

NEONATAL GASTRIC PERFORATION

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ABSTRACT

Gastric perforation in neonate is the least common type of gastrointestinal perforation. Here we report two such cases. Two boys of 4 days old presented with huge abdominal distention with signs of peritonitis and septicemia. Plain X-ray shows signs of perforation of gas containing hollow viscus. Emergency laparotomy revealed perforation of stomach with mid-gut volvulus and malrotation. Perforation repaired and malrotation corrected. Post-operative period was uneventful. The first patient after two years and second patient after one and half years follow up is well.

INTRODUCTION

Neonatal gastric perforation is a rare abdominal catastrophe with high morbidity and mortality. The first case of neonatal

gastric perforation was reported in 1825 by Siebold¹ Leger reported the first successful repair in 1950¹.

Though specific cause of gastric perforation may be difficult to determine because the infants are usually sick but most perforations are due to underlying pathology. There are three common possible mechanism of perforation: 1. Traumatic, 2. Ischemic, and 3. Spontaneous.² Obstruction distal to perforation, accidental gastric overinsufflation,³ drugs like indomethacine,^{4,5} Increased gastric acidity and gastric ulcer,^{6,7} congenital defects in muscular wall,⁸ etc are also the cause of neonatal gastric perforation.

Neonatal gastric perforations usually occur during the 4th to 5th day of life presented with huge abdominal distention of abrupt onset and rapidly progressive with sepsis and respiratory impairment. Gastric bleeding may occasionally coexist. Prematurity, low birth weight, episode of hypoxia are often present.¹¹

Plain x-ray abdomen is diagnostic. Other investigations are WBC and Platelet count, arterial blood gas analysis and pH measurement for assess the condition.

Initial management is rapid fluid resuscitation, blood transfusion, and correction of acidosis, antibiotics, nasogastric suction, maintenance of body temperature. Needle decompression of the abdominal cavity should be considered if respiratory compromise occurs. Definitive treatment is laparotomy followed by peritoneal toileting and repair of perforation.

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CASE REPORT

Case-1: A full term normally delivered four days old boy admitted in Chittagong Ma-Shishu and General Hospital with sudden abdominal distention, vomiting with reluctant of food for about 6 hours and no passage of stool for about 4 hours. No history of antenatal checkup. Patient was 2.5 kg in weight, non-icteric, no edema, no cyanosis, and moderate dehydration with mild respiratory distress without any cardiac anomaly.

Reflexes were normal Temperature was 101°F, Pulse 156/m, Respiratory rate-50/m.

Abdomen was distended, tender, tense and tympanic with obliteration of liver dullness. Plain X-ray shows collection of free gas under both dome of diaphragm. There were no hematological or biochemical abnormality.

After resuscitation by nasogastric tube, intravenous fluids and electrolytes, Intravenous antibiotics (Inj. Ceftriaxon and Metronidazol), Laparotomy performed under general anesthesia by supra umbilical transverse incision, which reveals volvulus of midgut and a linear perforation on posterior surface of the fundus of the stomach near greater curvature about 2 cm long with collection of fluids and gases in the peritoneal cavity. Perforation was due to distal obstruction. Volvulus was corrected by Lads procedure. Perforation repaired in single layer of interrupted stitch with 4/0 vicryl. After peritoneal toileting abdomen was closed layer by layer. Post-operative period was uneventful. On two years follow up patient is well.

Case-2: Another full term normally delivered male baby of four days old admitted in Chittagong Ma-o-Shishu and General Hospital with vomiting for about 10-12 hours and sudden abdominal distention for about 8 hours and absolute constipation. No significant antenatal history. Patient was 2.2 kg in weight, non-

icteric, no edema, no cyanosis, and moderate dehydration with moderate respiratory distress without any cardiac anomaly. Reflexes were normal. Temperature was 102° F, Pulse 180/m, Respiratory rate-60/m.

Abdomen was distended, tense and tympanic with obliteration of liver dullness. Plain X-ray shows collection of free gas under both dome of diaphragm. There were no hematological or biochemical abnormality.

After resuscitation by nasogastric tube, intravenous fluids and electrolytes, Intravenous antibiotics (Inj. Ceftriaxon and Metronidazol), Laparotomy performed under general anesthesia by supra umbilical transverse incision. There were volvulus of midgut and a large perforation on anterior surface of pyloric part of stomach of about 3 cm in diameter with huge collection of fluids and gases in the peritoneal cavity. Volvulus was corrected by Lads procedure. Perforation repaired in two continuous layer stitch with 5/0 vicryl. After peritoneal toileting abdomen was closed layer by layer. Post-operative period was uneventful. On one and a half years follow up patient is well.

DISCUSSION

The incidence of gastric perforation has been quoted as 1:2900 live birth⁹ but is probably lower as very few institution have reported treating more than one or two cases per year. There is a slight male preponderance M:F= 1.5:1. About 40-50% are premature infants.¹⁰ A history of perinatal asphyxia and hypoxia often available but we have no such history.

Most gastric perforations are due to iatrogenic trauma⁸ and the most common injury is caused by vigorous nasogastric or orogastric tube placement² but we have no such history.

Ischemic gastric perforations have been noted in conjunction with necrotizing enterocolitis. But the actual cause of ischemic

perforation is difficult to elucidate because these cases are associated with severe conditions of physiological stress such as extreme pre-maturity, sepsis, and neonatal asphyxia but our patient have no such conditions.

Spontaneous gastric perforation have been reported in other wise healthy infants, usually within first week of life which may be due to congenital defect in the musculature of the stomach.^{12,13,15,17} There are also reports of idiopathic gastric perforation in neonates.¹⁶ Perforation may occur due to distal obstruction by pyloric atresia²⁶ pyloric stenosis,²⁷ duodenal atresia^{28,29,30}, anular pancreas,³¹ midgut volvulus³² and diaphragmatic hernia²⁰. Both of our patients of gastric perforation were due distal obstruction by midgut volvulus. Though neonatal gastric perforation usually present with rapidly progressing abdominal distention, but it may present with Surgical Emphysema,¹⁸ Scrotal Pneumatocoles¹⁹. Linkner and Benson¹⁴ stressed rapid fluid resuscitation, blood transfusion, correction of acidosis, antibiotics, nasogastric suction, maintenance of body temperature, and performance of only essential diagnostic studies and then laparotomy followed by peritoneal toileting and repair of perforation. About 10% cases have multiple perforations²⁰ but we have no such. Though we have no mortality rate, on review of literature we see that mortality rate varies from 0-88%^{21,22,23,24,25}. Early diagnosis and management before clinical deterioration of the metabolic status may improve the outcome of neonatal gastric perforation.

CONCLUSION

Neonate presenting with abdominal distension may have gastric perforation. A high index of suspicion, early diagnosis, prompt referral and management before clinical deterioration are the key factors for the survival of these neonates.

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