Delayed laparotomy and gastric repair in gastric perforation: a case report in the neonatal patient

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ABSTRACT

Background: The incidence of gastric perforation in neonates is rare but has a high mortality rate of 15-70%. Spontaneous gastric perforation in neonates is estimated to occur in one of 2,900 live births and accounts for approximately 7% of all gastrointestinal perforations in neonates and children.

Case: We present a case of gastric perforation complicated by sepsis, anemia, thrombocytopenia, and hypoalbuminemia in a 3-day-old girl. The patient was brought to the emergency room of Dr. Soetomo Hospital Surabaya with blood vomiting complaints from 1 day. On physical examination, it was found that the general condition was weak, with an axillary temperature of 38.2°C. The abdomen looked distended from inspection of the abdominal region, with the dilated vein and periumbilical hyperemia. Its percussion revealed the loss of liver dullness. The results of laboratory examinations: Hb 10.7 g/dl, Platelet 14,000/mm³, Albumin 2.6 g/dl. Plain thoracoabdominal X-Ray study showed a pneumoperitoneum. Needle decompression was performed because there were signs of Abdominal Compartment Syndrome (ACS). After five days of optimization in the Intensive Care Unit, a laparotomy exploration and primary repair of gastric perforation were conducted.

Conclusion: Gastric perforation in neonates is a rare condition. The management principle for each patient is different depending on the patient’s condition. Needle decompression is indicated when ACS is present or the patient’s condition is not optimal for surgery. Definitive laparotomy can be done after the patient’s condition has been stabilized. Proper selection of treatment can improve the patient’s outcome and reduce the risk of morbidity and mortality.

Keywords: Gastrointestinal perforation, neonates, complicated.

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BACKGROUND

Gastric perforation in neonates is one of the rare cases of neonatal emergency. The mortality rate is 22.2% to 75%.1 Spontaneous gastric perforation is found in only 7% of all gastrointestinal perforations and is estimated to be 1 in 2900 births.2 In the Indian study, only about 5% of all pneumoperitoneum in neonates was caused by gastric and duodenal perforation.3 The Japanese study recorded only 11 cases of gastric perforation in neonates from 1991 to 2010.4 The gastric perforation in neonates results from ischemic, traumatic or spontaneous reasons. Ischemic necrosis is a side effect of the body’s defense mechanisms due to perinatal stress, hypoxia, or shock. Traumatic causes are mostly iatrogenic, from manual ventilation pressure via excessive bag-mask, esophageal intubation or gastric tube injury.5,6 Gastric perforation often occurs in premature infants with a history of asphyxia or hypoxia and emesis that may contain blood. Clinical symptoms may include sudden and progressive abdominal distension, respiratory distress, hemodynamic instability and eventually shock.7,8 The purpose of this writing is to report a case of gastric perforation with sepsis in a 3-day-old neonate.

CASE REPORT

A 3-day-old baby girl was brought to the Emergency Department of Dr. Soetomo Hospital from a private hospital because of generalized peritonitis. The patient vomited blood from the age of 1 day. She vomited after drinking breast milk, non-projectile and contained blood clot mixed with breast milk. Defecated 12 hours after birth. She was born by Cesarean Section because of her mother’s previous Cesarean history. Her birth weight was 2630 grams, gestational age 34 weeks, Apgar scores 5-6, not immediately crying, with a history of asphyxia.

On physical examination, it was found that the general condition was weak with a heart rate of 152 beats/ minute, rapid
CASE REPORT

Gastric perforation complicated by sepsis with pancytopenia, hypoalbuminemia, and electrolyte imbalance. The presence of respiratory distress syndrome’s sign, the tendency of abdominal compartment syndrome (ACS), severe sepsis and preshock condition did not allow this patient to undergo definitive exploratory laparotomy. Initial management for this patient consists of oxygenation, decompressive oral gastric tube and urinary catheterization, accompanied by broad-spectrum antibiotics: Ampicillin, Gentamicin, and Metronidazole intravenously. To prevent ACS reduced the intra-abdominal pressure, a needle decompression using a 16G needle with local anesthesia was conducted. Stabilization and optimization measures were continued in the Neonatal Intensive Care Unit (NICU).

After five days of treatment in the NICU, the optimal condition was achieved with heart rate 148 beats/ minute, respiratory rate 40 breaths/ minute, and temperature 36.7°C. Her full blood count also improved with Hb 13.1 g/dl and platelets 596,000/mm3. Her albumin level increased to 2.98, and her electrolyte went normal with sodium level of 140 mmol/L, potassium level of 5.2 mmol/L, and chloride level of 109 mmol/L. An exploratory laparotomy was then performed (Figure 2).

Intraoperatively obtained 200 ml reddish-brown peritoneal fluid, 3rd-grade inter-intestine adhesion, distended stomach covered with fibrin, and perforation in major curvature the stomach with 4 cm length, and 0.5 cm wide. Primary repair of gastric perforation was then conducted, and a subfascial drain at the incision site and an oral gastric tube were placed.

Postoperatively, the patient was treated at the NICU with a ventilator. Total parenteral nutrition was given parenterally via a central venous catheter. Medical therapy given includes the antibiotic ampicillin, gentamicin, metronidazole, paracetamol and omeprazole. On the second postoperative day, the ventilator was weaned off, and the patient could breathe spontaneously with oxygen support via a nasal cannula. On postoperative day 4, the patient was transferred to the incubator in the nursery room. The bowel movements were normal, her abdomen was not distended, and the oral gastric tube and drain were not producing. The patient’s condition remained stable until the 6th postoperative day, and then the patient began to have oral feeding gradually. During the treatment, there were no significant complications other than surgical wound infection found on postoperative day 6. The patient was sent home on postoperative day 17 in good condition and the surgical wound also improved (figure 3).

The patient was monitored up to 3 years post-surgery, and the patient had no complaints. Her weight was gained following the standard growth chart, with the weight of 15kg. The child was healthy and active, as seen in figure 4.

DISCUSSION

Gastric perforation in neonates is a rare case. Khan et al’s study in India stated that only 16.5% of patients in the pediatric surgery department were treated with pneumoperitoneum, and gastric and duodenal perforations caused only about 5% of all neonatal pneumoperitoneum. Research at the University Teaching Hospital Nnewi, Nigeria, from January 2009 Until November 2012, there were only 4 cases of gastric perforation out of 16 cases of neonates with gastrointestinal perforations. A study in Japan from 4 institutions in Chiba recorded that there were only 11 cases of gastric perforation out of 16 cases of neonates with gastrointestinal perforations. Another study at Fukuoka University Hospital Japan stated that only 3.1% of

Figure 1. Plain thoracoabdominal X-Ray

Figure 2. Surgical Procedure of the patient
all pediatric surgery patients were due to gastrointestinal perforation, with only 11.8% incidence of gastric perforation compared to other gastrointestinal perforations. Gastric perforation in neonates occurs due to ischemic, traumatic, or spontaneous reasons. Ischemic necrosis is a side effect of the body’s defense mechanisms due to perinatal stress, hypoxia, or shock. A single perforation of the major curvature is associated with gastric overdistention. Obstruction of the distal stomach can also cause gastric overdistention that can lead to perforation. In neonates, there might be a congenital disability of the stomach’s circular muscle layer, most notably in the major curvature fundus, which is often the site of perforation. In this patient who had a history of respiratory distress and received assisted-ventilation via Continuous Positive Airway Pressure (CPAP), the perforation location was in the major curvature, so the authors suspected the cause of gastric perforation in this patient was overdistention due to ventilation. The histopathological study from the patient’s stomach tissue at the perforation site revealed that there was erythrocyte thrombus in the gastric product that corresponded to spontaneous gastric perforation without any evidence of H. pylori.

Clinical symptoms usually appear between days 2-7. Often occurs in premature infants who have a history of asphyxia or hypoxia and emesis that may contain blood. Clinical symptoms may include sudden and progressive abdominal distension and continuing respiratory distress, hemodynamic instability and shock. Initial management consists of fluid resuscitation, oxygenation, gastric decompression and urinary catheterization, and broad-spectrum antibiotics administration if possible, immediately carrying out exploratory laparotomy. Needle decompression of the abdominal cavity may be considered if there is respiratory distress due to abdominal distension. Perforation closure can be performed with or without an omental patch, but resection is required if there is extensive necrosis. Even in extreme cases, a total gastrectomy, gastric replacement, or esophagoduodenostomy is required.

CONCLUSION
Gastric perforation in neonates is a rare emergency. Perforation can occur through ischemic, traumatic, or spontaneous mechanisms. Clinical manifestations include pneumoperitoneum accompanied by metabolic acidosis. The management principle for each patient is different depending on the patient’s condition. Exploratory laparotomy is the definitive diagnostic and therapeutic procedure in this case. However, if the patient is in respiratory distress or sepsis condition, temporary decompression is an option. This disease has a high mortality rate, which can be suppressed through rapid treatment.

DISCLOSURES
Author Contribution
All authors contributed in designing and concepting the research, analyzing the results, and preparing published manuscript.

Conflict of Interest
All authors stated no conflict of interest regarding this research.

Ethical Statement
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