

CHAPTER II

THE DIAGNOSIS AND TREATMENT OF LUNG ABSCESS IN VIEW OF CURRENT RESEARCH

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1.Introduction

Lung abscess is a cavitory lesion characterized by necrosis of the lung tissue caused by pyogenic microorganisms and is defined as a local suppurative process within the lung (1). This disease can be classified by its duration, etiology and passage of transmission. Lung abscess can be divided into two groups as acute (less than 6 weeks) and chronic (more than 6 weeks) (1). Etiologically, lung abscesses can be labelled as primary and secondary (1, 2). Primary lung abscess occurs in patients prone to aspiration or otherwise healthy individuals, with no underlying lung lesions. Secondary lung abscess develops in the presence of underlying lung lesions or on the basis of a systemic disease such as extrapulmonary infection, sepsis, immunosuppression or malignancy (1, 2). Considering the route of spread, the disease is grouped as bronchogenic (aspiration of oropharyngeal secretions, bronchial obstruction by the tumor, foreign body, enlarged lymph nodes, congenital malformation) and hematogenic (abdominal sepsis, infective endocarditis, septic thromboembolism) lung abscesses (1).

2.History

The clinical symptoms and treatment of lung abscess were first described by Hippocrates (1). In the pre-antibiotic period, one third of patients with lung abscess died, one third recovered completely, and the rest survived with sequelae such as chronic lung abscess, pleural empyema, or bronchiectasis (1, 3). While the mortality rate of lung abscess was quite high in the past centuries, this rate decreased significantly with open drainage and antibiotic treatment (4,5). Similarly, improvements in oral and dental hygiene have reduced the incidence of lung abscess (1).

3.Pathogenesis

Although abscess may occur in the lung due to reasons such as direct inoculation due to inhalation and trauma, contamination from the

diaphragm or mediastinum, and hematogenous transmission, the most important risk factor is the aspiration of the material in the oropharynx. In cases that cause confusion, sedative drug and alcohol use, epilepsy, head trauma, cerebrovascular diseases, diabetic coma and other diseases that disrupt the general condition, suppression of the gag reflex also facilitates the aspiration of the oropharyngeal flora and constitutes an important risk factor group. Aspiration during dental and periodontal sepsis also facilitates abscess formation. Adhesions in the esophagus that cause swallowing disorders and gastroesophageal reflux can facilitate aspiration. On the other hand, bronchial obstructions caused by malignancy, inflammation and foreign bodies in the lung can contribute to abscess formation by preventing the clearance of the aspirated oropharynx material (6). Conditions that suppress the functions of the respiratory system defense mechanics, smoking and pre-existing diseases that impair the natural immunity of the organism; factors such as viral infections, chronic liver and kidney disease, diabetes, corticosteroid, intravenous drug use and sepsis are other factors that facilitate abscess formation in the lung (7). Pulmonary abscess may occur with the initiation of suppurative infection in the lung parenchyma distal to the narrowing or obstruction of the bronchi for any reason. This situation is more common in tumors with bronchial obstruction and foreign body inhalation (8). Pneumonia foci may also become abscessed. Especially, necrotizing pneumonia caused by *Staphylococcus aureus*, *Klebsiella pneumoniae*, *Streptococcus* spp and *Pseudomonas aeruginosa* abscesses more frequently than other pneumonias (6-8). In the focus of consolidation, first small multiple abscesses and then large abscesses are formed by their combination. Staphylococcal abscesses are often complicated by empyema and pyopneumothorax (9). Septic embolism caused by an infection in the extra pulmonary focus can form lung abscesses by occluding the pulmonary artery branches. Such abscesses can be seen in staphylococcal infections such as osteomyelitis, furuncle, abdominal and pelvic sepsis, infective endocarditis, use of infected intravenous cannula, and septic thrombophlebitis. Hematogenous lung abscesses are usually multiple and localized in the peripheral parts of the lung (9, 10). Since the drainage bronchi of these foci are small, cellular infiltration and edema caused by inflammation cause blood pressure cysts, rupture into the pleura and empyema by obstructing the bronchi (10). Also, bronchogenic cysts can become infected, leading to lung abscess. Cystic lesions such as bulbs and blebs are rarely infected since they do not contain secretions (10). Infection of pulmonary hydatid cysts after perforation gives typical clinical findings of lung abscess. After the perforated hydatid cyst is completely excreted, the remaining cavity may become infected secondarily and shows a course like an infected lung abscess. If the cyst cannot be removed completely, it becomes infected easily and frequently as a result of the foreign body

reaction caused by the particles remaining inside, and gives symptoms and signs such as chronic lung abscess (6, 8). Abscesses in the upper part of the liver can penetrate the diaphragm and open into the lungs. As a result of perifocal reactions occurring in the diaphragmatic pleura, adhesion occurs between the upper face of the diaphragm and the lower face of the lung in the lesion area, thus lung abscess develops directly without empyema in the pleura. Sweep appendicitis and sweep cholecystitis cause lung abscess in this way. Rarely, perinephritic abscesses and sometimes together with nephrobronchial fistulas can lead to lung abscess. In addition, infections in the esophagus, mediastinum and vertebrae can spread to the lungs and cause abscesses, even if they are rare. Although tracheoesophageal fistula is a rare congenital formation, it can cause bronchiectasis and abscess formation in children. Amoebas should be considered in the etiology of abscesses in the lower right lung of patients with chocolate-colored sputum (11). The aspiration of mouth and throat flora to the lungs during general anesthesia and the difficulty in removing the aspirated septic material and bronchial secretion due to the ineffectiveness of cough in the post-operative period may cause abscess formation. In septic surgeries, bacteria-laden emboli from the operation site spread to the lung by hematogenous route and cause suppuration (9). Pulmonary abscess may occur as a result of the implantation of the infectious material into the lung, in injuries caused by firearms and piercing weapons, in traumatic events that cause the broken rib ends to stick into the lung. Infection of a traumatic hematoma may also cause abscess (9).

4.Pathology

Lung abscesses usually begin as small necrotic foci or microabscesses in a consolidated lung area. It can occur in any part of the lung, single or multiple. Abscess formation is mentioned when these suppurative microabscess foci combine to grow and reach 1-2 cm in diameter. If this pathological process is prevented by early antimicrobial treatment, recovery is achieved without any damage. However, if the treatment cannot be done or is not sufficient, the inflammation becomes chronic and continues. If the abscess is fistulized by eroding the neighboring bronchus, the purulent content of the abscess spreads to the bronchus and malodorous sputum expectoration begins (12, 13). Aspiration-induced pulmonary abscesses are more common in the right lung and are usually single, as the right main bronchus is more vertical (14). Abscesses that develop during the course of pneumonia or bronchiectasis are usually multiple, basal and diffuse distributed. Septic embolus and pyemic abscesses are numerous due to their irregular and random development and may involve any part of the lung (13). If a connection occurs between the abscess cavity and an airway, the exudate in the abscess is partially drained and a cavity

containing air forms. Added saprophytic infections tend to proliferate among the necrotic debris accumulated in the abscess cavity. In this way, continuous infection creates large multilocular cavities with bad odor, green-black color, with poorly defined edges, called lung gangrene. Cardinal histological change in all abscesses is suppurative destruction of the lung parenchyma within the central cavitation area. In chronic cases, abundant fibroblastic proliferation creates a fibrous wall (12-14).

5.Diagnosis

Patients typically show signs of upper respiratory tract infection. Early signs and symptoms of lung abscess are indistinguishable from pneumonia and include chills, cough, night sweats, dyspnea, weight loss and fatigue, chest pain, and sometimes fever with anemia (15). Hemoptysis is an indication of the evacuation of the necrotic contents of the abscess cavity. This first symptom is usually followed by the production of purulent, sometimes foul-smelling sputum in abscesses due to anaerobes. Abundant sputum production in the form of vomiting can be observed with the opening of the abscess to the bronchus. Staphylococcus aureus, gram-negative bacilli, and amoeba abscesses can show a rapid and severe course (16). In the late stage, amphoric or cavernous breathing, signs of pleurisy, and large abscesses are dull. Breathing sounds are attenuated. Clubbing may develop in chronic abscesses (1, 6). In the early stages of the disease there is leukocytosis and the erythrocyte sedimentation rate is increased. Hypochromic anemia can be seen with the toxic effect of the infection (17). Bacteriological examinations in sputum, examinations and cytological investigations in terms of mycobacteria, pathogenic fungi, parasites, routine aerobic and anaerobic cultures can be performed. Blood culture is useful in Staphylococcus aureus, gram negative bacilli and anaerobe infections. Aerobic and anaerobic culture in pleural fluid is especially useful before initiating treatment in empyema (16). Bronchoscopy is routinely performed in patients who do not respond to antibiotic treatment, have atypical clinical findings, and have abscess with malignant cavity, obstructive tumor and foreign body aspiration (1, 6, 18). In this way, if an endobronchial tumor is seen, a biopsy is taken, the foreign body is removed, drainage is provided with aspiration, an idea is obtained about the localization of the abscess according to the bronchus, and material is taken for bacteriological and cytological examination (6).

Chest x-rays and computed tomography (CT) are used in the diagnosis of lung abscess (1). Lung abscess is recognized by the cavity appearance showing air-fluid level on the chest x-ray (19). Radiography can also be taken in the lateral-decubitus position to better visualize the air-fluid level within the cavitory lesion. Sometimes accompanying conditions such as atelectasis, pneumothorax, pleural thickening may shadow the typical cavity appearance. In addition, if the abscess cavity has drained into

the pleural space, it is possible to see pneumothorax and pyopneumothorax on direct radiography. Computed tomography (CT) of the thorax, which enables the cavitory image to be detected more easily, has become more widely used today (1).

6.Treatment

If a lung abscess is suspected, empirical antibiotic therapy should be initiated. Empirical coverage should target organisms colonized in the upper airway and oropharynx, such as gram-positive cocci, respiratory gram-negative cocci, aerobic and anaerobic gram-negative bacilli (2, 20). Specific treatment is arranged considering the pathogenic microorganism obtained in microbiological research and other accompanying diseases of the patient (21). Catheter drainage of fluid in the abscess cavity and necrotic debris accompanied by bronchoscopy or radiology is another treatment method (22). The suggested combinations of antibiotics for lung abscess are a combination of β -lactam with inhibitors of β -lactamase (ampicillin-sulbactam, amoxicillin-clavulanate, piperacilin-tazobactam), chloramphenicol, second generation of cephalosporins (cefoxitin, cefotetan), imipenem or meropenem, fluoroquinolones-moxifloxacin that is proven to be as effective as ampicillin-sulbactam (1, 23). Empirical treatment is recommended with amoxicillin clavulanate, chloromphenicol or penicillin / metronidazole combination in community acquired acute lung abscesses, since the factors in the etiology are multiple anaerobes and rarely aerobic gram-positive microorganisms (21). Vancomycin or linezolid is preferred for methicillin-resistant *Staphylococcus aureus* (MRSA). Daptomycin has no efficacy against pulmonary infections (24). For methicillin-sensitive *Staphylococcus aureus* (MSSA), the choice is cefazolin 2 g IV every 8 hours or nafcillin 2 g IV every four hours or oxacillin 2 g every 4 hours. For methicillin-sensitive *Staphylococcus aureus* (MSSA) choice is cefazolin 2 g IV every 8 hours or nafcillin 2 g IV every four hours or oxacillin 2 g every 4 hours. Aminoglycosides are not recommended in the treatment of lung abscesses because they pass poorly through the fibrous pyogenic membrane of a chronic abscess. The duration of treatment depends on the clinical and radiological response of the patient (1, 2, 21, 22). Patients should be treated until the fever drops, the Palatinat odor in the sputum and the fluid level in the abscess disappears. The duration of treatment is at least two to three weeks, but usually patients require longer treatment (1, 22, 25). An effective response to antibiotic treatment can be seen after 3-4 days, the general condition improves after 4-7 days, but complete recovery can be seen with radiographic normalization after two months (1). The average closing time of the cavity is 3-4 weeks and can last up to 14 weeks. The average time it takes for the resolution of the infiltration is 8-10 weeks, but it can be up to 24 weeks. Small cavities (smaller than 3 cm) are resolved faster radiologically. The

change in cavity size slows down within 6 weeks after treatment. Cavity size may also indicate ongoing cystic (pneumotoceles) or bronchiectasis changes after treatment. These changes can be detected by bronchography and CT scans. These findings are not an indication for continued treatment or resection (21). Relapse of the abscess is common, especially in cases resistant to drug combinations used initially (22).

An important step in abscess treatment is drainage (1, 26). The presence of air in the cavity of the lung abscess usually indicates that it can be drained without mechanical intervention due to contact with a bronchus. Lung abscess often drains spontaneously into the airways, causing the infection to regress, but can also cause the infection to spread to other parts of the lung. Therefore, chest physiotherapy and postural drainage should be applied to the cases. If the abscess greater than 6 cm in diameter or the symptoms persist for more than 12 weeks with appropriate treatment, the chance of recovery with only medical treatment is very low, and surgical treatment (chest tube drainage or surgical resection of lung abscess with surrounding tissue) should be considered if the general condition allows (26). Endoscopic drainage of lung abscesses is performed during bronchoscopy as an alternative to chest tube drainage (1). Surgical resection of lung abscess is the treatment of choice in approximately 10% of patients. Indications for surgical resection of lung abscess can be divided into acute and chronic. Hemoptysis, prolonged sepsis and febricity, bronchopleural fistula, pyopneumothorax, empyema, abscess rupture are acute indications. Chronic indications are; lung abscess, which is treated unsuccessfully for more than 6 weeks, is defined as cancer suspicion, cavitory lesion larger than 6 cm, and persistent leukocytosis despite antibiotics (1).

7.Complications

Complications are secondary to delay in diagnosis, inadequate treatment, or untreated underlying cause of lung abscess. These include pleural space rupture, pleural fibrosis, restrictive lung disease, respiratory failure, bronchopleural fistula, and pleurocutaneous fistula (8, 12).

8.Prognosis

The mortality of lung abscess has decreased compared to the pre-antibiotic period. The size of the abscess (over 5-6 cm), progressive pulmonary necrosis, obstructive lesions, aerobic bacteria, immune system suppression, old age, systemic debility, delay in medical treatment are factors that increase abscess mortality. Other factors affecting mortality in the patient are anemia, low serum albumin, other diseases, abscess diameter, its location and placement in the right lower lobe (27). When the mortality was evaluated in terms of the agent, it was found that the prognosis was worse in abscesses caused by *P.aeruginosa*, *S.aureus*,

K.pneumoniae. Therefore, in abscess cases with such poor prognostic factors, treatment with a rapid aggressive approach is recommended in addition to antibiotherapy (1, 6). Overall mortality in the treatment of lung abscess is approximately 2.0-38.2% with the important role of patient age, malnutrition, comorbidity, immunity, appropriate and timely antibiotics, and supportive treatment (27, 28).

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