Chyloperitoneum: A Postoperative Complication After Repair of Tracheoesophageal Fistula

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Chyloperitoneum is a rare condition that can occur in response to various pathologic processes. The authors described a newborn baby who had chyloperitoneum after primary surgical repair of esophageal atresia with tracheoesophageal fistula (Gross type C). This probably resulted from iatrogenic damage of the thoracic duct during ligation of azygous vein resulting in leaking lacteal within the intestinal wall. Biochemical analysis of ascitic fluid, plasma, and milk formula for triglyceride and cholesterol level can differentiate neonatal gastric intestinal perforation.

INDEX WORDS: Chyloperitoneum, chylous ascites, complication, tracheoesophageal fistula, neonate.

Chyloperitoneum is a rare entity caused by accumulation of chyle within the peritoneal cavity after obstruction of the major lymphatic system and may occur postoperatively after an intraabdominal surgery or aortic surgery. Such condition has not been described previously after surgical repair of tracheoesophageal fistula during the neonatal period.

We report on a patient in whom postoperative chyloperitoneum developed after primary repair of the esophageal atresia with tracheoesophageal fistula (Gross type C).

CASE REPORT
A baby boy, with a birth weight of 2.235 kg, gestational age of 37 to 38 weeks, and history of maternal polyhydramnion, was delivered by cesarean section because of fetal distress. Apgar score at 1 and 5 minutes was 7 and 10, respectively. Associated physical anomalies include low set ear, cleft lip, cleft palate, asymmetric upper extremity, and campylodactyl of the second left finger. Diagnostic studies include chest x-ray, which showed the tip of orogastric tube at the level of second intercostal space. Echography results showed atrial septal defect type II, small patent ductus arteriosus, and normal abdominorenal echogram; brain echogram showed asymmetric lateral ventricle. Chromosomal study findings were normal. Transpleural approach primary anastomosis of the proximal and distal esophagus after division and ligation of the fistula with ligation of the azygous vein to facilitate identification of Gross type C tracheoesophageal fistula was done. Chest tube drainage was placed intraoperatively. Orogastric tube milk feeding was started on the fifth postoperative day. On the seventh postoperative day, abdominal distension occurred gradually followed later by thrombocytopenia and erythematous change of the abdominal wall. Chest tube persistently drained serous fluid, 5 to 10 mL/d. Plain abdominal film showed increased radio-opacity without pneumoperitoneum. Laparotomy showed milklike ascites of about 100 mL, grossly dilated, whitish in color, mesenteric, and small bowel lymphatic without gross rupture or leakage. No hollow organ perforation was found. The abdominal cavity was irrigated with normal saline, and a Jackson Pratt drain was placed. Biochemical analysis of ascites showed triglyceride level of 5,500 mg/dL and cholesterol of 100 mg/dL. Retrospective analysis of milk formula showed triglyceride level of 412 mg/dL and cholesterol level of 4 mg/dL. Ascites culture was negative. Oral feeding with medium-chain triglyceride–based milk formula was started on the fifth post abdominal laparotomy day without recurrence of chyloperitoneum during the 2 years follow-up period.

DISCUSSION
Chyloperitoneum is the accumulation of chyle within the peritoneal cavity resulting from various pathologic processes. The most common etiology of such rare condition in children is primary lymphatic abnormalities in which mechanical obstruction of the thoracic duct, cistern chyli, or intestinal lymphatic is the usual common factor in the pathogenesis. Three mechanisms in the formation of chyloperitoneum in the absence of thoracoabdominal surgery include (1) direct leakage of chyle through a lymphoperitoneal fistula associated with abnormal retroperitoneal lymphatic vessel, (2) exudation of chyle through the walls of the retroperitoneal megalymphatic without a visible fistula, (3) exudation or leakage of chyle after rupture of dilated lymphatic on the wall of the bowel and mesentery, caused by obstruction of the
lymphatic at the base of the mesentery, cisterna chyli, or thoracic duct.3

During surgical procedure of the esophagus especially in the midportion, there is an increase chance of injury of the thoracic duct,4 which probably occurred in our patient. During ligation of the azygous vein, the thoracic duct probably was iatrogenically ligated, but we are not able to prove it because a lymphangiography was not done. Obstruction of the thoracic duct results in retrograde increase in pressure in the major lacteal within the mesentery and intestinal wall thereby causing exudation and leakage of chyle. Lee5 in 1944 showed this mechanism experimentally in a dog after ligation of the thoracic duct. On the contrary, the presence of laceration or tear of thoracic duct will lead to chylothorax.

The preoperative diagnosis of chyloperitoneum is done rarely before paracentesis or operation6 because of nonspecific clinical manifestation and should be differentiated from neonatal gastrointestinal perforation. The diagnosis of chyloperitoneum depends on the character and composition of the ascitic fluid because physical examination, x-ray, and sonography cannot identify its composition.7 In all patients with ascites suspected to have such condition, a paracentesis should be done to determine the fat content (triglyceride and cholesterol), which is greater than the plasma6,7 and milk formula. Site of obstruction or leakage of the thoracic duct can be identified preoperatively by bipedal lymphangiography using lipiodol,8 but its role is quite limited in guiding the care of children or infants.

Optimal management is dependent on effective alleviation of the underlying etiologic process, which usually is being achieved only surgically.6 In the presence of retroperitoneal fistula, surgical closure or ligation is the most effective means.7,9 Other surgical means for intractable chyloperitoneum included peritoneovenous shunt10 and extracorporeal recirculation with a hemofilter incorporated into the circuit.11 When proved to be nonsurgical at exploration, establishment of a diagnosis permits the application of specific therapy, which may be either curative or palliative. Dietary manipulation by using medium-chain triglyceride–based formula was most satisfactory with exudation or leaking small bowel lymphatic as in our case. Conservative treatment by repeated paracentesis and supportive measures are of little value and may endanger the patient by delaying the institution of specific therapy.6 Parenteral nutrition appears to have decreased the hazard of medical therapy by maintaining an adequate nutrition intake while eliminating the obligate loss consequent to repeated paracentesis.6 In a case in which diagnosis is made preoperatively, conservative therapy for 3 to 4 weeks before the surgical intervention should be considered.2 When chyloperitoneum recur after resumption of diet, surgical exploration is mandatory according to a defined strategy that includes feeding the patient up to 6 hours before surgery to aid in visualization of chyle leak and visualization of the retroperitoneal origin of the superior mesentery for the presence of lymphoperitoneal fistula.6

After primary repair of esophageal atresia with tracheoesophageal fistula, the occurrence of abdominal distension accompanied by erythematous change of the abdominal wall, thrombocytopenia, and ascites should lead to the suspicious of chyloperitoneum. The condition can be differentiated simply from neonatal gastrointestinal perforation by comparative biochemical analysis of ascitic fluid, plasma, and milk formula for triglyceride and cholesterol level, thereby avoiding unnecessary laparotomy.

REFERENCES