

Gastric Perforation in Two Neonates:
Spontaneous? Secondary to Feeding
Tube: A Case ReportPatil Prashant S^{1*}, Gupta Abhaya¹, Kothari Paras L¹, Kekre Geeta¹, Patil Shalil H¹,
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CC-BY 4.0**Keywords** Gastric Perforation; Neonate;
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Abstract

Gastric perforation in neonates is an uncommon entity. Definite causes are found in few patients. Iatrogenic perforation secondary to introduction of a hard nasogastric tube has been reported in literature. Majority of cases have no obvious reasons.

We present two cases of gastric perforation in preterm babies probably caused by nasogastric tube. One baby had pneumoperitoneum while other one did not show gas under diaphragm. Etiopathogenesis of gastric perforation in preterm babies is discussed along with a comment on Spontaneous Intestinal Perforation (SIP).

Introduction

Gastric perforation in neonates is a rare, serious and life threatening condition. It is associated with prematurity, low birth weight, Neonatal Intensive Care Unit [NICU] stay, Necrotizing Enterocolitis (NEC) [1]. About 115 cases of gastric perforation have been reported in the world literature from 1943 to 2000 [2]. SIP is the second most common cause of bowel perforation in neonates. Trauma due to stiff nasogastric tube causing gastric perforation in already compromised neonates is not uncommon entity and a high index of suspicion can clinch early diagnosis.

Case Report

A preterm baby, 1.5 kg female child, delivered at 34 weeks, referred from outside was admitted in our NICU. Baby had a vaginal delivery. There was no history of birth asphyxia. Baby had cried after birth. Neonatal resuscitation was not required. In view of prematurity and low birth weight baby was started on maintenance intravenous fluids. Nasogastric feeds were started gradually. On day five of life baby developed abdominal distension which increased after feeds. Hence feeds were stopped. Baby's general condition gradually deteriorated, baby became lethargic, tachycardia and tachypnea developed. Vasopressors were requiring maintaining mean arterial pressure. X-ray abdomen showed nasogastric tube passing up to pelvis [Figure 1]. Flanks were fluid filled as shown by ground glass opacity. There was no pneumoperitoneum. Baby's total leucocyte count dropped to 1200/cmm, serum creatinine raised to 1.7 mg/dl and serum potassium was 7.1 mEq/L. A glove drain was put in peritoneal cavity under local anesthesia. About 20 ml of purulent fluid and gas drained, abdominal distension reduced. Baby's vital parameters improved. Serum K improved to 5.2. Baby was posted for emergency exploratory laparotomy after stabilization. Baby was incubated keeping nasogastric tube in situ. On exploration peritoneal cavity had contaminated fluid. Nasogastric tube was palpable in peritoneal cavity. It had come out through an opening near greater curvature of stomach in posterior wall [Figure 2]. Nasogastric tube was removed and gastric perforation was repaired in two layers. Rest of bowel was found to be healthy. A small sized [No.6] infant feeding tube placed as nasogastric tube. Baby was extubated on second post-op day and feeds were gradually started on fifth post-operative day. Baby was discharged on 11th post-operative day and is on follow-up for 1 month.

Second baby was a full term 2 kg male child who presented to us with history of abdominal distension since 1 day. Baby was admitted in NICU and nasogastric tube was put. However baby's abdomen distended more after feeding hence x-ray abdomen was done which showed pneumoperitoneum with feeding tube passing up to pelvis [Figure 3]. Baby was explored in emergency. Gastric perforation on body of stomach was found [Figure 4]. It was repaired in two layers. Rest of bowel was found to be healthy. Baby had an uneventful recovery and discharged on 7th post-operative day.

The histopathology reports in both cases (Frozen Section couldn't be done as it is not available in emergency at our institute) did not reveal Hirschsprung's disease or NEC.



Figure 1: X-ray abdomen showing nasogastric tube passing up to pelvis, no evidence of pneumoperitoneum.



Figure 3: X-ray abdomen showing gross pneumoperitoneum and nasogastric tube passing up to pelvis.

Discussion

Various mechanisms have been proposed for gastric perforation, but in most of cases, etiology is still unknown. Iatrogenic trauma by vigorous nasogastric or gastric tube placement has been described [3]. Prematurity, vigorous resuscitation, nasal CPAP, perinatal stress, perinatal hypoxia-ischemia, and distal obstruction might cause spontaneous perforation [4]. Though seen frequently in pre-term newborns with Very Low Birth Weight (VLBW) and Extremely Low Birth Weight (ELBW), only a few cases have been described in full-term newborns [5]. Ischemic gastric perforations occur in patients with necrotizing Enterocolitis. Spontaneous gastric perforation is more common in preterm baby. Maximum reported incidence of rupture is on 3rd day of life [6].

Pneumoperitoneum is usually an indication of perforated hollow viscera and requires urgent surgical intervention. NEC is the most common cause of pneumoperitoneum in premature neonates [7].

Stress, hypoxia, or shock may lead to regional hypo-perfusion and transient intestinal ischemia resulting in SIP. SIP is the second most common cause of neonatal intestinal perforation [8] and has been very well documented in the low-birth-weight neonates [9, 10]. Premature rupture of membranes, lower Apgar scores, and cardiovascular resuscitation in the perinatal period may prone the neonate to SIP. The terminal ileum is mostly affected site; however, SIP is also reported in the transverse and descending colons [11]. Its incidence is 1.1% in VLBW & 7.4% in ELBW neonates.

The clinical profile of first patient fits within the spectrum of SIP. The inciting trauma of nasogastric tube might have caused gastric perforation in a preterm, low birth weight baby. However stomach is an unusual site of perforation in SIP.

Second baby was a 2 kg full term neonate presenting with gastric perforation. However gastric perforation cannot be attributed simply to nasogastric tube as most babies tolerate feeding tubes well. There



Figure 2: Intra-operative finding of nasogastric tube coming out of gastric perforation.



Figure 4: Intra-op finding of gastric perforation.

must have been an episode of stress, hypoxia, or shock leading to regional hypo-perfusion and transient ischemia resulting in SIP in stomach, again an uncommon site of SIP in neonate.

Prompt surgical intervention with debridement and two layers closure of gastric perforation is recommended. Delay in surgery will result in higher mortality. Postoperative management includes broad spectrum intravenous antibiotics, mechanical ventilation, and Vasopressors support, transfusion of blood or blood products. In very sick infants, initial external peritoneal drainage, like in our case, followed by surgical repair of the perforation once the infant's condition stabilized improves outcome. Mortality rate of gastric perforation is high in premature infants due to the associated problems of sepsis and respiratory failure. For better outcome, interval between starting of symptom and definitive surgical intervention should be minimum.

Recently, Peritoneal Drainage (PD) has been used in very sick neonates with perforation caused by NEC, for whom general anesthesia and laparotomy are risky. PD may provide temporary stabilization and recovery, but most of these infants require subsequent laparotomy. [12,13]

PD has also been reported to provide successful and definitive treatment for many premature infants with isolated intestinal perforation. There is a small subset of patients with mild abdominal distension (e.g., less free air, less free fluid) and minimal or absent peritoneal signs similar to our patients, who are possible candidates for expectant line of treatment neither requiring laparotomy nor drainage. In the absence of peritoneal signs, we did not institute peritoneal drainage for our patients and observed them in the intensive care unit setting. Therefore, the neonates with pneumoperitoneum require a proper clinical and the radiographic correlation to establish the aetiology of perforation and the clinical picture should guide the therapy as happened in our patients. However, additional radiological evidence of air-fluid levels may also warrant a surgical intervention. Therefore, the absolute indications for surgery are established precisely when perforation is suspected, according to the abdominal signs, blood parameters including pH, and radiographic findings thereby avoiding some unnecessary explorations and peritoneal drainage.

These two cases are likely to be SIP because:

1. X-Ray: No evidence of portal venous gas or pneumatosis intestinalis.
2. Intra-operatively: Healthy bowel apart from the perforation site and no evidence of distal obstruction.
3. At Histopathology: No evidence of NEC.
4. Outcome: Good.

Conclusion

Any new born child, especially preterm or low birth weight, having progressive abdominal distension with or without pneumoperitoneum, diagnosis of gastric perforation should be kept in mind and early resuscitation, stabilization and surgical exploration is to be undertaken for better outcome. Primary peritoneal drainage can serve as primary management for a very sick neonate to gain time for stabilization or it alone can treat the patient. A distinction between SIP and NEC is important for management and outcome considerations.

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