Central Venous Catheter-Related Hydrothorax

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This report describes a case of 88-year-old women who developed central venous catheter-related bilateral hydrothorax, in which left pleural effusion, while right pleural effusion was being drained. The drainage prevented accumulation of fluid in the right pleural space, indicating that there was neither extravasation of infusion fluid nor connection between the two pleural cavities. The only explanation for bilateral hydrothorax in this case is lymphatic connections. Although vascular injuries by central venous catheter can cause catheter-related hydrothorax, it is most likely that the positioning of the tip of central venous catheter within the lymphatic duct opening in the right sub-clavian-jugular confluence or superior vena cava causes the catheter-related hydrothorax. Pericardial effusion can also result from retrograde lymphatic flow through the pulmonary lymphatic chains.

**Key Words:** central venous catheter; lymphatic system; pleural effusion.

Hydrothorax caused by central venous catheterization is rare, but a well-known complication. There have been numerous previous reports regarding this complication.[1-8] In most cases, the development of hydrothorax was attributed to the extravasation of intravenous infusion fluid through the eroded vascular wall of superior vena cava or innominate vein by the central venous catheter, even though the issue whether there was actually an erosion of vascular wall or not has never been clear. In the present report, we described a case of central venous catheter-related bilateral hydrothorax in which left pleural effusion developed, even while right pleural effusion, which developed before the left side, was being drained, so that there was no accumulation of fluid left behind in the right pleural space, indicating that there was neither extravasation of infusion fluid nor connection between two pleural cavities. The only explanation for bilateral hydrothorax should be the lymphatic connections. The tip of central venous catheter in the present case must have been positioned within the lymphatic duct opening in the superior vena cava, making retrograde lymph flow through the pulmonary lymphatic chains.

### Case Report

An 88-year-old woman underwent open reduction and internal fixation of right femur intertrochanteric fracture under spinal anesthesia. The surgical procedure was uneventful and the patient was admitted to the intensive care unit for the immediate postoperative care. Her previous medical history included hypertension, rheumatoid arthritis and new onset of senile dementia. She had been on aspirin, diazepam, and cinmetidine. In the intensive care unit, she was doing very well. Leukocyte count was 7,800 cells/mm³, hematocrit 32.3 %,
and platelet 273,000/mm³. Albumin was 2.7 g/dL, alanine aminotransferase 9 units/L, aspartate aminotransferase 15 units/L, and total bilirubin 0.6 mg/dL. BUN was 21.7 mg/dL and creatinine 1.17 mg/dL. CK-MB was 4.1 units/L and troponin I 0.03 mg/mL. N-terminal Pro-BNP was 276 pg/mL. Chest radiography was unremarkable (Fig. 1). The central venous catheter which was inserted via left jugular vein in the operating room was accidentally pulled out. In the afternoon of the first postoperative day, a 7F double lumen central venous catheter was inserted via left subclavian vein under ultrasonographic guidance in the intensive care unit, because of a poor peripheral venous access. After it was confirmed that blood could be aspirated easily from both two lumens, a crystalloid fluid was connected to the distal port to start infusion. After a few hours of delay, chest radiograph was taken to confirm the position of central venous catheter, which also showed a small to moderate amount of right pleural effusion (Fig. 2). Since she did not have any respiratory distress, it was decided to watch for the further progress.

On the second postoperative day, chest radiograph showed
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A collapsed right lung and more accumulation of fluid in the pleural space (Fig. 3). The patient was intubated orotracheally and placed on mechanical ventilation. Bronchoscopy was done to open up right lung and a pleural drain was inserted. A total of 2,500 mL of clear and slightly yellowish fluid was drained. Analysis of pleural fluid showed that protein was 1.02 g/dL, glucose 184 mg/dL, lactate dehydrogenase 78 units/L, amylase 5 units/L, chloride 117 mEq/L, pH 7.5 and specific gravity 1.015. Intravenous infusion of crystalloids was increased to catch up the fluid loss. Repeated chest radiograph taken on the third postoperative day showed clear lung field in the both sides (Fig. 4). A sum of 800 mL of fluid was drained from the right pleural space. On the fourth post-operative day, chest radiograph showed a left pleural effusion (Fig. 5). A left pleural drain was inserted and a sum of 800 mL was drained. A bedside transthoracic echo-cardiography was done to rule out the possibility of congestive heart failure as the cause of bilateral pleural effusions, which showed a normal range of left ventricular global systolic function. Total parenteral nutrition fluid with lipid was started via the other port, which also located near the tip of the catheter. In two and half hours, whitish chyle-like fluid was noticed in the right chest tube and chest bottle. About seven hours after starting total parenteral nutrition, chyle-like fluid was noticed being drained in the left side. In the morning of the fifth postoperative day, total parenteral nutrition fluid and lipid were discontinued. Chest radiograph taken in the early morning did not show any accumulation of chyle-like fluid in lung fields or mediastinum. The left subclavian central venous catheter was replaced with right internal jugular venous catheter. By the evening both drains stopped draining any fluid. On the sixth postoperative day, both drains were removed. She was extubated and discharged from intensive care unit.

Discussion

The unique feature of the present case is that the patient developed the left pleural effusion, even while right pleural effusion was being drained, so that there was no accumulation of fluid in the right pleural space. In addition, the chyle-like fluid, parenteral nutritional lipid solution, which was noticed being drained from the right chest tube without leaving any accumulation in the pleural cavity, was also noticed from the left pleural drain several hours later. When the infusion of lipid solution was discontinued, the pleural drain from both sides became clear rather quickly. These findings strongly suggest that there was neither the extravasation of intravenous infusion fluid including the parenteral nutritional lipid solution from the central venous catheter through the eroded vascular wall of vein, nor the perforation of vein in the present case. The diagnosis of central venous catheter-related hydrothorax was delayed in the present case. Since bilateral pleural effusions, more in the right than left, can develop in congestive heart failure,[9] considering the age of patient and the chest radiographic finding, the increased pulmonary markings in the right upper lung as shown in Fig. 2, the occurrence of pleural effusions was initially attributed to congestive heart failure. When congestive heart failure was ruled out and chyle-like fluid was noticed from the right pleural drain, the possibility of the delayed onset of chylothorax from the trauma, thoracic duct injury below the level of T6, the patient might have sustained was considered. However, considering the fact that thoracic duct crosses midline to the left side at the level of T6, bilateral chylothorax should have not developed unless it accompanied multiple chest traumas, which this patient did not have. This notion prompted to realize the occurrence of catheter-related hydrothorax.
There have been numerous previous reports regarding central venous catheter-related hydrothorax.[1-8] In most cases, the development of hydrothorax was attributed to the extravasation of intravenous infusion fluid through the eroded vascular wall of superior vena cava or innominate vein by central venous catheter, even though the issue whether there was actually an erosion of vascular wall or not has never been clear. A case of bilateral pleural effusions complicating left internal jugular venous catheterization has been reported, in which the tip of the catheter was positioned at the left brachiocephalic vein.[3] In this case, immediate chest radiograph which was taken after radiographic contrast was injected via the catheter, showed contrast in the mediastinum. However, a repeat chest radiograph taken 45 minutes later showed no contrast in the chest, suggesting that the extravasation was the least likely explanation in this situation. Another case, in which bilateral hydrothorax and cardiac tamponade developed simultaneously after right subclavian vein catheterization, has been reported.[4] In this study, it is not likely that the extravasated fluid could have accumulated in the pericardial space without the perforation of vascular wall. According to a previous report,[5] left subclavian vein cannulation, if a rigid dilator is passed over the guide wire with force, can lead to cardiac tamponade, usually in the acute setting. This occurs when the perforation is within the pericardial reflection, which ends at the level of the left brachiocephalic-caval confluence. If the perforation is extrapericardial, pleural effusion occurs, usually in the subacute or chronic setting. As such, neither the vascular wall perforation nor the extravasation can explain the simultaneous development of both pleural effusion and pericardial effusion in that case mentioned above.[4] In this case, particularly, right subclavian vein was cannulated and the distal portion of the catheter was making a loop with its tip sitting at the right subclavian-jugular confluence, according to chest radiograph. Thus, the possibility of either extravasation of infusion fluid or perforation of vascular wall is very low. Instead, the tip of catheter must have been caught within a lymphatic duct opening in the right subclavian-jugular confluence. Intravenous infusion of fluid and parenteral nutrition solution by infusion pump would make a retrograde lymphatic flow, causing hydrothorax in the right side and later in the left side, and pericardial effusion, because there are connections among lymphatics draining both lungs and heart as shown in Fig. 6. Lymph in the lung is drained into the main bloodstream via thoracic duct in the mediastinum but also more frequently via the lymphatic ducts of the peritracheobronchial lymph node chains directly into the jugulo-subclavian venous confluence. PTD denotes right paratracheal chain, ITB intertracheobronchial lymph nodes, BSG left superior bronchial lymph node, and RH beginning of the left recurrent chain (Adopted from Le Pimpec Barthes F, et al.[10] with permission from the authors and publisher).
dual connection can explain the development of bilateral pleural effusion with some delay in the left side, as shown in the present case report and the previous case reports.[3,4] It is most likely that the radiographic contrast in mediastinum, which disappeared within 45 minutes in the case mentioned above,[3] was not in the free mediastinal space as extravasate, but in the lymph node chains and lymphatic ducts, so that it drained quickly via both right lymphatic and thoracic ducts. It is expected that pericardial effusion would be drained in the same way, once the central venous catheter is removed as in the case mentioned above.[4]

A previous study has shown that when the right side internal jugular or subclavian vein is cannulated, central venous catheter-related hydrothorax is rare, thus strongly supporting the argument that the cause of central venous catheter-related hydrothorax was the extravasation of infusion fluid through the eroded vascular wall of superior vena cava by the left sided central venous catheter.[12] In contrary, the right side tension hydrothorax complicating right internal jugular venous cannulation has been reported.[8] In addition, the left side hydrothoraxes complicating left internal jugular vein and left subclavian vein cannulation have been reported.[6,7] Interestingly, in these cases the tip of left internal jugular vein catheter was positioned not even close to the right side lateral wall of superior vena cava. Thus, in these cases previously reported,[6-8] erosion or perforation of vena cava was least likely the cause of hydrothorax. In this situation, the most appropriate explanation is that the tip of catheter (distal port) must have been positioned within the lymphatic duct opening in the left brachiocephalic vein or innominate-caval confluence.

An interesting point in the present case is that there was such a long delay (longer than one full day) in the development of pleural effusion in the left side after that in the right side, despite the fact that there was only four and a half hours delay in the appearance of chyle-like fluid in the left pleural drain after that in the right drain. The most reasonable explanation is that the central venous catheter must have been out of the lymphatic duct opening in the wall of vena cava for a certain period of time and reentered again, and then the left pleural effusion developed. While the tip of the catheter was out of lymphatic duct opening, intravenous infusion fluid must have been infused into the vein instead of the right pleural cavity. This argument may explain the situation in which the patient was able to maintain an adequate intravascular volume, which was reflected by a good urine output, throughout the event of hydrothoraxes. Otherwise the whole intravenous infusion fluid would have been infused to the pleural cavities, thus leading to depletion of intravascular volume. The position of the tip of catheter in figure 4 appears to be hanging loose within the lumen of vena cava, when compared with that in other figures, more likely supporting this argument.

It has been well known that pleural effusion, unilateral or bilateral, occurs in patients with superior vena cava syndrome.[13] The effusion is either chylous or exudate. The occluded pulmonary lymphatic flow from increased hydrostatic pressure in the superior vena cava and left brachiocephalic vein probably contributes to the development of chylous pleural effusion. In the same way, infusion of intravenous fluid or parenteral nutrition fluid by infusion pump via central venous catheter, if the tip of catheter is positioned within the lymphatic duct opening in the vein, would make a retrograde lymph flow, causing hydrothorax in the right side and later in the left side, and pericardial effusion.

By no means is the possibility of vascular injuries by central venous catheter totally excluded. In some cases the erosion or perforation of superior vena cava or right brachiocephalic-caval confluence, or even pericardium by central venous catheter actually occurred, particularly when a somewhat more rigid or stiffer catheters made of polyethylene were used.[1,12,14] In a patient, in whom acute pericardial tamponade with cardiac arrest developed, immediately after a permanent hemodialysis catheter was inserted via right interval jugular vein, a median sternotomy revealed that the tip of catheter was within pericardium, having perforated the junction of the right brachiocephalic vein and superior vena cava.[6] A previous study[12] has suggested that prolonged use of the catheter for total parenteral nutrition may increase the chance of vascular wall erosion, resulting in hydrothorax. In any case, however, the vascular injuries, such as erosion or perforation, can explain neither the occurrence of bilateral hydrothorax, nor the occurrence of left hydrothorax by the left side vein cannulation.

In conclusion, although vascular injuries by central venous catheter may cause the catheter-related hydrothorax in some cases, it is most likely that the positioning of the tip
of central venous catheter within the lymphatic duct opening in the right subclavian-jugular confluence or superior vena cava causes the catheter-related hydrothorax, unilateral or bilateral, or pericardial effusion by making retrograde lymph flow through the pulmonary lymph node chains in most cases. It appears that the lymphatic duct opening in the superior vena cava should be located at the right spot at which the tip of catheter from the left side vein cannulation hits, in order for hydrothorax to develop. This may explain the rare incidence of catheter-related hydrothorax.

References