Empyema due to Ventriculopleural Shunt

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Empyema developing seven weeks after craniotomy in a 62-year-old black woman with an ipsilateral ventriculopleural (V-PL) shunt is described. Infection of the pleural space presumably resulted from transfer of organisms from a proximal V-PL shunt infection to the thorax. Empyema resulting from V-PL shunt infection has not previously been reported. Pleural effusions in patients with V-PL shunts must be considered as a potential site of infection with possible development of empyema.

(Chest 1991; 99:1538-39)

V-PL = ventriculopleural

Ventriculopleural shunting was introduced by Ransohoff for the treatment of hydrocephalus. In this early report, significant pleural effusions were not problematic, presumably due to the absorptive capacity of the pleural surfaces; however, later studies documented the frequent occurrence of pleural effusions, particularly in children. Antisiphon devices, as well as the inclusion of valves to ensure more regular CSF flow, were designed to enable more reliable absorption of CSF. These devices have resulted in significant decreases in the incidence of large pleural effusions complicating V-PL shunts. While infection of the V-PL shunt following such procedures is a known complication, occurring in 11 of 59 cases in a recent report, subsequent infection of the pleural fluid with development of empyema has not been described. We present a case of empyema which developed ipsilateral to an infected V-PL shunt.

Case Report

A 62-year-old black woman presented with altered mental status and fever of one week's duration. She denied any cough, sputum production, or pleuritic pain.

Nine years prior to admission, decreased right-sided auditory acuity led to the discovery of a cerebellar pontine angle tumor. Further evaluation was refused until four years prior to admission, when progression of symptoms occurred. Tumor enlargement was noted, which was believed to be consistent with an acoustic neuroma. A craniotomy was performed, with subtotal resection of tumor, because of the patient's desire to spare facial nerve function. After surgery, obstructive hydrocephalus developed, requiring V-PL shunting to the left hemithorax. Seven weeks prior to the current hospitalization, tumor recurrence was noted, and a second craniotomy was performed, again with subtotal excision of tumor.

On admission the patient was found to be in moderate respiratory distress, with a respiratory rate of 28/min and a temperature of 39.4°C (103°F). She was confused and disoriented. Decreased breath sounds were noted in the left hemithorax, with dullness to percussion. The findings from the remainder of the physical examination were unremarkable.

Laboratory examination revealed a white blood cell count of 33,600/cu mm, with 86 percent PMNs and 12 percent band cells. The hemoglobin level was 13 g/dl, and the platelet count was 450,000/cu mm. Findings from blood chemistries and urinalysis were within normal limits. Arterial blood gas analysis on room air...
revealed a pH of 7.51, PaCO₂ of 35 mm Hg, PaO₂ of 61 mm Hg, and 90 percent saturation. A chest x-ray film (Fig 1) demonstrated a V-PL shunt catheter in the left pleural space, with both free and loculated fluid. Pleural fluid revealed purulent material with a white blood cell count of 110,000/cu mm, with 78 percent PMNs, glucose level of 7 mg/dl, pH <7.0, LDH level of 7,600 units/L, and Gram-positive cocci in pairs and Gram-positive rods seen on Gram stain. Tube thoracostomy was performed, with removal of 1,800 ml of purulent material; and antimicrobial therapy with ceftriaxone and vancomycin was begun. No evidence of underlying pulmonary parenchymal infiltrates was noted. Culture of pleural fluid revealed Staphylococcus epidermidis and Streptococcus mitis. Thirty-six hours after the institution of antibiotic therapy, the patient was brought to the operating room for exteriorization of the proximal end and removal of the distal end of the V-PL shunt. A sample of turbid CSF with a white blood cell count of 490/cu mm was obtained at that time. Cultures of CSF, blood, and urine remained negative. The patient's condition improved after several days of pleural drainage and three weeks of ceftriaxone therapy. A small amount of loculated pleural fluid persisted until discharge.

DISCUSSION

The present report describes the development of empyma due to Staph epidermidis and Strept mitis ipsilateral to a V-PL shunt, presenting with altered mental status, fever, and leukocytosis. Pleocytosis of CSF was observed, although cultures were negative, presumably because this specimen was obtained after administration of antimicrobials. The absence of roentgenographically demonstrable pulmonary parenchymal infiltrates suggests that the pleural infection was not secondary to pneumonia. Furthermore, the organisms present on culture are among those frequently reported to cause V-PL shunt infections and are not often a primary cause of pneumonia. These factors suggest that the shunt catheter was the most likely source of pleural infection.

Ventriculopleural shunts are conduits from the ventricles to the pleural space. In particular, transfer of glioblastoma cells to the pleural space via a V-PL shunt has been reported in two patients. It would therefore seem reasonable that infection originating proximally in a V-PL shunt could also be transferred to the pleural space, producing empyma by direct delivery of organisms via the shunt catheter. To our knowledge, infection of pleural effusions resulting from V-PL shunts causing frank empyma has not been previously reported.

As shunt infection is a known complication of this procedure, a high index of suspicion must be maintained for infection in both the CSF and any associated pleural effusion when these patients present with fevers without another obvious source. Early sampling of the CSF and pleural fluid will direct prompt institution of antimicrobial therapy and shunt exteriorization, which may prevent the development of frank empyma.

REFERENCES


Chylothorax After Childbirth*

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We report a case of chylothorax which appeared in a mother after childbirth. Disruption of the thoracic duct occurred with the high intrathoracic pressures generated by the Valsalva maneuver used by the patient during labor to "push." No evidence of other trauma or malignancy were found and the patient did well after use of total parenteral nutrition, thoracotomy with thoracic duct ligation, and pleurodesis.

CASE REPORT

The patient was a 20-year-old nulliparous white woman who went into labor at the end of an uneventful pregnancy. More than three hours were spent in the second stage with the fetal head remaining at 1+ station despite repeated pushing by the mother. Variable deceleration of the fetal heart rate and meconium staining of amniotic fluid were noted, necessitating a low transverse cesarean section without complications. A chest roentgenogram after delivery documented a right pleural effusion (Fig 1) but no other diagnostic work-up was pursued. The patient was discharged home without further complications.

Three weeks after the cesarean section, the patient noted a dry cough and dyspnea. Chest roentgenogram demonstrated opacification of the right hemithorax. Thoracentesis yielded 3 L of milky fluid, which was found to be chylous with a protein level of 3.8 g/dl, triglycerides, 2,730 mg/dl, and large chylomicron band of 40 percent on lipoprotein electrophoresis. The fluid white blood cell count was 6,015/cu mm with 97 percent monocytes and 3 percent neutrophils. Pleural fluid glucose value was 96 mg/dl with an LDH level of 197 IU/L. Her dyspnea resolved, but three days later, she was admitted for recurrent effusion. Except for her cesarean section, the patient had no significant past medical history, no history of trauma, and no constitutional complaints. Physical examination revealed a healthy appearing female at 60.0 kg (132 lbs). There was no lymphadenopathy on examination and lung findings were consistent with a right pleural effusion. Admitting laboratory data included a normal white blood cell count, serum total protein value of 6.4 g/dl; albumin, 3.4 g/dl; and cholesterol, 184 mg/dl. A PPD was not reactive and cultures of pleural fluid were negative for Mycobacteria and bacteria.

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CHEST / 99 / 6 / JUNE, 1991