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Necrotizing Pneumonia and Conservative Treatment: A Case Report and Review of the Literature

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Abstract

We present a case of necrotizing pneumonia in an 87-year-old man without severe respiratory instability. Clinical suspicion arose due to the need for supplementary oxygen and persistent fever during treatment for community-acquired pneumonia. The diagnosis was confirmed by chest computed tomography. Although necrotizing pneumonia typically requires major surgical intervention upon diagnosis, we chose conservative management with antimicrobials and chest drainage alone. The patient experienced significant improvement and resolution of pneumonia with conservative management.

Keywords

Respiratory Tract Infections, Lung Disease, Bacterial Pneumonia

1. Introduction

Necrotizing pneumonia is a rare complication of community-acquired pneumonia (CAP) and it is associated with high mortality rates [1] [2]. It is a rare complication in adults and often is described as acute worsening of respiratory and infectious status in a person in CAP treatment. Management often necessitates intensive care unit (ICU) admission, antimicrobial therapy, and surgical intervention [3]. Since it is a rare disease, there are no guidelines to direct the correct medical care. Most case series present surgical treatment with lung debridement or decortication as the goal. Surgical interventions are commonly recommended due

to the life-threatening nature of necrotizing pneumonia and its poor response to antibiotics alone [4]. Historically, antibiotics were considered ineffective in penetrating necrotic tissue [5].

In this report, we present a case of necrotizing pneumonia in an 87-year-old man exhibiting mild respiratory deterioration, who responded well to conservative management (without extensive surgical intervention such as lung debridement or decortication).

2. Case Report

An 87-year-old male patient, from southern Brazil, a non-smoker, who was previously institutionalized, with a medical history significant for ischemic heart disease, hypertension, and type 2 diabetes mellitus He was previously hospitalized for meningoencephalitis secondary to subdural hematoma drainage six months prior to the current admission. On presentation to the emergency department, he was febrile with decreased consciousness. Clinical examination revealed hypotension (70/40 mmHg), febrile state (38.1˚C), lethargy, tachypnea (24 breaths per minute) and diffuse rhonchi on lung auscultation. Laboratory findings included a creatinine level of 1.85 mg/dL, potassium of 5.9 mEq/L, and elevated C-reactive protein (CRP) at 40 mg/dL. Complete blood count showed leukocytosis with 83% neutrophils, hemoglobin of 9.8 g/dL, and platelets within normal limits. Arterial blood gas analysis showed a pH of 7.43, pCO_2 34 mmHg, pO_2 60 mmHg, HCO_3 22.6 mEq/L, and oxygen saturation of 91%. Non-enhanced chest CT revealed consolidations and parietal thickening of segmental and subsegmental bronchi, more pronounced in the dependent portions of both lungs, particularly the basal segments of the left lower lobe, accompanied by pleural effusion, suggestive of an inflammatory/infectious process. Initial treatment with Piperacillin-Tazobactam 4.5 g 8/8 h was initiated for coverage against common pathogens in institutionalized patients, such as *Streptococcus pneumoniae* and *Pseudomonas aeruginosa*. The patient initially improved hemodynamics and respiratory status with Piperacillin-Tazobactam but continued to experience daily fevers. Antibiotic therapy was escalated to Meropenem 1 g 8/8 h due to suspected multidrug-resistant pathogens. Three days after the antibiotic switch, despite maintaining stable breathing patterns, normotension, and good general state, the patient became oxygen-dependent (1 to 2 L/m) and maintained low-grade fevers, prompting a repeat chest CT to assess infection complications and possible sources of persistence. The new image revealed increased consolidative opacity in the left lower lobe, with gas foci, suggesting necrotizing pneumonia (**Figure 1**). New opacities with ground-glass attenuation in the periphery of the right upper lobe suggested the possibility of coexisting inflammatory/infectious processes caused by atypical or viral germs. There was an increase in bilateral pleural effusion, with moderate-sized, free-appearing effusion on the right (consistent with congestion) and a lobulated appearance on the left, associated with pleural thickening, suggestive of pleural empyema. COVID and Influenza tests were negative. The case was deliberated with the

hospital's Infectious Disease department, resulting in the decision to continue Meropenem and supplement with Linezolid 600 mg 12/12 h and Azithromycin 500 mg/day to ensure coverage against *Methicillin-resistant Staphylococcus aureus* (MRSA) and atypical pathogens. Additionally, Furosemide was initiated to manage associated pulmonary congestion. Subsequently, radiologically guided drainage of the left pleural empyema was performed, yielding approximately 1 liter of purulent and citrine fluid (Figure 2). Microbiological analysis of the drained fluid revealed the presence of *Streptococcus viridans* (sensitive to Penicillin and Vancomycin) and MRSA *coagulase-negative Staphylococcus*. One day post-procedure, the patient was breathing ambient air, without further episodes of fever, progressing with clinical and laboratory improvement. A repeat non-enhanced chest CT after drainage showed regression of consolidative opacities and gas components in the left lower lobe, along with reduced bilateral pleural effusion and resolution of empyema. The patient was discharged after completing 14 days of the last antibiotic regimen. One month later he returned to scheduled clinical evaluation with significant improvement and without recurrence of infection (Figure 3).

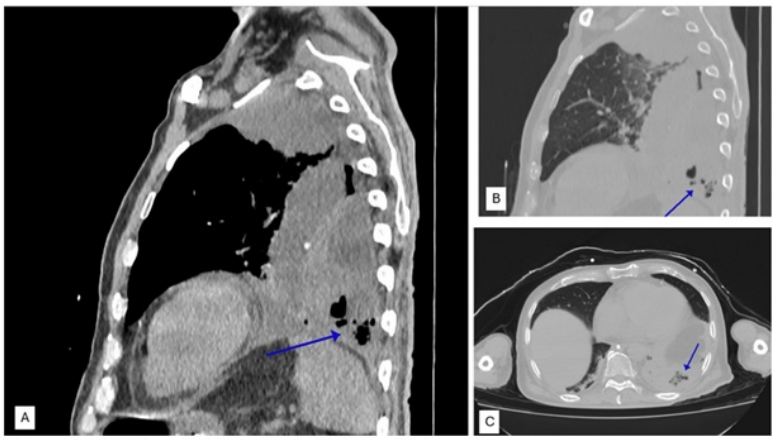


Figure 1. Chest computed tomography demonstrating pulmonary necrosis.

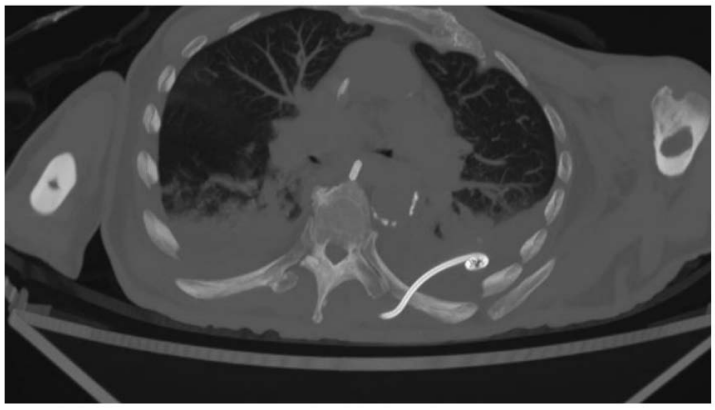
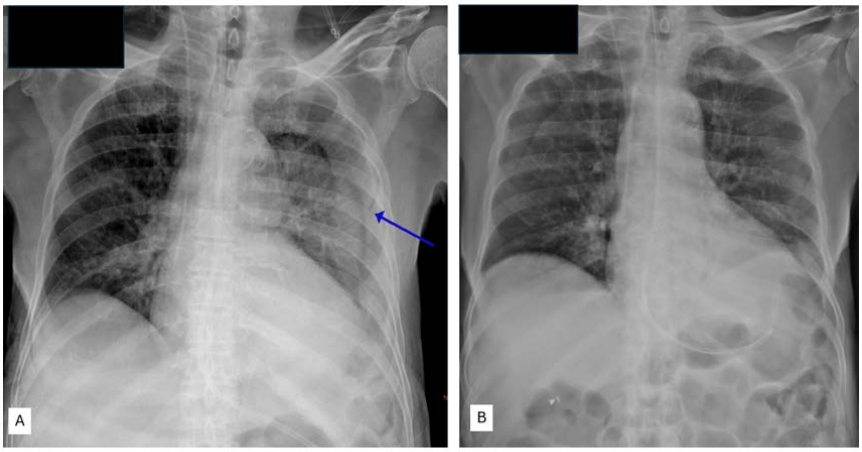


Figure 2. Chest drainage with pigtall drain.



3A Chest x-ray demonstrating loculated pleural effusion before drainage. 3B Chest x-ray after drainage and antibiotic treatment.

Figure 3. Follow up chest X-ray.

Patient perspective

The patient and family were informed about the treatment and it was consent. They also consented to the publication of this case report.

3. Discussion

Necrotizing pneumonia is a rare clinical condition commonly observed in pediatric populations. The incidence of necrotizing pneumonia in children, while low, appears to be increasing and accounts for approximately 5% to 10% of cases of community-acquired pneumonia [6]. Often, it is associated with CAP complications. Studies have reported necrotizing pneumonia in 0.8% to 7% of children diagnosed with CAP at tertiary centers in the United States. Conversely, its occurrence in adults is less frequent, estimated to be less than 1% [6] [7].

Necrotizing pneumonia is described as inflammation and dense consolidation within the lungs. This condition triggers a cascade of events where toxin release and cytokine response contribute to tissue necrosis and the formation of numerous small cavities. Furthermore, the pulmonary vasculature is frequently obstructed due to thrombus formation, leading to diminished blood supply and facilitating uncontrolled bacterial replication, often involving anaerobic bacteria [7]. This destruction and necrosis of lung parenchyma define necrotizing pneumonia as a complication of standard pneumonia [6]. Characterized by the development of multiple small, thin cavities, necrotizing pneumonia can progress to more grave complications, such as bronchopleural fistula, empyema, respiratory failure, and septic shock. Given the rapidly progressing nature of this condition, especially in previously healthy patients, there has been considerable focus on identifying host or pathogen factors contributing to its severity [7].

Necrotizing pneumonia usually initially presents as a typical bacterial pneumonia, with symptoms of cough, fever and dyspnea, often accompanied by elevated inflammatory tests, such as C-Reactive Protein and leukocytosis in the blood

count. However, this form of pneumonia becomes really severe, with high levels of clinical instability (respiratory failure and hypotension), and is associated with longer hospitalization, a greater risk of progression to sepsis and respiratory failure, and a higher incidence of local complications, such as pleural effusion, empyema and pneumothorax [8].

The diagnostic hallmark of necrotizing pneumonia lies in pneumonic consolidation accompanied by multiple areas of lung parenchymal necrosis. These necrotic regions may merge, forming a lung abscess when localized, or triggering pulmonary gangrene if involving entire lobes [9].

Plain chest radiographs are diagnostic in only approximately one-fourth of cases. Initially, cavitary lesions formed in necrotizing pneumonia are filled with fluid and therefore have the same density as the adjacent consolidated lung. Thus, it is difficult to differentiate necrotizing pneumonia from uncomplicated consolidations in chest radiographs. Cavitary necrosis is likely to be apparent on plain radiography, however, this finding occurs late in the course of the disease and therefore results in a delayed diagnosis [6].

Computed tomography (CT) is more sensitive than plain radiographs for detecting pneumonia complications and is indicated when there is no adequate response to antibiotics [6]. On a contrast-enhanced chest CT, initial findings of necrotizing pneumonia may include pleural effusion followed later by the development of multiple cavities. This exam usually shows diffuse or patchy consolidation in multiple lobes of the lung [10]. Loss of parenchymal enhancement, destruction of the lung parenchyma, thick-walled cavities, and air/fluid-filled thin-walled cavities can also be seen [9] [11]-[13]. Late findings of necrotizing pneumonia include pneumatoceles, bullae, and hydropneumothorax [6].

There is a well-documented association between necrotizing pneumonia and Panton-Valentine leukocidin (PVL)-positive *Staphylococcus aureus* strains [10]-[12]. PVL leads to local tissue necrosis as a result of phagocyte-mediated inflammation, playing an important role in antimicrobial strategies. [6] [14]. Concerning intensive care settings, Taffarel et al reported that community-acquired methicillin-resistant *S. aureus* (MRSA) represented 72% of cases [6] [15]. Other etiologic agents include *Streptococcus pneumoniae* and, less commonly, *Klebsiella pneumoniae*, *Haemophilus influenzae* and *Pseudomonas aeruginosa*. Besides that, pulmonary gangrene is more frequently seen in infections caused by gram-negative organisms like *K. pneumoniae* and *P. aeruginosa* [8] [12].

Influenza coinfection is a major risk factor for the development of necrotizing pneumonia. There is clear evidence that influenza increases host susceptibility to bacterial infections [6] [12]. In a case series of 43 cases of staphylococcal necrotizing pneumonia, 86% were PVL-positive, 28% had confirmed and 37% had suspected viral coinfection [13].

Taking into consideration the most common implicated pathogens, empiric treatment usually includes ceftriaxone [6] [13]. If suspicion of community-acquired MRSA pneumonia is high, vancomycin is the most frequently used agent,

but glycopeptide or linezolid are also recommended [9]. It is well-documented that poor response to vancomycin monotherapy, being combination therapy especially indicated in these cases [6]. In addition, agents such as antipseudomonal b-lactams, quinolones, and carbapenem may also need to be a part of the therapeutic arsenal [9].

The inclusion of antitoxin antimicrobials like clindamycin and linezolid may have a role in inhibiting the production of PVL by *Staphylococcal* infections [6] [9]. In patients with lung abscesses, it should be considered to use additional empirical anaerobic antibiotics, clindamycin, or metronidazole as possible treatment options in these cases [9]. Furthermore, there may also be a role for the use of intravenous immunoglobulin (IVIG) in necrotizing pneumonia, especially in S. aureus pneumonia [7] [9] [14]-[16].

It is recommended repeated radiographic evaluation every 3 - 4 days to verify antibiotic therapy's effectiveness or possible complications [14]. Moreover, in severe cases, intravenous antibiotic therapy should last at least 4 weeks after the last positive blood culture [14] [17].

It is a well-known fact that chest tube drainage souls be used for pneumonia with large pleural fluid collections. Nevertheless, this management can lead to the loculation of fluid collection and enhance the risk of fistulation. Therefore, patients with massive necrotic tissue, which prevents antibiotics from reaching the infected areas, benefit from surgical treatment. This kind of approach is strongly recommended for cases of persistent fever and leukocytosis, the occurrence of empyema, bronchopleural fistula, and hemoptysis or impaired respiratory function, despite the use of medications [6] [9].

Debridement, wedge resection, and segmentectomy are feasible when necrotic parenchyma is isolated in the periphery of the lung. However, if the affected parenchyma is too extensive, a lobectomy should be performed. During the procedure, it is important to maintain airways frequently suctioned, avoiding the spread of secretions to the contralateral side [9].

The most common postoperative complications are persistent air leaks, empyema, and ventilator dependency. Once residual space increases significantly the risk of empyema, pleural irrigation system, or obliteration with a muscle flap should be considered as preventive measures [9]. Since surgical management can lead to complications; conservative treatment should be considered depending on the patient's comorbidities and extension of lung damage [18].

4. Conclusion

Our patient was an institutionalized elderly man with extremely compromised functionality and moderate dementia. Due to these factors, it was determined that he would not support invasive measures such as endotracheal tube or thoracic surgery if there was significant clinical deterioration. Faced with the diagnosis of necrotizing pneumonia, the possibility of indicating surgical management for the patient, such as pleuroscopy, segmentectomy or pulmonary lobotomy, was

considered, based on the literature review carried out by the case's medical teams. However, the patient had no indication of measures such as mechanical ventilation, essential for the indicated procedures, which already constitute quite aggressive interventions in themselves. This led us to discuss the limits of therapy in this situation, as the patient remained symptomatic and dependent on oxygen even with optimized antibiotic therapy. Considering these factors, it was then decided to perform pleural effusion by radio intervention, even though it is not the most recommended procedure for cases of necrotizing pneumonia. As it is a more prevalent disease in children, management tends to be more aggressive in most cases seen with the same severity as our patient. However, our report presents an unusual situation in this type of pneumonia, in which the patient would probably not tolerate the indicated procedure or its potential complications, making it necessary to seek less invasive treatment alternatives. Fortunately, our patient responded well to management, being discharged asymptomatic and without oxygen therapy.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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