CLINICAL OBSERVATIONS

Cardiac tamponade and pericardial effusion due to venous umbilical catheterization

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Abstract

Aim: We present three cases of neonatal cardiac tamponade due to umbilical venous catheterization, a rare, but potentially fatal complication.

Methods: Timely diagnosis was made by echocardiography, and an urgent pericardiocentesis revealed TPN fluid. Perforation of the cardiac wall was proven by contrast X-ray showing contrast diffusing into the pericardial space.

Discussion: Most frequently, perforation has a delayed course and results from endothelial injury, caused by the hyperosmolar fluids, leading to transmural necrosis and thrombosis. Subsequently, the fluid diffuses transmurally across the myocardium into the pericardium. As migration of the catheter tip can occur, we suggest that its position should be checked immediately after insertion and twice a week thereafter.

Conclusion: Pericardial effusion and cardiac tamponade should be considered in any infant with a central venous line who develops a rapid, unexplained clinical deterioration. Timely diagnosis and drainage has been proven to be life-saving.

Key Words: Cardiac tamponade, neonate, pericardial effusion, venous umbilical catheter

Introduction

Central venous catheterization is frequently used in infants in the neonatal intensive care setting and is associated with several complications, including occlusion, infection, thrombosis, breakage, migration, and displacement of the catheter and perforation of the vessel wall [1,2]. Pericardial effusion and cardiac tamponade are rare but potentially fatal complications when not promptly diagnosed and treated [3]. In our neonatal intensive care unit, cardiac tamponade due to a venous umbilical catheter was diagnosed in three infants over a 10-y period.

Case 1

In a 1470-g female, born after a 32-wk twin gestation, a double-lumen umbilical venous catheter (Sherwood Argyle®, polyurethane, Ch 5) was inserted shortly after birth, with its tip projecting into the diaphragm on immediate X-ray. On day 5, she became progressively pale and tachypnoeic, with a metabolic acidosis (pH 6.91, base deficit −16.1 mmol/l). Because of severe dehydration, urgent intravenous rehydration was given through the umbilical venous catheter. Unexpectedly, apnoea, bradycardia and finally sudden cardiac arrest occurred within the next 30 min. Although CPR was started, vascular collapse persisted. An urgent echocardiography showed a large pericardial effusion and cardiac tamponade. The infusion of fluids through the venous umbilical catheter was immediately discontinued. By subxiphoid pericardiocentesis under echographic guidance, 25 cc of yellowish cloudy fluid was aspirated, resulting in a fast recovery of the circulation. Subsequent chest X-ray showed a pleural effusion, of which 51 cc was aspirated. Injection of contrast into the venous umbilical catheter showed the contrast diffusing into the pericardial space, proving...
a perforation of the atrial wall by the umbilical catheter (Figure 2). Biochemical analysis of the effusion fluid showed it to be similar to the composition of the hyperosmolar TPN solution infused through the umbilical catheter (glucose 884 mg/dl, sodium 146 mmol/l, protein 155 mg/dl and positive for lipids). Echocardiography 2 d later confirmed normal contractility with resolution of the pericardial effusion.

Case 2
A 1800-g girl was born after 33 wk gestation. A single-lumen umbilical venous catheter (Sherwood Argyle® polyurethane, Ch 5) was inserted within 1 h after birth. On X-ray, the catheter tip projected just outside the cardiac silhouette. On day 4 she suddenly became cyanotic with bradycardia and needed to be intubated and ventilated. She developed a metabolic acidosis (pH 7.01, base deficit $-19$ mmol/l) and hypotension. Chest X-ray showed an enlarged globulous heart and that the tip of the umbilical catheter was projecting into the right atrium. An urgent echocardiography revealed a large pericardial effusion with collapse of the ventricles (Figure 1). By pericardiocentesis, 35 ml of clear, yellow fluid was aspirated. The catheter was removed. The composition of the aspirated fluid was comparable to that of the hyperosmolar TPN solution administered through the catheter (glucose 1560 mg/dl and protein 200 mg/dl). Echocardiography on day 12 demonstrated resolution of the pericardial effusion.

Case 3
A 1380-g girl was born after 34 wk gestation. Postnatally, a single-lumen venous umbilical catheter (Sherwood Argyle® polyurethane Ch 5) was inserted. Because on X-ray the catheter tip appeared to be located in the right atrium, it was immediately pulled back by 2 cm. A control X-ray confirmed the correct position of the catheter tip outside the cardiac silhouette. On day 3 there was a sudden deterioration with bradycardia, gasping, and a respiratory and metabolic acidosis (pH 7.0, pCO$_2$ 80 mmHg, base deficit $-12$ mmol/l). She needed intubation and ventilation. Chest X-ray revealed a pleural effusion, and 45 cc of white, cloudy fluid was aspirated. Analysis showed it to be consistent with the intralipid emulsion she was receiving through the umbilical venous catheter. There was no clinical improvement. An echocardiography was performed, revealing cardiac tamponade. Pericardiocentesis yielded 10 cc of milky fluid, resulting in recovery of the infant. The venous umbilical catheter was removed. Echocardiography the next day showed resolution of the pericardial effusion.

Discussion
We describe an acute, life-threatening clinical deterioration in three infants due to cardiac tamponade, caused by perforation of the atrial wall by venous umbilical catheters. In two cases, the initial correct position of the catheter was confirmed by X-ray after insertion and in one immediately after repositioning of the catheter. All three received hyperosmolar TPN solutions through the catheter. Leakage of infusion
fluid into the pericardial space can be caused by direct perforation of the myocardium during the insertion of the catheter. More often, however, it occurs several days later due to endothelial damage, caused by repetitive hitting of the myocardial wall by the catheter, which creates local thrombosis and myosclerosis [4]. Hyperosmolar TPN solution additionally causes osmotic injury and diffuses transmurally into the pericardial space. Known risk factors for pericardial effusion are malpositioning of the catheter tip at the time of insertion, migration of the catheter tip afterwards and infusion of hyperosmolar fluids through the catheter [4–7]. The incidence of pericardial effusion caused by central venous catheters in neonates is estimated to be 5% [2,8]. Sudden onset of cyanosis, hypotension, bradycardia, tachycardia, neck vein distension, elevated central venous pressure, narrowed pulse pressure, distant heart sounds, paradoxical pulse, metabolic acidosis and respiratory distress are possible signs and symptoms [9]. Cardiac arrest may ensue if not recognized in time. The mortality rate of cardiac tamponade due to venous catheterization is reported to be as high as 34–95% [4,8,10], or 0.76–1% [1,5] of all inserted central venous catheters. The clinical suspicion of pericardial effusion can be confirmed by echocardiography or by a globulous heart on chest X-ray. Analysis of the aspirated fluid can prove it to be consistent with the composition of the fluid being infused through the catheter. Once the diagnosis has been established, urgent pericardiocentesis is warranted. The infusion of fluids through the catheter should be discontinued immediately and the catheter removed. Far more important is trying to avoid this life-threatening complication by verifying the correct position of the catheter tip outside the cardiac silhouette on X-ray immediately after insertion, if needed using contrast. Any coiling of the catheter should be avoided [1,3–10]. Its position should be checked at least twice a week by X-ray or ultrasound, as migration has been described [1,3,7,9,12]. We believe that retraction and mummiﬁcation of the cord remnant during the ﬁrst days of life can also cause inward migration of an initially correctly positioned catheter. Less severely ill infants are more prone to migration of and perforation by the umbilical venous catheter, because they are manipulated more frequently by medical staff and parents. The mobilization and manipulation of infants with umbilical venous catheters should be minimized in an attempt to prevent dislocation of the catheter tip [3,9]. Until now, no meta-analyses or EBM recommendations are published on this subject. Only a recent review article has suggested using single-lumen polyurethane or silicone lines with the catheter tip outside the cardiac silhouette conﬁrmed by X-ray or ultrasound. The duration of use should be shortened and the staff should be given thorough education on the matter [13]. Awaiting evidence-based recommendations, we suggest following these guidelines.

Conclusion

Pericardial effusion and cardiac tamponade should be considered in any infant developing a rapid and unexplained onset of bradycardia, cyanosis and metabolic acidosis, irrespective of an initially correctly positioned umbilical venous line. Timely diagnosis and drainage of the pericardial effusion has been proven to be life-saving. The occurrence of this life-threatening complication can be reduced, but not excluded [6,7], by regularly checking the catheter position on X-ray [1,3,7,9] and by minimizing infant mobilization as long as the catheter is in place [3,9].

References