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Atypical 'Aspiration Pneumonia: A Rare Case of Co-infection with Mycoplasma Pneumonia and Streptococcus Pneumonia

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Abstract Mycoplasma pneumoniae is known to cause atypical pneumonia, but not aspiration pneumonia, macrolides are still considered the first-line therapy. Typical antimicrobials used to treat aspiration pneumonia does not cover atypical pneumonia, here we report a case of a young, previously healthy female with no significant medical history, who was in her usual state of health before she was brought to the hospital with altered mental sensorium, preceded by benzodiazepine intake that she bought from the street. She had imaging and labs consistent with acute hypoxic respiratory failure due to aspiration pneumonia, was intubated and started on mechanical ventilation. She was found to be co-infected with mycoplasma pneumonia and streptococcus pneumonia, successfully managed with intravenous antibiotics, and eventually liberated from the ventilator and was safely discharged home.

Keywords: *aspiration pneumonia, mycoplasma pneumoniae, streptococcus*

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1. Introduction

Aspiration pneumonia is a disease recognized for ages, yet there are few conditions in pulmonary medicine as frequent in occurrence but as lacking in consensus regarding classification and treatment. Historically anaerobes are most commonly implicated in the pathogenesis of aspiration pneumonia. Mycoplasma pneumoiae is a respiratory pathogen that is primarily associated with 'walking pneumonia'. Macrolides are the first-line treatment, and the duration of therapy depends on the severity of the illness. To our knowledge, this is the first reported case of aspiration pneumonia in which both Streptococcal pneumonia and Mycoplasma Pneumoniae are implicated in the same patient of aspiration pneumonia.

2. Case Presentation

22 years old female with no medical problems was brought to the emergency department with altered mental sensorium of one-day duration. The patient was found slumped in the chair by her friend who reported that the patient overdosed on the "Xanax "that they bought on the street. The friend confirmed that the patient was all right and in her usual state of health, and they were having "fun on Saturday" night.

On presentation, the patient was hypoxic to 83%, tachycardia 133 /min, blood pressure 115/83 mm Hg, temperature of 99 F and a respiratory rate of 33/min. Physical examination was significant for a glas gow comma scale of 9/15 (E3M5V1) diffuse bilateral coarse crackles on bilateral lower lobes and right middle lobe; a neurological exam was hard to assess, and the rest of the physical exam was normal. Pertinent labs showed troponemia (77, 161 ng/l reference <12 ng/l) lactate dehydrogenase 564 units/l pro BNP 930 pg/ml WBC was 9 k/ul (84 percent neutrophils) hemoglobin/hematocrit 16 g/dl / 48%.Computed tomography(CT)of the head was normal. X-ray chest was significant for extensive right-sided infiltrate with probable effusion, and patchy left lower lobe infiltrate. Ct chest was consistent with diffuse bilateral consolidative airspace disease, right greater than left most consistent with multifocal pneumonia and/or ARDS (Figure 1).

Suspecting acute hypoxic respiratory failure due to aspiration pneumonia, patient was intubated and started on mechanical ventilation. The patient was started on intravenous piperacillin-tazobactam, and was transferred to the critical care unit for further management. The patient underwent bronchoscopy, bronchoalveolar lavage was purulent and respiratory cultures grew B streptococcus. Pneumonia work further revealed positive streptococcal antigen in the urine and serum analysis showed mycoplasma IgM with a titer of 1238U/ml on two consecutive serologies. COVID 19 was not detected. At

this point, infectious disease specialty was taken on board and piperacillin-tazobactam was deescalated to ceftriaxone with addition of azithromycin to the regimen. The patient was successfully liberated from the ventilator but remained dependent on noninvasive positive pressure ventilation first to Bi-level positive airway pressure to oxygen through the nasal cannula and finally weaned off to room air. The patient received a prolonged course of antibiotics, for a total of 14 days (intravenous and then oral levofloxacin) and was successfully discharged home.

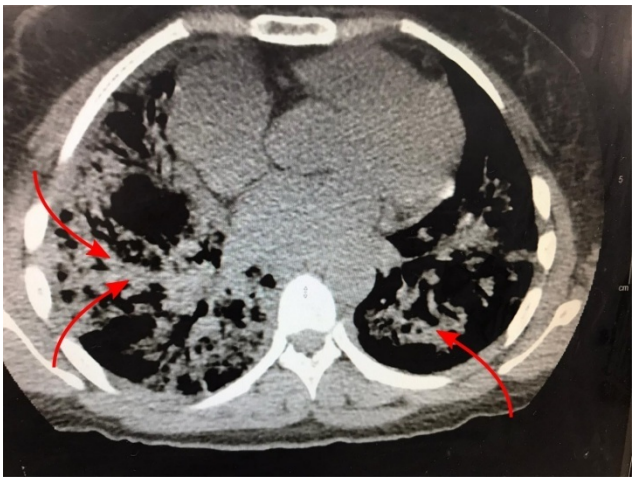

Figure 1. Ct chest showing bilateral consolidations (red arrows)

3. Discussion

Aspiration is not an uncommon event even in healthy individuals and is known to usually resolve without detectable sequelae. Studies show that at one-half of healthy adults aspirate during sleep [1]. Pulmonary sequelae depend upon the volume and contents of the inoculum and host defense mechanisms. Aspiration pneumonia refers to the pulmonary consequences resulting from this abnormal entry of fluid, particulate exogenous substances, or endogenous secretions into the lower airways. Predisposing conditions include reduced consciousness resulting in a compromise of the cough reflex and glottic closure that is most commonly seen with alcohol or illicit drug abuse or anesthesia and also occurs with generalized seizures; other significant risk factors include dysphagia, mechanical disruption of the glottic closure or cardiac sphincter due to tracheostomy, endotracheal intubation, bronchoscopy, upper endoscopy, and nasogastric feeding, pharyngeal anesthesia and miscellaneous conditions such as protracted vomiting, large-volume tube feedings, feeding gastrostomy, the recumbent position, and drowning [2]. Aspiration pneumonia is broadly classified into three categories, chemical pneumonitis, bacterial infection, and airway obstruction. The classification is based on the character of the inoculum, pathogenesis of the pulmonary conditions, presentation, and management [3]. Here, we will limit our discussion to bacterial infection only.

The most common form of aspiration pneumonia is caused by bacteria that normally reside in the upper airways or stomach. Historically, aspiration pneumonia has usually referred to an infection caused by less virulent bacteria, primarily oral anaerobes, and streptococci, which are common constituents of the normal flora in a susceptible host prone to aspiration [4]. The more recent work has questioned the dominance of anaerobic bacteria in aspiration pneumonia, with emphasis on the more common and more virulent bacteria encountered in hospital-acquired pneumonia, such as S. aureus, Pseudomonas aeruginosa, and other aerobic or facultative gram-negative bacilli [5]. Community-acquired aspiration pneumonia has a mixed infection that includes aerobes and anaerobes. In one report from Japan of 212 patients with lung, abscess showed streptococci to be the most common pathogens (60 percent of patients) and anaerobes to be the second most common (26 percent) [6]. In comparison, patients with hospital-acquired aspiration pneumonia commonly involve a mixture of anaerobes and gram-negative bacilli or S. aureus; therapy is usually directed at both the anaerobes and aerobes in this setting [7].

Mycoplasma pneumonia (M. pneumoniae) is an obligate parasite. It is most famous for causing walking pneumonia however it more commonly causes tracheobronchitis. The bacterium M. pneumonia has no cell wall, which renders it insensitive to Beta-lactam antibiotics. To the best of our knowledge, M.pneumoniae has never been implicated in aspiration pneumonia, and in our literature research we found that its confection with streptococcus pneumonia has been reported only once in case series of nine patients in pediatric patients admitted for community-acquired pneumonia

Recent studies have shown that the respiratory tract flora extends from the nose to the alveoli contrary to the prior belief that the respiratory tract is sterile below the larynx [8]. Two theories can be derived from this data firstly, micro aspiration is common and that aspiration pneumonia is a consequence of a large inoculum, the pathogenicity of the microbes aspirated, or both and secondly M. Pneumoniae being part of respiratory flora may get aspirated and cause aspiration pneumonia

The presenting findings in aspiration pneumonia due to bacterial infection are highly variable; most patients present with the common manifestations of pneumonia, including cough, fever, purulent sputum, and dyspnea. Cases involving anaerobes usually evolve over a period of several days or weeks instead of hours [9]. Antibiotics are the mainstay of the management of bacterial aspiration pneumonia. If anaerobic bacteria are suspected pathogens in aspiration pneumonia, and parenteral therapy is required, ampicillin-sulbactam is suggested as first-line therapy. The usual dose is intravenous 1.5 to 3 g every 6 hours, depending on the renal function. For patients who are not severely ill and who can tolerate an oral regimen, amoxicillin-clavulanate is an appropriate alternative. The dosage required is immediate release 875 mg orally twice daily or extended-release 2 g orally twice daily. The duration of antibiotics for aspiration pneumonia is not well studied. The usual duration of therapy for cases the cases that are not complicated by cavitation or empyema is seven days.

4. Conclusions

Our case emphasizes the importance of suspecting atypical organisms in the etiology of aspiration pneumonia and not just limiting the suspicion to more common ones

like anaerobes/gram negatives/positive aerobes. We would also like to stress upon sending complete pneumonia work up in critical patients.

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