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OCCULT PNEUMOCOCCAL SEPTICEMIA AND PNEUMOCOCCAL PNEUMONIA COMPLICATING PNEUMATOCELES IN A PREVIOUSLY HEALTHY CHILD*

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Fulminating pneumococcal septicemia without an obvious focus of infection is very rare in previously immunocompetent children older than two years. Furthermore, pneumatocele formation in pneumococcal pneumonia is uncommon. The majority of pneumatoceles are self-limited and disappear spontaneously. Here, we report a six-year-old healthy child with occult pneumococcal septicemia and pneumococcal pneumonia secondary to septicemia. Giant pneumatoceles causing respiratory insufficiency formed secondary to the pneumococcal pneumonia and were aspirated via needle under fluoroscopic guidance. *Key words: pneumococcal septicemia, pneumatoceles.*

Pneumococci are important agents of pneumonia, sinusitis, and otitis media, and are a syndrome of occult bacteremia and pyogenic meningitis in children[1]. Occult pneumococcal bacteremia is very rare in healthy children older than two years, but severe septicemia occurs in patients with functional or anatomical absence of the spleen[2].

Pneumococci are common causes of bacterial pneumonia over three months. They produce lobar involvement and may cause pleural effusion and sometimes lung abscesses. Pneumatoceles, however, are uncommon and have been discussed in only three reports to date[3-5].

We report a six-year-old previously immunocompetent child with pneumococcal septicemia and secondary pneumococcal pneumonia with pneumatocele formation.

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Case Report

A six-year-old boy was admitted to our clinic with the complaints of fever, tachypnea, abdominal pain and confusion, which had been present for one day. Medical history indicated a previously healthy child. On physical examination body temperature was 38.9 °C, pulse rate 128/min, respiratory rate 56/min and blood pressure 60/30 mmHg. He was hyperpneic, pallid and confused. Common moderate rales were heard on both lung fields. The spleen and liver were not palpable. Cutaneous petechiae and ecchymoses, peripheral cyanosis and coldness were noted. Other than lethargy, his neurologic findings were normal and his neck was not stiff. Laboratory studies revealed hemoglobin 8.2 g/dl, white blood cell count 1600/μl (63% polymorphonuclear neutrophils, 22% band neutrophils, 15% lymphocytes, immature/mature + immature neutrophil ratio = 0.26), and platelet count 97,000/μl. The peripheral smear showed toxic granulation in neutrophils. Prothrombin time was 35 seconds and partial thromboplastin time was 92 seconds. The erythrocyte sedimentation rate was 55 mm/h. Fibrinogen was 69 mg/dl (N: 125-300) and fibrin split products were present. Neutrophil functions, serum immunoglobulin levels, CD4/CD8 ratio and CH50 were normal. Sickledex test yielded negative result, and function of the spleen, as documented by radionuclide liver-spleen scans, was normal. Biochemical analyses yielded serum urea 28 mg/dl, creatinine 1.1 mg/dl, AST 198 U/L, and ALT 213 U/L. Streptococcus pneumonia was grown in blood culture. Arterial blood gasses showed metabolic acidosis. Chest x-ray, and abdominal and renal ultrasound were normal. The diagnosis of pneumococcal septicemia was made and the patient was treated with ceftriaxone (75 mg/kg/day) and amikacin sulfate (15 mg/kg/day), with adequate fluid replacements and monitoring of pulse, respiration, blood pressure and urine output.

On the fourth day of admission, he developed cough and dyspnea. On physical examination, breath sounds were diminished on the right hemithorax and there were rales on the left hemithorax. Plain chest x-ray revealed air bronchograms. consolidation in the right upper lung fields, right-sided pleural fluid and pneumatocele formation in the right paracardiac and peripheral lung fields (Fig. 1). Examination of pleural fluid revealed a cloudy appearance, pH 7.25, specific gravity 1019, glucose 46 mg/dl, protein 3 g/dl, lactate dehydrogenase (LDH) 1260 U/L, pleural fluid LDH/serum LDH ratio 18, WBC 1200/μl and pneumococci in gram staining. Streptococcus pneumoniae isolated in pleural fluid culture. An expectorant agent was added to the therapy and the dose of the ceftriaxone was increased to 100 mg/kg/day.

During the five-week period of hospitalization, his clinical findings (other than tachypnea and moderate dyspnea), and hematological and biochemical disorders improved, but arterial blood gasses showed moderate hypoxemia,

and pulmonary function tests showed restriction pattern. Plain x-ray and computed tomography of the chest showed multiple large pneumatoceles on the right middle and lower lung fields and there was also compressive atelectasia at the medial sites of the pneumatoceles (Figs. 2a, b).

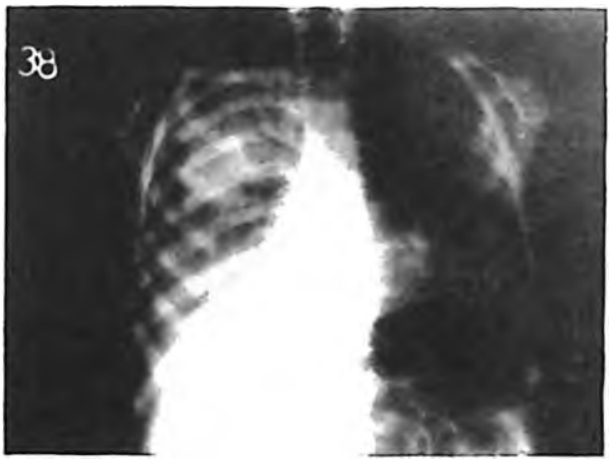
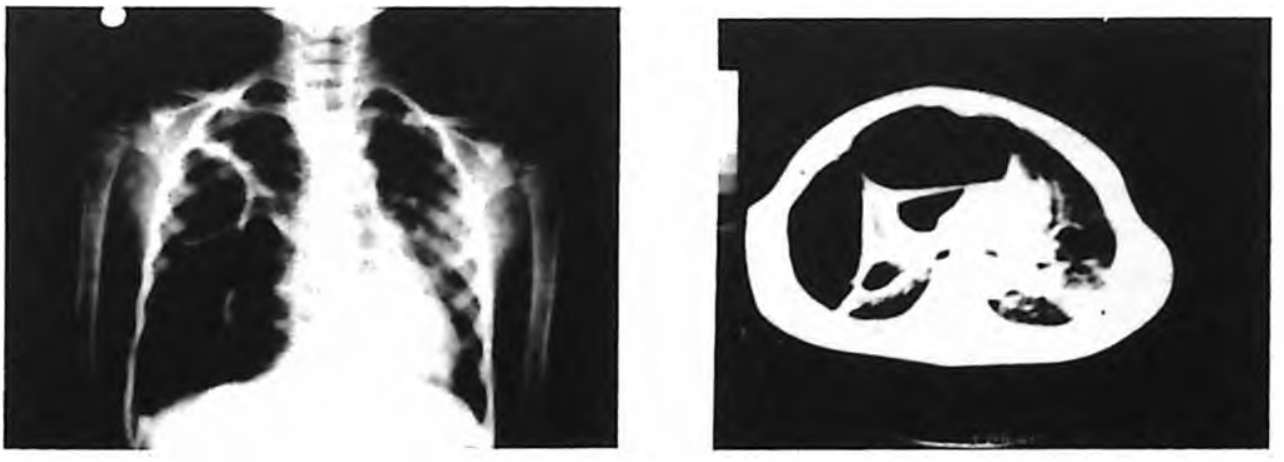


Fig. 1: Plain chest x-ray at fourth day of admission shows air bronchograms, consolidation in the right upper lung fields, right-sided pleural fluid, and multiple pneumatoceles in the right paracardiac and peripheral lung fields.



(a) (b)

Figs. 2a, b: Plain x-ray (a) and computed tomography (b) of the chest at the end of the fifth week of treatment and before needle aspiration show multiple large pneumatoceles in the right middle and lower lung fields, and compressive atelectasia at the medial sites of the pneumatoceles.

At the end of the five-week therapy, 18 Gauge intracat needles were inserted into the pneumatoceles by fluoroscopic guidance and the air in the pneumatoceles was aspirated by syringes, because respiratory difficulty persisted. Decompression via needles resulted in improved respiration, pulmonary functions and blood gasses. Immediately after the drainage, plain chest x-ray showed disappearance of pneumatoceles and reexpansion of the right lung field (Fig. 3).

Fig. 3: Plain chest x-ray after the needle aspirations shows disappearance of the pneumatoceles and reexpansion of the right lung field.

Discussion

Previously immunocompetent nonhospitalized patients may develop community-acquired bacteremia and/or sepsis from extension of local tissue infections, such as pneumonia due to streptococcus pneumoniae and Hemophilus influenzae type b, gastroenteritis due to salmonella, pyelonephritis due to Escherichia coli, etc. Alternatively, colonization and local mucosal invasion by a particularly virulent pathogen in a previously normal host may produce primary bacteremia and sepsis[6]. Occult bacteremia (without an obvious focus of infection) due to streptococcus pneumonia occurs in relatively well-appearing children between three and 24 month of age. The increased incidence of bacteremia among three to 24-month-old children may be due in part to a maturational immune deficiency in the production of opsonic IgG antibodies to the polysaccharide antigens present on these encapsulated bacteria[7].

Severe pneumococcal septicemia is extremely rare in healthy children older than 24 months[2, 6]. However, pneumococcal septicemia occurs in children with sickle cell anemia, asplenia, agammaglobulinemia, AIDS, etc.[2, 7-9]. This six-year-old case was previously healthy, and he doses not have immunodeficiency, splenic dysfunction, sickle cell disease, and/or local tissue infection.

Pulmonary pneumatoceles are uncommon but generally benign, thin-walled parenchymal air collections arising in association with acute pneumonia[10]. Although pneuomatoceles can be seen in the course of staphylococcal pneumonia, they also occur in pneumonia caused by other bacteria such as Hemophilus influenzae, streptococci, Klebsiella pneumoniae, Pseudomonas aeruginosa, Escherichia coli, etc[11]. Pneumatocele formation is unusual in pneumococcal pneumonia, there are only a few reports present in the literature demonstrating pneumatoceles following pneumococcal pneumonia[3-5]. The present case developed pneumatoceles following pneumococcal pneumonia.

The majority of pneumatoceles are self-limited and disappear spontaneously within a few days or months. Rarely, they may attain such size as to severely affect respiration and require treatment[10]. In the present case, the pneumatoceles were aspirated via needle under fluoroscopic guidance. Complete resolution of pneumatoceles and recovery of a normal respiratory pattern were achieved.

We conclude that streptococcus pneumonia may cause pneumatocele formation, albeit rarely, and that giant pneumatoceles may be easily treated by needle aspiration.

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