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Lung Abscess: Diagnosis, Treatment and Mortality

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ABSTRACT: *Lung abscess is the necrosis of pulmonary tissue with formation of cavities (more than 2cm)..Predisposing factors include bronchogenic carcinoma or other bronchial obstructions,bronchiectasis and pulmonary infarction. Diagnosis is by chest radiography and computed tomography(CT),Frequently isolated pathogens include anaerobes and nosocomial microorganisms,Staphylococcus aureus, Pseudomonas aeruginosa, mycobacteria,parasites and fungi.Antibiotics of choice include penicillin with β lactamase inhibitors., carbapenem, quinolones, amoxicillin-clavulanate and amoxicillin sulbactam. Metronidazole not so effective because of microaerophilic streptococci. Medical management failure often is secondary to undrained pleural collections, endobronchial obstruction caused by a neoplasm or foreign body. High rates of morbidity and mortality associated with lung abscess despite antibiotic therapy and supported care. Patients with predisposing conditions like a large sized abscess and right-lower lobe location, have the worst prognosis. The prognosis of lung abscess has not improved sufficiently since the introduction of antibiotics, other modalities should be considered for patients with prognostics signs.*

KEY WORDS: *Lung abscess, Etiologic agent, Radiography, Computedt tomography, and Treatment.*

I. INTRODUCTION:

Lung abscess is a type of liquefactive necrosis of pulmonary tissue and formation of cavities(more than 2cm) containing necrotic debris or fluid caused by bacterial infection[1].The term *necrotizing pneumonia* often is used to describe a similar pathologic process with multiple small (<2cm in diameter) cavities in contiguous areas of the lung. Lung abscess was much more common in the preantibiotic era, because of lack of treatment, bacterial pneumonia sometimes progressed to abscess formation, with or without empyema. Reduction in incidence also occurred in the late 1940s and 1950s,when it became clear that performing oral surgery and tonsillectomy in the sitting position was a risk factor for lung abscess,and this practice was discontinued[2].Classification of lung abscesses include(1)the causative organism(e.g.,anaerobic lung abscess or staphylococcal lung abscess;(2)the presence of a foul odor to exported sputum(putrid lung abscess);(3)the duration of symptoms before diagnosis(acute, symptoms present less than a 1 month chronic, symptoms present longer than 1 month);or(4);or the presence or absence of absence of associated conditions(e.g.,lung cancer, acquired immunodeficiency syndrome (AIDS),immunosuppression).The term *primary lung abscess* generally used when an abscess develops in individuals prone to aspiration or individuals in relatively good health. Secondary lung abscess indicates an obstruction in the airways neoplasma,a complication of intrathoracic surgery, or a systemic condition or treatment that compromises host defense mechanisms, such as human immunodeficiency virus(HIV) infection or transplantation immunosuppressive therapy. Approximately 80% of lung abscesses are primary, and roughly half of these are associated with putrid sputum [3]. In the preantibiotic era mortality rate of lung abscess was reported to have been 32% to 34% of all three therapies (e.g. conservative management, drainage by posturing or bronchoscopy) [4].Hirshberg and associates reported high rates of mortality associated with lung abscess despite appropriate antibiotic therapy and supported care[5].In a series of 62 patients with lung abscess, Nuri Tutar and associates reported a mortality rate of 12.9%[6].Common pathogens include *Staphylococcus aureus,Klebsiella* spp, *Pseudomonas aeruginosa ,Burkholderia pseudomallei*, group A streptococcus, *Streptococcus pneumonia, Nocardia, mycobacteria,* parasites and fungi [7]. Diagnosis is by chest radiography showing a lung cavity with an air-fluid level, computed tomography(CT) is more sensitive than radiography [8,9].Antimicrobials of choice include penicillin with a β lactamase inhibitors, carbapenem quinolones,amoxicillin-clavulanate and amoxicillin –sulbactam[10,11].Metronidazole does not appear to be particularly effective because of microaerophilic streptococci, [12].The paper reviews the current literature, diagnosis, treatment and association of lung abscess with mortality.

II. ETIOLOGIC AGENT

The predominant organisms responsible for lung abscess are bacteria, specifically mouth anaerobes that are normal flora in gingival cervices.[13].David Smith in his classic studies of lung abscess was able to produce typical lung abscesses with an inoculum containing 4 microbes that are thought to have been an anaerobic spirochete, *Fusobacterium nucleatum, Paptostreptococcus* species, and a fastidious gram-negative anaerobe(possibly *prevotella melaninogenicus*)[14].In the presence of periodontal disease the gingival crevice deepens and fills with anaerobic gram-negative organisms that can reach truly astronomical numbers(10^{12} colony forming units/g per gram of scraped gingival contents)[15].Studies using sample collection techniques that avoid contamination with oral flora combined with good anaerobic culture methods have shown that anaerobes are found in about 90% of lung abscesses and are the only organisms present in about half of cases[16].The most frequently isolated anaerobes are *Peptostreptococcus* spp, (now probably identified as *Finegoldia magna*), *Fusobacterium nucleatum*, and *Prevotella melaningenica*.The abscesses usually contain multiple anaerobe species, usually three to four per culture specimen; microaerophilic streptococci and viridian streptococci[17].

Monomicrobial lung abscess occasionally may be caused by bacteria, including *Staphylococcus aureus*, gram-negative rods such as *Klebsiella* spp., *Pseudomonas aeruginosa, Burkholderia pseudomallei*(melioidosis), *Pasteurella multocida*,group A *streptococc*i, *Haemophilus influenza* type b and c,Legoinella spp.,*Rhodococcus equi, Actinomyces* spp., and *Nocardia* spp., *Streptococcus pneumoniae*, particularly type 3,has been repor6ed to cause lung abscess, but cavitation in the setting of pneumococcal pneumonia may be caused by concomitant infection with anaerobes.Other organisms that can cause lung abscess include many fungi,mycobacterial spp, and parasites (e.g., *Paragominus westermani, Entamoeba histolytica*)[7].

A 2005 study from Taiwan [18] used transthoracic aspiration to obtain microbiology specimens from 90 consecutive adults with community acquired lung abscess. The authors suggested that the bacteriology of lung abscess had changed because the recovered anaerobes from only 31% of patients, but isolated *Klebsiella pneumonia* as the predominant bacterium from 33%.They further suggested that antibiotic selection for treatment of lung abscess should include coverage for *K.pneumoniae*.Other have argued caution in generalizing these results and suggested that surprising bacteriology may have been caused by (1) selection bias[19](2)antibiotic administration to 25% of patients before specimens were obtained[19] or(3)a geographic phenomenon, because *K.pneumoniae* in Taiwan is notably more virulent than strains from most other parts of the world[20].

Oropharyngeal colonization with *P.aeruginosa*,otheraerobic gram-negative rods and less often,*S.aureus* is common even in hospitalized patients, particularly patients who receive ventilator support.These bacteria are important pathogens when lung abscess or nectrotizing pneumonia develops during hospitalization and may produce infection as the sole pathogen or as component of mixed flora infection involving oropharyngeal organisms[3].In patients with impaired cell-mediated immunity (AIDS,transplantation immunosuppression), opportunistic parthogens, such as mycobacteria,*Nocardia*,*Aspergillus*,and *Rhodococcus*, are important cases of cavitary lung lesions.In patients with impaired host defenses caused by granulocytopenia(leukemia, chemotherapy),aerobic bacteria(*P.pseudomonas, S.aureus* and fungi including, *Aspergillus Zygomycetes*, are important pathogens[3].Judith and associates reported in a study of 42 patients with pulmonary Cryptococcosis without HIV infection, to elucidate the diagnostic and therapeutic approaches to patients with pulmonary cryptococcosis who are not HIV-infected[21].

III. CONTRIBUTORY FACTORS

Frequent contributory factors include:
(a) aspiration of oropharyngeal or gastric secretion

(b) septic emboli (c) septic pneumonia

(d) vasculitis:Wegener's granulomatosis or

(e) necrotizing tumors: 8 % to 18% are due to neoplasms across all age groups, higher in older people; primary squamous carcinoma of lung is the most common.

In the post-antibiotic era pattern of frequency is changing. In older studies anaerobes were found in up to 90% cases but they are much less frequent now [19].Onset of symptoms is often gradual, but in necrotizing staphylococcal or gram-negative bacillary pneumonias patient can be acutely ill. Cough fever with shivering and night sweats are often present. Cough can be productive with foul smelling purulent sputum(≈70%) or less frequent with blood(i.e. hemoptysis in one third cases).Affected individuals may also complain of chest pain, shortness of breath, lethargy and other features of chronic illness[22].Patients are generally cachectic at presentation.Fingger clubbing is present in one third of patients. Dental decay is common especially in alcoholics and children.On examination of chest there will be features of consolidation such as localized dullness on percurssion,bronchial breath sound etc.[22].

IV. CLINICAL PRESENTATION

Lung abscesses are divided according to their duration into **acute** (<6weeks) and **chronic** (>6weeks) [23].Presentation is usually non-specific and generally to a non-cavitation chest infection. Symptoms include fever, cough and shortness of breath. Peripheral abscess may also cause pleuritic chest pain. If chronic, symptoms are more indolent and include weight loss and constitutional symptoms. In some cases erosion into bronchial vessels may result in sudden and potentially life threatening massive hemoptysis [23].Primary lung abscess caused by mouth flora anaerobic bacteria usually present in a sub-acute or indolent fashion with symptoms present for several weeks or longer [13].Poor dental hygiene allows for a high-tittered inoculum with aspiration.Anaerobic abscess is uncommon in the **edentulous patient** unless there is a predisposing pulmonary disorder [24].Predisposing pulmonary disorders include bronchogenic carcinoma or other bronchial obstructions, bronchiectasis,and pulmonary infarction. COPD does not predispose to anaerobic lung infections[254.Fever,malaise,night sweats and cough with purulent sputum usually are present,pleuritic pain is common. Weight loss may be profound even in the absence of underlying malignancy.Shaking chills almost never are reported. Patients often seek medical attention when sputum production copious or develop pleuritic pain. The sputum has putrid smell in about 50% of cases and patients, or their close contact may complain of the foul sputum smell or of the patient's bad breath [25].Hemoptysis in some instances. There is often a history of antecedent loss of consciousness caused by seizure or intoxication. Physical examination include fever, poor dentition and gingival disease and abnormal lung findings consistent with parenchymal infection, plerural fluid, or both. Amphoric or cavernous breath sounds may be heard. Clubbing of digits may be seen. The gag reflex, a physiologic defense against a large-volume aspiration may be absent. Anemia of chronic disease and leukocytosis with approximately 15000 white blood cells/mm^3usually are present. Associated empyema is present in about one third of cases and may be seen with or without bronchoplerural fistula [26].Necrotizing pneumonia occasionally presents with a more rapid course, often presenting within 1 week of symptom onset. Early extension to other lobes and the pleural space, high fever, and pronounced leukocytosis)>20,000/mm^3) are common. Rapidly progressive pneumonia has been termed *pulmonary gangrene*[27].

V. DIAGNOSIS

The diagnosis is usually is made by **chest radiography** showing a lung cavity with an air-fluid level.Typically,the cavity wall is thick and irregular, and a surrounding pulmonary-infiltrate is often present. The infiltrate generally is localized to one pulmonary segment or lobe, and hilar adenopathy is not prominent.Multiloblar involvement suggests and underlying impairment in host defense mechanisms[8].**Computed tomography**(CT) is more sensitive than chest radiography and is useful to detect small cavities, provide evidence for obstructing endobronchial lesions, and distinguish lung abscesses from air-fluid levels in the pleural space[9].In the typical aspiration-prone patient with gingival disease, a subacute illness,and foul-smelling sputum, a putative diagnosis can be made,polymicrobial anaerobic infection can be assumed, and therapy can be instituted without microbiologic studies. Sputum gram stains in these patients show many neutrophils and mixed flora, with many morphologically different bacteria, routine cultures, usually grow normal respiratory flora. Because of expectorated sputum is contaminated by oral flora containing large numbers of anaerobes, special techniques for obtaining lower tract specimens are necessary to confirm the role of anaerobes [28].Patients without the classic presentation and patients with secondary lung abscess should have stains and cultures of expectorated sputum for aerobic bacteria, mycobacteria, fungi, in some instances parasites[3].

Differential diagnosis. Hospitalized patients routinely become colonized in the oropharynx with gram-negative rods[26];nosocomial lung infections, whether after aspiration or intrathoracic surgery,commonly involve virulent aerobic pathogens, such as *Klebsiella* spp, *Pseudoomona*s spp, and *S.aureus*.If a specific diagnosis is not clear(e.g., putrid sputum indicating anaerobic infection),patients may need to be placed in appropriate isolation until the possibility of tuberculosis is eliminated. Lung cancer masses may develop cavitation, even without secondary infection; more commonly, infections occurs behind a tumor-obstructed airway or within a large necrotic tumor mass.Fewer than 5% of pulmonary infarcts caused by bland embolism become secondarily infected; infection should be suspected when fever persists for more than few days, temperature is elevated(>103F[39.4]),or the leukocyte count is higher than 20,000/mm^3.Megastatic lung abscesses that occur by hematogenous dissemination from septic phlebitis or tricuspid valve endocarditis are distinguished by being multiple, bilateral, peripheral, and found in multiple lung fields; blood cultures are characteristically positive a rare finding in abscesses secondary to aspiration. Lung nodules in patients with Wegner's granulomatosis and rheumatoid arthritis may cavitate and are mistaken for abscesses, but the systemic features of these illnesses usually are apparent. Bullae and blebs are characteristically thin-walled and without surrounding infiltrate [26].

VI. TREATMENT

Antibiotic therapy. In a report by Smith of 1650 cases from period 1935 to 1945, when sulfonamides were available the report showed that the use of these agents had essentially no impact on the outcome [29]..Penicillin eventually won the favor, and William Weiss became the leading authority on medical management [5].For many years, penicillin was considered the drug of choice for anaerobic infections "above the diaphragm". In recent decades, however many mouth flora anaerobes, including *fusobacteria*, Prevotella, spp.,and non *fraglis Bacteroides* spp.,have been shown to produce penicillinase[30].Prospective studies have shown superiority of clindamycin over penicillin in the treatment of lung abscess as judged by time to defervescence time to resolution of putrid sputum, and relapse rates[31].Metronidazole use as monotherapy has been disappointing and is inferior to clindamycin.Metronidazole is not active against microaerophilic streptococci and some anaerobic cocci that are typical constituents of anaerobic lung infections. Metronidazole to be used with caution in alcoholics because of the potential for a disulfiram –like reaction [32.].Other agents that could be predicted to be useful for the treatment of lung abscess include combinations of a penicillin with a beta-lacatamase inhibitor, carbapenems, and quinolones with good anaerobic activity(moxifloxacin and gatifloxacin)[33].One study have shown excellent results using IV followed by oral amoxicillin-clavulanate[11].Another study has demonstrated that ampicillin-sulbactam is comparable to clindamycin plus a cephalosporin[34].Two recent studies have documented efficacy for moxifloxacin. a quinolone with good activity against the anaerobes and streptococci species commonly involved in lung abscess. Tetracycline should not be used because of widespread resistance across many anaerobes species[35].Oral therapy for lung abscess was shown to be equivalent to parenteral therapy many years ago, although few would choose that option for initial therapy now and no oral agent is approved for that use. If used, oral dosages for adults are (per 625 mg tab. Clavulanate K 125 mg, amoxicillin trihydrate 500 mg,per 1 g tab Calvulanate K 125 mg,amoxicillin trihydrate 875 mg) 8 hours ,300 to 600 mg every 8 hours for clindamycin, and 400 mg/day for moxifloxacin[5,35].

Treatment duration. There is no generally agreed-on duration for the treatment of lung abscess. Patients often are treated for 6 to 8 weeks or longer. One study using clindamycin to treat anaerobic abscess showed excellent efficacy, with no advantage of 6 weeks over 3 weeks of therapy, Many authorities recommend weekly or biweekly chest radiographs in patients showing clinical improvement, with discontinuation of therapy when chest radiograph is clear or there is a small stable residual lesion[36].

Bronchoscopy to be reserved for patients who do not respond to medical management and patients in whom an endobrochial tumor is suspected. Clinical features associated with an underlying malignancy and indications for early bronchoscopy to help diagnose a tumor include location of the abscess in and anterior lobar segment, lack of aspiration risk edentulous patient, age older than 50 years combined with strong smoking history, and lack of systematic symptoms [3].

Indication for surgery. Previously resectional surgery was the treatment for lung abscess. Today, almost all patients respond to appropriate antimicrobial therapy, and surgery is reserved for the 10% to 15% of patients who do not improve with appropriate medical management. Drainage is the most important step in the management of abscesses regardless of location. Lung abscesses, in contrast to other visceral abscesses, usually drain themselves through communication with large airways. This drainage is indicated by the presence of air-fluid levels. Causes of medical treatment failure and indication for consideration of lobectomy or pneumonectomy include large cavities(>8cm) abscesses caused by resistant organisms such as *P.aeruginosa*, obstructing neoplasm, and massive hemorrhage(rare).More recently, CT guided percutaneous drainage has been used safely and successfully in patients who were not responding to antimicrobial treatment [37].Another drainage option that has not been fully evaluated is endoscopic placement of a pigtail catheter through the airway into the cavity, with antibiotic irrigation[38].

Treatment failure and response to therapy. Patients have diminished fever and a subjective sense of change for better within few days of beginning of antimicrobial therapy Defervescence can be expected in 7 to 10 days. Persistence of fever beyond 2 weeks should lead to diagnostic tests to rule out complications, obstructions or both (CT scan, bronchoscopy), along with cultures for unusual pathogens, such as fungi and mycobacteria. When **medical management fails**, it most often is secondary to undrained pleural collections, endobronchial obstruction caused by a neoplasm or foreign body, resistant organisms, or large cavity size(>8cm in diameter).Chest radiographs commonly show worsening in approximately one third of patients during the first week of treatment[39].The median time to cavity closure is 4 weeks, and surrounding infiltrates may take twice that time to resolve. Radiographic improvement may lag behind clinical cure. In some patients with a clear clinical response to medical treatment, cavities resolve slowly and may take many weeks or months to disappear radiographically; a small percentage of patients are left with residual cavity[3].

Mortality and the predisposing factors. Hirshberg and colleagues in a study of 75 patients with lung abscess concluded that high rates of morbidity and mortality (20%) are associated with lung abscess despite appropriate antibiotic therapy and supportive care. In patients with several predisposing factors, such as a large abscess size and right lower- lobe location, the prognosis was worse. The patients infected with *S.aureus, K.pneumoniae*, and particularly *P.aeruginosa* had an ominous prognosis. As the prognosis for lung abscess has not improved sufficiently since the introduction of antibiotics, other modalities should be considered for patients with prognostic signs [4].In the preantibiotic era more than 45% of patients with lung abscess underwent surgery, and one third died. In more recent years, less than 15% of patients have undergone surgery, and overall mortality rate is approximately 10%.Death in patients with primary lung abscess or community- acquired abscesses (approximately 2 % to 5%)[22],but a fatal outcome is seen in more than 65% of cases associated with obstructive airway lesions, impaired host defenses, or nosocomial acquisition[40].

VII. CONCLUSION

With medical advancement, antibiotic therapy and supported care mortality of lung abscess has reduced to 20% a decline from 30 to 40 % rate of the preantibiotic era. Patients with predisposing factors and nosocomial infection have worst prognosis.

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