Chylous cardiac tamponade due to catheter-associated thrombosis of intrathoracic veins in a newborn infant

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A B S T R A C T
Placement of a central venous catheter (CVC) is a common procedure in Neonatology. Complications associated with CVCs include infections and thrombosis. We describe a case of fatal cardiac tamponade due to chylous effusion in a newborn infant with catheter-associated intrathoracic venous thrombosis.

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Placement of a central venous catheter (CVC) is a common practice in neonatology and facilitates lab draws and prolonged intravenous infusions into a central location. However, placement of these catheters is not without risk. Common complications associated with CVC include infections, thrombosis, and mechanical complications such as perforation, obstruction, and dislodgement [1]. A rare, but potentially fatal complication is pericardial effusion (PCE) and cardiac tamponade. Most of such cases in newborn infants occur following intracardiac CVC with or without myocardial perforation [2]. Chylous pleural and pericardial effusions associated with venous thrombosis have been reported in adults and older children with CVC [3–6], but not in newborn infants. Here we present a newborn infant with chylous pleural and pericardial effusions associated with thrombosis of the intrathoracic veins following CVC placement. Our purpose is to highlight upon occurrence of this rare but potentially fatal complication in newborn infants with CVC.

1. Case report

This preterm male infant was born at 34 weeks gestation to a 21-year-old primigravida mother. He was delivered vaginally with Apgar scores of 7 and 9 at 1 and 5 min, respectively. His birth weight was 5 lbs, 10 oz. He had an antenatal diagnosis of gastrochisis, which was confirmed postnatally. A silo was placed at 1 h of life, at which time a jejunoileal atresia was noted. Routine NICU and surgical cares followed, including antibiotics, total parenteral nutrition and daily reduction of gastrochisis.

On the sixth day of life, the skin surrounding the abdominal wall defect was noted to be erythematous; antibiotic coverage was broadened. The next day, the abdominal wall defect was surgically closed, however the skin was left open out of concern for cellulitis. A left subclavian 5-French 8 cm CVC was placed at the time of surgery. The atretic area of small bowel was left unrepaired, with plans to repair this in 2–3 weeks.

Postoperatively, the infant’s abdomen was erythematous, edematous, and shiny. Broad-spectrum antibiotics were continued. On the second postoperative day, the infant’s left arm was found swollen. An ultrasound revealed a large venous thrombus throughout the left internal jugular, innominate, subclavian and axillary veins with small collaterals present. A chest X-ray showed borderline cardiomegaly without pleural effusions and the CVC tip in satisfactory position in the low superior vena cava (SVC) (Fig. 1A). The infant was started on enoxaparin sodium (Lovenox®, Sanofi Aventis) injections and a coagulation work-up was initiated. He was extubated on the third postoperative day.

On postoperative day 5, the infant had acute clinical deterioration, characterized by progressive respiratory failure, hypotension, and bradycardia. He was intubated and resuscitated using positive pressure ventilation, chest compressions, bolus epinephrine and fluids. A chest X-ray revealed a large right pleural effusion,
moderate bilateral perihilar opacities, and the CVC tip position unchanged (Fig. 1B). Needle thoracentesis produced ~3 mL of cloudy fluid. The infant did not respond to resuscitation attempts and died. At autopsy, bilateral chylous pleural effusions (35 mL on right side, 25 mL on left side) and chylopericardium (25 mL) were present. Five mL of clear fluid was found in the peritoneum. The CVC tip was associated with white fibrin, but there was no disruption of the thoracic duct noted.

2. Discussion

Pericardial effusion and tamponade are associated with significant morbidity and mortality. PCE usually occurs in the setting of malignancy, thoracic surgery and trauma. PCE has been associated with CVC placement in newborn infants. In most of these cases, the effusion is secondary to myocardial perforation, as evidenced by biochemical analysis of the effusion being consistent with the composition of the fluid being infused [2]. Autopsy findings in these cases often reveal either an obvious perforation, or evidence of endocardial damage [7]. The risk of perforation increases with CVC placement within the heart, as opposed to the SVC [2]. In our infant, there was no evidence of myocardial perforation or endocardial wall damage on autopsy. Repeated chest X-rays showed the tip of the catheter to be placed in the low SVC, even after the acute clinical deterioration. Furthermore, the concurrent presence of pleural effusions suggests that obstruction to lymphatic flow, rather than myocardial perforation was the cause of pericardial effusion.

In the absence of myocardial perforation, significant thrombosis of the intrathoracic veins was likely the cause of PCE in our patient. The thrombus was likely due to the CVC, although there were multiple other contributing factors including recent gastrointestinal closure, prematurity, and possible infection. Incidence of CVC thrombosis in neonates varies from 0.07 per 10,000 neonates to 2.4 per 1000 NICU admissions [8,9]. There are isolated cases of catheter-associated thrombosis occluding the thoracic duct ostium and leading to chylous pericardial effusions in adults and older children [4]. While our patient had a similar thrombus, the thoracic duct was noted to be patent at autopsy. We contend that given the degree of thrombus, there was likely obstruction of lymphatic flow due to backpressure on the duct and communicating vessels without an overt occlusion. It has been reported that as little as 1.5 kPa pressure can obstruct lymphatic flow in adults [3,4].

The majority of infants with PCE and tamponade will present with acute cardiovascular collapse. There is significant mortality without pericardiocentesis [2]. Direct aspiration of the fluid from the CVC is inadequate. If the PCE recurs following pericardiocentesis, management strategies include leaving a drain in place and ligating the thoracic duct in cases of chylous effusions [3,6]. There is no consensus on whether the CVC should be removed in the setting of PCE. Both leaving the catheter in situ [2,5] and its removal [3,6] have been recommended.

The management of CVC-associated thrombosis is also not well established in the neonatal period. Suggested treatment modalities include supportive care, radiologic monitoring, and anticoagulant therapy for 6–12 weeks with low molecular weight heparin (LMWH), as well as removing the CVC after 3–5 days of anticoagulant therapy [8]. Three to five days of unfractionated heparin followed by an LMWH course is offered as an alternative. The safety and efficacy of thrombolytic therapy and surgical thrombectomy in neonates have yet to be established [8].

3. Conclusion

This case is unique in that the pericardial effusion was due to thrombosis causing relative obstruction of thoracic duct, whereas in previous case series the mechanism was felt to be gradual erosion of the catheter tip through the wall of the right atrium, or injury to the atrium/vena cava at the time of placement [10].

Our experience highlights the risk of PCE and cardiac tamponade due to CVC-associated thrombosis and suggests the importance of being vigilant for this complication in any neonate with a CVC who has a sudden clinical deterioration, regardless of catheter tip position. In this setting, if a neonate fails to respond to conventional resuscitation measures, urgent thoracentesis and pericardiocentesis should be considered. Early recognition and prompt treatment have the potential to be life saving.

Conflict of interest statement
The authors have no conflicts of interest to disclose.

References


