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CASE REPORT – OPEN ACCESS

A Male Infant with Congenital Chylous Ascites Accompanied by Hypertension

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ABSTRACT

Background: Congenital chylous ascites is a condition that arises from a disorder of the abdominal lymphatic system.

Case Report: In this case study, a nine-month-old infant diagnosed with congenital chylous ascites in the newborn period presented with hypertension and chylous ascites.

Conclusion: His hypertension was disappeared with the effective treatment of ascites. Patients with chylous ascites should be carefully evaluated concerning hypertension.

Keywords: Congenital, chylous ascites, hypertension, infant

INTRODUCTION

Congenital chylous ascites is a rare condition in infants that arises from malformation of the lymphatics. The presence of a milky, creamy appearing ascitic fluid with triglyceride content above 200 mg/dl is diagnostic for chylous ascites (1). There is no consensus on the treatment of this condition. Increased intra-abdominal pressure triggered by ascites may lead to abdominal compartment syndrome which is characterized by decreased renal blood flow and glomerular filtration rate in kidney and increased systemic vascular resistance and low blood pressure due to decreased cardiac output, and ultimately abdominal compartment syndrome can cause systemic hypertension. Systemic hypertension also improves after the factors causing abdominal compartment syndrome disappear (2). A nine-month-old infant diagnosed with congenital chylous ascites in the newborn period presented with hypertension and chylous ascites. His hypertension disappeared with the effective treatment of ascites.

CASE REPORT

A nine-month-old male infant born to first cousins' marriage was admitted to a local hospital for abdominal distension. On admission, his physical examination revealed ascites and biochemistry of peritoneal fluid was compatible with chylous ascites. Oral feeding of the patient was discontinued for two weeks, and he was supplemented with total parenteral nutrition (TPN). Since his symptoms did not improve and blood pressure was increased, he was referred to our hospital. It was learned that he was treated for two months due to chylous ascites in the newborn period. Paracentesis, short term octreotide and TPN treatment were administered in the newborn period. He had three healthy siblings, and his father had kidney stones. His body weight was 8 kg (SDS: -1.25), the height was 67 cm (SDS: -2.14), head circumference was 46 cm (SDS: 0.06), and arterial blood pressure was 140/100 mmHg (>95p). His physical examination was unremarkable except for abdominal distention (Fig. 1a). His femoral pulses were palpable, and there was no discrepancy in blood pressure of upper and lower limbs. His laboratory examination revealed the hemoglobin level as 11.7 g/dL, white blood count as 11260/mm^3 and platelet count as 465000/mm^3. Examination of the peripheral blood smear was normal. Urinalysis was normal. Biochemical parameters, including serum albumin and lipids, were within the normal range. Blood urea nitrogen levels ranged between 19–21 mg/dl (N: 4–19 mg/dl) for 14 days and then regressed to normal limits. Urine extraction concurrent with blood urea nitrogen elevation was oliguric (urine output was 0.5–0.8 ml/kg/h), then it returned to normal. Creatinine level was 0.2 mg/dl (N: 0.1–0.4 mg/dl) and never increased. Serum renin level was 26 ng/ml/h (N: 2–37 ng/ml/h) and aldosterone level was 1278 pg/ml (N: 118–1253 pg/ml). The patient's aldosterone level was mildly elevated. The paracentesis fluid was cream-milk colored (Fig. 1b). The triglyceride level was >2000 mg/dL, glucose was 196 mg/dL, albumin was 2.14 g/dL, protein was 4.8 g/dL and lactate dehydrogenase was 172 IU/L in the peritoneal fluid. It revealed no significant bacterial growth. Abdominal ultrasound demonstrated ascites and increased echogenicity of the kidney parenchyma. His echocardiographic examination revealed left ventricular hypertrophy. Captopril was administered

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Figure 1. (a) Abdominal distension of the patient. (b) Milky appearence of the paracentesis fluid. (c) Loss of abdominal distension of the patient

to control his blood pressure. Upper gastrointestinal endoscopy was normal. Thorax and abdomen computed tomography did not show any mass image. The lymphoscintigraphy was performed to show lymphatic leakage in abdomen, but no obvious lymphatic leakage was revealed by the lymphoscintigraphy. His oral feeding was discontinued and supplemented with TPN for four weeks. As a continuous infusion, octreotide was started a rate of 1 µg/kg/h which was gradually increased to 2,5 µg/kg/h. The amount of ascites in the abdomen was assessed weekly by ultrasonography, and the amount of ascites reduced from 50 mm to 10 mm. At the end of the four weeks, high medium chain triglyceride (MCT) content of formula (Pepti Junior®) was started, and the octreotide dose was gradually reduced and discontinued over the course of one week. As his blood pressure returned to normal limit of his age, captopril dose was tapered and finally discontinued. After discharge from the hospital, his feeding was continued mostly with the MCT formula and followed in our outpatient-clinic (Fig. 1c). The patient is now twelve-month-old and he has no ascites for one month. Consent of the patient's parents was obtained for this case report and figures.

DISCUSSION

Chylous ascites, the leakage of lipid-rich lymph fluid into the peritoneal cavity, is a rare form of ascites. The etiology includes congenital, traumatic, malignant, postoperative or inflammatory reasons. Congenital lymphatic dysplasia is primarily responsible in the etiology of lymphatic obstruction (1, 3).

In managing patients with chylous ascites discontinuing oral feeding, TPN and octreotide accelerate the treatment process by decreasing lymphatic flow (4, 5). Yang et al. (6) reported that chylous ascites improved more rapidly when TPN and octreotide were used together. Melo-Filho et al. (7) have surgically applied fibrin glue to a case with refractory congenital chylous ascites and reported that they achieved successful results. Mouravas et al. (5) have reported on four cases of congenital chylous ascites. They treated one with a conservative treatment (TPN for 125 days, octreotide 3 µg/kg/h for 30 days) and fixed the other three surgically (cyst excision, fibrin glue application). Karagol et al. (8) have reported that they increased the octreotide dose to 8 µg/kg/h in a case of congenital chylous ascites, and they fed the patient with MCT-formula and a fat-free formula when admitted with ascites again, after which the case improved. As can be seen, there is no standard treatment approach for the treatment of congenital chylous ascites. Our case was not fed orally for four weeks and successfully responded to octreotide treatment with TPN.

Our patient had arterial hypertension, which we thought was associated with increased intra-abdominal fluid. In situations that may cause an increase in intra-abdominal volume, the abdominal wall compliance reduces and when the intra-abdominal volume increases significantly, abdominal compartment syndrome develops. Reduced venous return, reduced cardiac output and increased systemic vascular resistance are among the cardiovascular effects of abdominal compartment syndrome. In addition, reduced glomerular filtration and oliguria are the renal effects of abdominal compartment syndrome (2). It is possible that the collapse of the inferior vena cava, portal vein and mesenteric vessels may result in the decreased cardiac output and renal blood flow in abdominal compartment syndrome secondary to increased intra-abdominal pressure. Therefore, catecholamines, angiotensin II and aldosterone are secreted and systemic vascular resistance is increased as a part of compensatory response to decreased renal blood flow. Treatment of the underlying cause may be enough to return blood pressure to normal limits (9). Elevation of serum blood urea nitrogen and aldosterone levels with oliguria of the patient at the initial evaluation was considered the renal effects of abdominal compartment syndrome due to chylous ascites. Serum blood urea nitrogen and aldosterone levels were mildly elevated, and creatinine and renin levels were normal in our patient. Mildly elevated blood urea nitrogen and aldosterone levels during hypertension were associated with effective paracentesis from the initial management of the patient. Since his hypertension was disappeared with effective treatment of as-

cites, we thought that the hypertension of our patient was interpreted as a sign of abdominal compartment syndrome caused by ascites. Although ascites causes hypertension as a reason for abdominal compartment syndrome, we aimed to emphasize the coexistence of congenital chylous ascites and arterial hypertension with this article.

In conclusion, we achieved a successful outcome in our case with congenital chylous ascites through long-term fasting, TPN, and octreotide treatment. In addition, the arterial blood pressure returned to normal secondary to the regression of ascites. Therefore, patients with chylous ascites should be carefully evaluated concerning hypertension.

Informed Consent: Written informed consent was obtained from patients who participated in this study.

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Conflict of Interest: The authors have no conflict of interest to declare.

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