CASE REPORT

Fatal, progressive dyspnoea with an unusual cause

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Abstract
Central venous catheters (CVCs) can cause severe complications, such as pneumothorax, bleeding or infection, and leakage of chyle. Checking the position by chest X-ray or ultrasound does not exclude the development of post-procedural complications. Leakage of chyle due to injuries to the thoracic duct during insertion of the CVC in the left subclavian or jugular vein can cause bilateral pleural effusion, resulting in severe respiratory distress. The need for placement of a CVC should be considered carefully because of the possible occurrence of potential lethal complications.

Introduction
Central venous catheters (CVCs) are commonly used for a number of medical and surgical indications.[1] Various complications of insertion of CVCs are known, depending on their position, such as pneumothorax, bleeding or infection and leakage of chyle.[1]

Pleural effusion secondary to vascular perforation is a rare complication of CVCs.[2] Pleural effusion due to injury of the thoracic duct is even rarer. We describe a case of bilateral pleural effusion after the uncomplicated placement of a CVC. We urge physicians to familiarise themselves with the possible complications of CVC placement for swift recognition and management of potential lethal complications.

Case
A 84-year-old woman presented to the emergency department with upper abdominal pain, vomiting for the last five days, dyspnoea, and dehydration. Her medical history revealed hypertension and chronic atrial fibrillation. In 1970 she had undergone an abdominal hysterectomy.

Physical examination revealed a cachectic woman with a heart rate of 92 beats/min, a blood pressure of 145/92 mmHg, and a body temperature of 37.7 °C. Oxygen saturation was 95% on room air. Auscultation revealed normal breathing sounds with mild rhonchi on the left side. Her abdomen was distended and tender on palpation. High-pitched bowel sounds were heard. Laboratory evaluation showed kidney failure (creatinine 266 μmol/l, ref. 50-90 μmol/l and urea 23.3 mmol/l, ref. 2.9-7.5 mmol/l), consistent with dehydration and an elevated CRP at 50 mg/l (ref. <10 mg/l).

A CT scan of the abdomen showed marked dilatation of the stomach and small bowel and a possible obstruction of the right lower colon. She was admitted to the ward with the diagnosis of ileus. After five days of conservative therapy and rehydration without clinical improvement a laparotomy was performed. It revealed herniation of the terminal ileum and caecum through the peritoneum, which was repositioned. Resection was not needed.

Two days after surgery, a CVC was positioned in the left subclavian vein. The placement procedure (without the use of ultrasound) was uncomplicated; it was achieved with a single puncture, blood was aspirated, and chest X-ray showed correct positioning of the tip in the superior caval vein (figure 1).

Figure 1. Chest X-ray after placement of left subclavian vein catheter

Parental feeding was commenced shortly afterwards. During the following days her clinical condition deteriorated and progressive
dyspnoea developed. Chest X-rays performed on several consecutive days showed progressively worsening pleural effusions, on the right more than the left. Because of the suspicion of hospital-acquired pneumonia of the left lower lobe, treatment with piperacillin/tazobactam was initiated nine days after surgery, and shortly thereafter the patient was admitted to the ICU because of respiratory insufficiency. Treatment with high flow nasal oxygen was started. The chest X-ray at the time of admission to the ICU showed a ‘white right lung’ and mild pleural effusion on the left side (figure 2).

The right pleural cavity was drained (seven days after placement of the CVC) yielding white fluid. Laboratory analysis showed a triglyceride concentration of 0.9 mmol/l, consistent with chyle. After drainage of 1000 ml of the effusion, ventilation improved, breathing frequency decreased and the oxygen saturation increased. The chest X-ray improved accordingly (figure 3), and high flow nasal oxygen could be replaced by oxygen through a nasal catheter. The CVC was removed since we believed that it had passed through the thoracic duct into the subclavian vein when it was inserted.

However, progression of left-sided pleural effusion was seen the following day. Therefore, another drain was positioned which also yielded white affluent (figure 4). Cultures of pleural fluid and blood showed S. epidermidis, and vancomycin was added. After initial improvement the clinical condition of the patient deteriorated. Seven days after admission to the ICU further treatment was withdrawn at her own request. She died due to respiratory failure.

Discussion
CVCs are used for a variety of reasons. Complications, such as pneumothorax, bleeding or infection, can occur immediately or shortly after insertion of a CVC. Complications might also develop over days. Our patient developed progressive respiratory distress in the days following insertion of the CVC, which was positioned in the left subclavian vein, while surprisingly the right pleural cavity became progressively ‘filled’ with effusion. That discrepancy was partly responsible for the delay in diagnosis and treatment. The location of the CVC tip on chest X-ray was described as normal, and the insertion procedure had been uncomplicated as described. The choice for positioning a CVC in the subclavian vein was according to the local protocol. This position is considered superior due to low infection rates, and lower occurrence of thrombotic complications.
Catheterisation of the subclavian vein versus the femoral vein provides better mobilisation opportunities. In retrospect, it could be argued that jugular vein catheterisation would have been a better choice; however the chance of thrombosis would then be slightly elevated. After admission to the ICU due to
respiratory failure, drainage of the pleural effusion showed milky white fluid, suspicious for chyle. Bilateral thoracocentesis was performed to relieve the respiratory distress. More than 3000 ml were drained, indeed improving respiration. The differential diagnosis of this white effusion was chyle, total parenteral nutrition (TPN), cholesterol effusion and empyema. Analysis of the milky white affluent showed a triglyceride concentration of 0.9 mmol/l. A triglyceride level below 0.56 mmol/l means that chyle can be excluded. Levels above 1.24 mmol/l prove the presence of chyle. In levels between 0.56-1.24 mmol/l the presence of chylomicrons can confirm the fluid to be chyle. We did not perform this test since other diagnoses were highly unlikely. In addition, the potassium concentration was 2.0 mmol/l which confirmed our suspicion that the fluid was not parenteral nutrition. We supposed the concentration of triglycerides might have been below 1.24 mmol/l due to previous low enteral intake. Chylothorax following introduction of a left-sided CVC in the subclavian or jugular position is caused by injuring the thoracic duct, which usually drains into the left subclavian artery (figure 5). Surprisingly, a chylothorax after CVC placement in the left jugular or subclavian position is rare, and the development of a bilateral chylothorax is even rarer. Unfortunately a correct positioning of the CVC, as shown by chest X-ray or ultrasound, does not exclude injury of the thoracic duct and development of chylothorax at a later stage. It could be argued that a peripherally inserted central catheter would have been preferable in this case. However, we believe that central venous access was not needed at all, since the only indication was the administration of parenteral nutrition. We believe it would have been preferable to feed this patient enterally.

The amount of chyle transported by the thoracic duct varies widely. It ranges from a few hundreds of millilitres in case of starvation and immobilisation to more than two litres in healthy, well-nourished adults. Treatment includes removal of the CVC and reducing the production of chyle. This can be achieved by keeping the patient fasting for a number of days. Parenteral feeding or a ‘medium chain triglyceride diet’ might be considered. There is no consensus regarding optimal feeding strategies. Bilateral pleural effusion after inserting a CVC is extremely rare, due to non-described anatomical routes between pleural cavities. Since the placement of a CVC is associated with potential lethal complications, its indication should be maintained strictly. One could argue that in our patient parenteral feeding was not strictly needed after surgery, since the ileus was resolved and enteral feeding could have been initiated. It remains unclear why parenteral feeding instead of enteral feeding was initiated on the surgical ward. Enteral tube nutrition is recommended in hospitalised patients with a lasting inability to take in sufficient nutrients. Parenteral feeding should only be considered in patients with strong contraindications to enteral feeding. In conclusion, a bilateral chylothorax is a rare but potentially fatal complication of left-sided placement of a CVC. A chest X-ray or ultrasound, which confirms a correct position does not exclude the development of chylothorax later. Before placement of a CVC other options should first be considered.

Disclosures
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References