

Gastric perforation in the newborn

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LA TUNISIE MEDICALE - 2013 ; Vol 91 (n°07) : 464-467

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R É S U M É

Prérequis : La perforation gastrique des nouveau-nés est une affection rare. Elle pourrait être spontanée, mais une cause contributive doit être recherchée.

But : Evaluer notre expérience dans le traitement de 8 nouveau-nés hospitalisés pour perforation gastrique au cours des 20 dernières années.

Méthodes : Nous avons examiné les dossiers de tous les nouveau-nés admis dans notre hôpital entre 1990 et 2010 en ce qui concerne le sexe, l'âge à l'admission, les facteurs contributifs, les anomalies associées, le site de perforation, le type de l'intervention et les suites opératoires.

Résultats : Sur les huit nouveau-nés, cinq étaient de sexe masculin. Le poids moyen était de 2130 g. Quatre bébés étaient prématurés. Trois enfants ont été ventilés pour des difficultés respiratoires. Cinq patients avaient des anomalies associées. La perforation a intéressé la petite courbure dans 4 cas et la grande courbure dans 3 cas. Tous les patients ont bénéficié d'une gastrorraphie. La mortalité était de 75% (6 enfants).

Conclusion : Une prise en charge périnatale avec un traitement précoce des pathologies primaires et la protection de l'estomac contre la distension des nouveau-nés à risque sont essentiels dans la gestion de la perforation gastrique néonatale.

S U M M A R Y

Background: Gastric perforation in neonates is an uncommon condition. It could be spontaneous but a contributing cause should be sought.

Aim: To review our experience of treating 8 neonates with gastric perforation over the past 20 years.

Methods: We reviewed the records of all newborns admitted to our hospital between 1990 and 2010 with regard to gender, age at admission, contributing factors, associated anomalies, site of perforation, type of operation, and outcome.

Results: Of the eight neonates, five were female and three male. The average weight was 2130 g. Four babies were premature. Three infants were ventilated for respiratory difficulty. Five patients had associated anomalies. Perforation occurred in the lesser curvature in 4, at the greater curvature in 3, and at the anterior antrum surface in 1. All patients were treated with gastrorrhaphy. Four neonates required additional gastrostomy. Mortality was 75% (6 infants).

Conclusion: Active perinatal management, early treatment of primary pathologies, and protection of the stomach against distension in neonates at risk are essential in the management of neonatal gastric perforation.

M o t s - c l é s

Perforation gastrique, nouveau-né

Key - words

Gastric perforation, Neonate

Gastric perforation in neonates is an uncommon condition. It could be spontaneous but a contributing cause should be sought. The high mortality rate in such patients could be improved by early diagnosis and prompt resuscitation, followed by surgery. The aim of this study is to report our experience of treating 8 neonates with gastric perforation over the past 20 years.

PATIENTS AND METHODS

A retrospective review was conducted of 8 cases of gastric perforation seen over a 20-year period on the pediatric surgical service of CHU-Fattouma-Bourguiba- Monastir Tunisia. Three babies were transferred from neonatal intensive care unit setting. At admission, the infants underwent serial physical examinations and laboratory and radiographic evaluation. All were treated with broad-spectrum antibiotics and nasogastric suction. Oral feeding was prescribed. Intravenous infusions to correct hypovolemia and electrolyte imbalance were immediately initiated and exchange transfusions were given to correct coagulopathies. Gastric perforation was diagnosed at operation, with the preoperative diagnosis being only perforation of a viscus.

RESULTS

The diagnosis of gastric perforation was made at an average age of 2.4 days (range, 1 to 4 days). The average weight was 2130 g (range, 1050 to 2950 g). There were 5 girls and 3 boys. Four babies were premature ranging in gestational age from 29 to 36 weeks and weighting 1050 to 2050 g. Three premature had one minute APGAR scores of less than five. They had had asphyxia and required mechanical ventilation at delivery after bag-mask ventilation. Two others infants (full-term baby and premature) underwent brief bag-mask ventilation for respiratory difficulty. Three premature were treated at neonatal intensive care unit for maternofoetal infection (2 cases) and for nosocomial infection (1 case). The initial clinical finding was abdominal distention in all cases. Two full-term neonates have hematemesis, it was associated with subcutaneous emphysema in one case. Symptoms begun on the first three days of life in 7 cases. X-ray examination showed massive pneumoperitoneum in all cases (figure 1) Surgery was performed urgently after a brief resuscitation. In all cases the exploratory laparotomy revealed intraperitoneal hemorrhage. Fresh thrombi were seen in the stomach. Necrosis was localized to the lesser gastric curvature (figure 2) in 4 cases, to the greater curvature in 3 cases and, in one case to the huge rent anterior antrum surface. In no case was there evidence of necrotizing enterocolitis. The perforation site was large more than 1 cm in diameter in 7 cases and situated within a large area of necrosis and muscular disruption. The surrounding gastric mucosa was congested, hemorrhagic, and showed sharply-defined demarcation lines. Associated malformations were noted such as duodenal atresia (2 cases), pyloric atresia (1 case), esophageal atresia with tracheoesophageal fistula (1 case) and malrotation (1 case). The patients who have duodenal atresia and pyloric atresia

presented a dismorphic face that evokes mongolism. The edges of the perforation were debrided and the defect closed in two or three layers. Four neonates had a gastrostomy and the associated malformations were managed at the same time. There were six deaths (3 premature and 3 full-term infants) in the early post-operative period (5 cases) and after five days of operation (1 case) as a result of multiorgan failure. No complication was seen between the survivors during 4 years of follow up. The histopathologic evaluation demonstrated areas of transmural necrosis surrounded by congestive processus muscular disruption at the perforation site. No bacteria were found in the submucosa and there was no inflammatory- cell infiltration.

Figure 1 : Upright film of the abdomen showing massive pneumoperitoneum

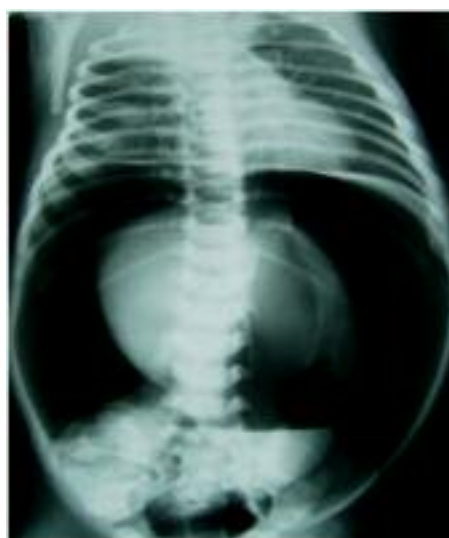


Figure 2: Gastric perforation of lesser curvature

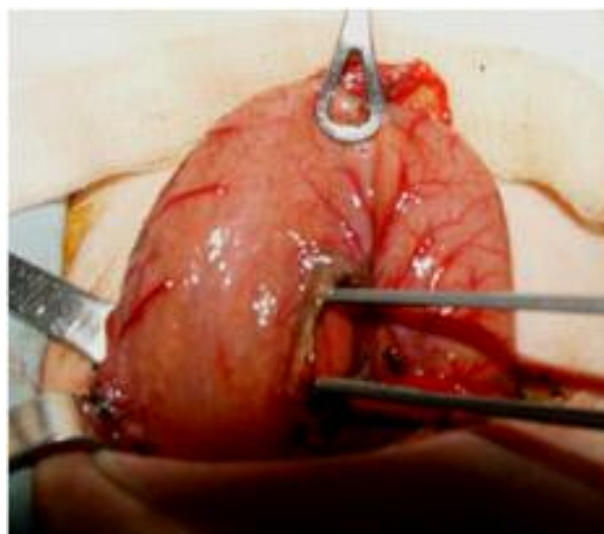


Table 1: Finding in 8 patients with Gastric Perforation

| Case N° | Age (week) | sex | Weight (g) | Day of life perforation | Mask Ventilation intubated | Location of Gastric Perforation | Associated Pathologies | Survival Died |
|---------|------------|-----|------------|-------------------------|----------------------------|---------------------------------|------------------------|---------------|
| 1 | 29 | F | 1200 | 2 | Yes | Lesser Curvature | No | Died |
| 2 | 31 | M | 1050 | 4 | Yes | Greater Curvature | Nosocomial Infection | Alive |
| 3 | 32 | M | 1900 | 3 | Yes | Anterior | MFI | |
| 4 | 36 | F | 2050 | 2 | Yes | Greater Curvature | No | Died |
| 5 | 38 | M | 2500 | 1 | No | Greater Curvature | Mongolism | Alive |
| 6 | 39 | F | 2950 | 3 | No | Lesser Curvature | Duodenal Atresia | Died |
| 7 | 39 | F | 2650 | 2 | No | Lesser Curvature | Mongolism | Died |
| 8 | 40 | F | 2800 | 2 | No | Lesser Curvature | Pyloric atresia | Died |
| | | | | | No | Lesser Curvature | MFI | Died |
| | | | | | No | Lesser Curvature | EA with TEF | |
| | | | | | No | Lesser Curvature | Mongolism | Died |
| | | | | | No | Posterior Gastric wall | Duodenal Atresia | |
| | | | | | | | Malrotation | |

DISCUSSION

Gastric perforation is a rare abdominal catastrophe with high mortality, usually occurring in neonatal intensive care unit setting (1, 2). Three mechanisms have been proposed for stomach perforation: traumatic, ischemic and spontaneous. The specific etiology of a gastric perforation may be difficult to determine because infants are usually sick and actual pathology yields few clues. Most gastric perforations are due to iatrogenic trauma. The most common injury is caused by vigorous nasogastric or orogastric tube placement. Traumatic gastric perforation may develop as a result of severe gastric distention during the course of positive pressure ventilation during bag-mask resuscitation or mechanical ventilation for respiratory failure (2). Increasing hydrostatic pressure due to various factors such as tracheoesophageal fistula or distal obstruction like pyloric atresia, duodenal obstruction from atresia, midgut volvulus, meconium and Hirschsprungs, have been cited by several authors (3-5). In more recent series we see increased numbers of gastric perforation occurring in cases of tracheoesophageal fistula or duodenal obstruction (5, 6). The mechanism of ischemic perforation has been difficult to elucidate because these cases of perforation are associated with conditions of severe physiologic stress such as extreme prematurity, sepsis and neonatal asphyxia. Ischemic gastric perforations have been noted in conjunction with necrotizing enterocolitis. Because gastric stress ulcers have been reported in a variety of critically ill infants, it has been proposed that these perforations result from the trans mural necrosis of such ulcers. Gastroduodenal perforation has been associated with postnatal steroid therapy to prevent or treat bronchopulmonary dysplasia

(1). Neonatal gastric perforation could occur spontaneously without any associated gastrointestinal conditions and was originally thought to be caused by a congenital absence of gastric musculature. It was the most common type of non obstructive perforation. Spontaneous gastric rupture was more common among premature, low birth weight, black male infants. Furthermore, there was usually a history of perinatal stress. The development of spontaneous neonatal gastric perforation is associated with the decreased quantity of interstitial cell of Cajal and damaged gap junction structure of the stomach wall (7). The usual location was higher on the anterior wall at or near the greater curvature of the fundus. The most common time for the perforation was within the first week of life particularly in between the first 2 and 7 days of life (8). In our series, four full-term infants have mechanical factors, such as distal obstruction (3 cases) and tracheoesophageal fistula (1 case). Three premature were treated in neonatal intensive care unit for maternofoetal infection and they were ventilated at birth for respiratory difficulty. They have one minute APGAR scores of less than five. Spontaneous gastric perforation in these patients couldn't be excluded despite they haven't muscular defect on the histological examination. Mechanical disruptions were reported to have predilection for the greater curvature like spontaneous gastric perforation. In our series, the perforation of greater curvature was noted in two premature and in a full-term baby affected with duodenal atresia. These patients were ventilated at birth. The perforation of lesser curvature was found in 4 cases. It was associated with mechanical perforation in three cases. The clinical presentation of neonatal gastric perforation was not exclusive. The features included vomiting, abdominal distention, and respiratory distress. Signs of hypovolemic shock and sepsis complete the clinical picture.

The most common feature is several abdominal distension, such as in our series, but this is not pathognomonic for gastric rupture. Diagnosis of the gastric perforation is based on the x-ray film of the abdomen. The upright film will show the "saddle" or "football" sign due to massive pneumoperitoneum (9). Gastric perforation in newborn represents immediate surgical emergency. A nasogastric tube should be placed while prompt resuscitation is undertaken.

These infants may have rapidly progressive pneumoperitoneum with associated cardiopulmonary compromise because of the large size and proximal nature of the perforation. Prior to definitive surgical intervention, during the evaluation and resuscitation of the infant, needle decompression of the abdomen with large intravenous catheter may be required (1). Aydin et al suggested that isolated gastric perforations in extremely low birth weight infant may be improved with percutaneous peritoneal drainage alone without need for primary surgical repair (9). The optimal treatment involves debridement of the necrotic edges and a two-layer closure. However, the extent of necrosis sometimes will necessitate a partial or total gastrectomy (10). The posterior wall of the

stomach should be explored and multiple areas of injury must be excluded. Distal obstruction should be searched. Neonatal gastric perforation was associated with a high mortality rate like in our study (75 %). Due to the associated problems of sepsis and respiratory failure often found in premature infants, mortality rates of gastric perforations are high, ranging from 45 % to 58% (1). More recent reviews have suggested that the early diagnosis of neonatal gastric perforation can affect outcome favorably, because many aspects of critical care have affected our ability to correct metabolic and electrolyte derangements before they become irreversible. Given early diagnosis and modern hemodynamic monitoring equipment, significant fluid resuscitation for intravascular hypovolemia also has been shown to improve outcome (6).

CONCLUSION

Gastric perforation of the newborn is a rare, serious and life threatening problem. The etiology should be sought. Success in treatment depends on early diagnosis, prompt resuscitation and immediate surgical intervention.

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