Spontaneous Linear Tears of the Stomach in the Newborn Infant

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Spontaneous linear tears in the stomach of the newborn infant can be lethal. While the etiology of this problem is unknown, pneumatic rupture of the stomach seems to be the most logical explanation for the gastric tear. The mechanism is much like Boerhaave's syndrome, the stomach being the target organ. Tremendous intragastric pressures may result because of incoordination and immaturity of the vomiting mechanism in the infant. The perforation occurs characteristically within the first seven days of life. Mortality is high, and surgical intervention is urgent. Three such patients have been successfully managed during the past 15 years. These patients are presented in detail, and the esophageal motilities in two of the survivors are presented. Pressure studies with rupture of cadaver stomachs and esophagi of newborn infants and adults are also presented in an effort to better understand the pathogenesis of this gastric catastrophy. Discussion of the diagnosis and management is also included in the presentation.

OVER 200 CASES of spontaneous tears of the gastric wall in the newborn have been reported in the American literature since Siebold reported the first case in 1825. This injury is highly lethal, and the pathogenesis is greatly debated. Congenital absence of the gastric wall musculature,[5] stress ulceration secondary to neurogenic difficulties,[10] and ischemia of the gastric wall secondary to vascular shunting[19] have been proposed as etiologic factors. Perforations of the stomach that occur proximal to obstruction, secondary to peptic ulceration, trauma associated with intubation, and accidental gastric insufflation, differ from the entity that we are discussing.

Over the past 14 years, three such patients have been cared for, and they form the basis of this report.

Clinical Observations

Two of the infants were full-term. The premature infant weighed 1,701 g. There were two female infants, and two of the infants were black. Premature rupture of the membranes occurred four days before delivery of the premature infant.

The two full-term infants were asymptomatic, but the premature infant was septic prior to rupture. Vomiting was the initial symptom in the two full-term infants. Respiratory distress, secondary to severe abdominal distention, was present in all of the infants.

The tear occurred between the third and fourth day of life and varied from 2 to 8 cm on length. It was single and anteriorly located in all infants. The greater curvature was involved in one infant and the midgastric wall in the remaining two. Roentgenograms of the abdomen demonstrated a pneumoperitoneum and confirmed the diagnosis of a ruptured viscus in all patients.

General anesthesia was used in all of the patients. Preoperative decompression of the abdominal distention with percutaneous catheterization was accomplished in one patient with relief of respiratory distress. The rupture was closed in two layers in all patients. The omentum was used to reinforce the repair in two. A gastrostomy tube was used to decompress the stomach postoperatively in one. Copious irrigation of the abdominal cavity with saline solution was carried out in all patients.

The postoperative course was uneventful in all patients. Perioperative antibiotics were used in all. Chalasia occurred in one infant but subsided in six months. Motility studies were obtained in two of the patients at 14 years and six months, respectively, and were all within normal limits.

Biopsy of the gastric wall adjacent to the rupture was accomplished in all patients. The microscopic changes were nonspecific. Edema, hemorrhage, necrosis, and inflammation were present in the two patients that were asymptomatic before the rupture. The mucosa was intact, and necrosis was present in the premature infant

Presented at the Annual Meeting of the Southern Surgical Association, December 8–10, 1980, The Breakers, Palm Beach, Florida.

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Submitted for publication January 26, 1981.

0003-4932/81/0600/0763 $00.80

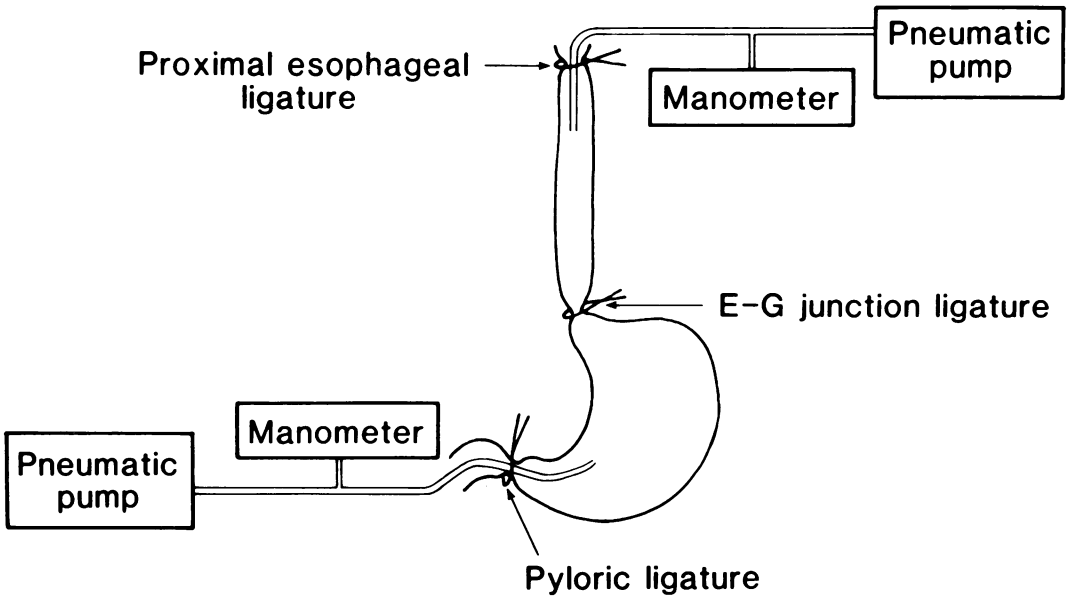

FIG. 1. Pneumatic rupture of cadaveric esophagus and stomach.

who was septic before the accident. Bacteria and microthrombi were not present in any of the biopsy specimens.

Experimental Observations

To elucidate the pathogenesis of neonatal gastric ruptures, 15 cadaver esophagi and stomachs were ruptured by inflation (Fig. 1). The ages ranged from one day to 26 years. Death in the 15 cases was unrelated to the upper gastrointestinal tract, and all specimens were tested within 24 hours of death. All newborn infants were full-term. The pressure needed to rupture the stomach varied from 3.4 pounds per square inch to 7 pounds per square inch (Table 1). Esophageal rupture occurred between 6.1 and 12 pounds per square inch.

TABLE 1. *Spontaneous Linear Tears of the Stomach in the Newborn*

Age	Pressure at rupture (lb/sq in)	
	Stomach	Esophagus
1 day	7.0	9.0
1 day	3.6	6.4
2 days	3.4	7.0
3 weeks	3.5	9.5
2 months	3.5	6.5
3 months	4.0	6.3
3 months	4.0	10.0
3 months*	4.0	6.0
3 months*	4.1	6.7
4 months	3.5	6.1
6 months	4.0	8.2
13 months	3.5	11.0
15 years	5.0	12.0
17 years	6.0	11.2
26 years	5.4	10.0

* Twins.

The pressure required to rupture the stomach was less than that needed to rupture the esophagus in all specimens.

Discussion

Pneumatic rupture of the stomach is the most logical explanation for the neonatal gastric tear.[14] Just how the elevated intragastric pressure develops is a puzzle. Some have proposed the "air-fluid trap syndrome."[8] The stomach that is affected by the air-fluid trap syndrome is much like a balloon that is distended with water and air and suspended between two fixed points. For rupture to occur as a result of this syndrome, several criteria are necessary: the infant must ingest a large volume of air and fluid, the infant must maintain a supine position, and angulation of the esophagogastric junction and duodenum must be severe enough to cause complete mechanical obstruction. It is theorized that if these criteria are met, progressive distention of the stomach will lead to rupture.

There is no question that the normal infant swallows a large volume of air during feedings. James and Shaker[8] aspirated 360 cc of air on an average from the infant's stomach in a six-hour period of time, and others[6] have collected as much as a liter of air over the same time period. In newborn infants who are supine, fluid accumulates in the fundus of the stomach and air collects in the antral end of the stomach, producing gastric distention. This distention is relieved when the infant is turned to a prone position or is positioned on his or her right side.[6] Eructation occurs more readily in this new position, and gastric distention is relieved.

Angulation of the esophagogastric junction occurs during gastric distention, but obstruction at this level is

less likely to occur in the newborn infant than in an adult, because the intraabdominal segment of the esophagus in the adult is longer and allows for more angulation.[6] In 80% of the newborn infants,[3,18] the intraabdominal segment of the esophagus is absent, and severe angulation of the esophagogastric junction is less likely to occur. Therefore, the esophagus enters the stomach at a more obtuse angle, and regurgitation occurs freely.[6] Since vomiting is the initial symptom in most infants with gastric rupture, occlusion of the esophagogastric junction does not seem likely.

Obstruction of the duodenum is thought to occur when the stomach is severely distended. Some have postulated that this condition is due to angulation of the duodenum over the vertebra, while others speculate that, with tension on the mesentary, the duodenum is secondarily compressed.[16] The obstruction that results is physiologic and not mechanical. This situation was demonstrated roentgenographically in one of our patients. On the day prior to rupture, a tremendous collection of air was noted in the stomach and small bowel. The obstruction created by angulation was not significant enough to impede gastric emptying, since gastric emptying is dependent upon the gradient between the intragastric pressure and the intraduodenal pressure.[9]

When gastric rupture occurs solely from overdistention, the tear characteristically is along the lesser curvature.[7] In a large series of newborn infants with gastric rupture, the greater curvature and anterior surface of the stomach were affected in 85–90% of the patients.[7,17] Kelly has shown that the stomach has the ability to accommodate to distention without an increase in intragastric pressure.[7]

The mucosa of the stomach may be injured[2] as a result of dilatation and the motility of the dilated stomach may be deranged,[16] but distention alone only contributes to and is not the primary cause of the high intragastric pressure in these infants. Incoordinated vomiting in the dilated stomach is postulated to be the most logical cause for the elevated intragastric pressure. To accept this theory, one must assume that the vomiting mechanism and swallowing mechanism are parallel in their development. The vomiting mechanism, which is complicated, is difficult to study in man. The peristaltic and emptying qualities of the stomach and esophagus have been studied extensively in the neonate.[3,18]

During normal vomiting, the cardia and upper stomach are dilated and displaced into the mediastinum.[1,20] The gastric contents are propelled through an open esophagus and out the oral cavity. Peristalsis in the esophagus of the newborn is incoordinated and does not become a coordinated movement until the third day of life.[3] Gastric peristalsis is abnormal in infants, and gastric emptying is slow.[4] Normal gastric motility does not occur until the third month of life, and full maturity may require 12 months.[3] The lower esophageal sphincter (LES) has a low tone at birth,[18] allowing for free reflux of gastric contents into the esophagus, and regurgitation occurs in over 40% of normal infants.[3] This problem is not overcome until the tone of the LES increases and this increase commences after the first week of life. Full competency, however, may not be evident until the first year of life.[3] The cricopharyngeal area is the least studied portion of the esophagus, but it normally contracts and opens during deglutition and vomiting. Any imbalance of its neurologic control may result in cricopharyngeal achalasia.[15]

It is postulated that because of immaturity and poor neurologic control in the newborn, the upper esophagus and cricopharyngeal areas fail to open properly during vomiting. Since the LES is incompetent, a tremendous pressure is exerted on the stomach (Fig. 2). Rupture occurs in that portion of the stomach that herniates into the mediastinum, where tension is greatest. The rup-

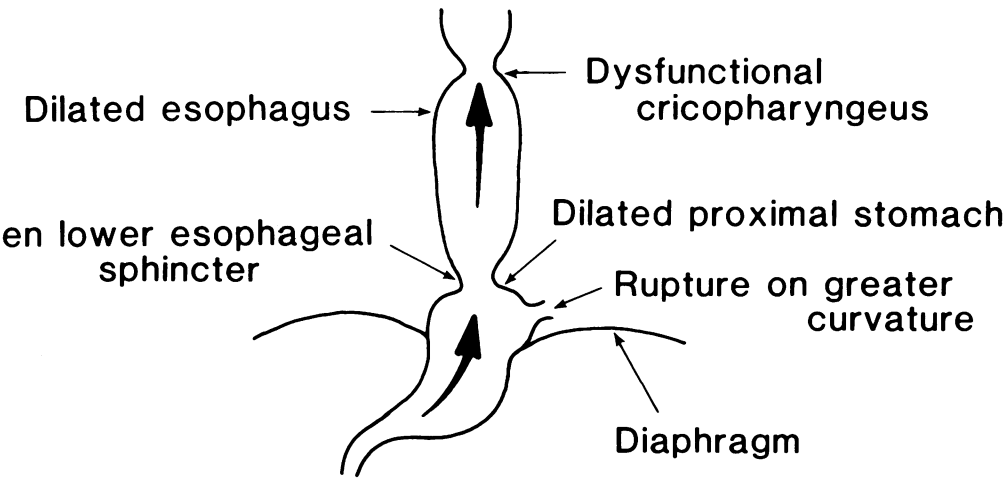

FIG. 2. Emetogenic gastric rupture in the newborn.

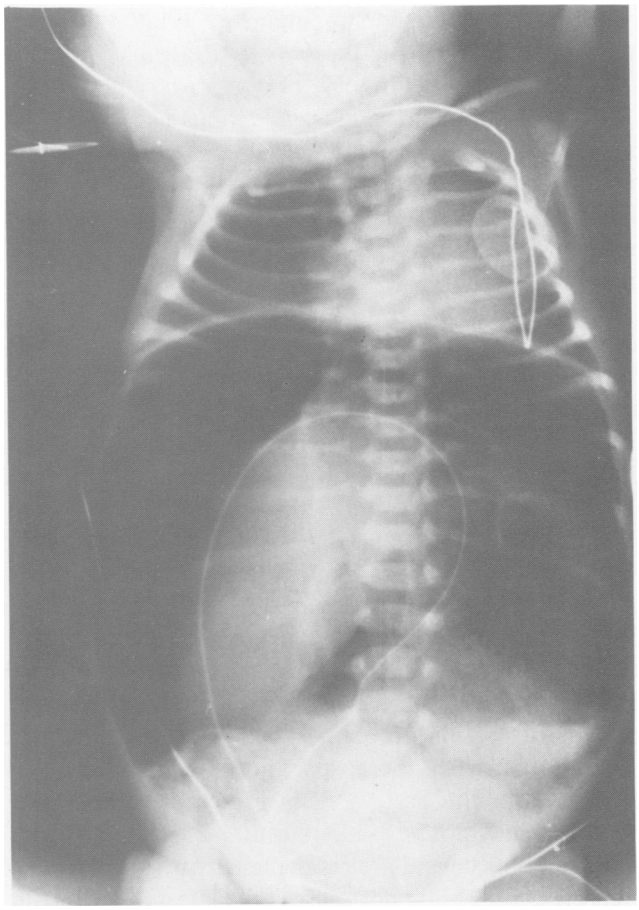

FIG. 3.

ture occurs characteristically along the greater curvature of the stomach, where the muscle is thinnest.[12]

Linear tears of the stomach seldom occur after the first week of life and are rarely seen in infants with pyloric stenosis. Some postulate that the cause for this discrepancy is maturing of the gastric wall musculature.[8] If the findings that were demonstrated in our cadaver studies are true *in vivo*, the stomach should be the organ to rupture in all emetogenic accidents of the upper gastrointestinal tract. The reverse, however, is true for esophageal ruptures. Esophageal ruptures characteristically occur in the adult, while gastric ruptures are rare. It is, therefore, postulated that the stomach is protected by maturing of the sphincter mechanisms and motility qualities of the esophagus and not by maturing of the muscular wall.

The newborn infant population that is affected by spontaneous gastric rupture is relatively characteristic in presentation. The majority of infants are premature but normal at birth.[7] Feedings are readily accepted and stooling is normal during the first few days of life. Vomiting is the initial symptom in most infants.[7,8] The illness is abrupt in onset and occurs within the first week of life in 96% of the involved infants.[17] The highest incidence of rupture is on the third day of life.[7]

Once the rupture occurs, the patient deteriorates rapidly, and abdominal distention ensues, with subsequent dyspnea, cyanosis, and shock. Subcutaneous emphysema of the abdominal wall and scrotal swelling may be present.[13] Roentgenograms of the abdomen will confirm the diagnosis (Fig. 3). The stomach is devoid of air. On the upright film of the abdomen, free air is present under the diaphragm. The abdominal viscera are displaced medially, producing the "saddlebag" appearance.

Rupture of the stomach may be preventable if decompression of the distended stomach is accomplished and feeding of the infant is delayed until normal motility is restored. When rupture does occur, prompt surgical intervention with repair of the gastric tear is paramount. Any delay in surgery will result in a higher mortality.[8,17]

At surgery, a two-layer gastric closure is accomplished and reinforced with a patch of omentum. The abdominal cavity is copiously irrigated with saline. Respiratory distress may exist prior to surgery because of the abdominal distention but can be relieved by needle aspiration. Postoperative intravenous fluids are administered until normal bowel sounds occur, and antibiotics are administered perioperatively.

Conclusion

Spontaneous linear tear of the stomach in the newborn is a virulent condition. It occurs most often in premature infants. The rupture characteristically occurs along the greater curvature of the stomach but may occur on the anterior surface of the stomach in the area of the cardia. The mechanism of the injury is postulated to be much like Boerhaave's syndrome, the stomach being the target organ. Tremendous intragastric pressures result because of incoordination and immaturity of the vomiting mechanism in the involved infant. The infants are asymptomatic before the rupture but deteriorate rapidly thereafter. Prompt surgical intervention is required.

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Discussion

DR. CLINTON M. CAVETT (Jackson, Mississippi): First, I need to just mention how indebted we are to Dr. Shaw, a member of this Association, who finally put to rest the idea that a congenital defect was responsible in these perforations.

I wish to point out another aspect of the considered cause in this type of perforation of the stomach. Dr. Touloukian and Dr. Lloyd have been most vocal in explaining the ischemic cause in this process; that is, the selective shunting of blood away from the splanchnic vascular bed as a response to hypoxic stress. As was pointed out by Dr. Houck, the vast majority of these patients fall into a category of the premature infant who is already undergoing some type of hypoxic stress, such as delivery suite resuscitation, respiratory distress, or severe congenital heart disease with shunting.

This hypoxic stress, when occurring in the newborn, sets the stage for the ischemic damage. Adding the ischemic damage to the hyperacidity known to occur in the first few days of life offers another explanation as to this catastrophic event.

I must mention that this sequence of events is not seen in the older child or adult. In Dr. Touloukian's lab—and we have begun to confirm this in our own laboratory—ischemic stress in the neonatal piglet leads to damage to the fundus and cardia of the stomach.

For completeness, I need to mention the pneumoperitoneum seen in this same group of patients who are on high-pressure ventilation for hyaline membrane disease. Ventilating these stiff lungs with high inspiratory pressures can produce pneumomediastinum, which will decompress by way of the peritoneal cavity. Therefore, a neonate on the high-pressure mechanical ventilation can develop pneumomediastinum and/or pneumothorax, and, later, pneumoperitoneum. And these are patients which must be identified, and not rushed to the operating room. As Dr. Houck pointed out, aspiration of the large pneumoperitoneum and close observation are indicated here.

As must be pointed out, these patients are also in the same group who can develop the spontaneous gastric perforation.

DR. ANTHONY SHAW (Charlottesville, Virginia): My interest in this uncommon problem was kindled by several cases of neonatal gastric rupture I encountered during my training in New York.

(slide) This is typical of seven cases we reported in 1965. Most of the infants we saw and whose case records we reviewed presented not with vomiting, but with respiratory distress, cyanosis, and abdominal distention. In this film you can actually see, quite well, the site of perforation on the greater curvature.

(slide) Grossly, these lesions look like tears, or blowouts, with ragged, thin edges. Histologically, the edges consist of mucosa and submucosa only, which you can detect in that infant.

On the basis of similar observations, Herbut, a pathologist at the University of Pennsylvania proposed a congenital gastric muscle defect as the cause of spontaneous gastric perforation in 1943. By 1962, when we saw this baby, this had become the prevalent theory of the cause of spontaneous gastric perforation. To us, these lesions look as if the stomach had blown out, or torn, with retraction of the seromuscular layer away from the mucosal perforation, which is a phenomenon familiar to all surgeons who have seen the results of distended, hollow viscera.

In the laboratory, we ran air into the stomachs of anesthetized newborn puppies, with cardia and pylorus occluded, until gastric rupture occurred. The lesions were not only identical grossly and histologically to those so-called spontaneous ruptures described by Herbut and others, but also to those seen by ourselves and others, in infants and adults where the cause of rupture was clearly pneumatic distention, such as situations where vigorous mask ventilation was carried out in babies with TE fistulas, babies with distal obstruction, and so on.

(slide) In the laboratory, this is a typical gastric perforation from overdistention. I might say that these perforations occurred in any part of the stomach. They were seen in the greater curvature, the lesser curvature, proximal, distal, anterior, posterior.

(slide) And note the curled edge of this defect. On the posterior surface of the same stomach is seen another area where there was a split in the seromuscular layer, but the mucosa was still intact.

(slide) Sections from the edge of the defect showed retraction of the muscle well back of the overhanging mucosal edge.

(slide) Histologically, this looked exactly like the specimen in our infants, with retraction of the muscle back here, and the overhanging mucosal edge.

(slide) This is from an area of thinning, where the mucosa had not disrupted, but the seromuscular layer had.

(slide) Another area of the stomach of one of the same dogs, where the retractions had occurred and the mucosa was beginning to give.

(slide) And these are the findings in a case—this slide was provided to me by Dr. Peter Kottmeier, at the State University of New York Downstate—of an infant he operated on, where the split had occurred in the seromuscular layer, and the mucosa hung through as a huge pseudodiverticulum. He had actually caught the rupture before disruption of the mucosa took place.

The purpose of these remarks is to indicate that the observations my colleagues and I reported 15 years ago strongly suggest that, whatever the exact mechanism—and Dr. Houch has told us an in-