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Spontaneous Idiopathic Rupture of the Stomach in the Neonatal Period

By

J. Dénes, F. V. Lukács, J. Léb

Apáthy Children's Hospital, Budapest

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Five cases of spontaneous idiopathic rupture of the stomach in the newborn period are reported. Three of the reported cases were premature infants with a birth weight less than 2000 g. All the babies but one were operated upon within six hours after the first symptoms had been noticed. Two infants, a full-term baby and a premature survived the intervention. The others died of intracranial haemorrhage and delay of admission, and one of a iatrogenic damage caused by a barium meal. Immediate surgical intervention is mandatory, with adequate preoperative and postoperative management with fluids, antibiotics, and eventual parenteral feeding.

Spontaneous idiopathic rupture of the stomach in the newborn period is a clinical and pathological entity manifesting with a broad perforation of the stomach. It develops without any evident cause such as birth or resuscitation injury, gastric dilatation above an obstruction, compression necrosis caused by catheterization, or peptic ulcer. This rare disease of the newborn period was first described by Siebold [11] in 1825. Stern et al. [12] in 1929 reported the first, but unsuccessful surgical intervention and Léger et al. [3] in 1950 the first successfully operated case. Since that time, and particularly in the last few years, the number of reports has increased [1, 4, 8, 9, 10, 14] and the number of reported cases is estimated by Wilson at 143 [15], by Saracli et al. at 191 [9], by Schürer at 203 [10], by Mye and Kenneth at 210 [6]. The incidence of such cases may justifiably be assumed to be much higher than these figures suggest, since most of the unsuccessful operations and the cases revealed post mortem have not been reported. Some of the reported cases cannot be included among the idiopathic spontaneous ruptures because of a demonstrable mechanical causative factor such as trauma, distention necrosis or catheterization.

Prematures are frequently involved, in 40 to 65%. The symptoms appear during the first week of life, usually on the third day. Morger [5] reported a case of gastric rupture that occurred in intrauterine life, and Mye and Kenneth [6] reported one in a six-week-old infant.

Pathology

The alteration is characterized by a few cm long linear defect on the stomach wall, with margins which

are often necrotic. The usual site of the rupture is at or near the greater curvature and on the anterior wall. The lesser curvature and the posterior wall are less commonly involved and the perforation occurs more often in the cardia and fundus than on the antrum. According to SARACLI et al. [9] the rupture is more frequent in male than female babies; 65% of the cases collected by them were males.

The facts that the majority of the cases were recognized only at autopsy and survival was poor in a considerable number of operated cases, has afforded sufficient material for histologic study. Besides an absence of the gastric musculature, histological examination may reveal submucosal oedema, leukocytic infiltration, venous stasis and thrombosis and necrosis.

AETIOLOGY

The aetiology is unclear, but several factors are known which may lead to a rupture. A congenital absence of the gastric musculature, observed by many authors, is the most widely accepted cause. Histological examinations performed by KNEISZL [2] showed, however, that an absence or weakness of the gastric musculature often occurs in prematures and does not always lead to rupture. Muscular absence of the gastric wall is a common, even physiological, condition which, however, does not necessarily lead to perforation. He observed a muscular gap on the gastric wall in 15 prematures who died of an illness unrelated to the stomach. The fact, however, that the gaps observed by him coincide with the usual sites of rupture (greater curvature, anterior wall and cardia), suggests the possibility of their being one of the aetiological factors.

Some other factors such as intracranial haemorrhage that may cause gastric wall lesions, circumscribed circulatory disturbances at the site of the rupture, high gastric acidity in the first days of life (stress effect?) and septicaemia, may also induce spontaneous gastric perforation.

DIAGNOSIS

The clinical symptoms appear usually on the 2nd to 5th day after birth. The presenting signs are unconvincing. Later, vomiting with increasing abdominal distention associated with cyanosis and respiratory distress appear. The circulation deteriorates rapidly, tachycardia and tachypnoea set in. A bandbox sound is heard, with absent liver dulness. As a rule, an X-ray examination made because of the respiratory disturbances clarifies the correct diagnosis preoperatively. In an upright film of the abdomen, the quantity of free air present in the abdominal cavity is much greater than in the case of bowel perforation. The typical localization of free air was rightly characterized by PARRISH [7] as having the shape of a saddlebag.

THERAPY

The only therapy is early operation. There are sufficient data to show that

the greatest part of the surviving cases was operated upon within six hours after the first signs of a perforation had appeared, and the probability of survival decreases with every hour. Before operation, emptying of the stomach is essential, this procedure may alleviate the abdominal distention and the respiratory distress. If necessary, the tension pneumoperitoneum is reduced by abdominal puncture, often a life-saving measure in cases of severe respiratory disturbance. After opening the peritoneal cavity and draining the fluid and gastric contents, the perforation is explored, the necrotic parts excised and the opening closed with a two-layer suture. In the postoperative period, gastric suction, drainage, parenteral feeding and antibiotics are essential. Oral feeding can be instituted only when regurgitation has stopped and peristalsis has been restored.

Prognosis

Besides the timing of the operation, the prognosis depends on the birthweight, on the overall state of the neonate, on the occasional accompanying anomalies (these, however, are rare), and on other complications such as intracranial haemorrhage, pneumonia and sepsis. Of the recorded cases, nearly 30% survived. Mortality rate has decreased in recent years as a result of early diagnosis and of better operative and postoperative therapy.

Case Reports

Case 1. N. J., a four-day-old full-term male infant. Birth weight was 3000 g. He was transferred to us from another hospital, with a diagnosis of acute abdomen. Before transfer the baby had vomited repeatedly and distention and tenderness of the abdomen had been noted. A barium meal had been fed without performing a plain X-ray examination. On admission there was muscular rigidity and the abdomen was extremely distended. An upright X-ray film of the abdomen revealed a tension pneumoperitoneum as well as contrast material in the stomach, in the intestines and a considerable quantity in the free peritoneal cavity, mostly in Douglas's pouch (Fig. 1). There were signs of respiratory and circulatory distress. After preoperative preparation, suction drainage of the stomach, correction of fluid and electrolyte imbalance and blood transfusion, laparotomy was performed under general anaesthesia. Free air and bile-stained contrast material flowed at high pressure from the peritoneal cavity. An oblique rupture, 5 cm long, that reached almost as far as the cardia, was found on the anterior wall of the stomach and its two sides were necrotic. The necrotic gastric wall was excised and partly inverted by a two-layer suture. The peritoneal cavity was flushed with antibiotics then closed. Parenteral feeding and antibiotic therapy were

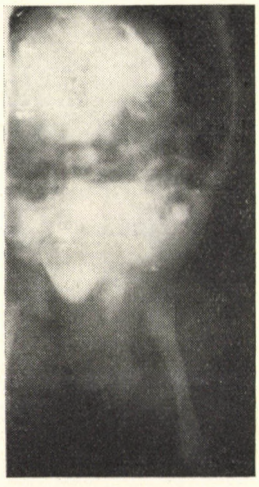

Fig. 1

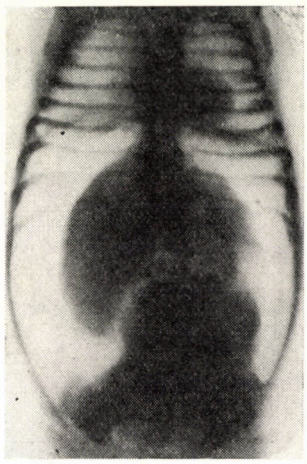

FIG. 2

introduced. The baby did well enough in the first few days, his abdomen became soft and he even passed a slight amount of faeces but later he developed a septic fever and died on the eighth postoperative day. At necropsy there was purulent peritonitis although the gastric closure was intact.

Case 2. N. T., a premature female infant birth weight 2200 g, was transmitted from the hospital for premature infants on the fourth day after birth. Her weight was then 1950 g and she was mildly icteric. She had vomited from time to time and,

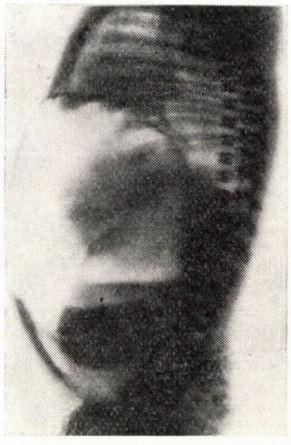

FIG. 3

five hours before admission, her abdomen suddenly became distended and as tense as a rubber ball. A plain X-ray film showed tension pneumoperitoneum (Figs 2 and 3). At laparotomy, a great quantity of free air, a slight amount of gastric contents and fibrin were found in the peritoneal cavity. There was a 1.5 cm rupture on the anterior wall of the stomach near the greater curvature, surrounded by a 3×2 cm ring of necrosis. The defect was closed in two layers and the peritoneal cavity was irrigated with a solution of antibiotics. On the first three postoperative days, the patient was fed parenterally. Jaundice decreased gradually and the postoperative course was uneventful.

Case 3. V. G., a male premature infant with scleroedema, birth weight 1300 g. At the age of two days, he had suddenly developed abdominal distention and tightness and his circulation and respiration deteriorated. A few hours later he was transmitted to our department where laparotomy was performed immediately. In the peritoneal cavity a large amount of free air and some gastric content was found. A tear of 1 cm in length with necrotic margins, was found on the anterior gastric wall near the greater curvature. The rupture was closed by a two-layer suture. The infant died on the next day of cardiorespiratory failure. At necropsy, diffuse peritonitis was found.

Case 4. O. L., a male premature infant with 1600 g birth weight was transmitted to us because on the third day of life abdominal distention had developed and the infant's condition deteriorated rapidly. On the first day of life there had been evidence of intracranial haemorrhage. A plain X-ray film showed free air in the peritoneal cavity with typical saddle-bag formation (Fig. 4). The baby was immediately operated upon. A rupture about 3 cm long and 1.5 cm wide with necrotic margins was noted below the cardia on the greater curvature and a large amount of free air and gastric content was found in the peritoneal cavity. The rupture was closed and the peritoneal

cavity was irrigated repeatedly with an antibiotic solution. On the third postoperative day the infant died with symptoms of peritonitis. Necropsy showed diffuse peritonitis and extensive intracranial haemorrhage.

Case 5. O. X., a full-term male newborn infant with 3500 g birth weight was sent to us from a country hospital. History of the confinement was as follows. An early rupture of the membranes caused amniotic fluid drainage and the mother had reported at the country hospital. A caesarean section had been performed because of protracted labour and a prolapsed umbilical cord. The newborn was born with algid asphyxia and had repeatedly convulsions. During the first day of life haematemesis had developed, with abdominal distention. After admission X-rays revealed a 3 cm layer of free air under the diaphragm (Fig. 5). Laparotomy was performed five hours after the rupture had been detected. Free air and bile-stained exudate was found in the peritoneal cavity and a 1.5 cm long rupture on the anterior wall of the stomach near the lesser curvature. The gastric wall was necrotic. Two-layer closure was done. On the third postoperative day the baby passed faeces and normal feeding could be instituted. Four days later severe septic symptoms presented, accompanied by jaundice and subcutaneous haemorrhages. Pneumonia and a second bout of abdominal distention and a small dehiscence in the incision developed. Ps. pyocyanea was cultured from the abdominal wound, from faeces and a throat swab. After prolonged antibiotic treatment the infant was discharged in full health at the age of seven weeks.

FIG. 5

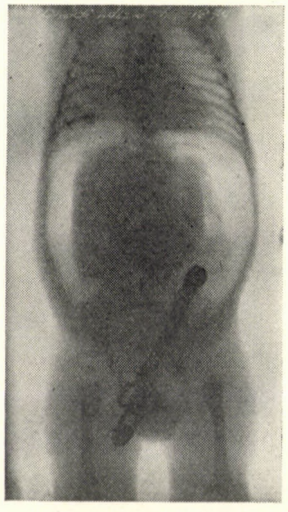

FIG. 4

DISCUSSION

The male versus female ratio was 4 to 1 in our case material and the ratio of prematures to full-term infants was 3 to 2. Birth weight of all the prematures was below 2000 g. The rupture ensued in two cases on the second day, in one case on the third day, in two other cases on the fourth day. In Case 5 it was noted as early as 16 hours after birth. All infants but one were admitted within six hours after the symptoms had been noticed.

The site of the rupture was in all cases on the anterior surface of the stomach near or on the greater curvature and nearer to the lesser curvature in one case only. The gap was on the proximal part of the stomach, in one case immediately below the cardia, and necrosis was seen in the close vicinity of the rupture in every case.

Of the five infants, two survived in spite of the fact that one patient was a premature infant and the other had a grave Ps. pyocyanea septicaemia in the postoperative period. One premature infant born with 1300 g who then displaced sclerooedema, died on the first postoperative day; the other premature (birth weight, 1600 g) had an extensive intracranial haemorrhage and died on the third postoperative day. In our opinion, the first baby could have been saved had he been transferred in time and had been fed no contrast material. This must have been the cause of the peritonitis that led to death eight days after the operation.

Concerning the aetiology, our cases failed to supply information. Intracranial haemorrhage was found in a deceased premature but also in a survivor. Early rupture of the membranes, and the possibility of amnionitis as the cause of septicaemia, occurred in Cases 3 and 5.

The only conclusion drawn from our cases is that early diagnosis and immediate surgical intervention may assure survival even in premature infants.

Dr. J. Dénes
Bethesda u. 3.
Budapest 14, Hungary

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