Pericardial Effusion Induced by Umbilical Venous Catheterization: Case Report

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ABSTRACT Pericardial effusion is a rare but life-threatening complication of umbilical venous catheterization in the newborn. Pericardial effusion is rarely seen as a complication of central venous catheterization. Use of central venous catheterization in neonatal intensive care units more commonly has caused increased incidence of pericardial effusion related to total parenteral nutrition. A 7-day-old male infant born prematurely was admitted to our clinic with the diagnosis of pericardial effusion. By guiding echocardiography, 40 ml milk-coloured total parenteral nutrition fluid was evacuated through subxiphoidal pericardiosynthesis. Following pericardiosynthesis, cardiorespiratory status improved significantly. Biochemical analysis aided in the diagnosis of catheter related etiology with possibility of parenteral nutrition fluid diffusion into the pericardial space. In a neonate with umbilical venous catheterization whose general status worsens and in whom cardiorespiratory signs develop pericardial effusion should be considered.

Key Words: Pericardial effusion; infant, newborn


Anahtar Kelimeler: Perikardiyal effüzyon; bebek, yenidoğan


Umbilical venous catheterization (UVC) is commonly used in neonatal care for administration of fluids, parenteral nutrition and drugs, taking sample for blood gas analysis, central blood pressure monitoring, and also for exchange transfusion.1 However, UVC may cause some complications like arrhythmia, thrombosis, myocardial perforation, endocarditis, pericardial and pleural effusion, and sepsis. Pericardial and pleural effusion compose 0.7% of the complications of central venous catheterization which may be fatal.2-5 We want to present a case who de-
veloped pericardial effusion as a complication of UVC performed for administration of parenteral nutrition.

CASE REPORT

The 7-day-old male infant born prematurely was admitted to our clinic with the diagnosis of pericardial effusion. In his history, he was born with 29 gestational weeks weighing 1440 g to a gravida 1, parity 1 woman by cesarean section due to fetal distress. 1st minute and 5th minute APGAR scores were 6 and 8, respectively. Following birth, the infant was hospitalized in neonatal intensive care unit, nasal continuous positive airway pressure (CPAP) was applied, UVC was inserted, and the infant was started to be fed with total parenteral nutrition (TPN). He received amikacin and penicillin G as antibiotherapy. At the 7th day of hospitalization, the patient became to get worse with tachycardia, tachypnea, and in his chest x ray, cardiomegaly was seen (Figure 1). Echocardiography revealed pericardial effusion (Figure 2). With these findings, the patient was referred to our clinic. In the physical examination, the general status of the infant was bad. Further examination revealed the body temperature to be 36.0 °C, breath rate 78/min, heart rate 188 bpm, blood pressure 78/38 (46) mmHg, O2 saturation 85%, and the weight 1370 g. On chest x ray, in addition to cardiomegaly, the catheter was seen to end within the cardiac silhouette.

In Doppler echocardiography, the tip of the umbilical venous catheter was seen to enter the pericardial space from the site where inferior vena cava opens into the right atrium, and massive pericardial effusion was noted. By guiding echocardiography, 40 ml milk-coloured TPN fluid was evacuated through subxiphoidal pericardiosynthesis. Following pericardiosynthesis, the pulse was 146 bpm, breath rate 40/min, blood pressure 78/41 (55) mmHg, O2 saturation 98-100%. The patient was weaned successfully from nasal CPAP. In pericardial fluid biochemistry analysis, glucose level was 502 mg/dL, triglyceride 449 mg/dl, albumin 20.8 mg/dl, total protein <1.0 mg/dl, and cholesterol was less than lower limit of the measurable range. In cell count, WBC was 15/mm³ (54% neutrophilic), RBC was 72/mm³. In the culture of the pericardial fluid, no growth occurred. Following pericardiosynthesis, pericardial fluid decreased and totally disappeared at the 3rd day in echocardiography.

DISCUSSION

Pericardial effusion is rarely seen as a complication of central venous catheterization. Use of central venous catheterization in neonatal intensive care units more commonly has caused increased incidence of pericardial effusion related to TPN.6 The reason of pericardial effusion development is not clear, however, various mechanisms have been
suggested according to clinical and autopsy findings.\cite{5,7} These are passage of the TPN fluid into the pericardial space due to direct perforation of the myocardium by catheter tip during the advance of the catheter, and more often, diffusion of the TPN fluid into the pericardial space a few days after catheterization owing to local thrombosis and myosclerosis developig as a result of endothelial damage caused the recurring hit of the catheter tip. Another mechanism may be the development of pericardial effusion through intramural diffusion as a result of osmotic damage resulting from the hyperosmolar TPN solution. The known risk factors for pericardial effusion are catheter malposition and migration, and hyperosmolar infusion.\cite{5,8} We think that the reason of pericardial effusion development in our case was the perforation of the atrial wall because the catheter tip was seen to be in the pericardial space.

Replacement of the catheter at the right position and site is important to avoid complications. The optimal catheter localization is the junction of right atrium and vena cava inferior (VCI), or thoracic vena cava inferior.\cite{9} The most common tool in the determination of UVC site is anteroposterior chest X-ray; the catheter tip should be just above the diaphragma, or its localization is determined according to the vertebral bodies. Greenberg et al stated that the localization of UVC tip at T8-T9 vertebrae level at X-ray corresponds to right atrium-VCI junctio in echocardiography.\cite{10} On the other hand, Ades et al revealed the tip of the UVC to correspond to T6-T11 vertebral space at chest X-ray when it is seen at right atrium-VCI junctio in echocardiography.\cite{7} Besides, it has been stated in many articles that direct X-ray graphy is not reliable in determining the place of the catheter tip accurately. In our patient, the catheter tip was beyond the right atrium and VCI junctio in echocardiography although it was at T9 vertebra level in X-ray.

In their series of 61 cases consisting of 14 cases with pericardial effusion from six different neonatology clinics within two years and 47 cases gathered from the literature between 1970 and 1999, Nowlen et al found that UVC had been performed in 21 patients whereas 40 patients had peripheral central catheter, and in 92% of the cases, the tip of the catheter was within the cardiac silhouette.\cite{5} In that study, the median duration between catheterization and development of pericardial effusion signs and/or diagnosis was found out 3 days, and the appearance of the fluid was in accordance with TPN in 53 (98%) cases among 54 cases in whom pericardiosynthesis had been performed. In 37 cases, the fluid was analyzed biochemistrically, and it was shown to be TPN fluid in 36 (97%) patients. In addition, the mortality rate was 34% in that study.\cite{5} In their 5-year-long study, Beardsall et al. showed that among 46,000 neonates in whom central venous catheter (CVC) had been performed, 82 developed pericardial effusion or tamponade, the frequency of pericardial effusion or cardiac tamponade being 1.8 for 1000 catheters and the mortality 0.7 for 1000 catheters.\cite{8}

The appearance of the pericardial fluid aspirated from our patient was milk-like (Figure 3). High glucose and triglyceride levels in biochemistry analysis supported that the fluid was TPN. The diagnosis was made at the 7th day although the symptoms had developed 3 days after the insertion of the catheter.

Weak heart beats, tachycardia or bradycardia, poor peripheral pulse, paleness, cyanosis, circulation disorder, increased inotropic support require-
ment and unexplained cardiopulmonary deterioration are nonspecific signs of pericardial effusion. In our patient, echocardiography was performed as the development of cardiopulmonary signs like tachycardia and tachypnea let us think that the causative pathology might be related to CVC, and abundant amount of pericardial fluid was seen. In our patient signs of tamponade were detected and pericardiocentesis was performed. However, in cases without evidence of tamponade it must be known that only withdrawal of the catheter lead to decline of effusion without pericardiocentesis.

In conclusion, we want to emphasize that in a neonate with UVC whose general status worsens and in whom cardiorespiratory signs develop pericardial effusion should be kept in mind, and that pericardial effusion might develop in spite of the appearance of the catheter at appropriate localization in direct graphy.

REFERENCES