of hypertension, previous kidney disease or heart disease. He had two COVID-19 vaccinations. There was no history of allergy.

There was a promiscuity history in the first marriage and he was divorced later. He now lives with his wife from the second marriage and a daughter. There is no history of lung tuberculosis in the family. He was an active smoker 12 cigarettes a day which he had stopped for 2 years (starting 30 years old). There was no history of recurrent dyspnea before and no history of COPD and asthma before.

A week before admission he visited UPIPI as an outpatient and sputum examination by Gene Expert was done which resulted as negative for *Mycobacterium tuberculosis*.

Physical Examination

General condition was weak and there was shortness of breath. He was alert with Glasgow Coma Scale E4V5M6 and hemodynamically stable: blood pressure 124/82 mmHg, heart rate 101x/min; respiratory rate 24x/min and axillary temperature 36.7^{o} C; peripheral oxygen saturation 94% room air. He had normal Body Mass Index (BMI) 21,79 kg/m2 (height 174 cm and body weight 68 kgs). Head and neck examination revealed there was pale conjunctiva, no scleral icteric; no lymph nodes enlargement in the head and neck area. Cardiovascular examination was normal, so was abdominal area, there was no liver or spleen enlargement and normal peristaltic sound. Lung examination revealed lagging left chest wall movement, with decreased fremitus palpation of left hemithorax and faint percussion at the lower 1/3 to mid left hemithorax. On auscultatory examination, there was a decrease in vesicular sound in the lower 1/3 of the left hemithorax and rhonchi was positive. The patient was consulted to a pulmonologist in the emergency room with suspected lung abscess, then further suggested thorax HRCT examination to localize anatomical lung affected and to differentiate with pure empyema process. The extremities was warm with CRT <2 seconds, and there was no edema.

Additional Supportive Investigation

Laboratory results: Hb 10.7 g/dL; Hct 30.6%; MCV 84.8 fL; MCH 29.6 pg; WBC 17710/µL; Basophils 0.3%; Eosinophils 0.2%; Neutrophils 82.9%; Lymphocytes 7.6%; Monocytes 9%; Platelets 630,000/µL; SGOT 49 U/L; SGPT 45 U/L; Albumin 3.11 g/dL; Random Blood Glucose 274 mg/dL. While the results of blood gas analysis using 4 lpm nasal cannula oxygenation: pH 7.5; pCO2 38 mmHg; pO2 69 mmHg; HCO3 29.6 mmol/L; BE 6.4 mmol/L; and SO2 95%. With serum electrolyte results Na 129 mmol/L (correction value in hyperglycemia: 132 mmol/L); K 3.6 mmol/L; and Cl 96 mmol/L with serum osmolarity calculation of 282 mOsm/kg (Normal 285-295 mOsm/kg).

X-ray of the thorax in the anteroposterior and left lateral positions found that the trachea was in the middle, the right anterior and posterior costophrenic sinuses were sharp while the left anterior was covered by a ridge. The heart was normal in size and shape, while the lung showed infiltrates on the right perihilar and paracardial and thick cavities on the left paracardial with air fluid levels projected on the lateral photograph. The right hemidiaphragm had no abnormality but the left was covered with a ridge. The retrosternal and retrocardiac areas were partially covered by a ridge, with no soft tissue or bone abnormalities.

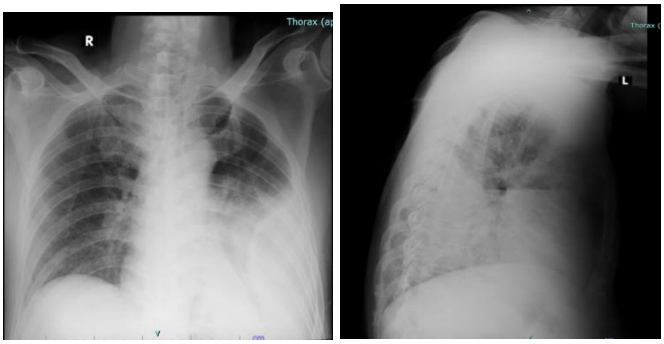

Figure 1. X-ray AP view (left) and lateral view (right) 1st day admision

Figure 2. Aspirated fluid reveal empyema

Diagnosis and Treatment

Patient then diagnosed as bacterial pneumonia PSI score 98 class risk IV with suspected lung abscess; hyperglycemia in type 2 DM; anemia of chronic disease; hypoalbuminemia related to catabolic conditions caused by infection; HIV infection on ARV treatment (FDC: Tenofovir-Lamivudine-Efavirenz); hypovolemic hypotonic hyponatremia related to decreased intake. Patient was given supplemental oxygen with nasal cannula 4 L/min; diabetic diet B1 2200 kcal/24 hour; crystalloid infusion of asering 500 ml/24 hour added by water consumption through drinking 1500 ml/24 hour; antibiotic: IV levofloxacin 750 mg/24 hour and IV metronidazole 500 mg/8 hour; fast-acting insulin aspart 4-4-4 unit pre-prandial subcutaneously; continuing ARV (FDC: Tenofovir-Lamivudine-Efavirenz) once daily; N-acetylcysteine 200 mg/8 hour. Patient was consulted to pulmonologist and HRCT was planned to differentiate lung abscess with lung empyema.

FOLLOW UP

In the second day of hospitalization there were no new complaints and new laboratory test was done revealed HbA1c value was 10,6%. Insulin was given on the first day of hospitalization at a dose of 4-4-4 units pre-prandial SC. Glargine insulin dose 1x10 units SC night was given since the 2nd day of treatment with a previous GDP (Fasting Blood Glucose) of 220 mg/dL (capillary stick). Pre-lunch blood sugar profile on 3rd day was 213 mg/dL and pre-dinner GDA 224 mg/dL. The dose of pre-prandial insulin aspart was increased to 6-6-6 units sc and blood glucose was controlled as targeted by adding glargine insulin (long-acting insulin) up-dosing gradually started from 0-0-10-unit SC until 0-0-18-unit SC through daily blood glucose profile monitoring (blood glucose achieved on the 6th day: fasting blood glucose 115 mg/dL; pre-lunch blood glucose 120 mg/dL; pre-dinner blood glucose 130 mg/dL).

In the 2nd day, MSCT (Multi Slice Computed Tomography) thorax with and without contrast was also done waiting for result up to 2 days afterwards. It revealed: a thick fluid density lesion (22HU) accompanied by air (-970 HU) with 7,2 cm in diameter, was found in the left pleural cavum which after contrast administration obtained rim contrast enhancement (57 HU). There was a bronchial branch track in the anterobasal segment of the inferior lobe of the left lung and approaching the pleura which was still a possible form of fistula (could not be excluded). Consolidation in the left lung lobe and ground glass opacity in the superior lobe of the left lung. There was no picture of atelectasis or lung collapse. Multiple lymph nodes were found with the largest size +/- 1.7 cm in the left parabronchial. Conclusion MSCT thorax were: pneumothorax and left empyema with suspicion of fistulation in the anterobasal segment of the inferior lobe of the left lung, left parabronchial lymphadenopathy and pneumonia. Procalcitonin result was 0.14 ng/mL (local infection) on the second day of treatment. Empirical antibiotic therapy was continued, waiting for culture result obtained in the first day.

HIV viral load and CD4 were also obtained in the 3rd day and there was no viral load detected and CD4 cell count was 464 cell/uL indicating successful ARV treatment.

Spontaneous expectorated sputum examination by gram staining and KOH was not obtained through hospitalization. Pulmonologist consultation suggested empirical antibiotic treatment for at least 3 days and chest x-ray follow up concluded that fluid-pneumothorax of left hemithorax was not improved. As MSCT concluded localization of pus build-up chest tub drainage was done in the 6th day of hospitalization. The drain produced 1,3 liters of pus in the first day, 200 mL the second day, and there was no pus production in the third day. Chest tube then inserted out at the 9th day. From pus obtained microbiological aerobic culture revealed ESBL (Extended Spectrum Beta-Lactamase) *Klebsiella pneumonia* sensitive to amikacin, cefoperazone-sulbactam, ceftriaxone, gentamicin; imipenem; meropenem; moxifloxacin; trimethoprim-sulfamethoxazole. In the 6th day after admission, antibiotic given was changed to intravenous moxifloxacin 400 mg/24 hour. There was clinical improvement (improve dyspnea; less cough production but only complaining pain at the chest tube insertion marks) following definitive antibiotic treatment for 3 days and lowered leucocyte count to 12.000/uL. Another follow up thorax x-ray was done before discharge revealed left loculated pleural effusion with pneumothorax and pneumonia and avascular lucent without lung parenchyma in the left lower hemithorax.

The patient was discharged after 12 days of hospitalization and scheduled for control 3 days later. The medication given after discharge were: Paracetamol 500 mg/8 hours PO; N-acetylcysteine 200 mg/8 hours PO; Glargine 0-0-18 units SC; Aspart insulin 6-6-6 units SC pre-prandial; Moxifloxacin 400 mg/24 hours and education to continue ARV FDC (tenofovir-lamivudin-efavirenz) 1tab/day. At the time of control there were complaints of minimal pus seepage in the area of the former chest tube without coughing. There was no fever or shortness of breath. The patient referred to pulmonologist for wound care and suture control at chest tube insertion mark.

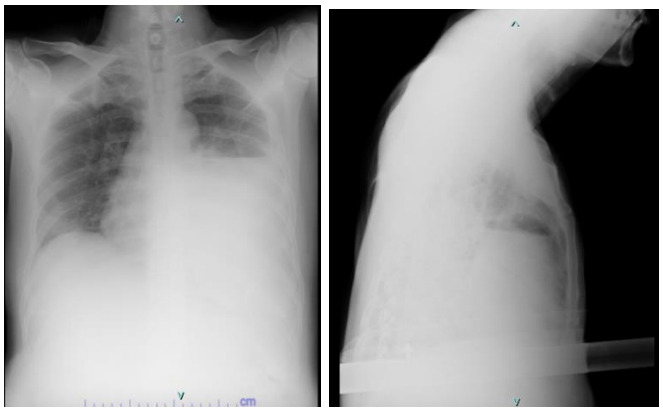

Figure 3. X-ray AP view (left) and lateral view (right) 3 days after empirical antibiotic showed no improvement

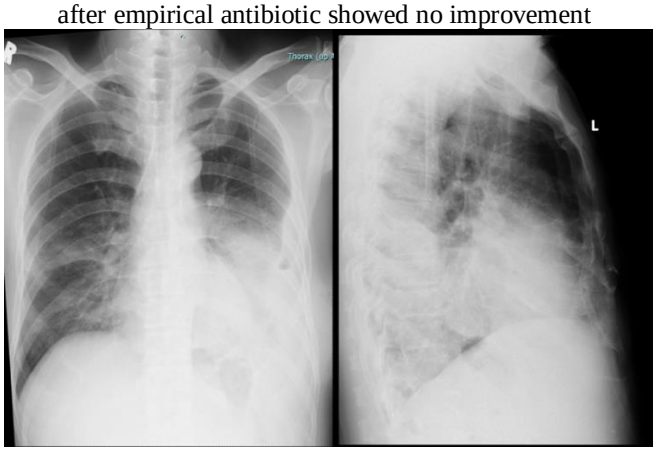

Figure 5. X-ray AP view (left) and lateral view (right) 3 days after chest tube removal

DISCUSSION

Klebsiella pneumonia is an Enterobacteriaceae, cause of pneumonia and one of mostly found pathogens associated with nosocomial pneumonia (27%). It is transmitted to the lungs by aspiration from upper respiratory tract and by inhalation of respiratory droplets[10]. Hence it also could be found in community acquired based population with multi-drug resistance characteristic. *K. pneumonia* is a negative rod bacterium which has big polysaccharide capsule impeding phagocytosis by macrophage. Its endotoxin could cause fever and shock with sepsis. The type of inflammation reaction after *K. pneumonia* infection is pyogenic formation involving neutrophils as predominant cells. In immunocompetent individual first occurrence should be cleared by neutrophil. Numerous innate immune systems play important role against Kp: alveolar macrophages, monocytes, and neutrophils. IL-17 is a critical cytokine mediator in the recruitment of neutrophils to the site of Kp infection. Adaptive immunity lineage with CD4+ T cell proliferation of Th17 is important. Other interleukin such as IL-23 and IL-22 were subsequently found to exert protective function against Kp through modulation of alveolar macrophages[2, 4].

The immune response of type 2 DM patients is impaired due to various risks related to the body's protection against microorganism invasion, such as neuropathic conditions that increase the risk of disruption of the natural barrier (skin/mucosa). Conditions contributing to infection susceptibility in type 2 DM patients are suppression of cytokine production (interleukin 1 β (IL-1 β); IL-6; interferon ɣ (IFN-ɣ); TNF-α by T lymphocytes) resulting in defective phagocytosis, failure of leucocyte recruitment to the focus of infection (Klebsiella pneumonia infection study: failure of neutrophil recruitment in alveolar capillaries in DM condition), immune cell dysfunction in recognizing pathogen receptor particles via toll like receptor I (TLR), neutrophil dysfunction (decreased production of ROS (reactive oxygen species) used to kill pathogens; neutrophil degranulation defects), failure to kill microbes. Which in this case neutrophil/polymorphonuclear activation to activate phagocytosis remain important for protective response in *K. pneumonia* infection[1,11].

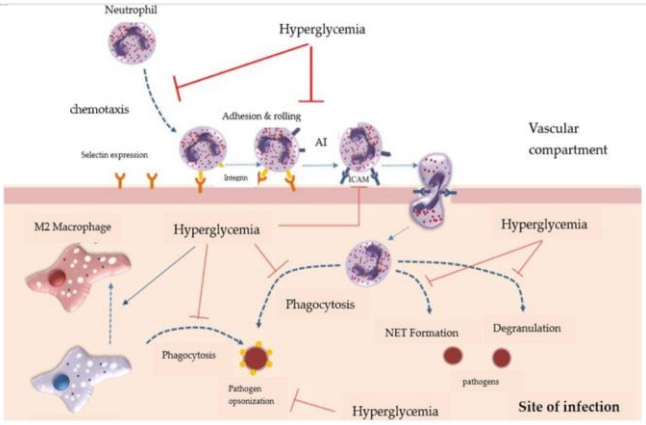

Figure 6. Impact of hyperglycemia on neutrophil dysfunction during *Klebsiella pneumoniae* infection.

Hyperglycemia impairs neutrophil adhesion, rolling, chemotaxis, phagocytosis, NET formation, and degranulation. Dysregulated macrophage activity and reduced bacterial clearance contribute to worsened infection severity and abscess formation in diabetic patients. Lung abscess most often occurs in men than women especially older age population. Immune system deficiency caused by chronic HIV infection or uncontrolled hyperglycemia in diabetes are risk factor contributing to lung abscess progression[13]. Air fluid level which is found radiologically through x-ray examination is a one of distinctive founding in lung abscess. Cavity from necrotic lung parenchyma caused by microbiological colonization manifests as single big cavity or multiple small cavities (<2cm) which is called as necrotizing pneumonia[14]. Mostly it's caused by anaerobic bacteria through bronchogenic spread (aspiration) and accompanied by purulent cough attacking mostly posterior segment of right superior lobe, followed by left lobe, superior segment of lower lobe right and left lung. Most often right hemithorax is affected due to its anatomical position which is straighter than the left bronchus. Other microbiological spreading is through hematogenic spreading. It is caused by septicemia or embolic septic or secondary to other focal infection such as tricuspid valve endocarditis or rupture of amoebic abscess from hepatic infection.

Multiple small abscess formation is mostly found in hematogenic spreading and caused by *Staphylococcus, Klebsiella pneumonia* and *Pseudomonas* group[13,8].

Predisposing factors for lung abscess are: 1) condition facilitating aspiration (impaired consciousness: CVD; coma; trauma;); 2) esophageal and gastrointestinal disorder: motility disorder; tracheal or nasogastric tube removing mechanical airway defenses; 3) mechanical airway defenses: tracheoesophageal fistula; 4) stasis of secretion through airway: Kartagener's syndrome; dysphagia; 5) iatrogenic causes; 6) periodontal cases; 7) poor oral hygiene; 8) bronchiectasis; 9) acute pneumonia; 10) lung cancer; 11) unresolved upper and lower respiratory tract infection; 12) immunocompromised patients: HIV with low CD4 count <50 cell/mm^3. Most occurrences have previous lung infection[13]. Click or tap here to enter text.

Case reported in 58-yo male with adequate ARV treatment (good viral control: undetected viral load and CD4 count 464 cel/uL). Patient had no bad oral hygiene nor condition facilitating aspiration. The affected segment was inferior lobe (anterobasal segment) in left hemithorax. He had history of cigarette smoking which he had stopped in the last 2 years. The glycemic control was not achieved (examined by random blood glucose check: 274 mg/dL). Air fluid level was found in the left hemithorax through CXR in lateral position and confirmed by MSCT examination with pyoneumothorax. Microbiological culture obtained from pus drainage revealed ESBL K. pneumonia with its virulence factor of antibiotic resistance which could worsen lung abscesses progression in this case.

Symptoms and signs could manifest as acute (<4-6 weeks) or slow progressing started after 1-3 weeks of general weakness, low appetite; weight lost; dry cough; cold sweat at nights; intermittent fever could achieve 39,4^{o}C or higher. After several days phlegm production may appear purulent and mixed with blood streak. Sputum production containing necrotic lung parenchyma may smell fishy with anchovy appearance which indicate anaerobe bacterial infection defined as putrid abscesses. Chest pain mostly accompanied by pleural process such as empyema and pneumothorax. Anaerobic pathogens are 89% causes of lung abscess in immunocompetent individual caused by aspiration (*Bacteroides fragilis; Bacillus intermedius; Prevotella melaninogenica; Clostridium perfringens* etc). Other aerobic bacterial could cause lung abscess in immunocompromised population: *Staphylococcus aureus; Streptococcus microaerophilic; Streptococcus pyogenes; Streptococcus pneumonia; Streptococcus viridans;* etc. Negative gram bacteria mostly caused by nosocomial bacteria: *Klebsiella pneumonia; Pseudomonas aeruginosa; Escherichia coli; Hemophilus influenza*. In cases such as immunocompromised population, atypical bacteria could be found: fungi such as *Histoplasma, Blastomyces; aspergilus species; cryptococcus; pneumocystis*; parasitic infection: *entamoeba histolytica*; *Mycobacterium tuberculosis* and non-tuberculosis. Most studies concluded that lung abscesses are a mixed bacterial infection: aerobic and anaerobic group. In HIV patient, most pathogens found are aerobic bacteria, fungi and *M. tuberculosis*[3,16].

Lung abscess is classified as primary and secondary. Primary lung abscess occurred through aspiration or complicated pneumonia in people with high risk of aspiration or even in general good condition or even in immunity suppression (HIV, transplant). 80% lung abscesses consist of primary lung abscess (50% of them had foul sputum) followed by 20% case of secondary abscess. Previous history of infection with underlying condition such as respiratory tract obstruction by neoplasm, bronchiectasis, intrathoracic complicated operation, or emboli septic were the causes of secondary lung abscess. Other classification included acute abscess (<1 month; or <4-6 weeks) and chronic abscesses if occurred more than that period of time. Foul breath odor in these patients was one characteristic of anaerobe organism with putrid abscesses sputum[9,8].

In this case, there was empyema which could result from parapneumonic effusion process which has 3 stages of development: started by exudative stage with sterile fluid accumulated in the pleural space infiltrated by inflammatory cell caused by pneumonia process; continuing to fibropurulent stage which predominated by more neutrophils cell and fibrin deposition (tend to be loculated) and lastly organized stage where fibroblasts grow into pleural walls and produce thick pleural peel, preventing lung expansion. Empyema primarily results from pneumonia but also could be the result of lung abscess, bronchopleural fistula, esophageal perforation, postsurgical complication, trauma etc. It could be detected through chest x-ray which appear as obliterated costophrenic angle caused by minimum of 175 mL fluid in upright position, but could be detected as little as 10 mL in lateral decubitus position. As most pleural effusion, symptomatic relief by evacuating fluid accumulated in pleural space is a therapeutic and diagnosis approach in this case. Removal of <1500 mL pleural effusion for the first time is recommended to avoid the risk of re-expansion pulmonary edema. After continuous daily empyema output of less than 50 mL in a day and fluid change to clear yellow, followed by re-expansion of lung and improvement clinical status, the chest tube could be safely removed[17].

Patient in this case had an acute lung abscess episode with previous history of unresolved productive cough for the last 3 weeks. He had no putrid abscess sputum but he felt feverish mostly at night accompanied by cold sweat. There was no hemoptysis but he felt shortness of breath during daily activities. Previous lower lung infection was detected 7 years ago after completion of anti-tuberculosis drug for 6 months. Empyema buildup accompanied by pneumothorax in this case were a result from lung abscess with uncontrolled

pneumonia infection. It was a primary lung abscess due to uncontrolled hyperglycemia in diabetes mellitus type 2 which worsen immune protective mechanism for infection without previously documented lung disease. Empyema was drained by chest tube insertion and total of 1500 mL fluid (1300 mL in 1st day; 200 mL in 2nd day; no fluid production in 3rd day) was obtained, and the tube was then removed after no further fluid accumulation and there was an improvement of clinical status.

Lung examination in lung abscess could reveal local chest tenderness accompanied by diminishing vesicular sound caused by consolidation process, faint percussion, and bronchial and rhonchi through auscultation. Big cavity could manifest as bronchial and amphoric sound indicating air coming out from bronchus and consolidation process surrounding adequate abscess drainage. If lung abscess closely located to pleura, it could burst and manifest as pyothorax which appear as lagging chest wall movement during respiration; lost vocal fremitus; faint percussion; lost breath sound and if not drainage externally it could push the heart contralaterally[9].

Laboratory test in lung abscess appear as high range leucocyte count from 10.000-30.000/mm^3 predominated by polymorphonuclear cells (mostly immature ones indicating shifting to the left). If the abscess is prolonged, anemia and elevated ESR are often found. Sputum examination can be helpful in finding microorganism causing the abscess but preferably obtained from transtracheal aspiration, transthoracic, thoracocentesis (percutaneous aspiration: high specificity compared to transtracheal) or bronchial rinses (diagnostic accuracy >80%) as spontaneous expectorated sputum will be contaminated with normal anaerobic organisms of the oral cavity and upper airway. These invasive procedures are done if response to antibiotic is inadequate. Gram staining, culture of aerobic and anaerobic organisms, fungi, *M. tuberculosis* screening through Gene expert are expected to be done for specific definitive antibiotic treatment. Gram staining to diagnose pulmonary infection due to anaerobic germs is preferred, because anaerobic bacteria often cannot thrive through culture. Eradicating anaerobic bacteria despite only aerobic bacteria found through microbiological culture is still considered. Though rarely positive, blood cultures can help to find etiology of infection[16].

Posteroanterior and lateral chest x-ray could help to localize lesion of lung abscess. Radiolucent images will be found in the shadow of dense infiltrates. Ruptured abscess with incomplete drainage into the bronchus could appear as irregular cavity with thick walls surrounded by infiltrates/consolidation with air fluid level in it (only visible in standing or sitting position). Most common location is in the superior segment of the lower lobe or posterior segment of the upper lobe, while the basilar segment of the lower lobe is often found in patients who aspirate in the standing position. Typical anaerobic lung abscesses have a solitary cavity which is usually found in primary lung infections, whereas secondary lung abscesses (aerobic, nosocomial or haematogenic) may have multiple lesions. One third of lung abscess cases may be accompanied by empyema. Localized empyema with bronchopleural fistula may be difficult to distinguish from lung abscess. CT scan could examine the endobronchial obstruction and abscess formation appeared as central cavity. Through this examination, abscess location in lung parenchyma could be differentiated from empyema. Lesions causing bacterial abscess including bronchogenic carcinoma with cavity, bronchiectasis, secondary empyema with bronchopleural fistulation, lung tuberculosis, lung mycosis, bullae or infected cysts, or subphrenic hepatic abscess. Cavity as radiologic founding should be examined carefully for another etiology. Pure empyema in CT reveals thinner walls and smoother lumen separating the thickening visceral and parietal pleura making the hallmark split pleura sign[14].Click or tap here to enter text.

The patient had asymmetric chest wall movement: lagging of left hemithorax, decreased fremitus palpation of left hemithorax and faint percussion at the lower 1/3 to mid left hemithorax. On auscultatory examination, there was a decrease in vesicular sound in the lower 1/3 of the left hemithorax and rhonchi was positive. Laboratory resulted as anemia due to chronic disease (infection) (Hb 10,7; Hct 30.6%; MCV 84.8 fL; MCH 29.6 pg), leukocytosis (17.710/uL; Neutrophils 82.9%). There was no airway obstruction but suspected fistulation through bronchial *branch track in the anterobasal segment of the inferior lobe of the left lung and approaching the pleura. Adequate drainage was achieved by chest tube insertion and production of pus 1500 ml in 3 days.*

The main management of lung abscess is eradicating source of infection with adequate antibiotic and drainage of empyema and prevent further complication. A diameter >4 cm of abscesses may well be treated in inpatient setting. Good postural drainage by maintaining affected lung in higher position could improve abscess drainage: such as lateral decubitus in healthy lung and abscess lung hemithorax above it; or Trendelenburg position if superior segment of lower lobe affected.

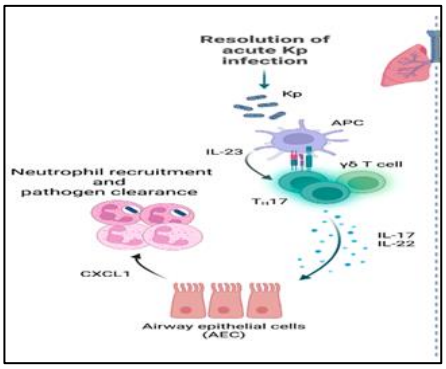

Figure 7. Immunological cascade involved in the resolution of acute Klebsiella pneumoniae infection.

The diagram illustrates antigen-presenting cell (APC) activation and IL-23-mediated stimulation of γδ T cells, leading to the release of IL-17 and IL-22. These cytokines trigger CXCL1 secretion from airway epithelial cells (AECs), promote neutrophil recruitment, and facilitate pathogen clearance in acute *K. pneumoniae* infection. Full resolution of abscess depends on effective antibiotic as early as possible aiming anaerobic bacteria or microorganism found in sensitivity culture test specimen. Small abscess and stabile general condition mostly improve by antibacterial treatment and effective postural drainage, other than that, around 10% needs operative approach. Empirically, clindamycin has a better coverage for anaerobe bacteria with initial dose 3x600 mg IV till clinical improvement then it can be switched to 4x300 mg orally/day or by giving 2x875 amoxicillin clavulanic acid. Other alternative regiment chosen is penicillin G 2-10 million unit/day, or combination with streptomycin and switch to penicillin oral antibiotic 4x500-750 mg/day. Combination of penicillin class antibiotic (amoxicillin 3x500 mg/day) with metronidazole 2 gram/day in divided dose: 500 mg orally or intravenously 2-3x/day (for anaerobe coverage) for 10 days can also be chosen. Some anaerobic bacteria (15-25%) such as *Prevotella*, *Bacteriodes spp*, *Fusobacterium* could produce penicillinase and beta-lactamase which make them resistant to penicillin. By combing β-lactamase inhibitor such as ticarcillin clavulanic, amoxicillin-clavulanic acid or piperacillin-tazobactam could also eradicate most anaerobic and negative gram bacilli bacteria. Other alternative antibiotic such as carbapenem or quinolone which could also be chosen, as they are actively suppressing anaerobe bacteria growth (moxifloxacin). Solely giving single dose metronidazole 15 mg/kg of weight IV followed by 7,5 mg/kg body weight divided 3-4x daily is not advised, cause most anaerobic cocci may have resistance and make the treatment to fail (50%). Definitively, antibiotic should be based on culture sensitivity result: *methicillin resistant Staphylococcus aureus* (may be caused by septic emboli complicated by nosocomial bacteria) should be treated with vancomycin; *nocardia* should be treated with sulfonamide 3x1 gram orally; amoebic lung abscess should be treated with metronidazole 3x750 mg, and if amoebic abscess is ruptured, emetine should also be added parenterally in the first 5 days. These antibiotics are given until the pneumonia has resolved and the cavities have disappeared (usually 5-21 days), leaving small residual lesions that stabilize over 2-3 weeks[8]. Complete resolution usually requires 6-10 weeks of oral antibiotics in outpatient setting, but reportedly studies showed 3 weeks of clindamycin were effective. Less duration of antibiotic treatment often leads to recurrence of infection complicated by resistant bacteremia appears as persisting fever >72 hours or no change in sputum production or no radiological feature improvement after 7-10 days (failure of treatment). Further examination should be done and may reveal bronchial obstruction by foreign body, neoplasm or infection with resistant bacteria, mycobacteria, parasites or fungi. Large cavities (>6 cm), poor general condition, ineffective antimicrobial selection, wrong diagnosis, empyema, abscess requiring drainage or even other organ complication such as brain abscesses were bad prognosis for lung abscess[13,17].

Surgical drainage is rarely necessary but it is a safe procedure in patients who fail short-term antibiotic treatment especially when the cavities are large, airway obstruction that prevents drainage is encountered when tumors or foreign bodies are present. It is a curative measure with minimal risk and complications and can prevent diminished lung parenchymal function and contamination of the pleural cavity. In the pre-antibiotic era 45% of patients required surgery, but in the current antibiotic era surgery is only required in less than 10-20% of cases. The indications for surgery are as follows: 1) Lung abscess that does not improve; 2) Complications: empyema, massive hemoptysis, bronchopleural fistula; 3) Treatment of underlying disease: carcinoma primary/metastatic obstruction, foreign body ejection, bronchiectasis, gastroesophageal motility disorders, congenital malformations or abnormalities; 4) Pulmonary infarction, massive necrosis (pulmonary gangrene) or rapidly progressive infection. Drainage of lung abscess needs cautious approach because it could result in multitude complications including development into empyema, pneumothorax, hemorrhage and the creation of bronchopleural fistula. The usage of intra-cavitary fibrinolytic agents (streptokinase, urokinase) is not recommended, previously thought to ease purulent necrotic with fibrin tissue to drain throughout the tube[8,3,17,16].

Rapidly progressing lung abscesses including those occurring in immunocompromised patients with a mucoraceae etiology require immediate lung resection in addition to antibiotics. Lung resection is also indicated in lung abscesses with minimal response to antibiotics, lung abscesses with large size (cavities >8 cm), lung infarction, obstructing neoplasms and massive bleeding. Lobectomy is the most common procedure, while segmental resection is usually sufficient for small lesions. Pneumonectomy is required for multiple abscesses or pulmonary gangrene refractory to drug treatment. The mortality rate after pneumonectomy is 5-10%. Patients with a high risk of surgery can be temporarily percutaneously drained via a catheter carefully to prevent leakage of abscess contents into the pleural cavity. Poorly drained lung abscesses can rupture into other segments with a tendency to spread *Staphylococcus* infection, while those that rupture into the pleural cavity become pyothorax (empyema) and pleural fibrosis. Other frequent complications include brain abscess, massive hemoptysis, visceral pleural rupture resulting in pyopneumothorax, bronchoplegeal fistula and pleurocutaneous fistula. Resistant (chronic)

lung abscess, which is resistant to treatment for 6 weeks, will cause irreversible lung damage and may result in bronchiectasis, corpulmonary and amyloidosis. Chronic lung abscess can cause anemia, malnutrition, cachexia, fluid and electrolyte disturbances and heart failure especially in the elderly[15,9].

In this case, with CXR examination and first encountered of pus formation should prompt chest tube insertion. But later CT scan revealed a suspected bronchopleural fistulation in anterobasal segment of inferior lobe in hemithorax. Unimproved clinical shortness of breath and sputum production for 3 days after empirical antibiotic levofloxacin 750 mg/day IV and metronidazole 3x500 mg IV accompanied by no resolution of chest x-ray showed no adequate postural drainage which could be improved by chest tube insertion to release lung restriction by fluid-pneumothorax buildup. After chest tube insertion for 3 days, 1500 ml of pus was collected and dyspnea was resolved after antibiotic switching to moxifloxacin 1x400 mg and metronidazole 3x500 mg based on microbiological sensitivity result. The poor prognostic factors were found in this case such as large cavity (7,2 cm diameter), compromised immunity (HIV and DM type 2), infection with aerobic pathogen (Klebsiella pneumonia).

The prognosis of simple lung abscess mainly depends on the inflammatory response and general condition of the patient, the location of the abscess and the extent of lung damage, and the treatment response. The mortality rate of lung abscess patients in the pre-antibiotic era was 33%-40% but half of the patients who survived would develop pulmonary complications including recurrent infections and abscesses, empyema and pleural adhesions, chronic bronchitis and bronchiectasis. In the antibiotic era the mortality rate of anaerobic lung abscesses was less than 10%, and approximately 10-15% needed surgery. When treatment is given over a long period of time the recurrence rate is low. Factors that make the prognosis poor are large cavities (more than 5-6 cm), severe underlying disease or comorbidities, immunocompromised status, very old age, empyema, progressive lung necrosis, obstructive lesions in the airway e.g. secondary to carcinoma, abscesses caused by aerobic bacteria (including Staphylococcus aureus and Gram-negative bacilli), and lung abscesses that have not received treatment for a long period of time. The mortality rate in these patients can reach 65%-75% and if cured, the recurrence rate is high[13]. Click or tap here to enter text.

Here we reported a case with improve clinical response after definitive antibiotic treatment and effective pus drainage after chest tube insertion. However, favoring factors for poor prognosis such as big cavity formation complicated with empyema and pneumothorax as lung abscess progression, compromised immune system due to previous episode of uncontrolled glucose level (DM type 2), multi-drug resistance gram negative involvement: ESBL K. pneumonia, adding to risks of recurrence abscess and infection. Controlling such risks, especially through adequate oral antibiotic completion for minimum of 6-10 weeks and close glucose monitoring could lower permanent lung damage and recurrence of infection. Resolution of infection through anamnesis by evaluating clinical symptoms (fever, productive/purulent cough, etc) and repetition of CXR (most X-ray resolution could take as long as 2 months) should be done, though in this case such follow up was lacking.

We reported a case of 58-years-old male living with HIV treated with ARV (FDC: Tenofovir-Lamivudine-Efavirenz), whom admitted to hospital due to complicated *Klebsiella pneumonia* infection (pneumonia) resulted in lung abscess and pyopneumothorax which previously not well responded to empiric antibiotic treatment and no adequate drainage of fluid-pneumothorax build up. He had multiple risk factors such as large cavities, empyema (fluid buildup accumulation), compromised immune condition which then managed by chest tube insertion and drainage resulted in improved clinical condition. Antibiotic switching based on microbiological sensitivity result also improved treatment respond. Treating underlying comorbidities such as diabetes and adequate antibiotic completion by monitoring clinical and chest x-ray resolution should be done to prevent further recurrent infection and abscess in already poor prognosis complicated lung abscess.

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Competing Interests

The authors have no competing interests to declare.

Ethical Approval

The study was approved by the appropriate ethics committee and conducted according to relevant guidelines and regulations.

Informed Consent

Not applicable.

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