ADDS

Dense packed fungal hyphae arranged in lamellae, morphologically resembling Aspergillus species (Fig 2). No evidence of fungal invasion into adjacent lung tissue was found.

DISCUSSION

Recurrent infections are common complications of ILS, and often are the first manifestations of this anomaly. They are usually caused by common bacteria, although tuberculosis and nocardiosis have been implicated occasionally. Most infections in ILS can be explained either by the presence of collateral ventilation between the airspaces of the ILS and adjacent lung parenchyma or by bloodstream seeding during bacteremia. Fungal mycetomas complicating ILS, as occurred in our patient, have been reported only rarely, and are more difficult to explain. Mycetomas are aggregates of fungal hyphae, most often Aspergillus species, occurring within previously abnormal, usually cystic areas of lung. The airways are thought to be the source of infection, since Aspergillus commonly resides in these structures even in normal individuals. The occurrence of a fungal mycetoma within an ILS indicates that the ILS must have some connection with the tracheobronchial tree, and in fact, such communications have been documented by bronchoigraphy in a few cases. Their presence supports Stocker's theory that ILSs are acquired lesions related to chronic infection, rather than being congenital abnormalities related to abnormal development of the lung bud (in which case connection with the airways would be absent). According to this theory, the systemic arterial supply of an ILS results from hypertrophy of normally occurring arteries in the inferior pulmonary ligament, and residual communication with the bronchial tree could occur depending on the extent and chronicity of the infection.

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Adult respiratory distress syndrome (ARDS) was described in 1971 by Petty and Ashbaugh. Since that time it has been reported in association with many disease entities. We report a case in which a patient with postanginal sepsis, also known as Lemierre's syndrome, had development of ARDS.

ARDS = adult respiratory distress syndrome; PEEP = positive end-expiratory pressure

Postanginal sepsis is characterized by persistent pharyngitis, toxic appearance, Pseudobacterium necrophorum bacteremia, empyema, pneumatoceles, septic arthritis, negative pharyngeal cultures for group A Streptococcus, and other distant sites of infection. First described by Schottmuller in 1918, it was best characterized by Lemierre in 1936. In the preantibiotic era, the mortality rate was 90 percent; a recent review by Moreno et al indicates a 12 percent mortality rate in the modern era. To our knowledge, adult respiratory distress syndrome (ARDS) has not been reported previously as a complication of postanginal sepsis.

CASE REPORT

A 19-year-old man with no medical history presented to our institution with 1-week duration of malaise, fever, sore throat, and 2 days of trismus. He was noted to be febrile to 38.6°C, have anterior cervical adenopathy; and an exudative pharyngitis; otherwise, results of his examination were unremarkable. His white blood cell count was elevated to 16.4 × 10⁹/l and his monospot was negative. He was believed to have bacterial pharyngitis and was treated with oral erythromycin and discharged from the hospital. Over the next 2 days, his clinical condition deteriorated, with left

Adult respiratory distress syndrome as a complication of postanginal sepsis

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ARD as Complication of Postanginal Sepsis (Cosgrove, Colodny, Pesce)

Figure 1. Computed tomographic scan of the neck demonstrating right parapharyngeal collection (arrow).
The cases his tenuous thorax support adequate expiratory sepsis not surgery.

FIGURE 2. Anteroposterior chest roentgenogram demonstrating ARDS and pneumatocele formation.

shoulder pain, tachypnea, tachycardia, fever to 40°C, persistent pharyngeal pain, and prostration; he was admitted to the hospital.

His initial course was stormy. The patient experienced worsening dyspnea. A chest roentgenogram at that time revealed increasing infiltrate and pseudotumor. On the third hospital day, the diagnosis of postanginal sepsis was made; this was confirmed by F. necrophorum bacteremia and computed tomographic (CT) scan of the neck (Fig 1). Operative drainage of a right parapharyngeal collection was accomplished as was drainage of a right empyema by tube thoracostomy. Appropriate antimicrobial therapy was initiated prior to surgery.

In the immediate postoperative period, ventilatory support could not be withdrawn due to persistent hypoxemia. Positive end-expiratory pressures (PEEP) of 10 to 12 cm H₂O and oxygen concentrations (FIO₂) of 50 to 100 percent were required to maintain adequate oxygenation. Neither central venous pressure nor pulmonary capillary wedge pressures were available, but there was no jugular venous distention, third heart sound, or peripheral edema. Chest roentgenogram revealed diffuse alveolar infiltration (Fig 2). The roentgenographic appearance combined with hypoxia despite high PEEP and FIO₂ and the lack of evidence for heart failure support the diagnosis of ARDS. Concomitantly he had development of thrombocytopenia, left shoulder septic arthritis, and left pneumothorax requiring chest tube expansion. His condition remained tenuous for 6 postoperative days; then over the next several days, his oxygenation improved, and ventilatory support could be weaned and terminated.

DISCUSSION

This case demonstrates the presentation and severity of postanginal sepsis, including ARDS. Others have described patients with septic arthritis, metastatic abscess formation, septic thoracoplebitis, and lung involvement—including septic pulmonary emboli, pleural effusions, pneumatoceles, and pyopneumomothoraces.1,4 Henry et al4 noted three deaths out of the 26 patients with lung involvement, but they did not specify the cause of death.4 Moreno et al3 reviewed 11 cases and found that two patients died of pulmonary complications. One patient died of complications following bronchoscopy and the other died of respiratory distress that was not characterized further.1 It is unknown whether these patients had ARDS associated with their illnesses.

In this report we describe a patient with postanginal sepsis complicated by ARDS. This complication needs to be considered when treating a patient with this disease process in order to plan diagnostic strategies and initiate therapeutic options in a timely manner, so as to further decrease the mortality of this syndrome.

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Flow Velocity Paradoxus and Pulsus Paradoxus in Obstructive Sleep Apnea Syndrome

Toshiaki Shiomi, M.D.; Christian Guilleminault, M.D.; Masato Maekawa, M.D.; Akira Nakamura, M.D.; and Kazumi Yamada, M.D.

Echo-Doppler monitoring was performed simultaneously with two-dimensional and M-mode echocardiography, polyvannography and blood pressure recording in an obstructive sleep apnea patient. Increase in tricuspid flow and decrease of mitral flow velocity was demonstrated during each diastole prior to pulsu paradoxus, while aortic flow velocity decreased with pulsus paradoxus during obstructed breathing during sleep.

(Chest 1993; 103:1629-31)

Pes = esophageal pressure

In a previous presentation, we reported the development of a significant leftward shift of the interventricular septum associated with obstructive sleep apnea or hypopnea.1 This observation was made using two-dimensional and M-mode echocardiography during sleep. We have indicated that this abnormal shift can also be seen in children and teenagers who develop abnormally negative peak inspiratory esophageal pressure (Pes) during heavy snoring, with the development of pulsus paradoxus.2

We recently had the opportunity to pursue an investigation of the mechanical effects of increased Pes nadir with sleep apnea or hypopnea in a 15-year-old boy who presented with

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