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Serendipitous Diagnosis of *B. pertussis*-Associated Empyema in Adult with HIV: A Case Report and Literature Review

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Abstract

The following report describes an extremely rare case of severe *Bordetella pertussis* pneumonia complicated by empyema and septic shock in a profoundly immunocompromised patient. Briefly, this was a 33-year-old male with a past medical history of advanced HIV/AIDS (CD4 count 15 cells/μL), disseminated MAC infection, and cryptococcal meningitis who presented with tachycardia, tachypnea, hypotension requiring norepinephrine infusion, and lactic acidosis. A CT scan demonstrated near-complete opacification of the left hemithorax secondary to a large pleural effusion with compressive atelectasis and infiltrate. A respiratory multiplex panel was positive for *B. Pertussis* and parainfluenza virus. Bedside thoracentesis yielded purulent fluid, consistent with empyema. Subsequently patient was treated with azithromycin for pertussis, intravenous ceftriaxone for empyema, pleural drainage for source control, vasopressor support, and supplemental oxygen. Antiretroviral therapy, and treatment for opportunistic infection prophylaxis were resumed.

Keywords: Serendipitous diagnosis; Lactic acidosis; Prophylaxis

Introduction

We present a clinical case where a HIV patient PMH of MAC, cryptococcal meningitis presented with tachycardia, pleuritic chest pain, shortness of breath and septic shock. Work up including respiratory panel, chest X ray and CT chest revealing the presence of empyema induced by *B. pertussis*. This is a very rare complication of *B. pertussis* in HIV patient. Searching of PubMed, Medline and other literature sources did not uncover any case report related to this complication and we present this case to highlight this unusual finding from this organism.

B. pertussis is a gram-negative aerobic coccobacillus which is causative organism of classic Whooping Cough. It mainly effects children [1]. It is spread by person-person transmission through respiratory droplets [1]. Its infection is characterized by a prolonged paroxysmal, spasmodic cough, postussive vomiting, and inspiratory whooping. In a few cases, it is complicated with pneumonia and encephalopathy. In unvaccinated children, it causes severe disease with increased mortality.

Vaccination provides substantial protection and is associated with a milder disease course in break-through infections [1]. It rarely effects adults who acquire infection from the children from close contact [1]. However, immunocompromised adult has increased risk of end-organ and systemic complications Specifically, spread to pleural space and infection of lung parenchyma. These clinical manifestations are rarely reported in the literature including PubMed and Google scholar thus far. Since being a very rare case, we report this clinical case of *B. pertussis* Empyema in older patient with severe immunosuppression. By reporting this case, we would like to emphasize the destructive and aggressive nature of *B. pertussis* infection in HIV patients. Specifically, we would like to highlight its clinical belligerence in its spreading and infecting lower airways and pleural space, thus instigating severe pneumonia, parapneumonic pleural effusion, and empyema. We will review the current evidence including transmission, pathophysiology, clinical features, diagnosis and treatment of *B. pertussis* infections in adult patients.

Case Presentation

A 33-year-old man with advanced HIV/AIDS (CD4 count 15 cells/μL; viral load 39,100 copies/

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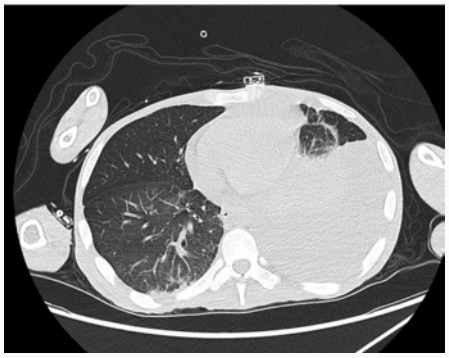

Figure 1: CT chest showing near complete opacification of left lung, thence signifying the presence of Bordetella Pertussis empyema of the left lung.

mL) and a history of medication nonadherence presented to the emergency department with progressive dyspnea and pleuritic chest pain. His medical history was significant for disseminated Mycobacterium avium complex infection, cryptococcal meningitis, chronic hepatitis B, chronic kidney disease (stage?), chronic cerebral venous sinus thrombosis, and severe protein-calorie malnutrition.

On presentation, the patient was tachycardic, tachypneic, and hypotensive. Laboratory evaluation demonstrated a lactate >4 mmol/L and cute kidney injury with a creatinine of 2.48 mg/dL. Persistent hypotension necessitated intravenous fluid resuscitation and norepinephrine for septic shock. Chest radiography demonstrated a moderate left sided pleural effusion with patchy left basilar opacities and consolidation concerning for pneumonia. Subsequent non-contrast chest CT demonstrated near-complete opacification of the left hemithorax secondary to a large pleural effusion with associated compressive atelectasis and infiltrate (Figure 1).

A respiratory panel was positive for *B. pertussis* and parainfluenza virus. Notably, the patient did not exhibit the classic paroxysmal cough typically associated with pertussis. Given the large pleural effusion and concern for complicated pneumonia, bedside thoracentesis was performed and yielded purulent pleural fluid, consistent with empyema. Initial pleural fluid cultures demonstrated gram-negative bacilli, while blood cultures remained negative.

The patient was diagnosed with ever pneumonia- associated with *B. pertussis*, complicated by empyema and septic shock in the setting of profound immunosuppression. Standard antibiotic therapy was initiated for complicated pneumonia with parapneumonic effusion was initiated which included azithromycin and ceftriaxone. Despite initial source control with thoracentesis, the patient remained dependent on supplemental oxygen and vasopressor support. His antiretroviral therapy and antimicrobial regimens for his underlying opportunistic infections were resumed. Repeat CT imaging demonstrated improvement with partial clearing of the left sided effusion (Figure 2). Due to the patient's immunocompromised state, an extended course of antibiotics was required.

Discussion

B. pertussis (BP) was first identified in 1906 and mainly responsible for whooping cough [2]. Despite the efficacy of pertussis vaccines, there has been an uptick in the amount of BP cases reported worldwide. As we pay attention to the pathophysiology of whooping cough, BP enters through the nasal passages from inhalation of respiratory droplets [3]. *B. pertussis* (BP) is a gram-negative bacterium that

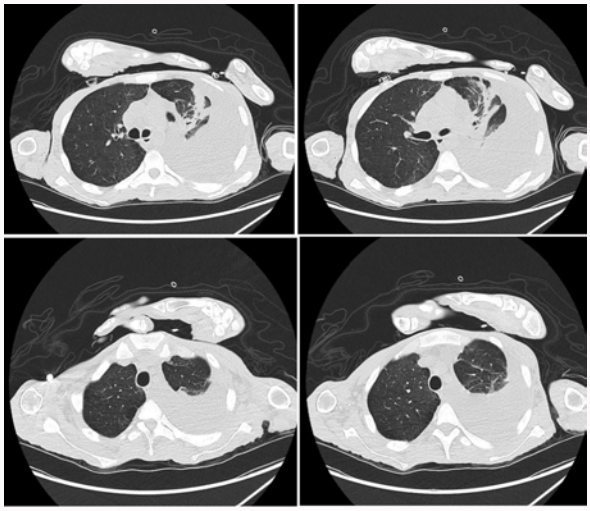

Figure 2: CT Chest showing partial resolution of Bordetella Pertussis empyema of the left lung after starting therapy.

usually effects respiratory airways. It principally affects children and is transmitted by respiratory droplets [3]. Close contact with infected patients can propagate the infections as these droplets perambulate for minimum of 3 feet. These respiratory droplets eventually land on the mucosal surfaces of close contacts thus providing a footing for these microorganisms to initiate and propagate the pathological disease process.

Once it gains entry to the susceptible host, it localizes to upper airways and binds to the cilia of respiratory epithelial cells with the help of fimbria and filamentous hemagglutinins [4]. After localization, it multiplies and releases various toxins [3,4]. Tracheal cytotoxin acts on the respiratory epithelial cells to provoke local apoptosis and tissue damage. Toxins such as adenyl cyclase toxin, and pertussis toxin work synchronously to suppress emigration of inflammatory epithelial cells as well as inhibit phagocytosis, thus fostering the progression of mucosal inflammation. This greases the wheels for perpetuation of mucosal congestion, mucosal edema and increased respiratory secretions. As a result of these transgressions, peri-bronchial lymphoid hyperplasia followed by necrotizing inflammation of larynx, trachea and bronchi can be appreciated. Further progression, migration to the lower airways and transition to pneumonia mainly depends upon the risk factors, bacterial co-infection and immunity of the patient. Complications that are reported in the literature include peri-bronchiolitis, emphysema and atelectasis.

After an incubation period of 7-10 days, children develop clinical symptoms including fever, nasal congestion, runny nose, and cough. The cough of BP is very characteristic. It is specifically characterized by severe hacking cough and culminates into a peculiar whooping sound. Unfortunately, some children progress to develop pneumonia. In this regard, it has been shown that children with BP infection frequently develop coinfections with rhinovirus, mycoplasma pneumonia, respiratory syncytial virus infection, streptococcal pneumonia and haemophilus influenza. Thus, physicians prefer to treat these cases with erythromycin/ciprofloxacin therapy, as they act through attenuating the protein synthesis and shrinking bacterial proliferation [3].

As we shift our focus to adult patients, the percentage of BP pneumonia cases that are reported in the literature thus far are very

scarce. In a recent study, percentage of BP infections reported in the hospitalized patients with community acquired pneumonia is around 3%, although serological evidence is much higher around 7% [5,6]. Moreover, it is estimated that BP infections are responsible for adult pneumonia in only 4% cases [7,8]. Even with history of recent BP infections, the capability of BP driving adult pneumonia is still debated as it might be coincidental and primarily acts as subservient to primary bacterial pathogens in instigating pneumonia. Nevertheless, we should not be convinced otherwise of its sufficing clinical virulence in sparking pneumonia. As a matter of fact, and beyond doubt, recent studies have corroborated its solo clinical efficacy in enkindling full-blown pneumonia in adult population.

With that being said, let's focus our attention on the clinical spectrum of BP in adult patients. The clinical symptomatology of BP pneumonia in adults are slightly variable and differs vastly from the children. Like children, most of the adult patients report cough that usually lasts for an average duration of 21 days [8]. However, whooping cough is rarely seen in adults. Instead, cough is usually followed by vomiting, which is specifically described as post-tussive emesis [8]. Alternate presentations including cough followed by choking and cough provoking sleep alterations. Other atypical symptoms reported in the adult patients include sweating, sinus pain, headache, influenza symptoms, pharyngitis and sneezing [8]. Complications in adult patients (28%) occur more frequently than pediatric patients (6%). Pneumonia is more likely to take shape in older patients (9%) as compared to younger adults (2%). Urinary incontinence is more predisposed to occur in elderly women (34%). In the present of risk factors like smoking and asthma, there is an increasing risk of protracted course for cough, sinusitis and cough related sleep disturbances. Empyema along septic shock is reported in our patient, a finding not reported elsewhere in the literature.

As mentioned before, BP induced empyema transpires when lung infection disseminates and sprawls into neighboring pleural space. This will smoothen the path for infection of adjacent pleural cavities, thus opening the doors for pleural colonization, pleural inflammation, fluid accumulation, bacterial multiplication and ultimately culminating into development of empyema.

BP empyema which literally indicates accumulation of pus inside the pleural cavity materializes due to heightened bacterial multiplication. There is only evidence of one case report published in the literature thus far showing BP mediated empyema in non-small cell lung cancer patient who presented with unilateral pleural effusion [9]. Two case reports, with unilateral pleural effusion and empyema caused by *B. bronchoseptica*, were reported in the literature [10,11]. Searching PubMed, Medline and Google Scholar did not find reveal any clinical case reports of empyema provoked by BP in adult patients.

In a typical clinical setting, older patients with underlying pulmonary impairment are more predisposed to develop BP mediated empyema in the presence of risk factors such as alcoholism HIV, immunosuppression, chemotherapy, radiation, CVOID-19, decompensated liver cirrhosis, lung malignancy, and hematological disorders [9-12]. A common denominator of above-mentioned risk factors is weakened and maladaptive innate and adaptive immunity, thus providing conducive environment for unrestrained BP multiplication, lung and subsequent pleural space invasion.

As BP empyema is suspected, then work up should be initiated for confirming the diagnosis. This should be started with platting nasopharyngeal secretions into charcoal blood sugar as well as performing gram stain and cultures [2]. Empirical therapy should be started immediately when diagnosis is suspected. However, antibiotic cultures and sensitivity should be taken for microbiological analysis to tailor the antibiotics for optimum clinical response. Confirmation of diagnosis can be pursued by identification of BP DNA by PCR utilizing primers utilized by various labs. Blood cultures should be ordered as bacteremia and sepsis is suspected. Chest-X ray, and CT chest should be ordered accordingly as per the clinical scenario to detect pneumonia, atelectasis, emphysema, pleural effusion and empyema.

Clinicians have relied on circulating titers of antibodies which might signify protection against infection. These circulating antibodies start to appear at 3 weeks after infection, and incline towards peaking at around 8-10 weeks post-infection. It is pertinent to note that, appearance of IgM and IgA in the blood indicates recent infection. Unequivocally, inception of serum IgA antibodies against pertussis toxin and filamentous hemagglutinin as well as secretory IgA in nasopharyngeal secretions within 2-3 weeks of infection symbolizes the activation systemic and local innate immunity against BP [2].

The antibiotics of choice for BP empyema includes a macrolide; Azithromycin, Clarithromycin, or Erythromycin [2,13,14]. If there is a sub-therapeutic response or intolerance to macrolides, then and Trimethoprim-Sulfamethoxazole, doxycycline or levofloxacin can be given to augment the bacterial clearance [2,15,16]. Since empyema induced by BP will have synergistic coinfection with staphylococcus, streptococcus and anaerobes additional empirical antibiotics with amoxicillin-clavulunate piperacillin-tazobactam and carbapenems is administered for 2-4 weeks [17]. However, an immunosuppressed patient might need longer duration of antibiotics based on their presence of risk factors and treatment response [17].

Viscous and adhesive purulent effusions or appearance of frankly purulent fluid in the pleural cavity will warrant chest tube placement for facilitating pus drainage, which will hasten earlier treatment response and faster patient recovery [17].

Hyperimmune pertussis immunoglobulin is sometimes used, although its efficacy is not verified in the clinical trials [2]. Acellular pertussis vaccines containing structural components of BP including pertussis toxin, filamentous hemagglutinin, pert-actin and fimbrial antigens are commonly used to boost immunity after recent infections [18]. Multiple studies reveal that humans mount an impeccable and flawless immune response to these bacterial toxins, thus affording remarkable protection against future infections. After completing primary vaccines in childhood, these acellular vaccines have been widely given as booster doses to augment immunity against BP [18]. These acellular vaccines have been widely employed in United States and Europe [18].

Adults with BP infections are recommended an adult tetanus, diphtheria, and acellular pertussis (Tdap) vaccine (3-dose series), which might help to reinforce the immunity after infection particularly in immunocompromised host [19]. Sometimes, chest tube placement for draining excess pus accumulation from the pleural space might be necessary.

Conclusion

- *B. pertussis* is a common cause of Whooping cough in children.

- The disease is transmitted by respiratory droplets which permeate within the distance of 3 feet from the patients and might come with close contacts.
- As *B. pertussis* enters the host, they circumnavigate to the upper airway respiratory mucosa, bind to ciliary respiratory epithelial cells, secrete toxins and instigates tissue necrosis.
- Adults do not present with classic whooping as described in children. In the contrary, they present with post-tussive emesis.
- *B. pertussis* infections are commonly affiliated with other bacterial coinfections including streptococcal pneumonia and hemophilus influenza.
- *B. pertussis* pneumonia is not uncommon in adults.
- In older patients with risk factors such as immunosuppression, these bacteria ramify and spread to the neighboring pleural space. This can be followed by bacterial multiplication, inflammation, pleural effusion, empyema and septic shock in rare cases.
- Nasopharyngeal gram stain, culture & sensitivity confirm the diagnosis should be pursued immediately.
- Imaging studies might uncover pneumonia and associated pleural effusion and empyema.
- Treatment with macrolides empirically and changing the antibiotic regimen based on cultures and sensitivity will improve clinical outcome to reduce morbidity and mortality.
- *B. pertussis* empyema is frequently linked with coinfections with staphylococcus, streptococcus and anerobes, thus warranting empirical antibiotic coverage with ampicillin, piperacillin-tazobactum or meropenem.
- Accumulation of frankly purulent fluid in the pleural space will need thoracentesis drainage with Chest tube placement for draining viscous and adhesive purulent effusions from the pleural space.
- Acellular pertussis vaccines can be administered to boost immunity after recent infections as they are very efficient in launching formidable immune response against future infections.

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