Necrotizing pneumonia in a young girl: a case report and literature review

Sou-Chi SIT
Department of Paediatrics, Princess Margaret Hospital, Hong Kong

Introduction

Pneumonia is a common reason for hospitalisation in paediatric population. Complications resulting from bacterial pneumonia have significantly reduced with the use of appropriate antibiotics. Unfortunately, severe complications including necrotizing pneumonia, lung abscess, pneumatocele, bronchopulmonary fistula and empyema do occasionally occur. Despite being widely reported in the adult literature, necrotizing pneumonia has been rarely described in children. Early diagnosis of this condition affects the intensity of patient monitoring, options and length of treatment.

Case Report

A 3-year-old girl, HTK, presented with 1 week history of cough and fever. She was diagnosed to have upper respiratory infection and given a course of azithromycin by the general practitioner. However, her fever persisted and she was subsequently admitted to our hospital for further management. Upon presentation, she was lethargic and dehydrated. Her chest examination showed that she was tachypneic with mild subcostal insucking. Bronchial breath sound was detected on the right chest and it was diminished. There was crepitation noted over the same region. Her oxygen saturation was only 91% in room air. Her heart rate was 145 per minutes and her blood pressure was low with the reading of 70/45 mmHg. There was metabolic acidosis with pH of 7.26 and base deficit of 9.3 mmol/L. The haemoglobin and platelet count was 105 x 10^9/L. The haemoglobin was 9.0 x 10^9/L and platelet count was 105 x 10^9/L. No abnormality was noticed on the blood smear. The renal function was impaired with creatinine and urea level of 230 umol/L and 19.5 mmol/L respectively. The serum sodium level was 131 mmol/L and potassium level was 3.3 mmol/L. There was metabolic acidosis with pH of 7.26 and base deficit of 9.3 mmol/L. The C reactive protein was raised to 368 mg/L. Chest drain was inserted on next day after admission. There was 340 ml yellowish fluid coming out from the drain on the first day. Analysis of the pleural fluid demonstrated lactate dehydrogenase of 12880 U/L, total protein of 41 g/L and glucose of 0.7 mmol/L. The bacterial culture of blood, sputum and pleural fluid all yielded Streptococcus pneumoniae which was intermediately resistant to penicillin. Fluid challenge with normal saline was given for several times and her blood pressure stabilised afterwards. She was treated initially with 200 mg/kg of cefotaxime per day. However, she developed progressive respiratory deterioration on day 2 of admission and was intubated. Her blood pressure also dropped. Dopamine and dobutamine infusion were started to maintain her haemodynamic status. Vancomycin was administered for additional coverage. CT scan of thorax on day 4 of admission (Figures 1 & 2) showed right upper and lower lobe consolidation and right pleural effusion. Her vital signs gradually improved over the next few days. The urine output was all along within normal range and her renal function test results returned to normal on day 3 of admission. The inotropes were successfully tailed off on day 6 of admission and she was weaned off from mechanical ventilation on the same day. The haemoglobin and platelet count normalised after packed cell was transfused for one time. However, there was still persistent fever on day 10 of admission despite the antibiotic therapies and chest drainage. CT scan was repeated on day 11 of admission (Figures 3 & 4). It demonstrated consolidation of right upper and lower lobe, multiple cavities without fluid collection or contrast enhancement, right pleural effusion and loculated right pneumothorax. The radiological features were suggestive of necrotizing pneumonia. She was transferred to the cardiothoracic unit with thoracotomy and decortication performed on day 14 of admission. The patient defervesced 1 day after the operation. Two weeks course of vancomycin and 3 weeks of cefotaxime were completed. Her recovery was uneventful and she was discharged on day 27 of admission. A repeat CT scan 2 months later revealed some degree of fibrosis and pleural thickening over the posterior segment of right lobe.

Discussion

Necrotizing pneumonia (NP) is a suppurative lung parenchymal complication of invasive pneumonia. It is

Email: sitsc@ha.org.hk
NP was defined as multiple small cavitations without enhancing margins within a necrotizing lung parenchyma. With more frequent use of CT scan of the chest, there have been increasing numbers of reports published on this condition. NP is generally regarded as a rare disease in childhood. A recent study reported that cavitatory pneumonia was present in twenty percent of all patients with empyema in their series. Necrosis of the lung parenchyma is caused by thrombotic occlusion of alveolar capillaries secondary to inflammation resulting in ischaemia. It is still unknown if the development of NP is related to inadequate therapy, invasive pathogen or exaggerated response of the host. Hacimustafaoglu et al suggested that hyperreactive inflammation of the host seemed to be a more likely explanation as NP patients in their series ran a longer clinical course despite optimal antibiotic therapies and negative results on repeated bacterial culture during the latter part of their illnesses. Immunodeficiency was not found to be a risk factor for NP in a recent series.

NP was well known to be associated with Staphylococcus aureus in the past. In recent years, Streptococcal pneumoniae is more frequently being identified as the pathogen of NP. In fact, Tan et al reported that there has been an increasing trend of complicated pneumococcal disease from 22.6% in 1994 to 53% in 1999 in their country. Locally, Streptococcus pneumoniae accounts for 50% of all 10 cases of NP in our center from 2001 to 2006 (unpublished data). Other microorganisms such as Haemophilus influenza, Mycoplasma pneumoniae and Streptococcus pyogenes have been reported to be the causative agents of NP. In adult, there is an established association between mixed anaerobic infection and NP. However, either mixed anaerobic infection or aspiration occurs only sporadically in paediatric cases.

Patients with NP typically present with pneumonia with or without pleural effusion which is not responded to adequate antibiotic therapy and closed chest tube...
The mean age of paediatric NP patients in the clinical series is between 2 to 5 years. In the present case, her pneumonia failed to show any response to a course of azithromycin and was complicated with empyema. Our case gives a good illusion of the progression of invasive pneumonia. Her first CT scan revealed only consolidation and pleural effusion. However, there were multiple cavitations, loculated pneumothorax identified in the second set of CT scan 1 week later. The loculated pneumothorax is probably due to the presence of bronchopleural fistula. Expansion of NP to periphery of the lung necrotizing adjacent pleura is the likely explanation for the development of bronchopleural fistula. In Hacimustafaoglu et al's series, bronchopleural fistula occurred in 63% of patients with NP and 80% of them required surgical treatment.

CT scan remains the golden standard for diagnosing NP. Chest radiograph is less sensitive than CT in detecting NP as consolidation or effusion may obscure the presence of multiple small lucencies. Only 5 out of 23 cases and 41% of NP cases detected by CT scan were demonstrated on chest X-ray in 2 studies. Hodina et al noticed a delay varying from 5 to 9 days between identification of NP by CT and visualisation by chest radiograph in their series. The diagnostic CT findings for NP include loss of normal parenchymal architecture, decreased parenchymal enhancement and presence of multiple thin-walled cavitary lesions. The decreased enhancement seen on contrast CT can be explained by the mechanism of NP that involves occlusion of the alveolar capillaries and ischaemia.

The mainstay of treatment for NP is prolonged course of antibiotic therapy. The recommended duration of therapy is 4 weeks or 1 week after resolution of fever. Wong et al advocated early use of appropriate antibiotics and closed tube drainage in NP patients without pleural complications. They claimed that this strategy of treatment was as effective as surgery. However, surgical intervention becomes necessary when there is presence of indications including bronchopleural fistula with large pneumothorax, lung entrapment, effusion not responding to simple chest tube drainage, extensive cavitary necrosis. The rate of surgery in the paediatric studies of NP ranges from 22% to 66%. All patients underwent thoracotomy for decortication, bronchopleural fistula repair or segmentectomy/lobectomy/pneumectomy. The present case had a stormy clinical course initially with respiratory failure and septic shock requiring inotropic and ventilatory support. Her disease process was not controlled with antibiotics and chest tube drainage and progressed to NP in several days time. Thoracotomy and decortication were performed to control the disease. Fortunately, she made a complete recovery from this dramatic illness.

In summary, NP is a serious complication of invasive pneumonia. This possibility should be considered when patient with complicated pneumonia fails to respond to optimal antibiotic therapy and closed chest tube drainage. CT scan is the standard diagnostic tool for NP and should be obtained without delay once this diagnosis is suspected. CT scan also plays an important role in the decision making for surgery because of the clearly definable findings. About half of the patients with NP can be successfully treated with medical therapy while the remaining patients require surgical intervention. Early diagnosis of this condition is crucial since it will influence the length and modalities of treatment.

References