Infected mediastinitis secondary to perforation of superior vena cava by a central venous catheter

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We describe the first case of infected mediastinitis associated with central venous catheter insertion. The rare occurrence of this complication may be explained by the fact that it results from central venous catheter-related bloodstream infection and catheter perforation of superior vena cava. The symptoms of this complication (chest pain, dyspnoea) are not specific. Diagnosis should be confirmed by chest x-ray and computerized tomography which show hydromediastinum and pleural effusion. Removal and subsequent culture of the catheter tip will confirm infection. Appropriate antibiotic therapy, guided by sensitivities of the cultured organisms, should be commenced. Any pleural effusion should be drained by thoracocentesis, and the pleural fluid cultured. In case of fever, bacteraemia or shock, a thoracotomy to drain mediastinal and pleural effusions may be considered.

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Central venous catheters are being used widely and increasingly in modern hospitals. We describe a case of iatrogenic infected mediastinitis after superior vena cava perforation by a central venous catheter in a patient with Crohn’s disease.

Case report

A 50-yr-old woman with a previous history of colectomy for Crohn’s disease, chronic obstructive pulmonary disease (COPD), obesity and pulmonary embolism (October 1998), presented in December 1998 with a relapse of her Crohn’s disease. She had a single lumen catheter inserted into the right subclavian vein in order to start parenteral nutrition. Full aseptic precautions were taken during insertion and blood flowed freely back through the catheter.

Three days later, the patient developed right upper chest pain of sudden onset, dyspnoea and a temperature of 38°C. A chest x-ray showed a right pleural effusion and a widened mediastinum. Pulmonary angiography ruled out a new pulmonary embolism. Computerized tomography (CT) showed a right pleural effusion and a widened mediastinum with enhanced density and ‘air pockets’ (Fig. 1). Later the same day, she became even more unwell with severe dyspnoea, hypotension and hyperthermia. Her trachea was intubated and mechanical ventilation of the lungs was started. Norepinephine was required to maintain her blood pressure.

She was then referred to the thoracic surgery intensive care unit (ICU) with the diagnosis of mediastinitis and septic shock. A triple lumen catheter was inserted into the left subclavian vein. The catheter in the right subclavian vein was not removed. Chest x-ray showed the catheter in the left subclavian vein but not in the right subclavian vein: there was a right pleural effusion and a widened mediastinum (Fig. 2). Repeated blood cultures were drawn and broad spectrum antibiotics started (piperacillin/tazobactam, isepamicine). Bronchoscopy and oesophagoscopy ruled out the likelihood of oesophageal or tracheal tear. A right thoracotomy was then performed. It showed a dissection of the mediastinum with an effusion of a milky fluid (about 2 litres). This fluid came from the tip of the right central venous catheter which was situated in the mediastinum. Mediastinal and pleural effusion were drained. Blood, mediastinal and pleural effusions cultures grew the same methicillin-resistant Staphylococcus aureus (MRSA).
Unfortunately, there was no catheter tip culture. The presence of triglyceride (128 mmol litre$^{-1}$) in the mediastinal fluid confirmed hyperalimentation fluid extravasation. We concluded that the patient developed a MRSA mediastinitis and a septic shock secondary to superior vena cava perforation by an infected central venous catheter. Piperacilline/tazobactam and isepamicine were replaced by vancomycin and fosfomycin. At first, the patient’s temperature increased intermittently. Bronchoalveolar lavage, urine and faecal cultures were negative for MRSA. CT revealed no deep abscess. Finally, the patient became afebrile, was weaned from the mechanical ventilator and discharged from the ICU on day 14. All antibiotics were discontinued on day 21. She was discharged 15 days later. Altogether, the patient had been in hospital for 6 weeks.

**Discussion**

This case history is the first that has been reported of infected mediastinitis associated with central venous catheter insertion (according to a Medline search, 1966–2001). Commonly, mediastinitis is secondary to cardiac surgery, oesophageal perforation or cervical infection.\(^1\) In this case, it is believed that the patient developed bacterial mediastinitis complicating perforation of the superior vena cava by a central venous catheter, because:

(i) blood, mediastinal and pleural effusions grew the same MRSA;

(ii) the tip of the central venous catheter was situated in the mediastinum;

(iii) the presence of triglyceride in the pleural fluid confirmed extravasation of the hyperalimentation fluid;\(^2\)

(iv) bronchoscopy and oesophagoscopy discarded the possibility of oesophageal or tracheal fistula.

The rare occurrence of this complication may be explained by the fact that it has to combine two mechanisms. First, there must be central venous catheter-related bloodstream infection, which is not infrequent (2–14 episodes for 1000 catheter days).\(^3\)\(^4\) Catheter-related bloodstream infections refer to the isolation of the same organism from a quantitative culture of the distal catheter segment and from the blood of a patient with clinical symptoms of sepsis and no other apparent source of infection.\(^4\) Unfortunately, there was no catheter tip culture and no parenteral nutrition culture. There are, therefore, four possible pathways for this catheter-related infection: external bacterial colonization; internal bacterial colonization; haematogenous seeding of the catheter during bloodstream infection of any origin; or contamination of the fluids or drugs administered intravenously (so-called intrinsic catheter-related bloodstream infection).\(^4\) Involvement of *S. aureus* suggests the likelihood of external or internal bacterial colonization.\(^4\) The attributable mortality of catheter-related bloodstream infection is about 25%.\(^5\)\(^6\) Gram-positive cocci are responsible for at least two-thirds of the infections. *Staphylococcus aureus* is responsible for 5–15% of the infections and is associated with a higher rate of complications.\(^7\)

The second mechanism involved is perforation of the superior vena cava by the catheter with extravasation of fluid into the mediastinum. Perforation of the superior vena cava by an i.v. catheter has been widely reported. This may induce haemothorax, hydrothorax, pneumothorax, hydromediastinum and pneumomediastinum.\(^8\)\(^–\)\(^12\) The incidence of this complication appears to be about 0.5% of catheter placements, but many catheter tip perforations remain undetected or unreported.\(^9\)\(^10\)\(^13\) Diagnosis is commonly delayed, which contributes to patient morbidity and mortality.\(^10\)

A few cases of chemically induced mediastinitis from central venous extravasation have been reported. No infectious source has been found in these cases. In one case report, mediastinitis was the result of extravasation of hyperalimentation fluid, after migration of a central venous catheter into the mediastinum.\(^2\) In this case, it was believed that mediastinitis was secondary to chemical irritation by
the hyperalimentation fluid, because all cultures (including catheter tip and pleural fluid) were negative. In three cases (one adult and two children), mediastinitis and venous thrombosis were secondary to catheter-related vesicant chemotherapy extravasation. In these cases, all the cultures were negative.

Our case emphasizes, once again, that central venous catheterization may induce potentially life-threatening complications. The prevention of catheter-related infected mediastinitis relies on the prevention of catheter-related infection and superior vena cava perforation. Perforation may be the result of mechanical trauma from the catheter tip or chemical damage from infused solutions (hypertonic or vesicant agents). The time from catheter insertion to vascular perforation has a range of 1–60 days. In our case, the delay of three days between the insertion of the catheter and the features of perforation (chest pain, dyspnoea) perhaps favours secondary damage.

There are many factors affecting the risk of perforation by central venous catheters. The more perpendicular the catheter tip to the wall of the vein, the greater the risk of perforation. Catheters inserted through the left subclavian or internal jugular vein are more at risk of perforation, because the innominate vein forms a right angle to the superior vena cava and the catheter tip may be positioned against the lateral wall of the superior vena cava with a wide angle. On insertion of a central venous catheter a return of blood through the catheter must be obtained in order to exclude venous perforation by the guidewire, but it does not exclude an extravascular position of the catheter tip, since most catheters have side holes. The best test to confirm correct tip placement is a chest x-ray, provided that the catheter is radiopaque (which was not so in our case).

In our case, the patient’s symptoms included chest pain and dyspnoea. Chest pain has been reported previously in patients with chemically induced mediastinitis, but also in patients with catheter malposition against the sidewall of the superior vena cava. Dyspnoea has been observed only in patients with mediastinitis and central vein perforation. The chest x-ray in our case showed a widened mediastinum, as in all but one of the cases of chemically induced mediastinitis. Contrast infusion through the catheter may help the diagnosis (demonstrating extravasation in the mediastinum). When mediastinal extravasation occurs, our case emphasizes the need to culture the catheter tip in order to exclude infection. Any pleural effusion should be drained by thoracocentesis, and the pleural fluid cultured. If fever, bacteraemia or shock occurs, a thoracotomy to drain the mediastinal and pleural effusions should be considered.

References