Paediatr Croat. 2025;69:252-9
PRIKAZ BOLESNIKA / *CASE REPORT*
journal.paedcro.com
http://dx.doi.org/10.13112/pc.1123

Neonatal chylopericardium: coincidence or complication related to vascular access?

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Introduction: *Chylopericardium is a rare entity characterized by the accumulation of lipid-rich, milky fluid in the pericardial cavity, which may evolve into life-threatening complications such as cardiac tamponade. In neonates, most cases are idiopathic, though secondary causes include surgical injury, trauma, or lymphatic malformations. Clinical presentation ranges from incidental findings to acute hemodynamic collapse, representing a diagnostic challenge in fragile populations such as preterm infants.*

Case report: *We report the case of a preterm male infant, born at 31 weeks' gestation by cesarean section for severe preeclampsia. Shortly after birth he developed respiratory distress requiring surfactant therapy, invasive ventilation, and multiple central line attempts, complicated by malpositioned catheters. On the fifth day of life, progressive tachycardia, worsening respiratory distress, and anuria prompted echocardiography, which revealed a significant pericardial effusion. Ultrasound-guided pericardiocentesis yielded 21 mL of chylous fluid with triglyceride levels of 691 mg/dL and sterile cultures, confirming neonatal chylopericardium.*

Conservative management was initiated, including drainage, total parenteral nutrition, suspension of enteral feeding, and gradual reintroduction of adapted enteral nutrition. The infant showed favorable progression, with no recurrence of effusion, and was eventually discharged to intermediate care in stable condition.

Conclusion: *This case highlights the diagnostic and therapeutic challenges of neonatal chylopericardium, particularly in preterm infants where vascular trauma may be suspected but not evident. Early recognition combined with conservative management can be effective, avoiding surgical interventions and improving outcomes in this vulnerable population.*

Key words: CHYLOPERICARDIUM; PREMATURE NEONATE; CENTRAL VENOUS CATHETERS; PERICARDIAL EFFUSION; NEONATAL INTENSIVE CARE (MESH TERMS)

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INTRODUCTION

Chylopericardium refers to the abnormal accumulation of chylous fluid – white, milky, sterile, and fat-rich – within the pericardial cavity. It is an exceedingly rare condition, with a reported incidence of 0.22%, and may be complicated by cardiac tamponade or constrictive pericarditis (1, 2).

Its etiology may be idiopathic, the most common form, or secondary to a variety of conditions. These include postoperative complications of cardiothoracic surgery, mediastinal neoplasms, trauma, infections, radiation, central venous hypertension, and subclavian vein thrombosis. Cases associated with congenital lymphangiectasia and abnormal connections between the thoracic duct and pericardial sac have also been reported (1, 3).

Clinical manifestations depend on both the rate and volume of fluid accumulation. Up to 60% of patients remain asymptomatic in early stages, while others may present with dyspnea, cough, chest pain, dizziness, or syncope. When progression leads to cardiac tamponade, classic signs such as hypotension, jugular venous distention, pulsus paradoxus, and muffled heart sounds become evident (3).

In asymptomatic patients, chest radiography may incidentally reveal cardiomegaly. Confirmation of the diagnosis relies on echocardiography, contrast-enhanced computed tomography, or lymphangiography, particularly in the setting of hemodynamic compromise. Pericardiocentesis is essential for biochemical analysis of the fluid: characteristic findings include a milky appearance, triglyceride levels >500 mg/dL, a cholesterol-to-triglyceride ratio <1, lymphocytosis, the presence of fat globules, and negative cultures (3).

Management typically begins with a conservative approach in clinically stable patients. This strategy includes pericardial drainage, dietary restriction of long-chain fats, total parenteral nutrition with medium-chain triglycerides, and the use of octreotide to reduce lymphatic flow. Nevertheless, failure rates may reach up to 50%. In refractory cases, thoracic duct ligation and pericardial window creation are recommended, reducing recurrence to less than 5%. When a secondary cause is identified, it should be addressed specifically (4).

The present case describes a premature newborn who developed chylopericardium during admission to the neonatal intensive care unit. Although the condition coincided with central catheter manipulation, a traumatic origin was considered unlikely, suggesting a spontaneous etiology.

CASE REPORT

A male preterm infant born at 31 weeks' gestational age, delivered by cesarean section due to severe preeclampsia, had a birth weight of 1,430 g, length of 40 cm, head circumference of 27 cm, and Apgar scores of 8 and 9 at 1 and 5 minutes, respectively. Initial resuscitation required tactile stimulation, airway suctioning with a rubber bulb, and positive end-expiratory pressure (PEEP) support using a Neopuff® device (PEEP 5 cm H_2O, FiO_2 0.21).

However, during transfer to the neonatal intensive care unit (NICU), the infant developed respiratory distress with a Silverman–Anderson score of 4. At that time, a lung ultrasound was unavailable to assess surfactant requirement, so the decision was made clinically, considering the absence of antenatal corticosteroid administration.

A single dose of surfactant was administered using the INSURE technique, followed by nasal continuous positive airway pressure "$CPAP_{(n)}$" (PEEP 6 cm H_2O, FiO_2 0.50). Despite this, respiratory distress persisted, prompting initiation of invasive mechanical ventilation in pressure-controlled, pressure-support mode (PEEP 5 cm H_2O, FiO_2 0.30, target tidal volume 5 mL/kg, respiratory rate 50/min).

Chest radiography revealed findings suggestive of mild hyaline membrane disease, with appropriate endotracheal tube positioning but malposition of the umbilical venous catheter, whose tip remained within the liver despite repositioning attempts (Figure 1).

Given this, a peripherally inserted central catheter (PICC) was placed but its tip projected toward the right cervical region, confirming malposition (Figure 2a). The line was removed and a new PICC was inserted via the left forearm, which radiographically extended into the right atrium; no looping was observed at that time, so the catheter was secured in that position (Figure 2b). The

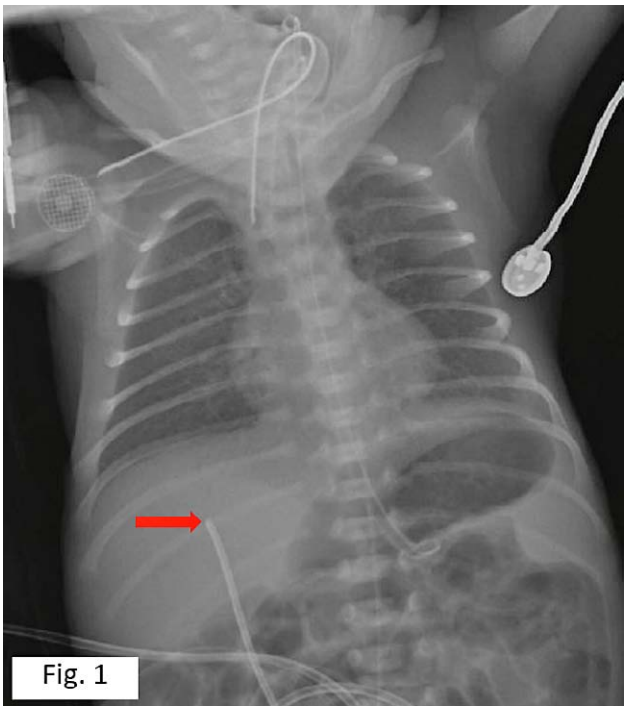

Figure 1. Chest radiography showed malposition of the umbilical venous catheter tip, which remained in the liver despite several attempts to reposition

patient subsequently received colostrum therapy, minimal enteral feeding, caffeine citrate, oxygen support, and parenteral nutrition.

On day 3 of life, total bilirubin levels exceeded the 95th percentile, and phototherapy was initiated.

During handling, the infant exhibited worsening respiratory distress and increased CPAP requirements. Later that day, gastric residuals (~6 mL of pre-digested milk) were noted, with abdominal girth increased by 2 cm, diminished bowel sounds, and abdominal radiography showing mild bowel loop edema. Laboratory results revealed leukopenia and thrombocytopenia, and gastric residuals increased to 18 mL, prompting suspension of enteral feeds, blood and cerebrospinal fluid cultures, and initiation of empirical antibiotic therapy.

By day 4, antibiotics were continued due to suspected septic ileus. Gastric residuals became bilious, with similar volumes to the previous day. While asleep, vital signs were: heart rate 169 bpm, respiratory rate 60/min, blood pressure 65/30 mmHg, positive fluid balance of +40.8 mL in 12 hours, and urine output 1.59 mL/kg/h.

After three hours – at the start of the night shift, on day 5 of life – the heart rate increased to 172–205 bpm, respiratory rate rose to 70–80/min, and respiratory distress worsened with increased CPAP oxygen requirements. Over the following six hours, urine output dropped to 0 mL/kg/h, peripheral pulses weakened, and blood pressure decreased to 43/20 mmHg (mean arterial pressure 28 mmHg).

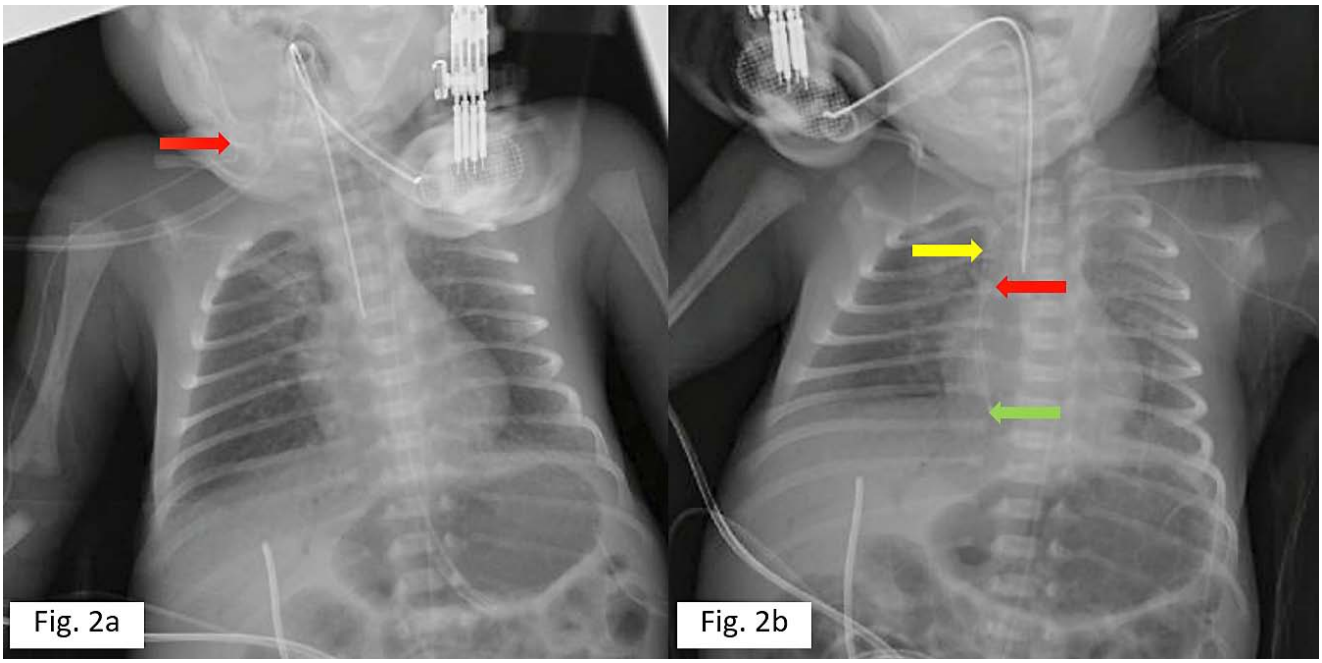

Figure 2. 2a. A peripherally inserted central catheter (PICC) was placed, but it migrated to the right cervical region; and 2b. A second PICC was inserted through the left forearm; the x-ray confirmed its location in the right atrium, but a loop was observed there.

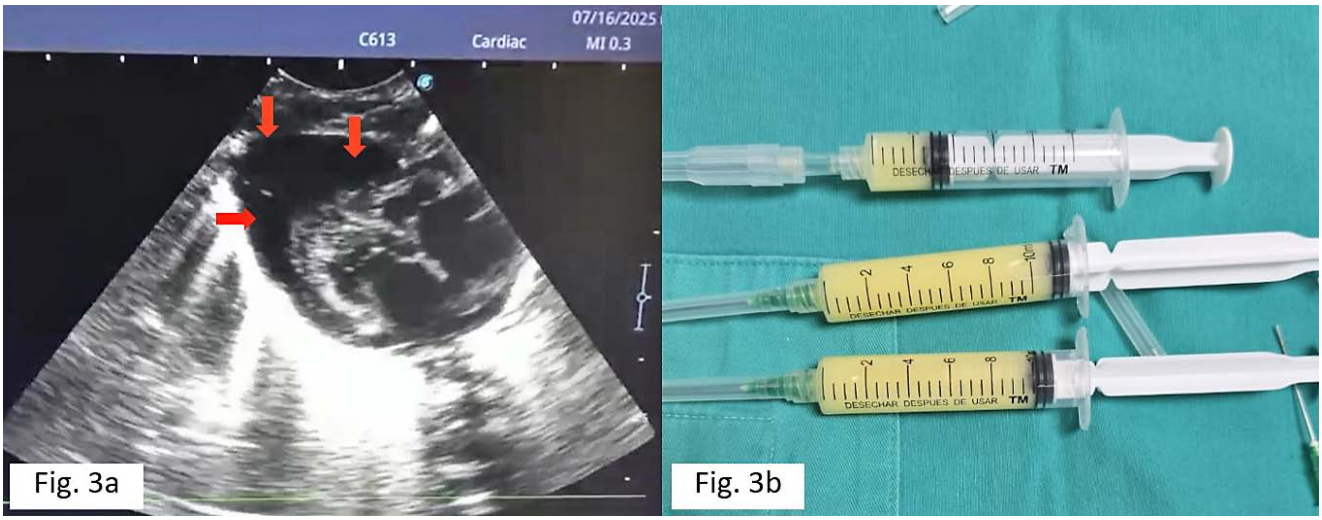

Figure 3. 3a. Echocardiography revealed a large pericardial effusion. 3b. An ultrasound-guided pericardial puncture was performed, draining ~21 mL of milky fluid.

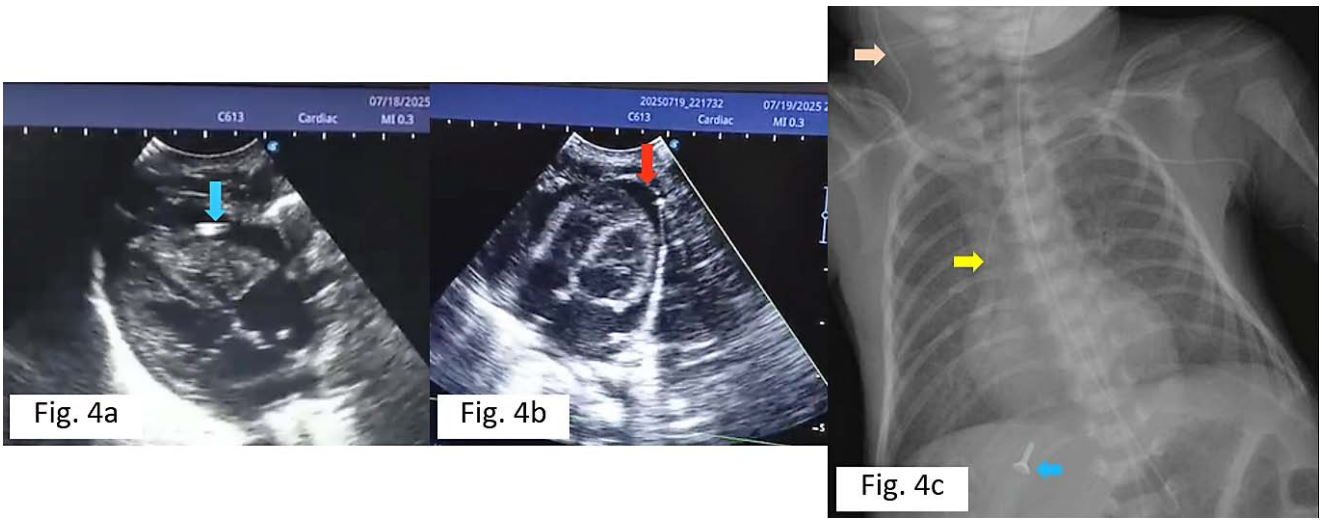

Figure 4. 4a. The branula tip is seen for pericardial drainage due to recurrent effusion. 4b. Pericardial effusion, indicated by the red arrow and evidenced by echovision in the NICU. 4c. PICC tip toward the right cervical region (gray arrow), second PICC tip in the right atrium (yellow arrow), branula attached close to the heart (light blue arrow), for possible pericardial drainage depending on the patient's progress.

Bedside echocardiography revealed a significant pericardial effusion (Figure 3a). An ultrasound-guided pericardiocentesis was performed, yielding ~21 mL of milky fluid (Figure 3b), which was sent for cytochemical analysis. After the procedure, a 10 mL/kg bolus of normal saline was administered, with gradual restoration of vital signs over the next three hours.

A pericardial drain was left in place but later became obstructed and was removed without echographic guidance, opting for close clinical monitoring instead.

On day 6 of life, a new pericardial fluid accumulation was observed, necessitating repeat drainage (Figures 4a, 4b). Fluid analysis revealed: 182 cells/mm³, proteins 3.23 g/dL, negative Gram stain, polymorphonuclear neutrophils (PMNs) 67.1%, glucose 576 mg/dL, triglycerides 691 mg/dL, lactate dehydrogenase (LDH) 46 U/L, and negative cultures. Due to suspicion of a traumatic etiology, second-line antibiotics (imipenem plus vancomycin) were initiated. Prior imaging demonstrated looping of the previous PICC tip, prompting catheter removal and attempted reinsertion, both unsuccessful despite different operators (Figure 4c). A new attempt was deferred to the next shift.

On day 7, central venous access could not be obtained via PICC, so pediatric surgery successfully

Table 1. Comparison of reported cases of chylopericardium through a narrative review during the first 2 months of life

Presented Case	Case 1: (5) Barili	Case 2: (6) Polic	Case 3: (7) Stringel	Case 4: (8)	Case 5: (2) López et al., 2010	Case 6: (9) Mohamad et al., 2003	Case 7: (10) López et al., 2000
Sex	Male	Not specified	Male	Male	Female	Male	Female
Age	Preterm, 31 weeks	Newborn, 29 weeks GA	Newborn, 29 weeks GA	Newborn	Newborn, 41 weeks GA	15 days	6 weeks
Clinical Presentation	Respiratory distress, tachycardia, anuria.	After crying episode: tachypnea, cyanosis, desaturation despite FiO_2 100%; followed by cardiorespiratory arrest, tachycardia, muffled heart sounds.	Bradycardia, distant heart sounds, hemodynamic deterioration.	Congenital anomalies including macroglossia, skin folds, overlapping sutures; progressed to cardiac tamponade.	Intermittent hypotension, tachycardia >200 bpm, decreased ECMO flow, high suspicion of tamponade. Cyanosis, grunting, tachypnea, O_2 sat 66%, shock; diagnosed with double-outlet RV, hypoplastic LV, mitral atresia, restrictive ASD, subaortic VSD.	Intermittent crying, perioral cyanosis.	Dyspnea and anorexia for 2 days, previously healthy, no history of trauma, heart disease, or surgery.
Biochemistry	Pericardiocentesis, milky fluid (21 mL): 182 cells/mm³, protein 3.23 g/dL, Gram negative, PMN 67.1%, glucose 576 mg/dL, triglycerides 691 mg/dL, LDH 46 U/L, negative cultures.	Pericardiocentesis, milky fluid (8 mL): high triglycerides, lymphocytosis, negative cultures.	Pericardiocentesis, milky fluid (10 mL): total protein 49 g/L, glucose 8.2 mmol/L, cholesterol 0.1 mmol/L, triglycerides 5.8 mmol/L, 4,900 cells/mm³ (1% neutrophils, 90% lymphocytes), negative cultures.	Pericardiocentesis: cloudy yellow fluid, WBC 21,650/mm³, RBC 20,000/mm³, PMN 4%, lymphocytes 93%, glucose 54 mg/dL, triglycerides 964 mg/dL, protein 4.1 g/dL, negative cultures.	Pericardiocentesis, milky fluid (20 mL): triglycerides 543 mg/dL, RBC 5%, segmented neutrophils 37%, lymphocytes 1%, Gram negative, negative culture.	Positive blood culture for Enterococcus faecalis.	Chylous pericardial fluid, triglycerides 2,313 mg/dL, Gram/culture/cytology negative.
Imaging	Echocardiography: pericardial effusion.	Echocardiography: pericardial effusion with tamponade, right atrial and ventricular compression.	Chest X-ray. Echocardiography: pericardial effusion.	Echocardiography: large pericardial effusion, poor ventricular function.	Echocardiography: complex congenital heart disease; pericardial effusion (11.5 × 8 mm) with right atrial collapse.	Chest X-ray: enlarged cardiac silhouette. Echocardiography: massive pericardial effusion. Lymphoscintigraphy: normal drainage.	Chest X-ray: enlarged cardiac silhouette. Echocardiography: massive pericardial effusion. CT/MRI excluded neoplasia or congenital malformation.
Etiology	Spontaneous	Spontaneous	Spontaneous	Spontaneous	Possible chylopericardium associated with total parenteral nutrition (TPN) via central catheter.	Complex congenital heart disease. Possible secondary chylopericardium post-surgery/intervention.	-
Treatment	Catheter removal, pericardial drainage, MCT-based nutrition and TPN.	Pericardial fluid drainage, simultaneous volume resuscitation.	Pericardial drainage, conservative management.	Thoracoscopic pericardial window + thoracic duct ligation after failure of drainage and medical therapy (fasting, TPN, octreotide).	Surgical pericardial window with immediate drainage. Prostaglandin E1, mechanical ventilation, balloon atrial septostomy, antibiotics, pulmonary banding with stent, pericardial drainage with pig-tail catheter, MCT-based diet.	Pericardiocentesis, Portagen® diet, catheter replacement, pericardial window and thoracic duct ligation.	Repeated pericardiocenteses, pericardial drainage, low-fat MCT-based diet, TPN.
Outcome	Favorable, no recurrence.	Favorable, no recurrence.	Favorable.	Favorable.	Favorable.	Favorable.	Favorable.

inserted a central venous catheter via venotomy after three attempts (Figure 4d).

Throughout this period, blood glucose and serum electrolyte levels remained within normal limits. On day 8, minimal enteral feeding (20 mL/kg/day) and parenteral nutrition were resumed, advancing as tolerated. By day 10, pericardial drainage volumes were minimal (1.3 mL in the morning, 1.7 mL in the afternoon), with no hemodynamic or ventilatory compromise.

After an additional five days, the infant was transferred to intermediate care on full enteral feeds and room-air oxygen, for ongoing nutritional recovery and routine preterm follow-up.

DISCUSSION

A review of the literature identified seven reported cases of chylopericardium in infants under two months of age, five of which involved newborns (Table 1). This rare accumulation of chylous fluid in the pericardial space may be complicated by cardiac tamponade. Although most cases are idiopathic, chylopericardium may also occur following surgery, trauma, or lymphatic malformations. Clinical manifestations range from asymptomatic presentations to signs of hemodynamic compromise. Diagnosis relies on imaging studies and pericardial fluid analysis, and management usually begins conservatively, though some patients require surgical intervention (1, 3–5).

From a pathophysiological perspective, chylopericardium may arise from congenital lymphatic dysfunction, abnormal connections between the thoracic duct and pericardial sac, or structural fragility associated with prematurity and anatomical immaturity (6, 7, 10, 11). In postoperative or traumatic cases, it is attributed to direct injury of the thoracic duct or lymphatic vessels (2, 8), including reports following, extracorporeal membrane oxygenation (ECMO) cannulation (9).

In our patient, despite multiple attempts at central line placement by different operators, there was no evidence of vascular injury. The use of PICC, made of biocompatible materials such as silicone or polyurethane, reduces the likelihood of traumatic damage. Furthermore, the premature myocardium, characterized by increased rigidity due to a higher proportion of type I collagen, is more resistant to mechanical trauma (12). These factors support the hypothesis of spontaneous lymphatic leakage related to immaturity of the lymphatic system.

Clinical presentation is highly variable. In the published cases reviewed (Table 1), chylopericardium often manifested as an acute event, with sudden hemodynamic collapse due to tamponade, without preceding warning signs (6–9). By contrast, some reports describe more progressive courses, with clinical signs of hemodynamic deterioration following cardiovascular interventions (2), while others document silent presentations, diagnosed incidentally as cardiomegaly on imaging (10, 11). Our patient experienced a progressive clinical decline, characterized by sustained tachycardia, worsening respiratory distress, and reduced urine output. Although nonspecific, these findings reflected systemic deterioration and prompted early suspicion of an underlying cardiac cause. This highlights that beyond acute forms, chylopericardium may evolve insidiously, warranting close monitoring, particularly in preterm infants.

Diagnosis requires a combination of imaging findings and biochemical analysis of pericardial fluid. In acute cases, echocardiography is crucial for rapidly identifying effusion and guiding pericardiocentesis. The fluid typically exhibits a milky appearance, triglycerides >500 mg/dL, lymphocytosis, and sterile cultures, consistent with classical diagnostic criteria (6–9). In less symptomatic cases, suspicion may arise from chest radiography, later confirmed by pericardiocentesis (10, 11). Other reports describe diagnosis in the context of postsurgical deterioration, likewise confirmed by fluid analysis (2).

In our patient, suspicion arose from progressive clinical worsening and echocardiographic detection of pericardial effusion. The aspirated fluid displayed a milky appearance, triglycerides of 691 mg/dL, a cholesterol-to-triglyceride ratio <1, and negative cultures, confirming the diagnosis. Treatment varies according to severity: some patients recover with a conservative approach after initial drainage, restriction of long-chain triglycerides, total parenteral nutrition, and, occasionally, octreotide (6, 7, 9–11). However, recurrence or persistent effusions often require surgical in-

terventions, such as thoracic duct ligation or pericardial window creation (2, 8).

In this case, a conservative approach was chosen, consisting of ultrasound-guided drainage, withdrawal of enteral feeding, total parenteral nutrition, and gradual reintroduction of adapted enteral nutrition. The clinical outcome was favorable, without recurrence, demonstrating that in stable patients, conservative management can be effective and prevent unnecessary surgery.

We consider a traumatic origin unlikely, as PICC and central venous catheter (CVC) placement proved technically difficult despite being performed by experienced operators, suggesting underlying vascular or lymphatic anomalies. In addition, intrinsic features of the premature myocardium – namely structural rigidity due to predominance of type I collagen – make it less vulnerable to mechanical injury (12). Finally, PICCs, being highly flexible and biocompatible devices, significantly reduce the risk of direct trauma (13).

CONCLUSION

Neonatal chylopericardium is an uncommon but clinically significant complication, as it can progress to cardiac tamponade. In the present case, the absence of direct vascular trauma suggests a spontaneous origin, likely related to lymphatic immaturity in the premature infant. Diagnosis was established through echocardiography and biochemical confirmation of pericardial fluid characteristics, which enabled timely initiation of conservative treatment with ultrasound-guided drainage and nutritional support. The favorable outcome underscores the importance of considering this entity in the differential diagnosis of cardiovascular compromise in neonates and highlights the value of early, noninvasive management in clinically stable patients.

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SAŽETAK

Neonatalni hiloperikard: slučajnost ili komplikacija povezana s vaskularnim pristupom?

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Uvod: Hiloperikard je rijedak entitet karakteriziran nakupljanjem lipidima bogate, mliječne tekućine u perikardijalnoj šupljini, što se može razviti u po život opasne komplikacije poput srčane tamponade. Kod novorođenčadi je većina slučajeva idiopatska, a sekundarni uzroci uključuju kirurške ozljede, traume ili limfne malformacije. Klinička slika varira od slučajnih nalaza do akutnog hemodinamskog kolapsa, što predstavlja dijagnostički izazov u osjetljivim populacijama poput prijevremeno rođene djece.

Prikaz slučaja: Izvještavamo o slučaju prijevremeno rođenog muškog dojenčeta, rođenog u 31. tjednu trudnoće carskim rezom zbog teške preeklampsije. Ubrzo nakon rođenja razvio je respiratorni distres koji je zahtijevao terapiju surfaktantima, invazivnu ventilaciju i višestruke pokušaje postavljanja centralnog venskog puta, komplicirane nepravilno postavljenim kateterima. Petog dana života progresivna tahikardija, pogoršanje respiratornog distresa i anurija indicirali su ehokardiografiju, koja je otkrila značajan perikardijalni izljev. Ultrazvučno vođena perikardiocenteza rezultirala je s 21 mL hilozne tekućine s razinom triglicerida od 691 mg/dL i sterilnim kulturama, što je potvrdilo neonatalni hiloperikard.

Započeto je konzervativno liječenje, uključujući drenažu, potpunu parenteralnu prehranu, obustavu enteralne prehrane i postupno ponovno uvođenje prilagođene enteralne prehrane. Dojenče je imalo povoljan klinički tijek, bez ponovne pojave izljeva te je na kraju otpušteno na intermedijarnu skrb u stabilnom stanju.

Zaključak: Ovaj slučaj ističe dijagnostičke i terapijske izazove neonatalnog hiloperikarda, posebno kod nedonoščadi kod kojih se može sumnjati na vaskularnu traumu, koja često nije očita. Rano prepoznavanje u kombinaciji s konzervativnim liječenjem može biti učinkovito, izbjegavajući kirurške intervencije i poboljšavajući ishode u ovoj ranjivoj populaciji.

Ključne riječi: HILOPERIKARD; NEDONOŠČAD; CENTRALNI VENSKI KATETERI; PERIKARDIJALNI IZLJEV; NEONATALNA INTENZIVNA NJEGA