Clinical analysis of spontaneous gastric rupture in non-neonatal children

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Research Article

Keywords: spontaneous gastric rupture, non-neonatal, children, diagnosis, treatment

Posted Date: June 10th, 2022

DOI: https://doi.org/10.21203/rs.3.rs-1693173/v1

Abstract

Background

Spontaneous gastric ruptures are rare in non-neonatal children, but once gastric rupture occurs, it results in rapid progression with high mortality. This study aimed to summarize the treatment experiences of 10 cases of spontaneous gastric rupture in non-neonatal children. We further investigated the etiology and treatment of spontaneous gastric rupture in non-neonatal children to reduce complications and improve efficacy.

Methods

A retrospective analysis was performed on 10 cases of spontaneous gastric rupture in children admitted to the Shanxi Children's Hospital between January 2009 and December 2020. Abdominal radiography showed a large amount of free gas in the abdominal cavity, which confirmed gastric rupture. Therefore, surgical treatment was performed in all the patients.

Results

Of the 10 patients, 2 died postoperatively, 3 had anastomotic leakage after the second surgery following the primary operation, and 5 were treated by the primary operation. All living patients had no complications at the 1 year follow-up.

Conclusions

Acute gastric dilatation is the primary cause of gastric rupture. A large amount of free gas in the abdominal cavity on abdominal radiographs and extraction of gastric contents on abdominal puncture are valuable for diagnosing the disease. Preoperatively, fluid infusion is performed to correct shock as well as water, electrolyte, and acid-base imbalances. Gastric rupture should be treated surgically, and a simultaneous gastrostomy should be performed as it is key to ensure anastomotic healing.

Background

Spontaneous gastric rupture commonly occurs in neonates and adults [1] but rarely in non-neonatal children. The pathogenesis of gastric rupture in non-neonatal children remains unclear. Once gastric rupture occurs, rapid progression follows, resulting in high mortality rates. Due to the low incidence of gastric rupture, there is a lack of public awareness for the early diagnosis of the disease and reasonable treatment of spontaneous gastric rupture in children. In the current study, a retrospective analysis was performed on cases of non-neonatal spontaneous gastric rupture admitted to our hospital, to investigate its diagnosis and treatment in children and further explore its etiology.

Methods

General information

Data of 10 patients of spontaneous gastric rupture who were admitted to the Shanxi Children's Hospital between January 2009 and December 2020, were analyzed. Three of the 10 patients were hospitalized for gastric dilatation; after admission, gastric rupture occurred. Two patients had a history of gastric dilatation, whereas 5 patients had no history of gastric dilatation. Among the patients, 3 were male and 7 were female, and their ages ranged from 8 months to 7 years.

The study protocol complied with the Declaration of Helsinki and was approved by the Research Ethics Committee of Shanxi Provincial Children's Hospital. The requirement for written informed consent was waived because of the retrospective nature of the study.

Among the cases, spontaneous gastric rupture had no specific clinical manifestations but presented with vomiting, abdominal distention, abdominal pain, and irritability. Notably, 2 patients had a history of binge eating and drinking. Physical examination revealed a poor mental response, abdominal distention, abdominal muscle tension, direct and rebound abdominal tenderness, and peritonitis. Signs of dehydration were evident in some cases. Regarding laboratory examinations, 1 case had decreased, 2 cases had normal, and 7 cases had elevated white blood cell counts; sodium levels were lower than normal; and blood gas analysis showed metabolic acidosis. Radiography revealed a large amount of free gas in the abdominal cavity (Fig. 1), and abdominal ultrasonography revealed a large amount of ascitic fluid. Turbid abdominal fluid was extracted via peritoneal puncture.

[Figure 1 near here]

Treatment

All patients were treated with gastrointestinal decompression and fluid rehydration to correct the water–electrolyte imbalances and acidosis. Rapid fluid replenishment and dilatation were performed in patients who experienced shock. Emergency surgery was then performed in all patients. A large amount of gas and liquid mixed with food residue was observed in the abdominal cavity (Fig. 2A). The abdominal cavity was flushed with normal saline until the saline was clear. Exploration of the gastric rupture site (Fig. 2B) revealed that the gastric wall around the rupture site was either weak or necrotic. The weak gastric wall tissue was subsequently trimmed. In 5 cases, absorbable 4 − 0 Vicryl (Ethicon Inc, New Jeysey, USA), was used for continuous inversion suture of the whole seromuscular layer and intermittent suturing of one seromuscular layer. In the other 5 cases, necrotic tissue around the rupture site was trimmed and sutured in two layers. A "mushroom head" rubber drainage tube was inserted for gastrostomy. Additionally, in 2 cases, a nasojejunal feeding tube was inserted through the stomach under direct visualization, and an abdominal drainage tube was inserted in all patients. Postoperatively, gastrointestinal decompression, dietary prohibition, fluid infusion, and parenteral nutrition support were continued. In cases with an indwelling nasojejunal feeding tube, enteral nutrition support was administered after recovery of

intestinal function. Upper digestive tract angiography, which was performed one week postoperatively, revealed no anastomotic leakage, and the fistula was clipped (Fig. 3). The clinical data for each patient are presented in Table 1.

Table 1
Clinical data of the 10 children with gastric rupture

Serial No.	Age	Gender	Gastric dilation or not	Surgical approach	Anastomotic leakage	Results
1	5 years	Female	No	Trim the perimeter of the rupture and suture the rupture	—	Died
2	8 months	Female	No	Trim the perimeter of the rupture and suture the rupture	Yes	Cured
3	4 years	Female	Yes	Trim the perimeter of the rupture and suture the rupture	No	Cured
4	7 years	Female	No	Trim the perimeter of the rupture and suture the rupture	Yes	Cured
5	1 year	Female	Yes	Trim the perimeter of the rupture and suture the rupture	Yes	Cured
6	6 years	Male	Yes	Suture the rupture and perform gastrostomy	No	Cured
7	4 years	Female	Yes	Suture the rupture, perform gastrostomy, and place a naso-jejunal nutrient tube	No	Cured
8	2 years	Male	No	Suture the rupture, perform gastrostomy, and place a naso-jejunal nutrient tube	No	Cured
9	3 years	Female	Yes	Suture the rupture and perform gastrostomy	No	Cured
10	1 year	Male	No	Suture the rupture and perform gastrostomy	—	Died

[Figure 2 near here]

[Figure 3 near here]

[Table 1 near here]

Results

Among the 5 children who underwent simple gastric wall trimming and suturing, 1 died 15 h postoperatively, and 3 suffered from anastomotic leakage. These 3 patients underwent a second surgery

for fistula repair and suture on the 7th, 8th, and 10th postoperative days, respectively. However, only 1 patient had primary healing. Given the high incidence of anastomotic leakage, gastrostomy was performed in the other 5 cases with suture repair. Among these cases, fistulas in 4 cases were clipped 1 week postoperatively, and the clips were removed 1 month later. Meanwhile, 1 patient who had experienced shock upon admission, died 22 h postoperatively.

All 8 patients were followed-up for 1 year and no complications had occurred. All gastric rupture sites were located at the greater curvature, with seven on the anterior wall and three on the posterior gastric wall.

Discussion

Etiology

Tumors and peptic ulcers are the leading causes of gastric rupture in adults. In women, binge eating and drinking are the most common causes, with ruptures often occurring on the side of the lesser curvature [2]. Gastric ruptures are primarily observed in premature neonates with asphyxia, low birth weight infants, and children with food intolerance. Other causes of gastric rupture include esophageal atresia, small bowel obstruction, and annular pancreas [3]. Libeer reported that spontaneous gastric rupture could be attributed to gastric dilation [4]. In the current study, 5 children presented with gastric dilatation or had a history of gastric dilatation. Gastric distension in children causes weakened gastric emptying, resulting in flatulence and residual food in the stomach cavity. Residual food rots and ferments, enlarging the stomach and distending its walls, which makes them thinner. In addition, blood vessels in the gastric wall become elongated, narrowed, or even embolized, leading to local ischemia. Once gastric pressure exceeds 14 mmHg (20 cm H_20) and the gastric wall venous pressure, resulting in gastric ischemia [5]. Coughing or vomiting may cause a sudden rupture of the stomach. In addition, when the stomach overdilates, gastric lumen pressure increases, the angle of His sharply decreases, and the fundus of the stomach compresses the esophagus and lessens the right diaphragm angle, forming a one-way valve [6]. When children cry, gas enters the stomach cavity under esophageal peristalsis, and residual food and gastric expansion stimulate the stomach to secrete more gastric juice, further causing ischemic necrosis. Therefore, any further increase in stomach pressure or a sudden increase in abdominal pressure may cause rupture in the weakest part of the stomach wall. Similar to neonates, gastric rupture sites in non-neonatal children occur in the greater curvature of the stomach. As it is the area of the gastric wall that readily dilates, the greater curvature of the stomach is prone to ischemic necrosis, perforation, and rupture. In this study, all patients had gastric rupture in the greater curvature of the stomach [7].

Among the 10 patients, 5 had no history of gastric dilatation, and gastric ruptured in these patients had an acute onset. Additionally, abdominal distension appeared after a short period of vomiting, and abdominal radiography revealed large amounts of free gas in the diaphragm. In this study, only 5 cases showed necrosis of the gastric wall tissue with infiltration of inflammatory cells. Meanwhile, 1 patient had a gastric wall muscle defect as detected by intraoperative pathological examination of the gastric

wall tissue around the site of gastric rupture. In addition to gastric dilatation, gastric rupture can also occur due to developmental defects in the congenital gastric wall muscle. Fukata et al. reported that the etiology of spontaneous gastric rupture in children was similar to that in neonates, as revealed by the pathological findings of a muscle defect [8]. Additionally, Salerno et al. revealed abnormal gastric wall development in a 5-year-old girl during pathological examination [9]. However, in 1965, Shaw et al. explored this theory by tying the esophageal and duodenal ends of the stomach in dogs and inflating them until the organ perforated. Their study revealed that all perforations occurred on the greater curvature, and histological examination showed that all specimens were deficient in muscle tissue adjacent to the perforation [10]. Therefore, spontaneous gastric rupture in children cannot be attributed solely to congenital defects of the gastric muscle wall.

Several studies have reported [4] that gastric rupture in children is mainly characterized by the following findings: 1) has a high incidence among girls; 2) most commonly occurs at the greater curvature and anterior wall of the stomach; and 3) the rupture is circular. All cases in our study showed consistency with the above characteristics.

Diagnosis

In this study, gastric rupture in children had clinically nonspecific signs and symptoms in the early stages such as vomiting, abdominal pain, and abdominal distension. However, obvious peritonitis, abdominal distention, direct and rebound abdominal tenderness, muscle tension, positive percussion mobility dullness, and weakened or absent bowel sounds were noted in the patients. After gastric rupture, the disease progressed rapidly resulting in the deterioration of their condition, as manifested by poor mental response, pale complexion, and shallow breathing due to abdominal distention and diaphragmatic elevation. Gastric rupture results in mechanical damage and rapid hemodynamic changes [11]. Furthermore, once gastric contents enter the abdominal cavity, toxin absorption causes shock, manifesting as irritability or prostration in children, tachycardia, hypotension, skin spots, cold extremities, and poor peripheral circulation. Owing to the severity of gastric rupture, simple and rapid methods are preferred as auxiliary examinations for diagnosis. Therefore, abdominal radiography was the preferred examination method. An upright abdominal radiograph showed the presence of large amounts of free gas below the diaphragm or atmospheric fluid across the entire abdomen with a gastric bubble; these signs indicated a diagnosis of gastrointestinal perforation or rupture. Abdominal ultrasonography revealed a large amount of peritoneal fluid in the abdominal cavity, and an abdominal puncture, which revealed the presence of gastric contents, was performed to confirm the diagnosis.

Treatment

The mortality rate associated with gastric rupture surgery caused by acute gastric dilatation is reported to be as high as 33% [12]. In the absence of prompt surgical intervention after the diagnosis of gastric rupture, the mortality rate can reach up to 100% [5]. Preoperatively, a gastrointestinal decompression device should be used, and potent broad-spectrum antibiotics should be administered. In the current study, all children with gastric rupture had hyponatremia and acidosis. Thus, fluids were administered to

correct dehydration, electrolyte disturbances, and acidosis. However, owing to the rapid progression of the disease, children often present with shock upon admission. In this study, two patients died due to shock. After gastric rupture, gastrointestinal bacteria enter the abdominal cavity and multiply in large numbers, releasing exogenous toxins. Under such circumstances, the body releases various mediators that inhibit heart function and dilate blood vessels, resulting in a lack of effective circulating blood volume. Therefore, rapid fluid infusion and dilatancy should be performed for children experiencing shock. However, vasoactive drugs should be used reasonably while monitoring urine volume using an indwelling catheter, and surgery should be performed simultaneously. In children with dyspnea caused by severe abdominal distention, abdominal puncture decompression before surgery should be performed. A syringe should be used to extract gas at the highest point of the abdominal wall and below the xiphoid process to reduce intra-abdominal pressure [13].

Different surgical approaches have been reported in children with spontaneous gastric rupture. Libeer [11] performed sleeve gastrectomy in a non-neonatal child with spontaneous gastric rupture; however, the child had difficulty eating postoperatively. He reported that simple closure of a ruptured stomach would have been better for children than sleeve gastrectomy due to the high propensity of feeding problems after sleeve gastrectomy. Some surgeons believe that total gastrectomy is the safest option for treating spontaneous gastric rupture because of ischemia of the gastric wall tissue, resulting in poor healing of the residual gastric tissue [14, 15]. However, total gastrectomy is a relatively complicated and long procedure. In addition, children who undergo total gastrectomy may experience abnormal growth and development later.

Children with gastric rupture are often admitted in a critical condition. Therefore, as much as possible, simple surgical methods should be selected to shorten the surgery time and preserve the organ function in children. In this study, repair of gastric rupture and gastrostomy were performed. A nasojejunal feeding tube was also inserted whenever the patient's condition allowed it, which contributed to satisfactory healing.

Intraoperatively, an incision was made in the upper abdomen through the rectus abdominis or in the midline of the abdomen. After gaining access to the abdomen, a large amount of food residue, gastric juice, and other gastric contents became visible in the abdominal cavity. An aspirator was used to aspirate abdominal fluid. Physiological saline solution was used to flush the abdominal cavity until the saline was clear. The ruptured mouth of the stomach was explored, and the contents were removed. In most cases, omental necrosis was resected. Intraoperatively, thin and suspected necrotic tissue were observed around the rupture; these were trimmed until normal gastric wall tissue was reached as evidenced by blood flow. Whenever the condition of the patient permitted, nasojejunal nutrition was administered at the rupture site. Based on the age of the children, 4 – 0 or 5 – 0 absorbable sutures were selected for continuous suturing of the entire gastric wall to avoid gastric mucosal evagination at the incision margin. The seromuscular layer was sutured intermittently with reinforcement. Meanwhile, a "mushroom head" drainage tube perforating the left abdominal wall was indwelled and fixed for gastrostomy. A peritoneal drainage tube was also placed in the pelvic cavity.

Continuous gastrointestinal decompression and fluid infusion were performed postoperatively to maintain water and electrolyte stability and acid-base balance. Decompression also helped to maintain patency of the gastrostomy and abdominal drainage tubes to prevent anastomotic rupture and abdominal infection. Moreover, parenteral nutritional support therapy and albumin supplementation were administered to promote anastomotic and incisional healing in patients with hypoalbuminemia. High-dose gamma-globulin therapy was administered to children with septic shock. Enteral nutrition was provided if intestinal function returned after the insertion of an indwelling nasojejunal feeding tube.

Severe gastric rupture in children is usually accompanied by numerous complications such as high mortality and postoperative anastomotic leakage. Based on the current findings, the following outcomes are summarized: 1) to reduce mortality, surgical intervention is crucial before severe toxic shock and multiple organ failure occur, thereby preventing further disease deterioration. 2) Postoperative gastric rupture is associated with a high incidence of anastomotic leakage. In the early stages, direct anastomosis should be performed after trimming necrotic tissue around the rupture. Aside from 1 patient who died, 3 of the other 4 patients had anastomotic leakage. Gastrostomy was performed using an improved surgical method, and no anastomotic leakage was observed, except for 1 child who died of severe shock at the time of admission. As gastric rupture is mostly caused by gastric dilation, postoperative gastric dilation will persist, and gastric function cannot return to baseline levels. Conventional gastric decompression by nasogastric tube has proven inefficient [16], whereas simple gastrointestinal decompression fails to effectively relieve gastric pressure, resulting in anastomotic leakage [17]. After revising the surgical method and performing a gastrostomy, gastric drainage improved, and the anastomosis healed without any complications. 3) An indwelling nasojejunal nutrition tube can effectively isolate the lesion site, and early enteral nutrition treatment is conducive for enhancing the overall nutritional status of children. The normal physiological function of the digestive tract should be maintained, and immunity should be improved by reducing postoperative infection and anastomotic leakage. Additionally, intravenous fluid administration should be reduced to lower medical costs.

Conclusions

In conclusion, spontaneous gastric rupture is a rare clinical condition in non-neonatal children, and it is believed that spontaneous gastric rupture in non-neonatal children is caused by gastric dilation. Therefore, early diagnosis and surgical intervention are crucial to reduce mortality. Abdominal radiographs should be obtained to determine the presence of free gas in the abdominal cavity, and an abdominal puncture should be performed to check for the presence of gastric contents to confirm the diagnosis. Intravenous fluids should be administered to correct shock as well as water, electrolyte, and acid-base imbalances. If feasible, a nasojejunal feeding tube can be inserted and left in place. Furthermore, simultaneous gastrostomy should be performed, and gastrointestinal decompression should be continued postoperatively. Gastrostomy is key to ensure proper anastomotic healing.

Declarations

Ethics approval and consent to participate: The study protocol complied with the Declaration of Helsinki and was approved by the Research Ethics Committee of Shanxi Provincial Children's Hospital (approval number IRB-KY-2018-001). The Research Ethics Committee of Shanxi Provincial Children's Hospital waived the requirement for informed consent due to the retrospective nature of the study.

Consent for publication: Informed consent for the publication of the data and figures was obtained before surgery from all of the participants in the study.

Availability of data and materials: The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests: The authors declare that they have no competing interests.

Funding: Not applicable.

Authors' contributions: ZY and HX conceptualized and designed the study, drafted the initial manuscript, and reviewed and revised the manuscript. ZS carried out the initial analyses, reviewed and revised the manuscript. All authors read and approved the final manuscript.

Acknowledgements: The authors thank the staff of the Department of General Surgery and Department of Pathology at Shanxi Children's Hospital. We would also like to thank Editage (www.editage.cn) for English language editing.

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Figures

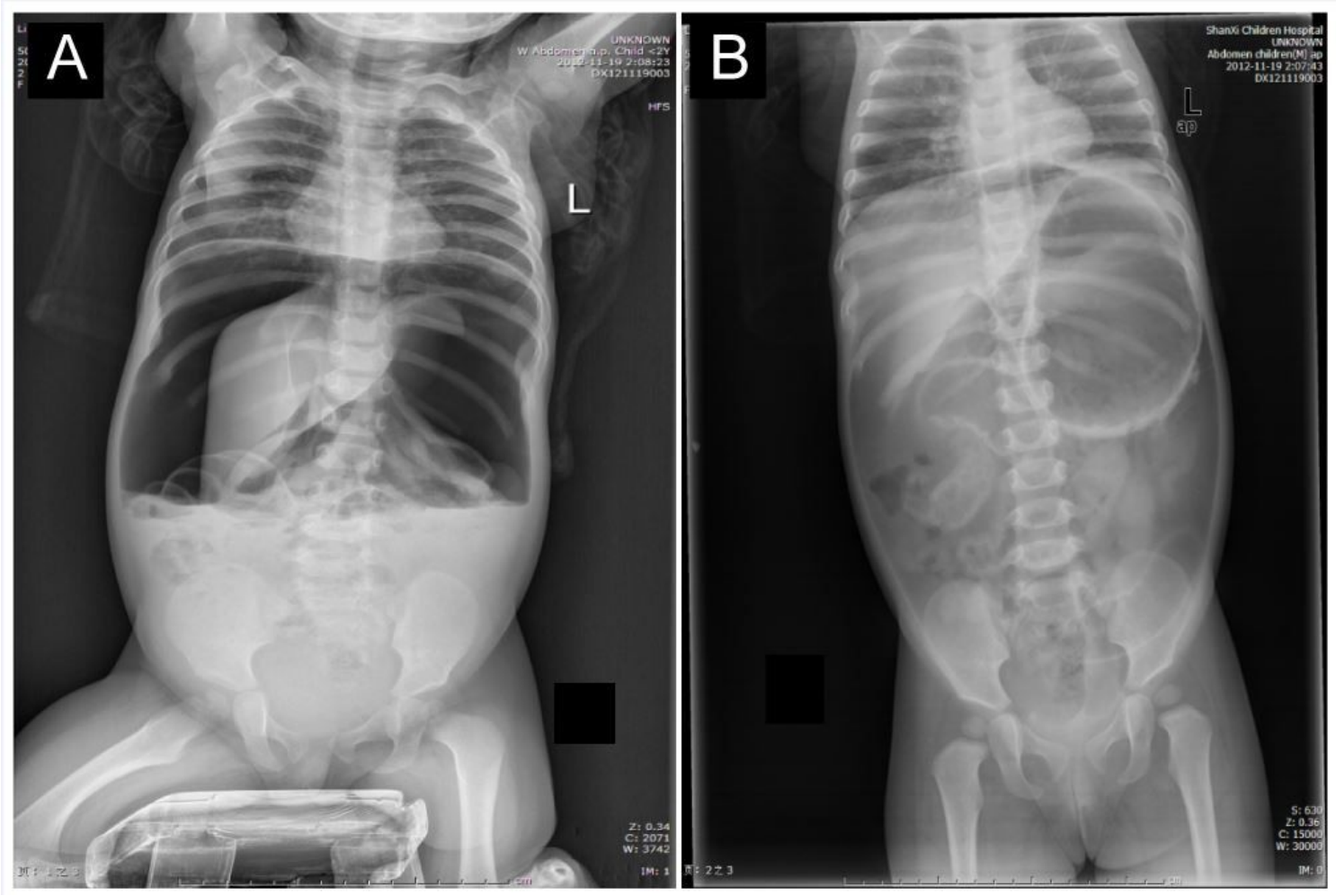

Figure 1

A. Upright abdominal radiograph shows a large amount of free gas under the diaphragm. The gastric bubble is not noted; B. A supine abdominal radiograph showing a large amount of free air in the mid-abdominal cavity.

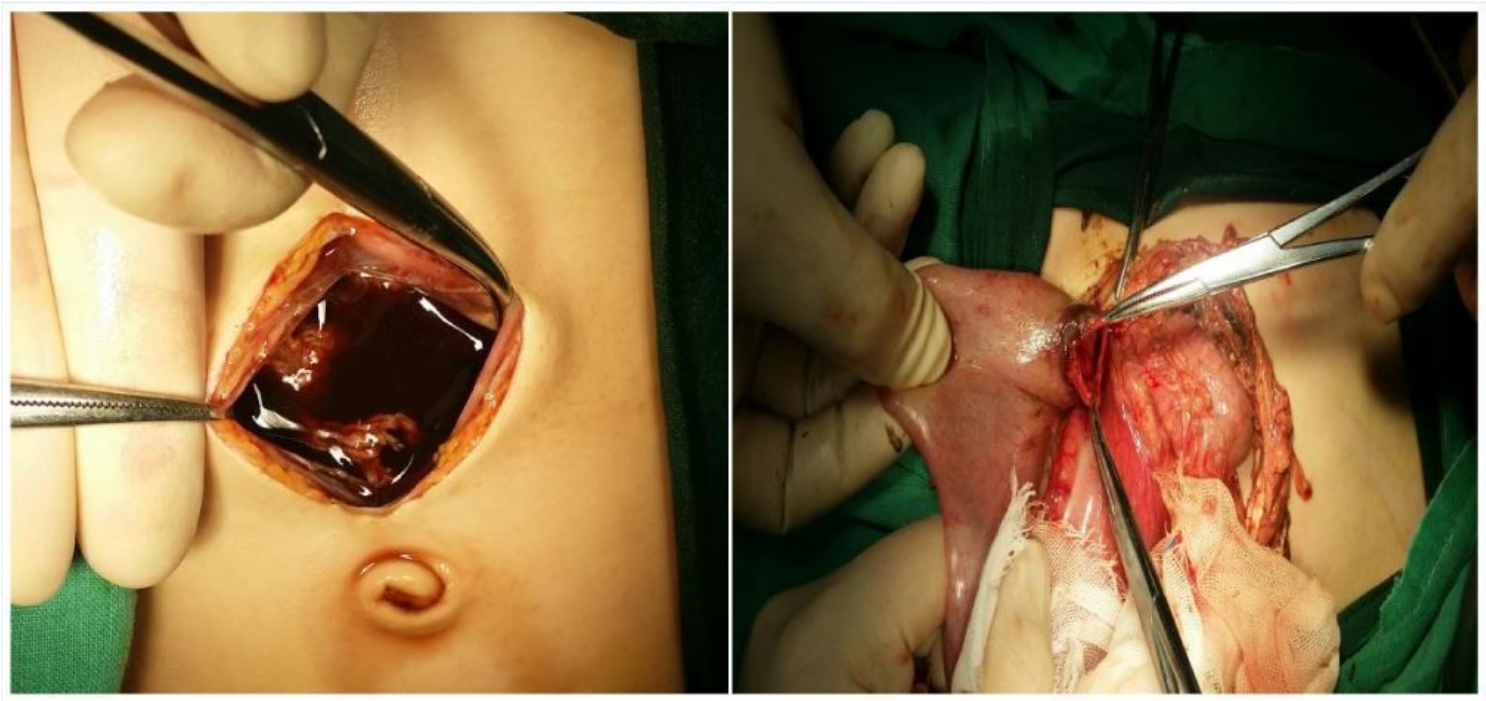

Figure 2

A. A large amount of brown fluid mixed with food residue in the abdominal cavity; B: Rupture of the stomach wall on the greater curvature.

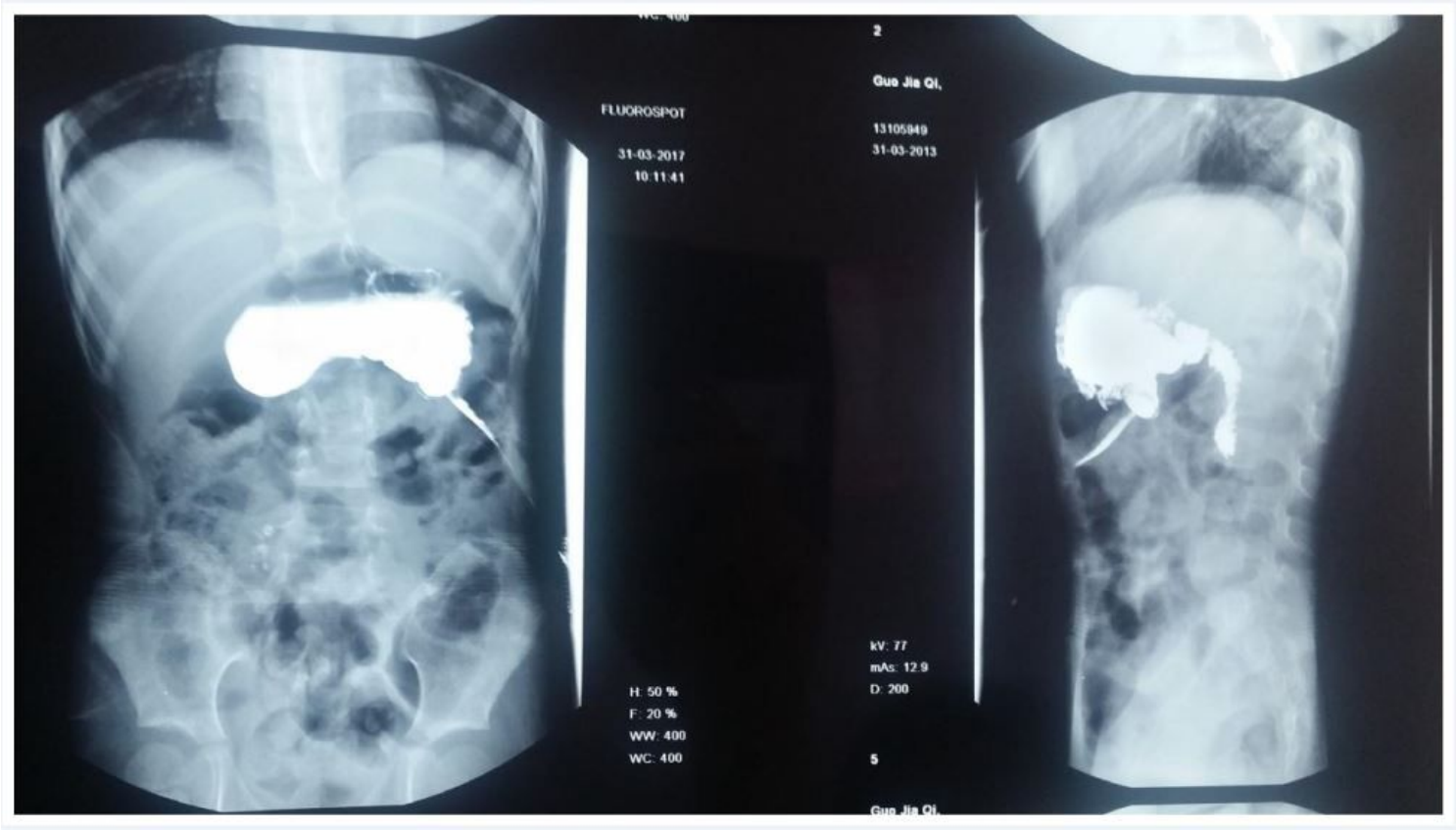

Figure 3

No anastomotic leakage as visualized by upper gastrointestinal angiography at 1 week postoperatively.