Pediatrics and Neonatology (2019) 60, 634–640

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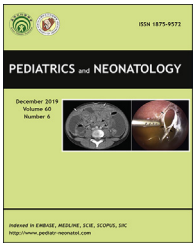

Original Article

Pediatric gastric perforation beyond neonatal period: 8-year experience with 20 patients

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Received Jul 25, 2018; received in revised form Mar 8, 2019; accepted Mar 8, 2019
Available online 21 March 2019

Key Words
pediatric gastric perforation;
prognosis;
treatment

Background: To describe the characteristics, treatments, and prognosis of pediatric gastric perforation patients beyond neonatal period.
Methods: Twenty pediatric patients beyond neonatal period were included in this study. Medical records were reviewed and clinical characteristics were analyzed. According to the outcomes, patients were divided into the survival group and the death group. Death time was documented, and survival patients were followed up. The degree of severity was calculated using pediatric critical illness score (PCIS). Differences between the two groups were analyzed by the Student's t-test, Mann-Whitney test and Chi-square test appropriately.
Results: Gastric perforation was diagnosed in 20 pediatric patients beyond neonatal period, including 6 males (30%) and 14 females (70%), with the age of 37.18 (15.90, 107.12) months, and the range was from 4.30 months to 14.17 years old. They had different manifestations, etiologies, sites of perforation and surgery procedures. Among the 20 cases, 14 (70%) survived and 6 (30%) died. Age, gender, length and number of perforation had no statistically difference between the two groups. However, PCIS, ischemia of gastrointestinal wall, and transmural necrosis of gastric wall were statistically different. For the survival group, during a follow-up period of 50 (36, 68) months, ranging from 2 months to 8 years and 7 months, one patient had a second-time perforation, another 3 patients had brain injury symptoms, and the rest 10 patients had good quality of lives.

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https://doi.org/10.1016/j.pedneo.2019.03.006

Conclusions: Gastric perforation of pediatric patients beyond neonatal period causes a mortality of 30% on this study. Spontaneous great curvature of gastric wall perforation has the highest morbidity. Low PCIS predicts for unfavorable prognosis. Most of the survival patients have satisfactory living quality after operation.

1. Introduction

Gastric perforation is uncommon[1,2] and life-threatening in infants and children.[3,4] Large amounts of studies have explored neonatal gastric perforation, which has been characterized[5,6] as onset within the first week of age and is complicated with alimentary malformations. The mortality is up to 30%—70%,[7–10] and the most common cause is congenital gastric muscular wall defects. However, gastric perforation in pediatric patients beyond the neonatal period has rarely been reported, and they have different characteristics from neonatal patients. In general, the clinical characteristics, treatments, and prognosis of such patients are not well understood.

Patients with gastric perforation may present with critical symptoms,[11] and their prognosis varies according to the severity of the condition. Pediatric critical illness score (PCIS) has been found to be an objective and quantification method to evaluate the severity of diseases.[12–14] However, no such study applying PCIS in evaluating the severity of pediatric gastric perforation has ever been reported.

The purpose of this study is to review our experiences with 20 gastric perforation cases beyond neonatal period over eight years, describe the clinical characteristics, treatments, and prognosis, and analyze the differences between the survival group and the death group to provide a foundation for primary prognosis prediction.

2. Methods

2.1. Patients

Medical records of 20 pediatric patients beyond neonatal period, diagnosed with gastric perforation, admitted to the general surgery department of our center from January 2009 to July 2017 were retrospectively reviewed. Medical records of these patients were reviewed with regard to age, gender, clinical manifestations, degree of severity, surgical procedures, site and length of perforation, number of perforation and gastric samples. Depending on the outcomes, patients were divided into two groups, the survival group and the death group. Death time was documented, and all the survival group patients were followed up.

2.2. PCIS (pediatric critical illness score)

The degree of severity was calculated before surgery by PCIS,[15] which synthesized patients' age and ten physiological indexes including heart rate, breath rate, systolic blood pressure, oxygen partial pressure and pH of arterial blood, serum sodium and potassium, creatinine or urea nitrogen, hemoglobin and Glasgow coma scale (Supplementary Table 1).

2.3. Statistic analysis

All the data were analyzed using SPSS for Windows version 17.0. Normal distribution data was presented by (mean $\pm$ standard deviation), and was analyzed using the Student's t-test. Non-normal distribution data was presented by median (first quartile, third quartile), and was analyzed using Mann-Whitney test. Categorical variables were presented by frequencies and percentages, and were studied using Chi-square test. $P < 0.05$ was considered statistically significant.

3. Results

Gastric perforation was diagnosed in 20 pediatric patients beyond neonatal period over eight years in our institution, including 6 males (30%) and 14 females (70%), with the age of 37.18 (15.90, 107.12) months, and the age range was from 4.30 months to 14.17 years old. General condition and outcomes of the patients are displayed in Table 1. Fig. 1 shows a case of posterior gastric wall perforation during surgical exploration. Besides, four patients presented with definite ischemia of gastrointestinal wall during operation. Pathological results of five cases showed transmural necrosis of gastric wall. Fig. 2 is a great curvature picture taken from one of them, which shows the disappearance of normal gastric wall layer, exfoliation of mucosal epithelium, degeneration of muscular layer, and transmural necrosis of gastric wall.

Among the 20 patients, 14 patients (70%) survived, and 6 patients (30%) died. Time of death was 3.00 (1.00, 10.25) days after surgery, and the time range was from 7 h to 27 days. Table 2 shows a comparison between the survival group and the death group. Age, gender, length, and the number of perforation had no statistically significant difference between the two groups, while PCIS, ischemia of gastrointestinal wall, and transmural necrosis of gastric wall showed a statistically significant difference between the two groups. Specifically, the death group had lower PCIS, more ischemia and transmural necrosis of gastric wall cases compared to the survival group.

In the survival group, during the follow-up period of 50 (36, 68) months, ranging from 2 months to 8 years and 7 months, only one patient suffered from a second-time

Table 1 Pediatric gastric perforation patients beyond neonatal period between 2009 and 2017.

No.	Age (month)	Gender	PCIS[a]	Clinical manifestation	Site of perforation	Number of perforation	Length of perforation (cm)	Gastrointestinal wall ischemia	Surgical procedures	Transmural necrosis of gastric wall	Etiology	Outcome	Second-time gastric perforation	Time of death after operation (day)	Living condition
1	33.50	F	84	Dyspnea	Anterior wall	1	0.3	No	Repairation of gastric wall and left diaphragm	No	Left diaphragmatic hernia	Survive	No	—	Good
2	137.93	F	72	Vomiting, abdominal distension and abdominal pain	Posterior wall	>1	6	No	Gastric wall repairation and gastrostomy	No	Spontaneous	Survive	Yes	—	Good
3	43.94	F	90	Vomiting, abdominal distension	Posterior wall	>1	1	No	Gastric wall repairation and gastrostomy	No	Spontaneous	Survive	No	—	Good
4	14.53	F	82	Vomiting, abdominal distension, melena	Greater curvature	1	2	No	Gastric wall repairation and gastrostomy	No	Spontaneous	Survive	No	—	Low intelligence
5	170.13	M	100	Abdominal pain	Anterior wall	1	0.5	No	Gastric wall repairation and gastrostomy	No	Gastric wall non-hodgkin lymphoma	Survive	No	—	Good
6	4.30	F	92	Nausea, abdominal distension	Greater and lesser curvature	>1	5	No	Gastric wall repairation	No	Spontaneous	Survive	No	—	Good
7	40.86	F	90	Vomiting, abdominal distension and abdominal pain	Anterior wall	1	1	No	Gastroplasty and enterostomy	No	Spontaneous	Survive	No	—	Epilepsy
8	76.02	F	96	Vomiting, abdominal pain	Greater curvature	1	6	No	Gastric wall repairation and gastrostomy	Yes	Spontaneous	Survive	No	—	Good
9	16.08	M	90	Dyspnea	Anterior wall	1	0.3	No	Repairation of gastric wall and left diaphragm	No	Left diaphragmatic hernia	Survive	No	—	Good
10	57.98	F	100	Foreign bodies intake	Lesser curvature	1	0.6	No	Gastric wall repairation	No	Foreign body intake	Survive	No	—	Good
11	98.72	M	96	Abdominal pain, melena	Anterior wall	1	0.5	No	Gastric wall neoplasm resection and gastrostomy	No	Gastric wall non-hodgkin lymphoma	Survive	No	—	Good
12	22.17	F	80	Vomiting, abdominal distension	Posterior wall	1	10	No	Gastric wall repairation and gastrostomy	No	Spontaneous	Survive	No	—	Good
13	15.36	M	90	Abdominal distension, abdominal pain	Greater curvature	1	0.5	No	Gastric wall repairation	No	Spontaneous	Survive	No	—	Good
14	31.12	M	88	Abdominal trauma	Greater curvature	>1	4	No	Gastric wall repairation	No	Abdominal trauma	Survive	No	—	Claudication
15	6.06	F	64	Vomiting, abdominal distension	Greater curvature	1	2.5	Yes	Gastric wall repairation and gastrostomy	Yes	Spontaneous	Die	No	27	—
16	132.31	F	72	Dyspnea	Greater curvature	1	3	Yes	Gastric wall repairation	Yes	Left diaphragmatic hernia	Die	No	5	—
17	21.43	F	80	Vomiting, abdominal distension	Posterior wall	>1	0.5	No	Gastric wall repairation	No	Spontaneous	Die	No	12	—

18	144.00	F	68	Abdominal distension, abdominal pain	Greater curvature	1	20	Yes	Subtotal gastrectomy and gastrostomy	Yes	Gastric ulceration	Die	No	1	—
19	164.43	M	48	Dyspnea	Greater and lesser curvature	>1	15	Yes	Repairation of gastric wall and left diaphragm	No	Left diaphragmatic hernia	Die	No	1	—
20	11.40	F	87	Abdominal distension	Greater curvature	1	4	No	Gastric wall repairation and gastrostomy	Yes	Spontaneous	Die	No	1	—

[a] PCIS = pediatric critical illness score.

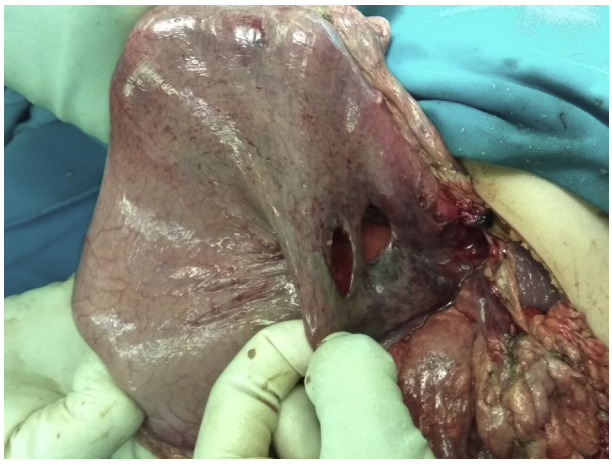

Figure 1 A surgical exploration picture showed posterior gastric wall perforation.

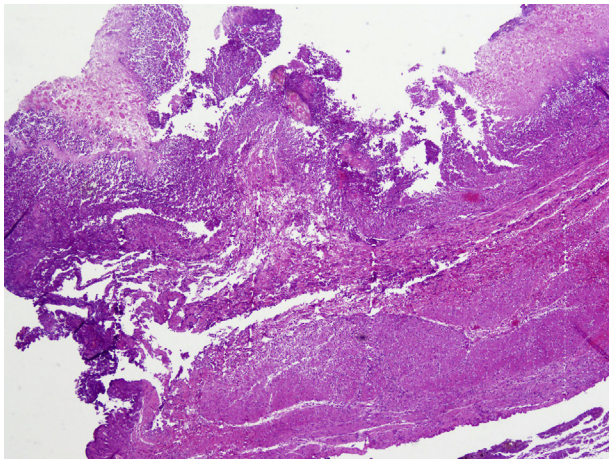

Figure 2 A pathological picture of great curvature perforation under light microscopy (HE × 4), which showed the disappearance of normal gastric wall layer, exfoliation of mucosal epithelium, degeneration of muscular layer, and transmural necrosis of gastric wall.

perforation at the posterior gastric wall one month after the first operation, and gastric wall repair, as well as gastrostomy, were done once more. The patient had a satisfactory feeding condition post operation during the follow-up of 3 months. Another three patients had neurologic symptoms, such as epilepsy, claudication, and learning ability deficits, which may be due to anoxia caused by shock and late fluid resuscitation. The remaining 10 patients had no abdominal distension, adhesive intestinal obstruction, second-time perforation or growth retardation.

4. Discussion

Clinical characteristics of gastric perforation in pediatric patients beyond the neonatal period are not well documented, and this is the first consecutive case study up to the present.

After retrospectively analyzing the clinical data, we found that most patients had a spontaneous gastric

Table 2 Comparison between survival group and death group.

Variables		Survival group	Death group	Results	*P*
Age (month)		37.18 (17.60, 71.51)	79.94 (13.91, 141.07)	−0.165	0.869
PCIS[a]		89.29 ± 7.83	69.83 ± 13.54	−4.086	<0.001
Length of perforation (cm)		1.00 (0.50, 4.75)	7.50 (2.63, 12.25)	−1.410	0.159
Gender	Male	5	1	0.726	0.394
	Female	9	5		
Number of perforation	1	10	4	0.045	0.831
	>1	4	2		
Gastrointestinal wall ischemia	Yes	0	4	11.667	<0.001
	No	14	2		
Transmural necrosis of gastric wall	Yes	1	4	7.937	<0.001
	No	13	2		

[a] PCIS = pediatric critical illness score.

perforation, which was in accordance with other case reports.[8,16,17] *Helicobacter pylori* infection is thought to be highly associated with peptic ulcers, which might induce gastric perforation. However, gastric ulcer-induced perforation is uncommon in pediatric patients. Hua et al.[18] reported a 20-year experience on 52 pediatric patients with perforation induced by peptic ulcers, and only 11 cases (21.2%) were induced by gastric ulcers. Secondly, patients with gastric ulcers usually have periodic epigastric pain, and some may have typical postprandial pain. When perforations occur, they are manifested as an acute attack of the chronic course of the disease, but this was not observed in patients included in this study. Thirdly, Hua et al.[18] also revealed that perforation induced by gastric ulcers was mostly found in the pylorus area, which accounted for up to 63.6% (7/11). However, this study concludes that pediatric gastric perforation is mostly found in the great curvature of the gastric wall. Fourthly, one patient in the death group had *Helicobacter pylori* examined, while the result showed weakly positive. He had a gastric perforation at the great curvature, with a length of 20 cm, which was different from that caused by gastric ulcers. Fifthly, patients infected by *Helicobacter pylori* should undertake regular medical treatments post operation, else they would have symptoms again. However, in the follow-up period of the survival group, most patients had no symptoms. Even the patient who had a second-time perforation had a satisfactory feeding condition without abdominal pain after the second operation. Thus, we excluded the possibility of *Helicobacter pylori* infection and gastric ulcers in this study. Other reported causes, such as trichobezoars,[19,20] gastric volvulus,[21] corrosive ingestion,[22–24] iatrogenic injuries,[3,25,26] childhood dermatomyositis[27] or child abuse,[28] were not found in this study. The decrease in the venous outflow, which is caused by increased intragastric pressure above 30 cm H_2O, could lead to ischemia and infarction of the gastric wall, which most likely contributed to the "spontaneous" gastric perforation.[29]

Gastric perforation in pediatric patients beyond the neonatal period mostly occurred at the greater curvature, which is the same as that observed in neonatal cases,[8] accounting for eight cases in this study. This may be explained by the gastric receptive relaxation theory. Because the great curvature is the most distensible area of the gastric wall, it tends to be the most prone place for ischemic necrosis, which results in desperate perforation.

Several surgical procedures were chosen depending on the primary disease and gastrointestinal condition in this study. If the perforation was small with no gastrointestinal ischemia or complications, then simple gastric repair was adopted. However, in cases where the perforation was large or accompanied by large areas of gastrointestinal ischemia or necrosis, simple suture was difficult to perform because of the edematous and fragmentary status of the gastric wall, and delayed perforation would possibly occur, thus more aggressive procedures should be required,[20] and in this study, gastric wall repair accompanied by gastrostomy were done in many cases. Other surgery procedures, such as repair of the diaphragm or neoplasm resection, were performed where appropriate. Unlike our study, Singh et al.[30] suggested a video-assisted thoracoscopy as a diagnostic and therapeutic method for gastric perforation induced by a diaphragmatic hernia.

All these efforts contributed to a survival rate of 70%, but 30% of patients who manifested with critical conditions died. The death time varied from 7 h to 27 days after surgery, and 66.7% of patients died within one week. High mortality of the disease may be a result of the following reasons. Firstly, the intense chemical stimulation of gastric fluid in the peritoneum causes severe peritonitis. Secondly, bacteria erupted from the alimentary tract, entering into the sterile peritoneal cavity, causes abdominal infection. Besides, gastric fluid is not easily limited to form a package, resulting in diffuse abdominal infection. Thirdly, gastric acid and pepsin not only produce strong stimulation to the abdominal viscera but also have a corrosive effect,[31,32] thus accelerating the occurrence of organ failure.

To quantify the severity, PCIS was applied. It is a comprehensive value generated by the emergency group of Chinese medical association pediatric society and a pediatric group of Chinese medical association emergency society and has been proven to be an effective tool that can best assess the severity of diseases.[33,34] PCIS value of lower than 70 indicates extreme severity, 70–90 demonstrates severity, and higher than 90 stands for non-severity.[35] Compared with the survival group, the death group had lower PCIS value, more ischemia and transmural necrosis of gastric wall cases. However, the length and number of perforation showed no difference between the two groups.

One patient in the survival group had two perforations at the anterior and posterior wall, reaching up to 6 cm in length, and had a shock onset. The patient's PCIS was 92 with no ischemia and necrosis of gastric wall, respiratory failure or MODS (multiple organ dysfunction syndrome). Another patient in the death group had a 3 cm long perforation at the great curvature, and a PCIS value of 72, with respiratory failure, shock, MODS, and ischemia and necrosis of gastric wall, which indicates that the severity and prognosis had no correlation with length and number of perforation. However, ischemia and transmural necrosis of gastric wall aggravated the infection and led to lower PCIS. Thus, low PCIS value may serve as a prediction for unfavorable prognosis.

Severe as it had been, to our relief, we had bright sight. Despite of the three patients with brain injuries and one who has a second-time perforation, the rest 10 survival patients are in good health and have a good quality of lives after appropriate operations. It inspires us to maintain a positive attitude towards such patients.

5. Conclusion

Gastric perforation in pediatric patients beyond neonatal period may lead to fatal clinical outcomes, which causes a mortality rate of as high as 30%. Spontaneous great curvature of gastric wall perforation has the highest morbidity. Low PCIS, caused by ischemia and transmural necrosis of gastric wall predicts unfavorable prognosis. Most of the survival patients will be in good health after receiving appropriate operations, and only few of them will have second-time perforation or sequela of brain injury after shock.

Conflict of interest

The authors have no conflicts of interest relevant to this article.

Acknowledgment

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. The authors thank the entire staff of the Department of General Surgery, Department of Pathology, and Center for Clinical Epidemiology and Evidence-based Medicine, Beijing Children's Hospital.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.pedneo.2019.03.006.