Laceration of the Left Pulmonary Artery during Removal of a Bronchogenic Cyst by Right Thoracotomy*

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Major vascular involvement by a bronchogenic cyst is rare; most large cysts cause respiratory symptoms. We present a case in which a large bronchogenic cyst was adherent to the left pulmonary artery. During the removal of the cyst by right thoracotomy, the artery lacerated. Repair of the artery in such a situation taxes the surgeon’s ingenuity and decision-making process. (Chest 1991; 100:267-68)

Laceration of the left pulmonary artery during excision of a bronchogenic cyst by right thoracotomy presents a formidable surgical challenge. Inflow occlusion of both venae cavae provides a bloodless field but only allows a short time for repair of the pulmonary artery. To the best of our knowledge, this is the first reported case where this particular set of circumstances occurred.

CASE REPORT

A 32-year-old patient with chronic renal failure who was receiving hemodialysis was hospitalized in December 1989 with the chief complaints of chest pain, cough, and some difficulty in breathing. He was known to have a “cyst of the lung” noted on chest roentgenogram two years ago for which surgery was recommended but refused. Routine chest roentgenogram as compared with the previous one showed considerable enlargement of the cystic mass. This was confirmed with a computed tomographic (CT) scan (Fig 1).

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FIGURE 1. Computed tomography shows large bronchogenic cyst located to right of midline.

Because of symptoms and the increased size of the cyst, surgery was recommended. A right posterolateral thoracotomy was used to approach the cyst that was freed with great difficulty from the carina, right main bronchus, and the esophagus. While attempting to free the cyst from the left lateral aspect structures, brisk hemorrhage ensued that proved to be a laceration of the left pulmonary artery. Since we were in the right side of the chest, even with single lung anesthesia with a double lumen tube that allowed for a complete collapse of the right lung, repair of the pulmonary artery in the depths of the mediastinum was not feasible. With digital compression of the laceration controlling the hemorrhage, the pericardium was widely opened anterior to the right phrenic nerve and encircling loops were placed around both venae cavae. After inflow occlusion, the operative field was sufficiently dry to allow for repair of the left pulmonary artery by continuous polypropylene suture. The inflow occlusion time was approximately 1½ minutes. The cyst was removed piecemeal and a remaining piece of the wall had its lining ablated by electrocautery. The patient had an uneventful postoperative course.

DISCUSSION

Most of the symptoms of bronchogenic cysts are due to compression and irritation of the airway.1 Although compression of vascular structures has been previously described,2,3 to the best of our knowledge, a lacerated left pulmonary artery during removal of a bronchogenic cyst by right thoracotomy has not been reported.

There are several lessons to be learned from this case: (1) Bronchogenic cysts in an adult are liable to be adherent to neighboring structures because of intense inflammatory reaction. (2) Total removal may be hazardous and one should opt for partial excision and ablation of the lining by electrocautery. (3) If CT shows adherence to the left pulmonary artery, one should remember that the artery is anterior to the left main bronchus and hence, quite inaccessible by right thoracotomy when the subcarinal location makes such an incision appropriate (4) One-lung ventilation provides ideal exposure by collapse of the right lung. (5) Tear of the left pulmonary artery is a formidable problem. Should this occur, the surgeon should immediately proceed to inflow occlusion by snaring both venae cavae after generous pericardiotomy.

REFERENCES


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Overwhelming Necrotizing Tracheobronchitis due to Inadequate Humidification during High-Frequency Jet Ventilation*

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Necrotizing tracheobronchitis (NT) associated with conventional mechanical ventilation or high-frequency jet ventilation (HFJV) is a lesion reported most often in neonates. In most cases, a specific cause is not identified. We describe a case of NT in an adult that occurred during HFJV and was attributable to inadequate humidification.

(Chest 1991; 100:268-69)

HFO = high-frequency oscillation; HFJV = high-frequency jet ventilation; BPF = bronchopleural fistula; iPEEP = intrinsic positive end-expiratory pressure; NT = necrotizing tracheobronchitis; DP = driving pressure; psig = pounds per square inch gauge; IT = inspiratory time; CMV = conventional mechanical ventilation

High frequency jet ventilation (HFJV) is an alternate mode of ventilatory support for patients with bronchopleural fistulae (BPF) or for use in the operating room for bronchoscopy or thoracic surgery. Complications of HFJV include pneumothorax, pneumomediastinum, subcutaneous emphysema, intrinsic positive end-expiratory pressure (iPEEP), and depression of cardiac output. Necrotizing tracheobronchitis (NT) is another complication of HFJV that has been observed primarily in neonates. The etiology of NT has not been fully elucidated, but NT may be due to high operating pressures or flows, inadequate humidification, excessive airway temperature, or ischemia.1 We report a case of NT that was directly attributable to inadequate humidification.

CASE REPORT

A 41-year-old woman was admitted to an outside hospital with a two-week history of fever, myalgia, dyspnea, and cough. A chest radiograph revealed diffuse bilateral alveolar infiltrates. Because of severe hypoxemia, her trachea was intubated and mechanical ventilation with PEEP was begun. A sputum Gram stain revealed Gram-positive diplococci and Streptococcus pneumoniae subsequently grew from a culture. Cefuroxime therapy was started, but when there was no improvement in the patient’s condition at 48 hours, her antibiotic regimen was changed to imipenem/cilastatin, gentamicin, and erythromycin. Her clinical course was complicated by the need for high inspired oxygen concentrations and high PEEP. Bilateral pneumothoraces, pneumoperitoneum, and a BPF developed. Heparin therapy was begun for deep vein thrombophlebitis. Subsequent cultures were negative except for Candida albicans in the urine. Dobutamine and dopamine were intermittently required for hypotension.

On the 20th hospital day, she was transferred to our institution. Breathing 100 percent oxygen at a rate of 10 breaths per minute, tidal volume of 700 ml, and PEEP of 14 cm H₂O, her arterial blood gas values were as follows: pH, 7.48; PaO₂, 35 mm Hg; PaCO₂, 56 mm Hg, with a calculated saturation of 91 percent. Her static lung/ chest wall compliance was 15 ml/cm H₂O. After obtaining informed consent, HFJV (APT1010, Model 5, Advanced Pulmonary Technologies, Glastonbury, CT) was begun with a driving pressure (DP) of 33 pounds per square inch gauge (psig), inspiratory time (IT) of 36 percent, frequency of five breaths per second (Hz), and inspired oxygen concentration (FIO₂) of 1.0. Forty-eight hours later, while receiving dobutamine 5 μg/kg-min and ventilated with a DP of 35 psig, FIO₂ of 0.52, IT of 40 percent, and frequency of 5.2 Hz, her arterial blood gas values were as follows: pH, 7.39; PaO₂, 33 mm Hg; PaCO₂, 70 mm Hg, with a calculated saturation of 90 percent. On this same day, the nurse discovered the endotracheal tube temperature had decreased from >36.6°C to 30 to 32.7°C for 3 h. We found the bias flow humidifier (Inspiron Vapophase Plus, model 0096300, Intertech Resources, Inc, Bannockburn, IL) had shut off because the servo-temperature probe, which shuts the humidifier down when the airway circuit temperature exceeds a specified limit, was inserted in the wrong portion of the circuit. Thus, the patient had been ventilated for approximately 3 h with cold, dry gas. Two days after this incident, the nurses started to suction thick plugs from the endotracheal tube. Refractory hypercarbia and hypoxemia developed and the patient died five days later.

Postmortem examination demonstrated severe organizing diffuse alveolar damage (adult respiratory distress syndrome [ARDS]) and NT that began just distal to the cuff of the endotracheal tube and extended into all major bronchi with obliteration of the airways by

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