FIGURE 2. Posteroanterior chest x-ray film, obtained during lymphangiography four days after patient's admission with no evidence of extravasation of contrast medium. Produced marked improvement in the patient's dyspnea. Analysis of the fluid revealed the presence of chylomicrons. Triglyceride values were 3.62 gm/100 ml, cholesterol levels were 149 mg/100 ml, total lipid values were 4.25 gm/100 ml, and total protein values were 3.1 gm/100 ml. The Sudan stain for fat was positive. Routine and acid-fast cultures were negative. Bilateral pedal lymphography, performed four days later, demonstrated opacification of multiple, small radicles of the thoracic duct and the duct crossing the midline at the thoracic inlet to terminate in the region of the right subclavian and jugular veins. The termination of the duct was larger than normal. There was no extravasation, however (Fig 2).

The patient was given a medium-chain triglyceride diet to diminish the flow of chyle. She was discharged one week after admission and maintained on this diet for three weeks; she has been totally asymptomatic for six months. Follow-up x-ray films of the chest, obtained two months and six months later, showed normal findings, except for those in the left upper lobe, which were unchanged from the roentgenogram made one and one-half years before admission.

DISCUSSION

Although the site of extravasation was not demonstrated, it is postulated that the sudden violent motion accompanied by a Valsalva's maneuver caused either a rupture of one of the small radicles of the thoracic duct or a leakage from the main thoracic duct. The duct may have been affected by the prior tuberculosis, making it more susceptible to rupture. Thoracocentesis and a medium-chain triglyceride diet were successful conservative management.

In an appropriate clinical situation, the clinician should include chylothorax in the differential diagnosis of pleural effusion. This case demonstrates that chylothorax can be a benign disease not associated with intrathoracic tumor or severe trauma.

REFERENCES


Near-Drowning Presenting as the Adult Respiratory Distress Syndrome*

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We present a patient who was submerged in fresh water resulting in severe hypoxemia and requiring mechanical ventilation on a volume respirator with positive end expiratory pressure. Near-drowning should be considered as a possible cause of the adult respiratory distress syndrome.

Recent reviews concerning the subject of near-drowning, while recognizing the role of aspiration and chemical pneumonitis, have not presented the clinical state resulting from submersion as a possible etiology of the adult respiratory distress syndrome.1,2 We present a patient who suffered submersion in fresh water resulting in severe refractory hypoxemia, as seen in the adult respiratory distress syndrome, requiring mechanical ventilation on a volume respirator with positive end expiratory pressure.

CASE REPORT

A 16-year-old Caucasian boy with no previous major medical or surgical problems was one of a group of young adults in a car when it veered off the road and into a fresh water pond. He was submerged for approximately six minutes. The patient was unconscious after removal from the pond and mouth-to-mouth resuscitation was given. When brought to the emergency room 25 minutes later, he was noted to be in acute respiratory distress. He was cyanotic, dyspneic and tachypneic receiving nasal oxygen at 9 liters per minute. The blood pressure was 140/80 mm Hg, pulse rate 104 per minute and regular, respiratory rate 44 per minute, rectal temperature 37.7778°C. There was no evidence of head injury. There was retraction of the intercostal and supraclavicular muscles. Diffuse rales and wheezes were auscultated over both lung fields. Heart sounds were diminished in intensity, but the heart rate was regular at 104 per minute.

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There was cyanosis of nail beds. Peripheral pulses were barely palpable.

The patient’s initial blood gas determinations on 9 liters of nasal oxygen revealed an arterial oxygen pressure (Pao₂) of 22 mm Hg, arterial carbon dioxide pressure (Paco₂) of 67 mm Hg and a pH of 7.08. Intubation was initiated and ventilation with a volume cycled ventilator (MA I Bennett) was initiated. Immediately following intubation and controlled ventilation on the MA I volume respirator with 100 percent inspired oxygen the Pao₂ rose to 40 mm Hg. The addition of 10 cm of positive end expiratory pressure resulted in a further increase in the Pao₂ to 75 mm Hg on a lower inspired oxygen percentage of 60. A tracheostomy was performed on the second day after admission.

An admission chest roentgenogram disclosed bilateral alveolar infiltrates (Fig 1). Complete blood cell count on admission indicated a hematocrit reading of 52 percent, hemoglobin of 18 gm, 100 ml, leucocyte count of 13,300 cells per mm³ with 67 percent polymorphonuclear cells, 1 percent band, 13 percent lymphocytes and 2 percent monocytes. Other laboratory values on admission were: urea nitrogen (BUN), 17 mg percent; carbon dioxide, 19 mEq/liter; chloride, 102 mEq/liter; sodium, 133 mEq/liter; and potassium 3.3 mEq/liter. Within 24 hours the BUN and electrolyte levels were all within normal limits. Clotting and bleeding parameters were normal and there was no evidence of hemolysis. Initial sputa cultures were negative. However, after four days of high doses of steroids, Escherichia coli was cultured from tracheal secretions. Blood cultures were persistently negative. The electrocardiogram showed sinus tachycardia.

The patient initially required 10 cm of positive end expiratory pressure (PEEP) in order to achieve adequate oxygenation; six hours after admission on an inspired oxygen percentage of 50 with PEEP the arterial Pao₂ was 75 mm Hg but fell to 53 mm Hg without PEEP. Control on PEEP required use of morphine and diazepam (Valium). Hydrocortisone (400 mg) was given in the first 24 hours, with 300 mg administered daily for the next two days to decrease inflammatory reaction secondary to aspiration of pond water and associated particulate matter. The patient received antibiotic therapy, with sodium cephalothin (Keflin) and kanamycin. He was also treated with furosemide (Lasix) to prevent a positive water load while on positive end expiratory pressure. PEEP was required for the first three days to maintain adequate oxygenation with an arterial Pao₂ of over 80 mm Hg, with an inspired oxygen percentage of 40. On the fourth through the sixth days in the hospital, mechanical ventilation, without control on PEEP, on assisted ventilation he achieved a range of arterial Pao₂ of over 80 mm Hg, with an inspired oxygen percentage of 30. On the seventh through the eighth days the patient was weaned off the MA I and received supplemental oxygen through a blow by tubing attached to the tracheostomy tube. The tracheostomy tube was removed on the tenth day. Significant hypoxemia, with an arterial Pao₂ of 60 mm Hg on room air at sea level, persisted through the 12th day, necessitating the use of supplemental oxygen through a nasal cannula. The patient had only mild hypoxemia, with an arterial Pao₂ of 75 mm Hg, on room air on the 15th day and was discharged on the 18th hospital day.

A followup pulmonary function study four months after discharge disclosed normal lung volumes, ventilatory parameters, and rest and exercise blood gas values. A chest roentgenogram taken 13 days after admission still showed residual infiltrate. A followup roentgenogram four months later showed normal findings.

**DISCUSSION**

The more recent literature regarding near-drowning victims contains references to fresh and salt water submersion as a form of aspiration pneumonia resulting in a chemical pneumonitis.³,⁴ Modell et al.¹ have demonstrated that the critical problems in cases of submersion are severe hypoxemia, acidosis, and respiratory insufficiency rather than electrolyte imbalance or hemolysis. They emphasize that emergency therapy should be geared to afford effective ventilation with adequate oxygenation and that the patient should be treated with steroids and antibiotics for aspiration pneumonitis.

The particulate matter in both fresh and salt water coming into contact with the alveolar membrane produces an inflammatory reaction with exudation of protein-rich fluid into the alveoli. The concomitant loss of surfactant results in alveolar instability, the collapse of alveoli on expiration, and subsequently atelectasis with stiff, noncompliant lungs. Positive end expiratory pressure will maintain alveolar patency, increasing the functional residual capacity and the number of alveoli open for gas exchange thereby providing for improved oxygenation.³

Recently it has been pointed out that there has been no major advocacy of the use of positive end expiratory pressure for treatment of near-drowning.⁴ The concept that near-drowning may result in a type of chemical pneumonitis with severe refractory hypoxemia, as seen in the adult respiratory distress syndrome, has important therapeutic implications. Utilization of the therapeutic modalities, including mechanical ventilation with positive end expiratory pressure, outlined for the adult respiratory distress syndrome resulted in a satisfactory outcome in our patient.⁵,⁶

**REFERENCES**

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Complete Heart Block Caused by the Swan-Ganz Catheter

William S. Abernathy, M.D.*

Complete heart block was caused by passage of a Swan-Ganz catheter into the pulmonary artery of a patient with acute myocardial infarction and complete left bundle branch block. This complication could be easily treated with the prophylactic insertion of a temporary pacing catheter.

The Swan-Ganz catheter has been recently introduced for the bedside monitoring of pulmonary artery and pulmonary artery wedge pressures in acutely ill patients. Although Forrester et al reported no serious complications in approximately 200 patients in whom this catheter was used, a number of complications were reported, including: thrombosis on the catheter; intra-cardiac knotting of the catheter; perforation of the pulmonary artery; rupture of the balloon; and ventricular beats induced by the catheter. Passage of the Swan-Ganz catheter in the patient reported here caused complete heart block. Knowledge of this potentially fatal complication is important because it is readily treatable with a pacemaker.

CASE REPORT

A 64-year-old white man was transferred to the cardiac intensive care unit because of cardiac arrest at another hospital. He had progressive angina pectoris for nine months prior to admission, and pneumonia and pulmonary edema developed during the five days before admission.

On examination the blood pressure was 75 mm Hg by palpation, and the skin was cool, clammy, and cyanotic. The neck veins were distended, rales were present throughout the lungs, and a third heart sound was heard at the apex.

An electrocardiogram seven days before admission revealed complete left bundle branch block (QRS = 0.18 second, PR = 0.20 second). On admission, the electrocardiogram showed sinus tachycardia and persistence of the complete left bundle branch block (QRS = 0.18 second, PR = 0.17 second). Determinations of creatine phosphokinase and isograms of lactic acid dehydrogenase were consistent with an acute myocardial infarction.

After initially responding to treatment, the patient developed progressive shock on the third day. As an emergency measure, a Swan-Ganz catheter was passed at the bedside with continuous monitoring of the pressure at the catheter tip. As the catheter reached the pulmonary artery, complete heart block developed (Fig 1). The catheter was immediately withdrawn into the right arm, cardiopulmonary resuscitation was begun, and sinus tachycardia reappeared shortly. The catheter was again advanced into the pulmonary artery with the same sequence of events: complete heart block, resuscitation, then sinus tachycardia. Despite the fact that the catheter was not passed again and despite successful institution of pacing with a bipolar electrode catheter in the right ventricle, multiple cardiac arrests occurred over the next hour, and the patient died. No autopsy was performed.

DISCUSSION

It is well known that cardiac catheterization may produce right bundle branch block and complete heart block. The presence of left bundle branch block, as in this patient, increases the chance of complete heart block resulting from the procedure. It would seem prudent to insert a pacing catheter prior to passage of the Swan-Ganz catheter in patients with left bundle branch block.

REFERENCES