□ Case Report □

Umbilical venous line-related pleural and pericardial effusion causing cardiac tamponade in a premature neonate : A case report

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Cardiac tamponade with pleural and pericardial effusion is a rare but life-threatening complication of umbilical venous catheterization in the newborn. It requires a timely diagnosis and urgent treatment, such as pericardiocentesis, to save lives of affected patients. Recently, we experienced a 7 day-old, very low birth weight infant, who developed a cardiac tamponade with pleural and pericardial effusions complicated by umbilical venous catheterization. The patient was successfully treated with pleural and pericardial drainages. Here, we report this case with a review of literature, since there has been no such previous case reported in Korea. **(Korean J Pediatr 2006;49:686-690)**

Key Words : Umbilical venous catheterization, Cardiac tamponade, Pleural effusion, Pericardial effusion

Introduction

Central venous catheterization (CVC) is frequently used in neonatal care, and umbilical venous catheters (UVCs) are relatively easy to insert and widely used in sick newborns. However, they have been associated with several complications, including occlusion, infection, thrombosis, breakage, migration, and displacement of the catheter and perforation of the vessel wall[1].

A cardiac tamponade with pericardial effusion is a rare but potentially fatal complication, and requires a prompt diagnosis and treatment for life-saving[1, 2]. Possible mechanisms are direct puncture by the catheter tip, or most frequently, delayed perforation which results from endothelial damage caused by hyperosmolar infusates, followed by transmural necrosis[3].

In Korea, there have been a few case reports of cardiac tamponade complicated by CVCs, but none by UVCs in neonates so far[4-8]. Therefore, we present a case of umbilical venous line related pleural and pericardial effusion causing cardiac tamponade which was successfully treated with pleural and pericardial drainages.

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Case Report

A 1,008 g male infant was born at 34 weeks of gestation, to a 30 year-old mother who was admitted for severe preeclampsia and treated with $MgSO_4$. He was delivered by emergency cesarian section because of fetal distress and oligohydramnios. Apgar scores were 6 and 9 at 1 and 5 minutes, respectively.

On admission to the neonatal intensive care unit (NICU), he was placed on the infant flow nasal continuous positive airway pressure for mild respiratory distress with cyanosis. A UVC (5 Fr polyvinyl chloride catheter, Sherwood Davis & Geck medical company) was inserted without difficulties and the position of catheter tip was on T9 at the level of diaphragm by X-ray (Fig. 1A). On hospital day 2 (HD#2), total parenteral nutrition (TPN) was started through UVC.

On HD#4, since abdominal distension with purpuric discoloration was noted and abdominal X-ray showed a gaseous distension of bowels, he was intubated and placed on mechanical ventilation for the management of possible nec-

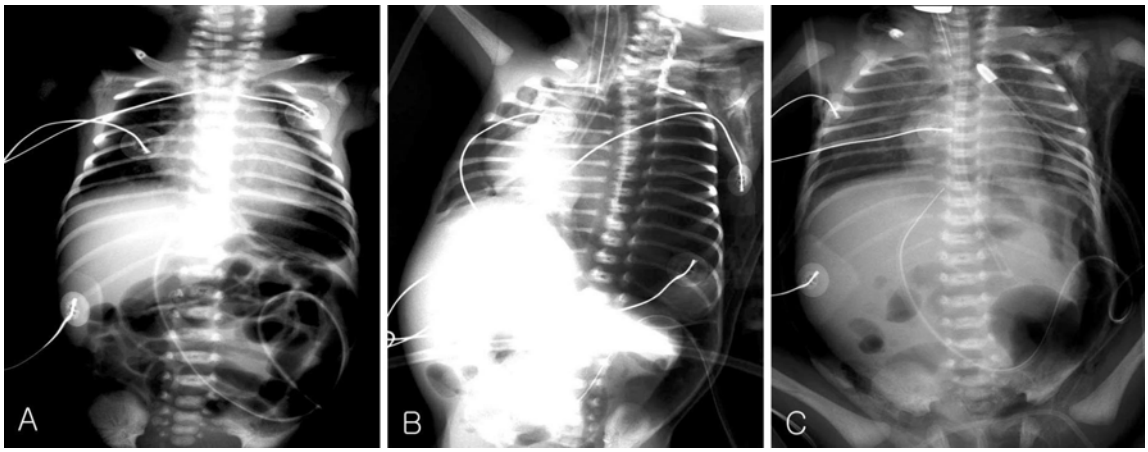

Fig. 1. Infantograms taken before the collection of effusion showed UVC tip was in a desirable position, at the level of diaphragm (T9). **(A)** On HD#1, UVC was inserted. **(B)** On HD#5, left pneumothorax developed. **(C)** On HD#5, left chest tube was inserted.

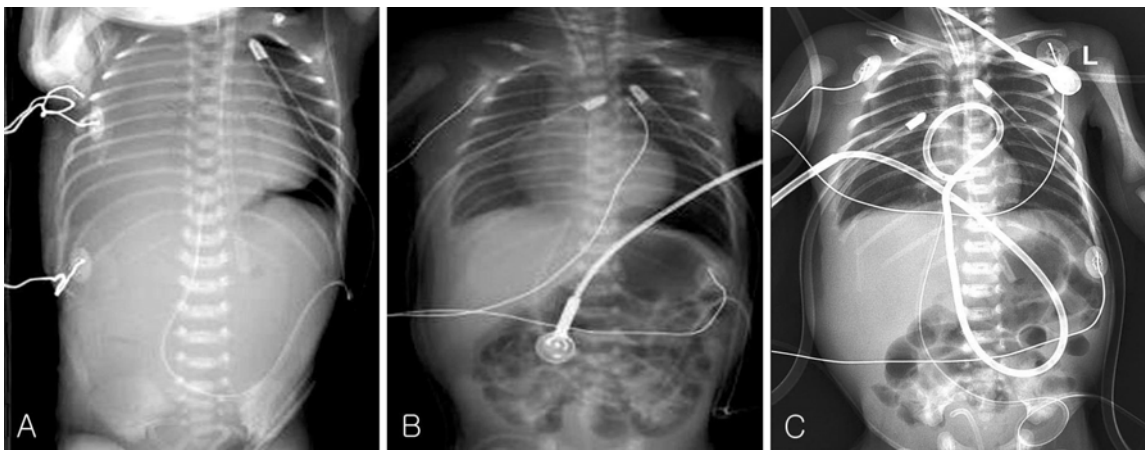

Fig. 2. Infantograms taken during the development of pleural and peritoneal effusion showed UVC tip was still in a desirable position without migration. **(A)** On HD#7, large right pleural effusion was shown on right lateral decubitus film. **(B)** On HD#8, right pleural effusion was drained by chest tube placement, but cardiomegaly newly developed. **(C)** On HD#9, pericardiocentesis with drainage was performed.

rotizing enterocolitis (NEC). On HD#5, he was self-extubated and became hypoxic and bradycardic. Cardiopulmonary resuscitation (CPR) including chest compression was performed. Chest X-ray (CXR) revealed left-side tension pneumothorax, which was decompressed with needle aspiration followed by chest tube insertion (Fig. 1B & 1C). He was placed on the high frequency oscillatory ventilation. TPN was discontinued and high dextrose solutions with electrolytes were infused through UVC.

On HD#6, he showed occasional desaturations with hypotension. CXR revealed pulmonary hemorrhage, pulmonary edema, and progressive right pleural effusion (Fig. 2A). On following days, sono-guided pleural taps were done twice with 24 and 22 mL serohemorrhagic fluid removed, and chest tube was inserted with 100-150 cc/day of serous fluid drained (Table 1).

On HD#9, he developed an intractable hypotension with desaturations, unresponsive to volume expanders and vasopressors. CXR showed cardiomegaly (Fig. 2B) and echocardiography revealed a pericardial effusion. Fluoroscopically guided pericardiocentesis was performed with 7 mL of serous fluid removed, and then pericardial catheter (6.3 Fr multipurpose pigtail catheter, Boston Scientific) was placed with additional 15 cc drained in next 12 hours (Fig. 2C & Table 1). After the drainage of pericardial fluid, his blood pressure went up and his oxygen requirement came down from 100% to 50%.

Because the tip of UVC was at correct position without

Table 1. Clinical Courses of Pleural and Pericardial Effusion

HD	Amount of drainage (mL/day)			Clinical findings and treatment
	Left thorax	Right thorax	Pericardium	
#5	2	–	–	Left pneumothorax-Left chest tube
#6	20	–	–	Hypoglycemia, Right pleural effusion
#7	20	24+22	–	Hypotension, Sono-guided aspirations
#8	10	62+50 (ooz)		Right chest tube insertion
#9	10	70.0	7 (centesis)	Hypotension, tachycardia, oliguria
			+15 (drain)	Pericardial effusion- Pericardiocentesis and drainage
#10-2	10-20	140-150	2.8-6.5	
#13	10	130	4.0	UVC removal
#14-5	0	0	1-3	
#16	0	0	8	Both. chest tube removal
#22	–	–	<2	Pericardial drainage removal

Table 2. Analysis of Pleural and Pericardial Fluids

Test item Hospital day (HD)		Pleural fluid		Pericardial fluid HD#9
		HD#7	HD#12	
Cell count	RBCs/mm^3	2,320	8,640	60
	WBC/mm^3	25	180	65
	Poly/Mono (%)	45/55	90/10	2/98
PH		7.5	7.5	7.5
Specific gravity		1.015	1.028	1.016
Protein (mg/dL)		203	994	237
Glucose (mg/dL)		1,950	1,227	1,221
Gram stain/Bacterial culture		No bacteria	No bacteria	No bacteria

any migration in all follow-up X-rays, we initially did not suspect this event as a complication by UVC. However, since the analysis of pleural and pericardial fluids came back with high glucose level of 1,220-1,950 mg/dL (Table 2), UVC was removed on HD#13 and the amount of fluid drained from right pleural and pericardial space was markedly decreased. Drainages were removed without reaccumulation of fluid on HD#16 and #22, respectively (Table 1). The infant was fed and thrived, and discharged on HD#63, at 40^{+6} weeks of postconceptional age.

Discussion

Umbilical catheters have been implicated in the causation of many problems, including arrhythmias, intracardiac thrombosis, systemic and pulmonary embolization, endocarditis, myocardial perforation, pericardial effusion, pleural effusion, pulmonary infarction, hemorrhage and catheter related infections. Recently, it has been stressed that umbilical catheters should only be used when equivalent care cannot be provided by safer alternative methods, and yet they are still widely used in the care of the sick neonates, as shown in a survey where 99% of surveyed NICUs in the United States of America (USA) reported placement of UVCs and UACs[9, 10]. Therefore, a main concern of umbilical catheter use is identification and prevention of associated complications.

Cardiac tamponade with pleural and pericardial effusion is a rare but life-threatening complication of UVCs in the newborn. The incidence of pericardial effusion caused by central venous catheters in neonates is estimated to be 1-5 %, and mortality rate is reported as high as 34-95%[11, 12] which is secondary to the lack of warning signs and sudden decompensation[13]. It requires a timely diagnosis and urgent treatment such as pericardiocentesis for life-saving. Pericardiocentesis was associated with significantly reduced mortality and must be considered in any patient who has sudden cardiovascular or respiratory decompensation and has a central venous catheter[13].

Nowlen et al[13] recently described 14 cases in six Texas neonatal units and reviewed 47 in the literature, detailing their clinical characteristics. In his report, pericardial effu-

sion occurred in 0.5-2% of CVCs, with its composition similar to the infusate in the vast majority. The median time from CVC insertion to presentation was 3 days (range 0-37), nearly two thirds presenting as sudden cardiovascular collapse and most of the rest having unexplained cardiorespiratory instability. However, in Korea, there have been a few case reports of cardiac tamponade complicated by CVCs, but none by UVCs in neonates so far[4-8]. In this report, we describe a cardiac tamponade with pleural and pericardial effusion complicated by UVC in a very low birth weight neonate that was successfully treated with pleural and pericardial drainages.

Pleural and pericardial effusion can be caused by direct perforation of a vessel or myocardium during the insertion of the catheter. However, more often, it occurs several days later and probably results from erosion of the vascular or cardiac wall[14]. In our case, pleural and pericardial effusion developed 5, and 8 days after initial UVC insertion, respectively, suggesting rather the latter mechanism be the better explanation for the fluid accumulation than direct perforation by the catheter tip.

Known risk factors for pericardial effusion are malpositioning or migration of the catheter tip, infusion of hyperosmolar fluids through the catheter, and prematurity[1]. Thoracic surgery such as ligation of patent ductus arteriosus or chest tube placement for pneumothorax/pleural effusion were also considered as a predisposing factors for a catheter related pericardial effusion[14]. In our case, catheter tip was always at correct position without migration. However, high dextrose fluid with electolytes infused through UVC, and pneumothorax, chest tube insertion, and CPR preceeded pleural and pericardial effusion, which might be contributed to the damage of the vascular or cardiac wall to lead to pericardial and pleural effusion by both chemical and mechanical irritations.

In order to avoid this life-threatening complication, it is essential to verify the correct position of the catheter tip outside the heart[1, 16]. In practice, it is recommended that the position of catheters tip should be checked immediately after insertion and monitored regularly by serial CXRs or ultrasound and fixation of the catheter should be secure to avoid migration of catheter[1]. By the current survey, anteroposterior CXR with or without using contrast media is clearly the imaging study of choice for catheter placement because a real-time ultrasound, although it has been shown to reduce complications during insertion, is not always accessible or cost-effective[9, 16]. Desired location is for the tip of the catheter to be just above the right diaphragm or at thoracic vertebrae 8 or 9 corresponding to the junction of the right atrium and inferior vena cava by echocardiography[8, 9].

However, as shown in our case as well as in Onal et al's, extracardiac positioning does not abolish the risk of pericardial effusion probably because the pericardial reflection extends some way along the caval veins[8, 14]. In fact, we initially did not suspect a complication of UVC until the analysis of fluids came back with high glucose concentration, since the tip of UVC had been always at correct position in every X-rays taken from the time of the insertion to the event. Based on our experience, we strongly support the suggestion by Onal et al[8] that in any infant with a central venous catheter in situ who deteriorates clinically, pericardial effusion/cardiac tamponade must be considered. Once the diagnosis has been established, urgent pericardiocentesis is warranted. The infusion of fluids through the catheter should be discontinued immediately, and the catheter should be removed as well[1].

한 글 요 약

제대정맥도관으로 인한 흉막삼출 및 심장압전을 동반한 심낭삼출 1례

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심장압전을 동반하는 흉막 및 심낭삼출은 신생아, 특히 극소저출생체중아에서 흔히 시행되는 제대정맥도관의 드물게 발생하는 치명적인 합병증으로서 신속한 진단과 치료를 요한다. 저자들은 극소저출생체중아에서 제대정맥도관의 합병증으로 발생한 흉막삼출과 심장압전을 동반한 심낭삼출을 흉관삽입과 심낭도관술로 치유한 1례를 경험하고, 아직 국내에는 보고된 적이 없어 문헌고찰과 함께 보고하는 바이다.

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