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Congenital Chylous Ascites Associated with Polycystic Kidney Disease: A Case Report

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Abstract: Congenital chylous ascites is a rare condition caused by abnormalities in the lymphatic system, leading to the leakage of triglyceride-rich lymphatic fluid into the peritoneal cavity. The etiologies include congenital lymphatic malformations, lymphatic obstruction, or increased intra-abdominal pressure. Polycystic kidney disease (PKD) is suspected to impair lymphatic drainage due to elevated intra-abdominal pressure, causing chylous fluid accumulation.. We reported a 16-day-old female neonate born via cesarean section at 38 weeks of gestation due to hydrops fetalis. The infant presented with progressive abdominal distension. Abdominal ultrasound revealed massive ascites, a cystic mass in the left adnexa, and bilateral polycystic kidneys, suggesting secondary lymphatic obstruction. Babygram imaging confirmed significant abdominal distension without intestinal obstruction or skeletal anomalies. Ascitic fluid analysis confirmed its chylous nature, with no signs of malignancy or active infection. Laboratory tests revealed mild anemia, hypoalbuminemia, and elevated inflammatory markers.The underlying pathophysiology in this case suggests that PKD led to secondary lymphatic obstruction due to increased intra-abdominal pressure. The patient underwent ascites drainage using a pigtail catheter, fluid and electrolyte management, analgesics, and antibiotics. After 10 days post-intervention, the infant's condition showed gradual improvement, with reduced abdominal distention, stable respiratory function, and no signs of infection or further complications. In conclusion, congenital chylous ascites in neonates may be secondary to conditions like PKD, which cause lymphatic drainage impairment. Early diagnosis and comprehensive management are essential to prevent long-term complications and optimize outcomes in affected neonates.
Keywords: congenital chylous ascites; polycystic kidney disease; neonate; lymphatic obstruction; ascitic fluid analysis

INTRODUCTION

Congenital chylous ascites is an uncommon neonatal condition characterized by the accumulation of triglyceride-rich lymphatic fluid in the peritoneal cavity.[1] It may arise from primary lymphatic abnormalities such as hypoplasia or aplasia of intra-abdominal lymphatics, or from secondary lymphatic obstruction due to elevated intra-abdominal pressure, trauma, or underlying pathological conditions such as polycystic kidney disease (PKD).[2] In this case, the patient experienced progressive abdominal distension, most likely due to secondary lymphatic obstruction from enlarged polycystic kidneys that impeded lymph flow and led to fluid leakage.[3]

This condition has significant clinical implications, including malnutrition, hypoalbuminemia, immunodeficiency, and electrolyte imbalances resulting from protein and lymphocyte loss via ascitic fluid.[4,5] Management can be challenging due to the lack of standardized treatment guidelines, making the approach highly dependent on the underlying cause and individual patient response.[6]

This case report aims to describe a rare instance of congenital chylous ascites associated with PKD in a neonate and explores the underlying pathophysiological link between PKD and lymphatic leakage. Increasing reports of such cases may help refine diagnostic strategies and therapeutic options in neonatal care.

CASE REPORTS

A 16-day-old female neonate was referred to our institution with progressive abdominal distension since birth. She was born via cesarean section at 38 weeks of gestation due to hydrops fetalis. In examination, the infant exhibited abdominal distension but no signs of severe respiratory distress, only mild tachypnea (Figure 1). There was no history of maternal infection, medication use during pregnancy, or other notable antenatal complications. Initial clinical suspicion was congenital chylous ascites.

Abdominal ultrasonography showed massive anechoic ascites, a cystic mass in the left adnexa, and bilateral polycystic kidneys. The ascitic fluid measured approximately 4.0 cm in depth and caused significant abdominal distension. No solid masses or skeletal abnormalities were noted (Figure 2).

Anteroposterior babygram revealed significant abdominal enlargement, displacement of bowel loops to the periphery, and normal skeletal structure. These findings supported the presence of massive ascites with increased intra-abdominal pressure (Figure 3).

Based on clinical and ancillary findings, the diagnosis was consistent with congenital chylous ascites secondary to polycystic kidney disease (PKD). Complete blood count revealed mild anemia with a hemoglobin level of 7.6 g/dL (normal range: 12.5–20.5 g/dL). The white blood cell count was within normal limits (8.80 x10^3/uL), yet inflammatory markers were significantly elevated—C-reactive protein (CRP) at 48.00 mg/L (normal <6.00 mg/L) and procalcitonin at 3.27 ng/mL (normal <0.5 ng/mL)—suggesting a systemic inflammatory response, although not specific for active infection.

Figure 1. Clinical appearance of the patients

Table 1. Laboratory test results

Test Category	Test	Result	Unit	Reference range
Complete Blood Count				
Hemoglobin (Hb)	7.6	g/dL	12.5 – 20.5	Decreased (secondary anemia)
Leukocytes	8.80	x10³/uL	6.00 – 22.00	Increased (inflammatory reaction)
Albumin	3.42	g/dL	3.50 – 5.70	Decreased (mild hypoalbuminemia)
Inflammatory Markers				
C-Reactive Protein (CRP)	48.00	mg/L	<6.00	Increased (Systemic inflammation)
Procalcitonin	3.27	ng/mL	<0.5	Increased (Potential infection/severe inflammation)
TORCH Panel				
Anti-Toxoplasma IgG	650,000	IU/mL	<900.0	Positive (Past infection)
Anti-Toxoplasma IgM	0.254	IU/mL	<0.900	Negative (No active infection)
Anti-CMV IgG	170,100	U/mL	>3.000	Positive (Past exposure)
Anti-CMV IgM	0.201	U/mL	-	Negative (No active infection)
Anti-Rubella IgG	30,830	IU/mL	>10.000	Positive (Rubella immunity)
Anti-Rubella IgM	0.204	IU/mL	<1.000	Negative (No active infection)
Microbiology				
Ascitic fluid culture	Negative	-	-	No bacterial growth
Ascitic fluid cytology	No malignant cells found	-	-	No evidence of malignancy

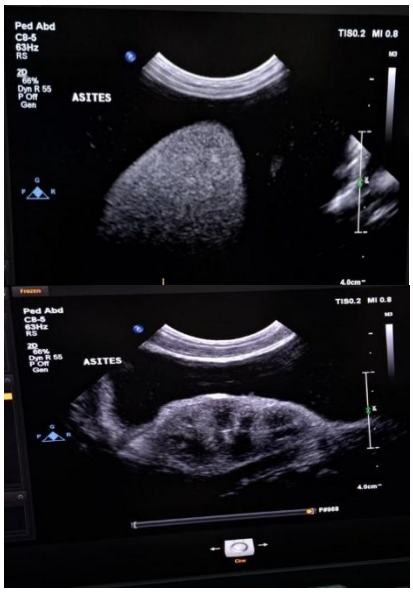

Figure 2. Abdominal ultrasonography

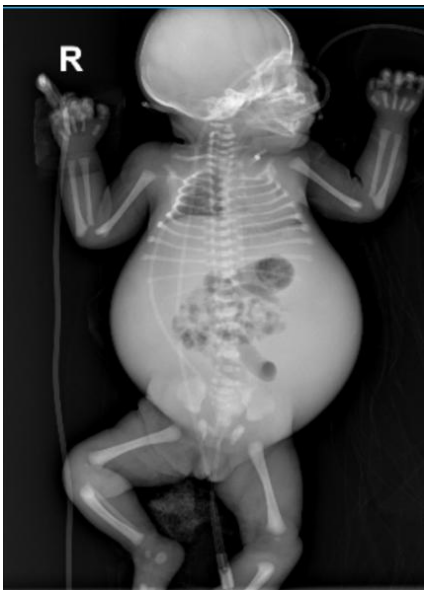

Figure 3. Anteroposterior babygram

Serum albumin was mildly decreased at 3.42 g/dL (normal range: 3.50–5.70 g/dL), potentially contributing to ascitic fluid leakage due to reduced plasma oncotic pressure. The hypoalbuminemia was likely attributable to protein loss through ascitic fluid and impaired renal function associated with PKD.

TORCH screening showed positive IgG for Toxoplasma (650,000 IU/mL), cytomegalovirus (CMV, 170,100 U/mL), and rubella (30,830 IU/mL), with negative IgM results across all tested agents. These findings indicate previous exposure to these infections without evidence of active congenital infection. Therefore, TORCH-related infections were excluded as the underlying etiology of the chylous ascites in this patient.

The patient underwent therapeutic paracentesis with the insertion of a pigtail catheter for ascitic fluid drainage. As observed in the drainage fluid over several days (Figure 4), there was a

noticeable progression in the appearance of the fluid. Initially (Day 1), the fluid was clear, consistent with typical chylous ascites. However, by Day 5, the fluid began to take on a yellowish hue and became increasingly viscous by Day 6. By Day 10, the drainage fluid had further thickened and maintained a yellowish appearance, which could have raised concerns for peritonitis. However, the negative bacterial cultures and lack of clinical signs of infection confirmed that this change in appearance was likely due to the progressive nature of the chylous ascites rather than an infectious process. Cytological analysis of the ascitic fluid revealed no malignant cells, thereby excluding malignancy as a potential cause.

Figure 4. Changes in drainage fluid

DISCUSSION

Congenital chylous ascites is a rare but clinically significant condition in neonates, characterized by the accumulation of triglyceride-rich lymphatic fluid in the peritoneal cavity. The pathogenesis commonly involves either primary lymphatic malformations or secondary causes such as increased intra-abdominal pressure or lymphatic obstruction. In this case, the underlying etiology was likely related to bilateral polycystic kidney disease (PKD), which can exert mechanical compression on abdominal lymphatics and impede lymphatic drainage, leading to chylous fluid accumulation.

The enlargement of polycystic kidneys increases intra-abdominal pressure and may disrupt the normal lymphatic flow, a mechanism previously described in literature involving patients with abdominal masses or massive organomegaly.[7–9] In our patient, this was supported by imaging findings showing bilateral polycystic kidneys and massive ascites without evidence of intestinal obstruction or structural anomalies.

Laboratory analysis revealed mild hypoalbuminemia (3.42 g/dL), which may exacerbate ascites through reduced plasma oncotic pressure. This is consistent with previous findings in peritoneal dialysis patients, where hypoalbuminemia due to protein leakage is associated with increased fluid accumulation.[10,11] Infections are known contributors to ascitic fluid buildup, particularly congenital infections such as CMV or toxoplasmosis, which may induce inflammation or lymphatic damage. However, in this case, TORCH screening indicated past exposures (positive IgG) but no active infection (negative IgM), thus excluding congenital infections as the cause of ascites.[12,13] Malignancy is another differential diagnosis in cases of chylous ascites, particularly lymphomas that may obstruct lymphatic flow. Nevertheless, cytological analysis of the ascitic fluid showed no malignant cells, and culture results were negative, ruling out infectious or neoplastic etiologies.

Given the changes observed in the drainage fluid and the clinical presentation, there was initial concern for peritonitis, which could have led to the consideration of a false laparotomy. However, several factors contributed to the decision to avoid unnecessary surgical intervention. First, the negative bacterial cultures from the ascitic fluid ruled out bacterial peritonitis, which is a common indication for laparotomy.[14] Second, the absence of any clinical signs of infection such as fever,

leukocytosis, or worsening systemic inflammation supported the decision.[15] The yellowish, viscous nature of the fluid over time was consistent with the natural progression of chylous ascites and not indicative of an infectious or septic process.[6,16] Furthermore, imaging and cytological studies did not show any evidence of malignancy or other surgical conditions that would warrant an exploratory laparotomy. Therefore, with these findings, the decision was made to continue conservative management, monitoring the fluid changes, and avoiding unnecessary surgery.

Management of neonatal chylous ascites is highly dependent on the severity and underlying cause. The mainstay of treatment begins with conservative approaches such as dietary modification using medium-chain triglyceride (MCT)-based formulas. MCTs are directly absorbed into the portal system, bypassing the lymphatic system and thereby reducing lymph production.[17] If unresponsive, total parenteral nutrition (TPN) and pharmacologic therapy with octreotide, a somatostatin analog, can be considered to reduce intestinal lymphatic flow.[18,19]

In more severe or refractory cases, interventional procedures such as serial paracentesis or pigtail catheter placement may be necessary to decompress the abdomen and alleviate respiratory or hemodynamic compromise.[20,21,22] These interventions must be closely monitored to prevent complications such as hypovolemia, electrolyte imbalance, and secondary infections.[23]

For persistent or recurrent cases, surgical interventions including thoracic duct ligation or peritoneovenous shunting may be indicated.[24] However, spontaneous resolution with conservative therapy has been reported in many neonates, underscoring the importance of individualized care based on clinical progression.[3,25] In our patient, the ascites was attributed to impaired lymphatic drainage secondary to PKD, with no evidence of infection or malignancy. This case highlights the importance of thorough diagnostic workup to identify potentially reversible or manageable causes in neonatal chylous ascites.

CONCLUSION

Congenital chylous ascites is a rare but potentially serious condition in neonates, requiring a multidisciplinary diagnostic and therapeutic approach. In this case, polycystic kidney disease (PKD) was identified as the likely underlying etiology, causing secondary lymphatic obstruction due to increased intra-abdominal pressure. The diagnosis was supported by clinical findings, imaging studies, and ascitic fluid analysis. Early recognition of the condition and identification of the underlying cause are essential to guide appropriate management and prevent long-term complications. This case underscores the importance of considering structural renal anomalies such as PKD in the differential diagnosis of neonatal ascites, particularly when associated with lymphatic involvement.

Conflict of Interest

The authors affirm no conflict of interest in this study

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