

Rupture and displacement of umbilical arterial catheter: Bilateral arterial occlusion in a very low birth weight preterm

Dilek Dilli, M.D.^a, Elif Özyazici, M.D.^a, Nurdan Fettah, M.D.^a, Özkan Kaya, M.D.^b, Melek Pala Akdoan, M.D.^c, Ayşegül Zenciroğlu, M.D.^a, Nurullah Okumuş, M.D.^a and Nilüfer Güzoğlu, M.D.^d

ABSTRACT

Umbilical vessel catheterization is a common procedure in Neonatal Intensive Care Units, especially in very low birthweight infants. Rarely, umbilical artery catheters break, and the retained fragments can cause thrombosis, infection, distal embolization, and even death. Herein, we describe a neonate with clinically significant bilateral limb ischemia developing after removal of a broken umbilical artery catheter. He was under vasodilator treatment in addition to fibrinolytic and anticoagulants. The evolution was favourable.

Key words: premature infants, catheter, thrombosis.

<http://dx.doi.org/10.5546/aap.2015.eng.e283>

INTRODUCTION

Umbilical vessel catheterization is a common procedure in Neonatal Intensive Care Units (NICU), especially in very low birthweight infants. Indications for umbilical artery catheters (UAC) include the need for blood gas or laboratory sample analysis and continuous blood pressure monitoring.^{1,2} Rarely, UAC break, and the retained fragments can cause thrombosis, infection, distal embolization, and even death.³⁻⁶ Besides, neonatal thrombotic disease is most commonly diagnosed in association with indwelling intravascular catheters.⁷⁻⁹

Herein, we describe a neonate with clinically significant bilateral limb ischemia developing after removal of a broken UAC. He was under vasodilator treatment in addition to fibrinolytic and anticoagulants. The evolution was favourable.

CASE

A 28-week male neonate weighing 1000 g was referred from another hospital to our NICU because of a broken umbilical arterial catheter on the 3rd day of life. He was admitted for prematurity and respiratory distress to a regional NICU where umbilical vein and artery catheters were placed. On the 3rd day of life, the umbilical vein catheter was removed due to dripping of the fluid. The UAC was sectioned by a scalpel at the skin level while removing the retaining suture. Several attempts to retrieve the broken fragment by using surgical forceps had been made, but it was not visible. The patient was immediately referred to our unit. A plain X-ray showed an 11 cm of broken fragment of the UAC lying from the umbilical artery origin to distal aortic arch. Pediatric cardiologists were consulted regarding the optimal management. The patient was taken to the angiography room where a trans-femoral approach failed. Then, the right umbilical artery was used to insert a gooseneck snare catheter under fluoroscopic guidance. After grasping the tip of the broken UAC, it was removed successfully. The time between the break of the catheter and the beginning of de endovascular procedure was exactly, six hours.

The patient was transferred back to the NICU and remained stable on the first day after the removing procedure. The following day, he developed bilateral lower limb ischemia; pulses of the femoral and distal arteries were not detectable (*Figure 1*). Doppler studies revealed bilateral external iliac artery occlusion extending into the common femoral, popliteal, and crural arteries. Because of rapidly increasing limb ischemia, it was decided to treat with a tissue plasminogen activator (rt-PA) protocol (an initial

-
- Department of Neonatology, Dr. Sami Ulus Maternity and Children Research and Training Hospital.
 - Department of Pediatric Cardiology, Dr. Sami Ulus Maternity and Children Research and Training Hospital.
 - Department of Radiology, Dr. Sami Ulus Maternity and Children Research and Training Hospital. Ankara, Turkey.
 - Department of Neonatology, Faculty of Medicine, Kırıkkale University.

E-mail Address:
Dilek Dilli, M.D.: dilekdilli@yahoo.com

Funding: None.

Conflict of interest: None.

Received: 2-2-2015
Accepted: 5-4-2015

bolus of 0.7 mg/kg over 30-60 minutes followed by infusion of 0.2 mg/kg/h and low dose heparin drip (10 IU/kg/h) in addition to fresh frozen plasma infusions.⁸ Serum fibrinogen level and activated partial thromboplastin time (aPTT) were within normal limits. Since, no significant change in colour of the limb was observed it was started intravenous nitroglycerin infusion (0.5 µ/kg/min) and topical 2% nitroglycerin ointment (4 mm/kg three times in a day) was applied to all over the ischemic areas. There was a gradual improvement in colour and capillary refill over the next several days. The area of ischemia was limited to the tips of the fingers. At the 72nd hour of the treatment, doppler USG showed the presence of pulsatile arterial flow within the major vessels and collateral circulation. At the same day, the patient presented convulsive movements. A cranial ultrasound obtained prior to fibrinolytic treatment was normal. However, control cranial ultrasound showed grade 3 intracranial hemorrhage. Therefore, intravenous nitroglycerin infusion and rt-PA were stopped, heparin was tapered to 5 IU/kg/h. On the 7th day of the treatment, he was transitioned from heparin drip to low molecular weight heparin (LMWH) with the dose titrated to goal levels of 0.5 to 1 IU/mL of anti-factor Xa. Topical nitroglycerin was stopped after full recovery of the fingers, on the 15th day of the treatment (Figure 2). Cranial ultrasound performed on the 8th week of life showed grade 2 hemorrhage and periventricular leukomalacia. The patient

did not have any further complications and was discharged at about 10 weeks of age as our NICU protocols. He was continued on LMWH for 6 weeks with close monitoring. His motor neurodevelopment was consistent with his age at 3 months of age.

DISCUSSION

Breaking and embolization of umbilical catheters are rare, but can be a dreadful situation. The literature includes few reported cases of transvascular or surgical retrieval of a broken UAC.^{4,6} Choi et al.⁴ first reported the breakage of UAC in two cases in 1977. They were able to retrieve the broken piece via dissection of the right common iliac artery. Murphy et al.⁶ used a transumbilical approach. In our case, the broken fragment was embolized in the aorta and was retrieved successfully via a transumbilical approach.

On the other hand, preterm neonates have a small arterial diameter relative to the catheter size, and they are therefore more susceptible to complications such as vasospasm and thrombosis with subsequent tissue ischemia and necrosis of the involved limb. In this case thrombosis may be related to the umbilical access, femoral artery intervention or the diameter of the equipment used for the procedure to remove the catheter.

The usual treatment of ischemic injuries includes immediate removal of the catheter, elevation of the affected limb, and application of warm compresses to the opposite limb (reflex vasodilation). Anticoagulants such as unfractionated or LMWH and thrombolysis with rt-PA are primarily employed when complete thrombotic occlusion of the vessel occurs with rapidly progressive ischemia, and

FIGURE 1. Bilateral lower limb ischemia developed after removal of the broken fragment of the catheter



FIGURE 2. Complete resolution 15 days after the incident



the risks associated with surgery are considered substantial or even detrimental.⁷ Nitroglycerine is a known smooth muscle relaxant which is readily absorbed through intact skin.¹⁰ It forms free radical nitric oxide which activates guanylatecyclase and increases cellular guanosine 3'5'-cyclicmonophosphate. This dephosphorylates smooth muscle myosin which regulates the contractile state and results in vasodilation. The usual starting dose of 2% nitroglycerine ointment is 4 mm/kg, which is equivalent to 0.2-0.5 mg/kg/min administered intravenously.¹⁰ In the presented case, repeated doses of topical nitroglycerin relieved distal ischemia and resulted in complete resolution and normal perfusion of the fingers.

Finally, the breakage of the UAC is a rare complication; immediate removal of broken portion is lifesaving. The physicians should be aware of the risk for developing limb ischemia even after removal of the retained fragment. Topical nitroglycerin application can be considered if distal ischemia persists despite thrombolytic and anticoagulant treatment. Thrombolytic and anticoagulant treatment may cause intracranial hemorrhage. ■

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