Hydropneumomediastinum and Bilateral Hydropneumothorax as Delayed Complications of Central Venous Catheterization*

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A case of hydropneumomediastinum and bilateral hydropneumothorax from a central venous catheter is described. The complication is related to the position of the catheter after placement through the left internal jugular vein. Proper placement of a central venous line into the superior vena cava is essential to avoid this serious complication. 

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*CVC = central venous catheter; SVC = superior vena cava

Central venous catheters (CVCs) are commonly used in the intensive care unit to administer fluids, medications, and blood products. Central venous catheters are associated with complications occurring at the time of placement, such as pneumothorax and carotid artery puncture,1 as well as delayed complications such as hydrothorax and catheter-related infection.1 We report the findings in a patient who developed a bilateral hydropneumothorax and hydropneumomediastinum, previously unreported delayed complications of central venous catheterization.

CASE REPORT

A 48-year-old white man who was receiving prednisone for Crohn's colitis was admitted to the medical service with an ileus. Parenteral hyperalimentation was initiated through the left internal jugular vein, which was cannulated using the central approach with a single-lumen 16-gauge silicone elastomer (Silastic) catheter and a J-tip guide wire. The catheter was placed by an experienced resident and supervised by an attending physician. Insertion of the catheter was without difficulty, and blood was freely aspirated through the catheter, at which time it was sutured into place. A chest radiograph confirmed placement of the tip of the catheter at the junction of the left brachiocephalic vein and superior vena cava (SVC) (Fig 1), and hyperalimentation through the catheter was initiated.

The patient had no complaints until 7 days after CVC insertion, when he noted substernal chest pain and dyspnea. Physical examination revealed diminished breath sounds and dullness to percussion bilaterally. The patient was hemodynamically stable. A chest radiograph demonstrated a large bilateral hydrothorax without apparent pneumothorax, a widened mediastinum with a suggestion of a rim of mediastinal air, and migration of the tip of the catheter into the mediastinum away from its site of initial placement. A thoracentesis performed from the left pleural space yielded 700 ml of thin milky fluid which appeared to be identical to the patient's hyperalimentation fluid. The hyperalimentation infusion was immediately terminated and the CVC removed. A thoracentesis from the right pleural space yielded 1,000 ml of fluid, again identical in appearance to the hyperalimentation fluid. Both thoracentesis procedures were uncomplicated, and chest radiographs done after each showed smaller bilateral pleural effusion and absence of a pneumothorax. Analysis of the pleural fluid was consistent with the hyperalimentation fluid (glucose level, 500 mg/dl; triglyceride level, 1,900 mg/dl; no erythrocytes, and cultures were sterile. The patient's condition remained stable, and he reported marked symptomatic improvement after the pleural fluid drainage. It is estimated that 10 L of hyperalimentation fluid had been infused through the CVC.

A computed tomographic (CT) scan of the chest was obtained on the day after removal of the CVC, due to a concern that the CVC

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FIGURE 1. Chest radiograph immediately following placement of CVC using left internal jugular venous approach. Note position and angle of tip of catheter (arrow) at junction of left brachiocephalic vein and SVC.

FIGURE 2. CT scan of chest taken 1 day after onset of dyspnea and chest discomfort. Note bilateral effusions (squares), bilateral pneumothorax (arrows labeled 1), pneumomediastinum (arrows labeled 2), and fluid accumulation in mediastinum (arrow labeled 3). Subcutaneous air is also present.
had ruptured through the central veins into the mediastinum. The CT scan showed a bilateral hydropneumothorax, a hydropneumomediatinum, and subcutaneous air (Fig 2). An echocardiogram on the same day as the CT scan revealed only a small posterior pericardial effusion. Subsequent chest radiographs showed complete resolution of the bilateral pleural effusions, mediastinal widening, and mediastinal air. The patient was discharged several days later without sequelae.

**Discussion**

Several delayed complications associated with CVCs have been reported, including cardiac tamponade, catheter-related infection, unilateral and bilateral hydrothorax, and hydromediastinum. To our knowledge, this report describes the first documented case of a bilateral hydropneumothorax and hydropneumomediatinum as delayed complications of a CVC. Although the bilateral pneumothorax was not clearly evident on the chest radiograph taken at the time when the complication became clinically manifest, it was clearly seen on the CT scan, a more sensitive method. The suggestion of mediastinal air on the chest radiograph was confirmed by the CT scan, which demonstrated a hydropneumomediatinum, subcutaneous air, and a bilateral hydropneumothorax (Fig 2).

The events leading to the development of hydrothorax or hydromediastinum (or both) related to CVCs have been speculated upon in previous case reports. Placement of the CVC through the left internal jugular vein is thought to predispose to rupture through the SVC, due to the nearly right angle of the junction of the left brachiocephalic vein and the SVC, which potentially places the tip of the catheter at a nearly perpendicular angle to the SVC. This right angle can cause the tip to create mechanical forces on the wall of the SVC, leading to rupture of the vein. The movements of the patient's head and neck, as well as respiratory and cardiac motion, are thought to contribute to the mechanical forces exerted by the tip of the catheter onto the SVC if the tip lies at or near a right angle. The chest radiograph taken immediately after placement of the catheter (Fig 1) demonstrates the tip of the catheter at a nearly right angle to the SVC. In retrospect, it would have been safer to advance the catheter further into the SVC so that the tip of the catheter would lie parallel to the long axis of the SVC, thus minimizing mechanical forces on the wall of the vein. Rupture of the vein by the tip of the catheter leads to the accumulation of fluid in the mediastinum, resulting in a hydromediastinum. The fluid can then migrate into the pleural space, resulting in a unilateral or bilateral hydrothorax. Although some authors have noted the absence of serious sequelae or hemorrhage from this type of CVC complication, others have described it as life-threatening due to respiratory compromise. It should be noted that the complication of a hydrothorax from a CVC has been reported with insertion into both the right and left central veins.

The type of fluid infused in this case, hyperosmolar hyperalimentation fluid, may cause intimal injury, thus predisposing the tip of the catheter to rupture through the vein. The high flow rate of blood within central veins should normally minimize this risk due to rapid dilution of the infusate; however, abutment of the tip of the catheter at a nearly right angle to the SVC, as occurred in this patient, may not allow for optimal mixing of the infusate, thus concentrating the hyperosmolar solution along the wall of the SVC. The patient was receiving prednisone for his Crohn's colitis. Glucocorticoids are known to lead to vascular fragility, possibly aggravating the endothelial damage of the malpositioned tip of the catheter as it generated mechanical forces on the wall of the SVC, predisposing to rupture. Finally, the stiffness of the material of the CVC is believed to be related to vascular damage. In this case the catheter was composed of relatively flexible silicone elastomer, which is believed to be less likely to cause vascular damage than the stiffer polyethylene catheters; however, silicone elastomer CVCs have been associated with vascular perforation.

Understanding the pathogenesis of the hydropneumomediatinum and bilateral hydropneumothorax described in this case poses a challenge. Clearly, the tip of the catheter ruptured through the central vein sometime after placement of the catheter, allowing the infusate to accumulate in the mediastinum. The pressure generated by the mediastinal fluid accumulation may have caused rupture of the mediastinal pleura, with drainage of the fluid from the mediastinum into one or both pleural spaces. In addition, it has been speculated that congenital or acquired interpleural communications exist between the right and left layers of posterior mediastinal pleura, which could allow dissection of fluid that reached one pleural space directly into the contralateral pleural space without traversing the mediastinum. The presence of air in both pleural spaces and the mediastinum could be caused, for example, by the inadvertent addition of air through the catheter into the mediastinum when the bags of hyperalimentation fluid were changed. The air in the mediastinum could then migrate into the pleural spaces along similar pathways to those described for the mediastinal fluid.

Another explanation of the bilateral hydropneumothorax and hydropneumomediatinum is that the catheter ruptured through the vascular space into a pleural space. The mechanical force of the tip of the catheter or the accumulation of irrigating hypertonic infusate could then have caused rupture of the visceral pleura, leading to the accumulation of air in the pleural space. The air and fluid may then have migrated along the interpleural communications described earlier to directly involve the contralateral pleural space or may have perforated the mediastinal pleura to cause a hydropneumomediatinum (or both). The accumulation of fluid and air in the mediastinum could have ruptured the contralateral mediastinal pleura, leading to or aggravating the contralateral hydropneumothorax. Although the chest radiograph showed the tip of the catheter in the mediastinum when the complication became clinically evident, it is possible that it had migrated from the involved pleural space back into the mediastinum before the chest radiograph was obtained.

It seems highly unlikely that the bilateral thoracentesis performed to remove the fluid resulted in the bilateral pneumothorax. Each thoracentesis was done without complications and confirmed by chest radiographs. In addition, the suggestion of mediastinal air on the radiograph taken at the onset of the patient's complaints makes it seem even more likely that the air was present in the thorax prior to the drainage of pleural fluid.
Finally, although the initial placement of the catheter seemed to be uncomplicated, the free return of blood and the apparent radiographic location of the tip of the catheter within the central vein do not guarantee that the catheter was inside the vascular space immediately upon insertion; however, the fact that the patient was asymptomatic for 7 days after insertion of the catheter while hyperalimentation fluid was being infused makes an immediate complication of CVC insertion even less likely.

The patient’s bilateral hydrothorax contained no red blood cells. Past case reports of hydrothorax as a delayed complication of CVCs are conspicuous for the absence of hemorrhage into the pleural space. It is difficult to understand the absence of a hemotheroax associated with delayed perforation by the catheter. One would expect that since the CVC eroded through the vascular space, bleeding at least into the mediastinum would occur. Perhaps a thrombus forms around the site of central venous perforation, thus preventing clinically evident hemorrhage.

In summary, to our knowledge, this case report is apparently the first to describe a hypodermomediastinum and bilateral hydropneumothorax as delayed complications of a CVC. The left internal jugular venous approach and the hypotonic infusate likely predisposed to the complication, although central venous rupture has been described with other approaches. When a CVC is inserted, particularly for parenteral nutrition, one must be certain that the tip of the catheter is placed into the SVC, lies parallel to the long axis of the SVC, and is proximal to the right atrium. This position should minimize the possibility of rupture by the catheter. A catheter which lies at an angle to the SVC is at risk for delayed perforation, which may have life-threatening consequences. In retrospect, in placing the CVC in this patient, it would have been safer to advance the tip of the catheter further into the SVC and parallel to the long axis of the vein, thus minimizing mechanical forces on the venous wall. Finally, even a catheter composed of relatively flexible material such as silicone elastomer can rupture through the vascular space.

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Chylous Pleural Effusion Associated With Primary Lymphedema and Lymphangioma-like Malformations*

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We describe a patient with a chylous pleural effusion associated with primary lymphedema of his right leg and abdominal wall. On evaluation a generalized, severe hypoplasia of the lymphatic system turned out to be associated with hyperplastic, lymphangioma-like malformations.

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Although primary lymphedema of the extremities is known to be associated with pleural exudates,1,2 chylous effusions are extremely rare. To our knowledge, only three well-documented cases have been published.3-5 We report a fourth case, not only remarkable because of the response to therapy, but also because of a thus far unreported association of a generalized hypoplasia of the lymphatic system and lymphangioma-like malformations.

CASE REPORT

A 28-year-old man was referred to our hospital because of a gradually progressive lymphedema of his right leg, which had started at the level of the knee after a minor sports-induced trauma at the age of 15 years. Thereupon, his lower leg became edematous. In later years, his thigh, scrotum, left upper thigh, and abdominal wall also became affected. The growth of the right leg lagged behind. Complaints about shortness of breath on exertion started 3 months before admission. The complaints were most prominent in the early morning and diminished during the day. He had no other respiratory symptoms, fever, night sweats, weight loss, or gastrointestinal complaints. There was no family history of congenital lymphedema, and no history of infection or trauma during pregnancy. The delivery had been normal, as was the patient’s development until his 15th year.

On physical examination, the right leg was shorter than the left and more voluminous. On palpation, there was no temperature difference between both legs. The edema was nonpitting. His arms and nails showed no abnormalities. The findings from physical examination of the chest were remarkable for dullness and decreased breath sounds at the right lung base. Being most prominent in the early morning, this area was noticed to become smaller during the daytime.

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