□ Case Report □

대 한 주 산 회 지 제26권 제4호, 2015
Korean J Perinatol Vol.26, No.4, Dec., 2015
http://dx.doi.org/10.14734/kjp.2015.26.4.360

Recurrent Gastric Perforation in a Neonate Recovered after Primary Surgical Repair and Percutaneous Peritoneal Drainage

Hye Rim Park, M.D.[1], Ji Eun Ryu, M.D.[1], Sung-Hoon Chung, M.D., Ph.D.[2], and Yong-Sung Choi, M.D., Ph.D.[1]

Department of Pediatrics[1], Kyung Hee University Medical Center, Seoul, Korea
Department of Pediatrics[2], Kyung Hee University Hospital at Gangdong, Seoul, Korea

Neonatal gastric perforation is a rare but fatal disease, occurred mainly in preterm infants. In general, primary surgical repair is the main treatment. To the best of our knowledge, there has been only one report of improvement of gastric perforation in neonates after percutaneous peritoneal drainage alone. We describe a case of gastric perforation in a premature extremely low-birth-weight infant girl of 25^{+4} weeks gestational age. We present this case to emphasize that gastric perforation may improve with percutaneous peritoneal drainage.

Key Words: Extremely Low Birth Weight Infant, Gastric Perforation, Drainage

Neonatal gastric perforation is a very rare, but serious condition. It is associated with a high mortality rate. Its incidence is reported to be 1 in 5,000 live births. In most cases, the underlying cannot be precisely determined. Gastric perforations occur most often on the 3rd day of life but can occur at any time in early infancy.[1] Several contributing factors have been reported. Prematurity is the most common facilitating factor.[2] Some mechanical factors are also considered as risk factors, such as mechanical pressure exerted by nasogastric catheters, and excessive gastric distension due to positive-pressure ventilation.[1, 3-7] Chorioamnionitis, necrotizing enterocolitis, asphyxia and postnatal corticosteroid administration could also be the risk factors.[1, 5, 6]

Early diagnosis and treatment could be the most important factors to achieve a good outcome. Characteristic clinical manifestations may include poor activity, abdominal distension, respiratory distress, feeding intolerance, vomiting, and cyanosis.[2] The major defects are proximal linear tears along the greater curvature that occur as a seromuscular laceration, resulting in mucosal ballooning with a pinhole perforation on the top.[5, 6]

Primary surgical repair is the principal mode of treatment. The first successful surgical repair of gastric perforation was accomplished by Kellogg et al in 1951.[8] Several subsequent reports of successful repair have been published, and improved survival rates have been documented.[7, 9] An improvement of gastric perforation in neonates after percutaneous peritoneal drainage has not been described as much previously.

Received: 8 September 2015
Revised: 16 October 2015
Accepted: 21 October 2015
Correspondence to: Yong-Sung Choi, M.D.
Department of Pediatrics, Kyung Hee University Medical Center, 23 Kyungheedae-ro, Dongdaemun-gu, Seoul 02447, Korea
Tel: +82-2-958-2921, **Fax:** +82-2-958-8304
E-mail: feelhope@gmail.com

The Korean Journal of Perinatology · pISSN 1229-2605 eISSN 2289-0432 · e-kjp.org

Case Report

A female, 2nd baby of twin, was born at 25^{+4} weeks of gestation by emergency cesarean section, due to oligohydramnios and fetal distress. Her birth weight was 900 g. She had a prenatal history, of preterm premature rupture of the membranes lasting about 37 days, with intermittent maternal fever. *Enterobacter cloacae* were detected in her mother' s cervical smear, which indicated chorioamnionitis. At birth, because of poor spontaneous breathing and heartbeat (<100/min), the baby was intubated shortly, had 1 vial of surfactant via endotracheal tube for rescue therapy, and had 2 minutes of positive pressure ventilation. Apgar score was 3 and 5 at one and five minutes. On admission to NICU, mechanical ventilation was applied to her. Subsequently, an additional dose of 0.5 vial of surfactant was given within the first 24 hours of life due to low oxygen saturation levels and insufficient aeration of the lungs on auscultation and follow up X–rays. Symptomatic patent ductus arteriosus was diagnosed on the 48th hour of life and intravenous ibuprofen was used for 3 days. A nasogastric tube was inserted shortly after birth and kept; the tip located in the upper stomach, and there was no discharge coming through the tube. At 5 days after birth, she ate a preterm formula once. However, abdominal distension was observed and the feeding was stopped. On the 7th day of life, her general condition deteriorated suddenly with a distended abdomen and increased respiratory distress. At that point, complete blood

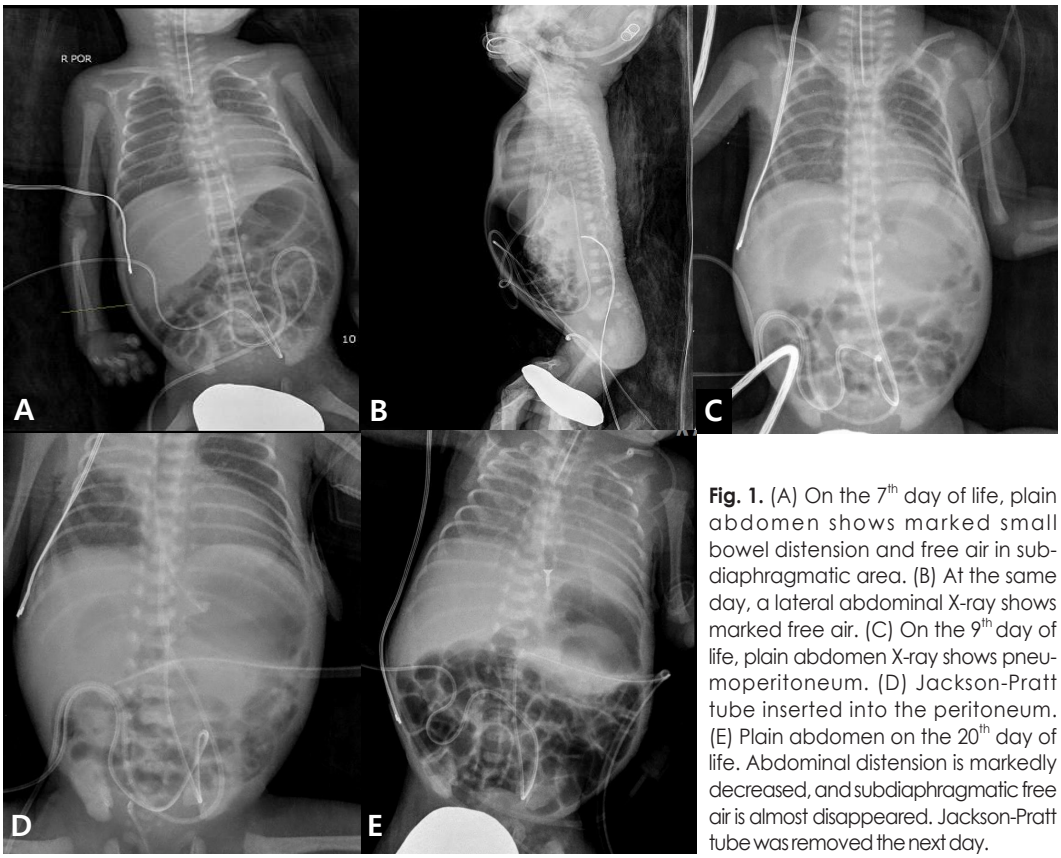

Fig. 1. (A) On the 7th day of life, plain abdomen shows marked small bowel distension and free air in subdiaphragmatic area. (B) At the same day, a lateral abdominal X-ray shows marked free air. (C) On the 9th day of life, plain abdomen X-ray shows pneumoperitoneum. (D) Jackson-Pratt tube inserted into the peritoneum. (E) Plain abdomen on the 20th day of life. Abdominal distension is markedly decreased, and subdiaphragmatic free air is almost disappeared. Jackson-Pratt tube was removed the next day.

count, biochemical analyses, and C-reactive protein levels were within normal limits. An abdominal X-ray demonstrated a large amount of free intraperitoneal air (Figs. 1A, B).

Primary surgical repair was performed, and gastric perforation was diagnosed. Multiple perforation lesions were visible in the lesser curvature, and gastric tissue was extremely weak. Several bubbles were found nearby the perforated portion, bleeding and inflammation were observed at the perforation site. After surgery, urine output was decreased and systemic edema was observed. Disseminated intravascular coagulation (DIC) syndrome[10] occurred with acute renal failure symptoms. Three days after surgery at hospital day 9, she vomited intestinal content with blood. Abdominal X-ray showed free air in the peritoneal cavity. At that day, her body temperature was 36.2-37.0℃, the heart rate was 140-200 beats / min, respiratory rate was 35-50 breaths / min, mean blood pressure was 21-25 mmHg (systolic, 27-30 mmHg; diastolic, 17-22 mmHg) and oxygen saturation was 95-100%. On hospital day 9, peripheral blood results as follows: white blood cell count 18,760/ μL, hemoglobin 6.8 g/dL, platelets 70,000/μL, PT 118.5 s, aPTT 21 s, D-dimer 8.01 μg/mL, and C-reactive protein 2.26 mg/dL. However, re-operation was impossible because of severe hypotension and DIC.

Ultrasonography-guided percutaneous insertion of a Jackson Pratt tube was performed into the moderate-sized cyst. Hemo- and pneumoperitoneum at the posterior aspect of the stomach lower body and antrum were identified by abdominal sonography (Fig. 2). Drainage of the free air, saliva, and gastric content was observed from the intermittent suction by nasogastric tube. On the 14th day of hospitalization, with recovery in the general statue of the patient, a follow-up abdominal X-ray demonstrated complete resolution on the free air and the presence of gastric gas shadow. Percutaneous drainage was terminated on the 22nd postnatal day. Overall, the patient was mechanically ventilated for 33 days. Feeding with mother's milk was started on the 36th postnatal day. The patient tolerated oral feeding, showed an increase in weight, and remained in good health. She was transferred to other center for operation of progressing hydrocephalus with severe intraventricular hemorrhage on the 59th postnatal day. The patient was 33^{+5} weeks of gestation, had 1,756 g of body weight.

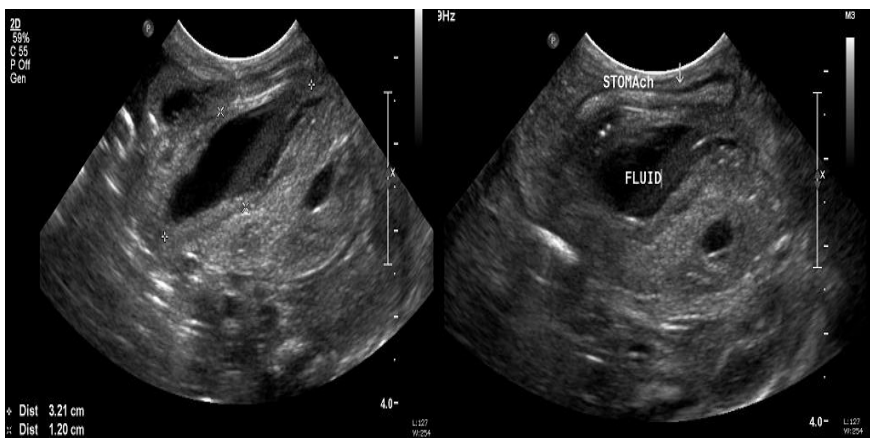

Fig. 2. Hemo- and pneumoperitoneum at the posterior aspect of the stomach lower body and antrum were identified by abdominal sonography.

Discussion

Neonatal gastric perforation was first described by Siebold in 1825.[9] The first successful surgical repair was reported by Kellogg et al in 1951.[8]

Three mechanisms have been proposed for stomach perforation: traumatic, ischemic, and spontaneous.[9, 13–15]

Ischemic necrosis due to hypoxia, perinatal distress and shock, and extensive resuscitation performed postnatally may cause gastric perforation. Dexamethasone and indomethacin may also cause gastric perforation in newborns.[3, 4, 15] *E. cloacae* was detected in the cervical smear of our patient' s mother, and she was diagnosed with chorioamnionitis. Also mechanical injury by a rigid nasogastric tube could not be excluded as the cause. In our case, immaturity of the patient, positive pressure ventilation, intravenous ibuprofen use, perinatal distress may have acted as a combination of factors.

The peak incidence of gastric perforation is reported to be from the 2nd to the 7th day of life.[16, 17] Miller found that gastric acidity in a newborn child was maximal at 24 h of age; the acidity decreased over the following 9 days, by which point it approached the normal level for a child.[12] Thus, gastric acidity is exceptionally high during the first week of life, which is the period when the incidence of gastric perforation peaks. While this temporal association does not prove causality, it is possible that high gastric acidity in the early newborn period contributes to gastric perforation.[8] The most common site of gastric perforation is the greater curvature (more than 50% of all cases), followed by the lesser curvature and anterior wall of the stomach. In our patient, the perforation was at the lesser curvature.

In the present case, intestinal perforation can be excluded for 2 points. A marked free air and pneumoperitoneum with small bowel distension in the subdiaphragmatic area was observed on both plain and lateral abdominal X–ray films (Fig. 1A, B). Also, when the Jackson–Pratt catheter was inserted, free air, gastric content, and blood were drained. Bile or intestinal content was absent, similar to the case reported by Aydin et al.[18]

The mortality rate of gastric perforation varies between 27% and 83%.[12] Sepsis and respiratory distress frequently accompany clinical conditions in premature infants, which increases the mortality.[13] There are no gender differences in newborns. Almost 60% of gastrointestinal perforation cases have been reported in premature infants. The mortality of infants with gastric perforation is also higher among premature and low–birth–weight neonates than in full–term babies.[2]

Sudden abdominal distension and respiratory distress are the most common findings in gastric perforation. These two clinical findings are seen in our case. Other symptoms and signs such as feeding intolerance, acidosis, and shock also can be found.18 On the abdominal X–ray, subdiaphrgmatic free air and small bowel distension is seen in Fig. 1.

The time between diagnosis and operation affects the survival rate. After the onset of symptoms of the disease, the time to finish a large impact operation is limited. Because of the rapid progress of gastric perforation, early diagnosis and treatment are very important.[9] Primary surgical repair is the most important treatment in gastric perforation. Mortality and morbidity increases as the time to begin the surgery delays. Perforations in other parts of the intestines may occur as sequelae in the first few days after surgery.[6]

As mentioned above, primary surgical repair is the major treatment of neonatal gastric perforation, there is a case report of improved isolated gastric perforation without surgical repair.[18] In this case, when the second gastric perforation occurred, the patient had generally unstable vital signs and under DIC status. She had high risk for re-operation. Conservative management was chosen, Jackson-Pratt tube was inserted. As in this case, ultrasono-guided needle insertion could also be a method of treatment when loculation is found at the site where a pseudocyst was located.

In conclusion, gastric perforation in our patient probably developed because of gastric distension as a result of positive pressure ventilation; prematurity, perinatal distress, intravenous ibuprofen, and mechanical injury by a rigid nasogastric tube may have also played a role. Gastric perforations in newborn infants could be improved by percutaneous peritoneal drainage.

References

1) Jung YS, Kim SY. Neonatal spontaneous gastric perforation caused by Group B streptococcus (GBS) chorioamnionitis. Korean J Perinatol 2007;18:438-42.
2) Lin CM, Lee HC, Kao HA, Hung HY, Hsu CH, Yeung CY, et al. Neonatal gastric perforation: report of 15 cases and review of the literature. Pediatr Neonatol 2008;49:65-70.
3) Winn HN. Group B streptococcus infection in pregnancy. Clin perinatol 2007;34:387-92.
4) Duran R, Inan M, Vatansever U, Aladag N, Acunas B. Etiology of neonatal gastric perforations: review of 10 years' experience. Pediatr Int 2007;49:626–30.
5) Kawase Y, Ishii T, Arai H, Uga N. Gastro-intestinal perforation in very low-birthweight infants. Pediatr Int 2006;48: 599-603.
6) Rhim SY, Jung PM. Clinical study of neonatal gastric perforation. J Korean Assoc Pediatr Surg 2005;11:123-30.
7) Leone RJ Jr, Krasna IH. Spontaneous neonatal gastric perforation: is it really spontaneous? J Pediatr Surg 2000; 35:1066-9.
8) Kellogg HG, Abelson SM, Cornwell FA. Perforation of the stomach in the newborn infant; a report of survival. J Pediatr 1951;39:357-62.
9) Thelander HE. Perforation of the gastrointestinal tract of the newborn infant. Am J Dis Child 1939;58:371-93.
10) Fischer D, Schloesser R, Buxmann H, Veldman A. Recombinant activated factor VII as a hemostatic agent in very low birth weight preterms with gastrointestinal hemorrhage and disseminated intravascular coagulation. J Pediatr Hematol Oncol 2008;30:337-42.
11) Miller RA. Observation on the gastric acidity during the first month of life. Arch Dis Child 1941;16:22-30.
12) Yoon JM, Lim JW, Cheon EJ, Ko KO, Mok WK. The case of pseudocyst formation after spontaneous neonatal gastric perforation. J Korean Soc Neonatal 2006;13:273-7.
13) Chouteau W, Green DW. Neonatal gastric perforation. J perinatol 2003;23:345-7.
14) Pochaczevsky R, Bryk D. New roentgenographic signs of neonatal gastric perforation. Radiology 1972;102:145-7.
15) Nagaraj HS, Sandhu AS, Cook LN, Buchino JJ, Groff DB. Gastrointestinal perforation following indomethacin therapy in very low birth weight infants. J Pediatr Surg 1981;16:1003-7.
16) Gillesby WJ, Wheeler JR. Acute gastric dilatation. Am Surg 1956;22:1154-62.
17) Saracli T, Mann M, French DM, Booker CR, Scott RB. Rupture of the stomach in the newborn infant: report of three cases and review of the world literature. Clin pediatr (Phila) 1967;6:583-8.
18) Aydın M, Zenciroğlu A, Hakan N, Erdoğan D, Okumuş N, İpek MS. Gastric perforation in an extremely low birth weight infant recovered with percutaneous peritoneal drainage. Turk J Pediatr 2011;53:467-70.