Infected chylothorax caused by Streptococcus agalactiae: A Case Report


Abstract: Chylothorax is bacteriostatic in nature. Bacterial infection rarely develops in chylothorax and has never been reported in a non-immunocompromised host. A 33-year-old woman was admitted to National Taiwan University Hospital because of fever and right pleuritic pain. Chest roentgenography and computed tomography revealed right pleural effusion. Examination of the pleural effusion revealed a profile compatible with empyema and chylothorax. Culture of the pleural effusion yielded Streptococcus agalactiae. The woman was not immunocompromised. This is the first report of infected chylothorax caused by Streptococcus agalactiae in a non-immunocompromised host.

Case Report

A previously well 33-year-old woman was admitted to National Taiwan University Hospital because of fever, chills, and right pleuritic pain that had developed on the previous day. She had suffered a contusion on the right side of the chest 10 years before this admission, but her medical history was otherwise unremarkable. At the time of admission, her body temperature was 38.2°C. Auscultation of the chest revealed decreased breath sounds over the right hemithorax and a systolic heart murmur over the apical area. No cervical lymphadenopathy or breast lesion was found. The hemogram revealed leukocytosis (total white blood cell count 11,120 x10⁹/L) with 94.7% neutrophils and 3.7% lymphocytes. Radiography and computed tomography of the chest showed loculated pleural effusion in the right side of the thorax (Figure). The pleural effusion had a milky appearance. The total white blood cell count of the pleural effusion was 11,300 x10⁹/L, with 96% neutrophils and 4% lymphocytes. Biochemical

Figure. Computed tomography of the chest reveals loculated pleural effusion over the right side of the chest.

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studies of the pleural effusion yielded the following data: total protein, 43 g/L (normal, 65 g/L); lactate dehydrogenase, 847 U/L (320 U/L); glucose, 0.56 mmol/L (5.44 mmol/L); triglyceride, 1.47 mmol/L (0.63 mmol/L); and cholesterol, 2.79 mmol/L (3.21 mmol/L). Bacterial culture of the pleural effusion sample taken on the day of admission yielded \(S. agalactiae\) 2 days later, but the culture of the blood sample collected at the same time showed no growth. Infected chylothorax with empyema formation due to \(S. agalactiae\) was diagnosed.

The minimum inhibitory concentration (MIC) of penicillin for the isolate was 0.023 g/mL by the E-test (AB Biodisk, Solna, Sweden). Intravenous penicillin G (3,000,000 units every 4 hours) and gentamicin (60 mg every 8 hr) were administered; a chest tube was inserted for drainage; and a medium-chain triglyceride diet was given. Technetium-99m (Tc-99m) lymphoscintigraphy revealed extravasations of the thoracic duct into the right pleural cavity.

Because of persistent fever and chyle loss, surgery was performed to ligate the thoracic duct and to decorticate the affected area on the 15th day of hospitalization. Two days after the operation, the fever subsided and the chyle loss stopped. Transthoracic echocardiography revealed vegetation over the anterior mitral leaflet. The results of gastrointestinal and genitourinary tract examinations, including abdominal and pelvic computed tomography, were unremarkable. The patient was treated with penicillin G for 6 weeks and gentamicin for 2 weeks, and remained well 2 years after discharge.

**Discussion**

The case reported here differs from the only other reported case of infected chylothorax in several important respects [6]. First, this is the first description of infected chylothorax due to \(S. agalactiae\). Second, our patient did not have preceding pneumonia, any history of penetrating injury of the chest, or underlying immunosuppressive status. Third, the association of \(S. agalactiae\) infection with possible \(S. agalactiae\) infective endocarditis is also unique.

The etiology of chylothorax in our patient is unclear. She had suffered chest trauma 10 years previously and might have developed chylothorax as a result. However, she had experienced no discomfort and no chest radiographs had been taken since the trauma.

The portal of entry of \(S. agalactiae\) into the pleural cavity in this patient remains obscure. This organism usually colonizes the gastrointestinal and genitourinary tracts, but is rarely found in the airway [7]. According to Duke's criteria for infective endocarditis, our patient may also have had infective endocarditis due to \(S. agalactiae\) [8]. Although \(S. agalactiae\) is considered an uncommon pathogen of infective endocarditis in non-pregnant adults [9–12], about 6% of non-pregnant adults with \(S. agalactiae\) infection present with infective endocarditis [12]. In our patient, the organism might have invaded the bloodstream from the diseased gastrointestinal or genitourinary tract to cause endocarditis, although no obvious abnormalities in these sites could be found. Interestingly, large systemic emboli have been reported to occur in at least 40% of patients with \(S. agalactiae\) endocarditis [9], which suggests that \(S. agalactiae\) might have directly inoculated the preexisting chylothorax via systemic emboli in our patient.

In conclusion, this report describes the first case of infected chylothorax due to \(S. agalactiae\) in an immunocompetent host. The patient was successfully treated with intravenous antibiotics, surgical intervention, and diet control. This report emphasizes the need for awareness of the possibility that chyle may be subject to bacterial infection.

**References**