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Postprandial proximal gastric acid pocket and its association with gastroesophageal acid reflux in patients with short-segment Barrett's esophagus*

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Abstract: Objective: To determine the characteristics of postprandial proximal gastric acid pockets (PPGAPs) and their association with gastroesophageal acid reflux in patients with Barrett's esophagus (BE). Methods: Fifteen patients with BE (defined by columnar lined esophagus of ≥1 cm) and 15 healthy individuals that were matched for age, gender, and body mass index, were recruited. The fasting intragastric pH and the appearance time, length, lowest pH, and mean pH of the PPGAP were determined using a single pH electrode pull-through experiment. For BE patients, a gastroesophageal reflux disease questionnaire (GerdQ) was completed and esophageal 24-h pH monitoring was carried out. Results: The PPGAP was significantly longer (5 (3, 5) cm vs. 2 (1, 2) cm) and the lowest pH (1.1 (0.8, 1.5) vs. 1.6 (1.4, 1.9)) was significantly lower in patients with short-segment BE than in healthy individuals. The PPGAP started to appear proximally from the gastroesophageal pH step-up point to the esophageal lumen. The acidity of the PPGAP was higher in the distal segment than in the proximal segment. In short-segment BE patients, there were significant correlations between the acidity and the appearance time and length of the PPGAP. The length and acidity of the PPGAP were positively associated with gastroesophageal acid reflux episodes. The acidity of the PPGAP was associated with the DeMeester scores, the GerdQ scores, and the fasting intragastric pH. Conclusions: In patients with short-segment BE, a PPGAP is commonly seen. Its length and acidity of PPGAP are associated with gastroesophageal acid reflux, the DeMeester score, and the GerdQ score in patients with short-segment BE.

Key words: Short-segment Barrett's esophagus; Postprandial proximal gastric acid pocket (PPGAP); Gastroesophageal acid reflux
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1 Introduction

Barrett's esophagus (BE), defined by an extension of salmon-colored mucosa into the tubular esophagus ≥1 cm proximal to the gastroesophageal junction (Fitzgerald et al., 2014; Falk, 2016), is considered to be an adaptive change in the esophageal mucosa following prolonged exposure to an acidic environment (Kahrilas et al., 2013). It has been demonstrated that BE is associated with a high risk of the development of esophageal and cardia cancers. Thus, extensive attention has been paid to the etiology, physiology, and pathogenesis of BE (Jankowski and Odze, 2009). BE is a complication of chronic esophageal injury from gastroesophageal reflux disease

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(GERD), as a consequence of the replacement of reflux damaged esophageal squamous cells by mucous-secreting columnar cells. The length of BE with intestinal metaplasia positively correlates with the degree of acid reflux (Fitzgerald et al., 2014). Therefore, BE originates from acid reflux. However, the role of acid reflux in the physiology of BE has not been fully elucidated.

A postprandial proximal gastric acid pocket (PPGAP), defined by the presence of acidity (pH<4) in a segment of the proximal stomach between distal (food) and proximal (lower esophageal sphincter or distal esophagus) nonacid segments, occurs in healthy individuals and patients with GERD (Fletcher et al., 2001; Herbella et al., 2012; Kahrilas et al., 2013; Mitchell et al., 2016). Previous studies have reported that patients with GERD have a higher incidence of PPGAPs with a longer length, a higher position, and a higher probability of acid reflux, than those observed in healthy individuals (Clarke et al., 2008; Beaumont et al., 2010; Kahrilas et al., 2013). Recently, we published a study that analyzed the characteristics of PPGAPs in patients with GERD, which indicated that PPGAPs play an important role in GERD (Wu et al., 2018). However, BE was not included in that study, and thus, the characteristics of PPGAPs in patients with BE remain unknown.

PPGAP occurs in the part of the esophagus in which mucosal injury first appears and is most severe (Fletcher et al., 2004). Considering this observation and the close association between PPGAP and GERD, we hypothesized that the occurrence of BE is due to constant acid exposure caused by a PPGAP in the gastroesophageal junction, and that PPGAPs play an important role in the physiology of BE. Therefore, the aim of the present study was to determine the characteristics of PPGAPs and the association between PPGAPs and gastroesophageal acid reflux in patients with BE.

2 Materials and methods

2.1 Subjects

Patients aged ≥18 years, undergoing upper gastrointestinal endoscopy and diagnosed with BE according to the endoscopic appearance (i.e., columnar lined esophagus of ≥1 cm without histological evidence of intestinal metaplasia) between May 2014 and July 2015, were recruited for the study. According to the results of upper gastrointestinal endoscopy and upper abdominal ultrasound examination, patients with hiatus hernia, chronic atrophic gastritis, gastrointestinal tumors, peptic ulcer, diabetes mellitus, gallbladder diseases, or mental disorders, or those that had a history of surgery for upper gastrointestinal tract diseases, were excluded. In addition, healthy individuals matched for age, gender, and body mass index (BMI) were recruited from the community.

2.2 GerdQ

At recruitment, all BE patients completed a GRED questionnaire (GerdQ), which is a questionnaire consisting of six parts providing an assessment of the degree of gastroesophageal reflux symptoms (Jones et al., 2009). The GerdQ score is the sum of the scores from the six parts and ranges from 0 to 18. A patient was classed as GerdQ-positive if their GerdQ score was ≥8.

2.3 Detection of PPGAPs

The method used for the detection of PPGAPs was similar to that described in our previous study (Wu et al., 2018). Before examination, patients with BE and healthy individuals fasted for more than 8 h. Any drugs that might affect gastrointestinal motility or the pH of gastric juice were stopped for an individually determined amount of time: proton pump inhibitors (PPIs) were stopped for 7 d, H2 receptor blocking agents for 3 d, prokinetic agents for 2 d, and antacids for 6 h. An Omega dynamic impedance pH monitoring system (Medical Measurement Systems (MMS), the Netherlands) with a connected pH electrode, a portable recorder, and monitoring software was used. The pH electrode was calibrated with buffers of pH 4 and 7, separately, and distilled water was used to rinse the electrode before, or in between, immersion in a buffer.

2.3.1 Detection of pH changes in the distal esophagus and proximal stomach on fasting

While the subject was in a seated position, the calibrated single channel pH electrode was pernasally inserted into the gastric cavity and the intragastric pH value was recorded. The electrode was then gradually withdrawn from the stomach to the esophagus. The gastroesophageal pH step-up point, the point at which

the pH reading increased from the gastric pH level to the esophageal pH level (the pH step-up), was identified. The distance between the pH step-up point and the nasal tip was measured. Following this, the pH electrode was repositioned 5 cm below the pH step-up point, and pulled proximally at a rate of 1 cm/min, and the pH value at each detection point was recorded until the esophageal pH reading was observed. The electrode was then again positioned 5 cm beneath the pH step-up point.

2.3.2 Postprandial pH changes in the distal esophagus and proximal stomach

Each individual subject was given 10–15 min to consume 150 g of bread, 30 g of ham sausage, and 100 mL of water. Ten minutes after consumption, the pH electrode was again pulled proximally at a rate of 1 cm/min. The pH value at each detection point was recorded until the esophageal pH was observed. The pH electrode was then once again repositioned 5 cm beneath the gastroesophageal pH step-up point.

Postprandial pH changes at each detection point in the distal esophagus and proximal stomach were detected by repeated pull-through of the pH electrode. The distance from each detection point to the nasal tip was recorded with the time of detection and the pH value. The measurement was continued until the maximum range and peak acidity were detected. Then, the appearance of a PPGAP (i.e., when the pH value of the region between the distal esophagus and proximal stomach was ≥4 preprandially and <4 postprandially), and its appearance time (i.e., the period of time from the end of a standard meal to the time of PPGAP appearance), length (i.e., the length determined by the maximum acidified region during PPGAP appearance), lowest pH, and mean pH (i.e., the mean of the pH values detected at all the acidified points during PPGAP appearance) were calculated.

2.4 Esophageal 24-h pH monitoring

For patients with BE, the pH electrode was placed 5 cm above the gastroesophageal pH step-up point after the detection of a PPGAP. Patients were then allowed to go home with a portable pH recorder. They were required to keep a record of meal time, postural changes, and gastroesophageal reflux symptoms, and were not allowed to have sweet, acidic, cold, or hot food, or carbonated beverages. After 24-h pH monitoring, the data were uploaded into the MMS system. Gastroesophageal acid reflux episodes and DeMeester scores were analyzed automatically. Pathological gastroesophageal acid reflux was defined by a DeMeester score of ≥14.72 in the esophageal 24-h pH monitoring.

2.5 Statistical analysis

Data obtained from the present study are not normally distributed and thus are presented as the median and percentiles (first and third quartiles). Mann-Whitney *U* tests and Pearson's correlation analysis were used to analyze data with a non-normal distribution, and Chi-square tests were performed to compare the categorical data between the two groups. The statistical analyses were performed using the statistical software SPSS 17.0 (Chicago, IL, USA). A P value of <0.05 was considered to be statistically significant.

3 Results

3.1 Comparison of PPGAPs between patients with BE and healthy individuals

Fifteen patients with short-segment BE with a length range of 1.0–3.0 cm (7 males and 8 females, ranging from 32–65 years of age) and 15 healthy individuals (7 males and 8 females, ranging from 31–66 years of age) were recruited. A PPGAP was detected in all subjects, but the PPGAP was significantly longer (P=0.001) and the lowest pH was significantly lower (P=0.013) in patients with short-segment BE than in the healthy individuals (Table 1). There was no significant difference in the mean pH of the PPGAP between the two groups, although the reading tended to be lower in patients with short-segment BE than in healthy individuals.

3.2 PPGAP in patients with BE

Observationally, the PPGAP started to appear proximally from the gastroesophageal pH step-up point to the esophageal lumen. The acidity of the PPGAP was higher in the distal than in the proximal segment. After the PPGAP was extended to the maximum length, its acidity continued to increase until a peak was reached. In patients with BE, there was no food buffering after meals within the 5-cm

Table 1 Comparison of indicators for PPGAP between patients with short-segment BE and healthy individuals

Group	Time of PPGAP appearance (min)	Length of PPGAP (cm)	Lowest pH of PPGAP	Mean pH of PPGAP
Patients with short-segment BE (n=15)	14 (13, 19)	5 (3, 7)*	1.1 (0.8, 1.5)*	1.8 (1.6, 2.2)
Healthy individuals (n=15)	19 (16, 21)	2 (1, 2)	1.6 (1.4, 1.9)	2.1 (1.7, 2.3)

Data are expressed as median (25th percentile, 75th percentile). * P<0.05. PPGAP: postprandial proximal gastric acid pocket; BE: Barrett's esophagus

region beneath the gastroesophageal pH step-up point and the PPGAP: the acidity remained the same pre- and postprandially. The acidity in the esophagus above the PPGAP declined postprandially to a weak acid level (pH≥4).

Fig. 1 shows the PPGAP profile of one patient with short-segment BE, as an example. In this patient, the pH values varied in the distal esophagus and the proximal stomach on fasting and postprandial duration. In this short-segment BE patient, the gastroesophageal pH step-up point was located 49 cm away from the nasal tip on fasting. The acidified part of the PPGAP was present at the start of the PPGAP detection cycle and extended proximally from the gastroesophageal pH step-up point, reaching its maximum range at 40 min and maximum acidity at 70 min after a meal.

3.3 Correlations among the appearance time, length, and lowest pH of PPGAP in patients with BE

There was a significant correlation between the lowest pH and the appearance time (r_s=0.788, P< 0.001; Fig. 2a) and the length of the PPGAP in patients with short-segment BE (r_s=−0.915, P<0.001; Fig. 2b).

3.4 Correlation between PPGAP and gastroesophageal acid reflux in patients with BE

There were significant correlations between the length (r_s=0.969, P<0.001; Fig. 3a) and acidity (i.e., lowest pH, r_s=−0.876, P<0.001; Fig. 3b) of the PPGAP and gastroesophageal acid reflux episodes.

There were significant correlations between the acidity (i.e., mean pH) of the PPGAP and the GerdQ (r_s=−0.854, P<0.001; Fig. 4a) and the DeMeester scores (r_s=−0.914, P=0.007; Fig. 4b) in patients with BE.

The lowest pH of the PPGAP was significantly associated with the fasting intragastric pH in patients with BE (r_s=0.765, P=0.001; Fig. 5).

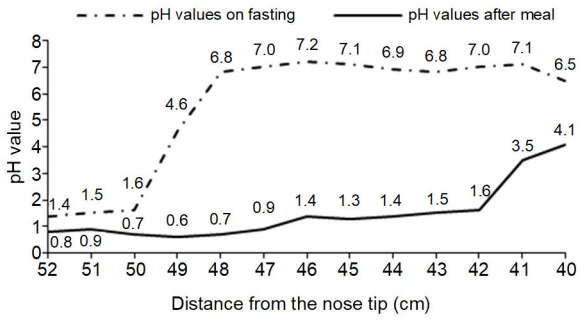

Fig. 1 pH values on fasting and 70 min after meal between the distal esophagus and proximal stomach in one patient with short-segment Barrett's esophagus (BE) as an example case

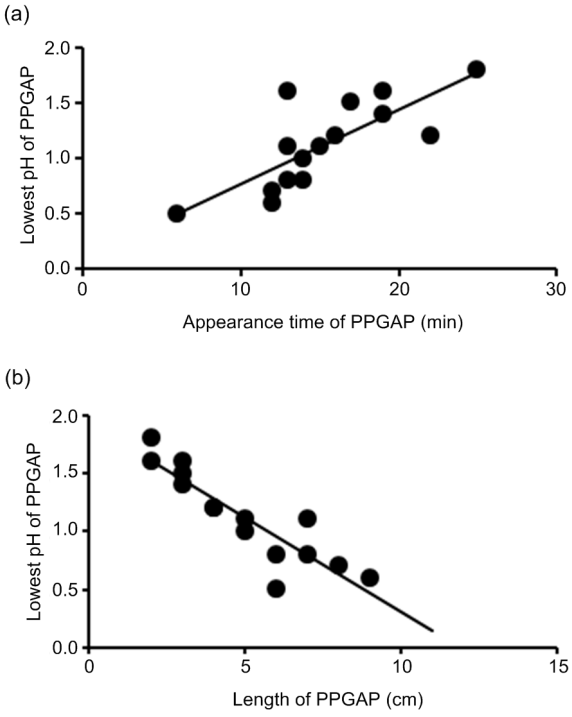

Fig. 2 Correlations between the lowest pH and the appearance time (a) and length (b) of postprandial proximal gastric acid pocket (PPGAP) in patients with short-segment Barrett's esophagus (BE)

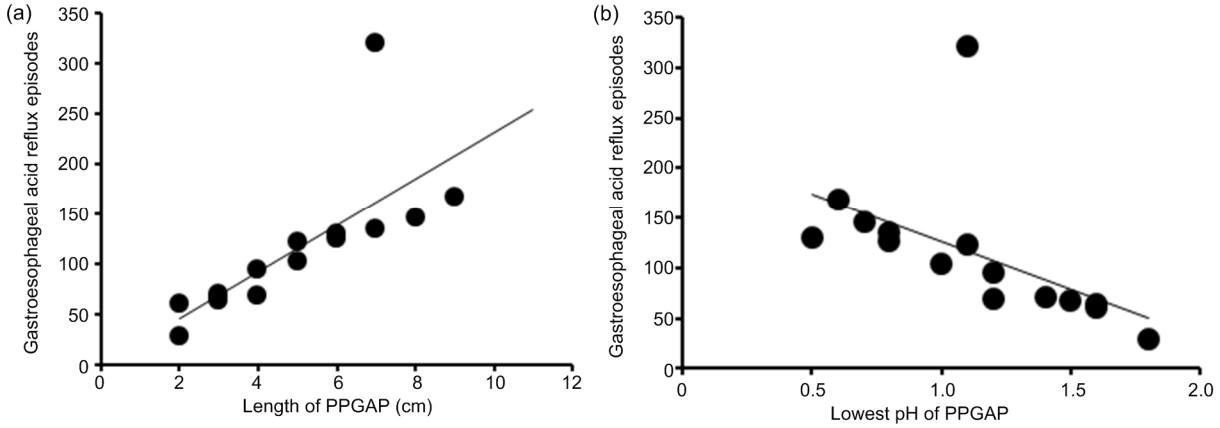

Fig. 3 Associations of gastroesophageal acid reflux episodes with the length (a) and lowest pH (b) of postprandial proximal gastric acid pocket (PPGAP) in patients with short-segment Barrett's esophagus (BE)

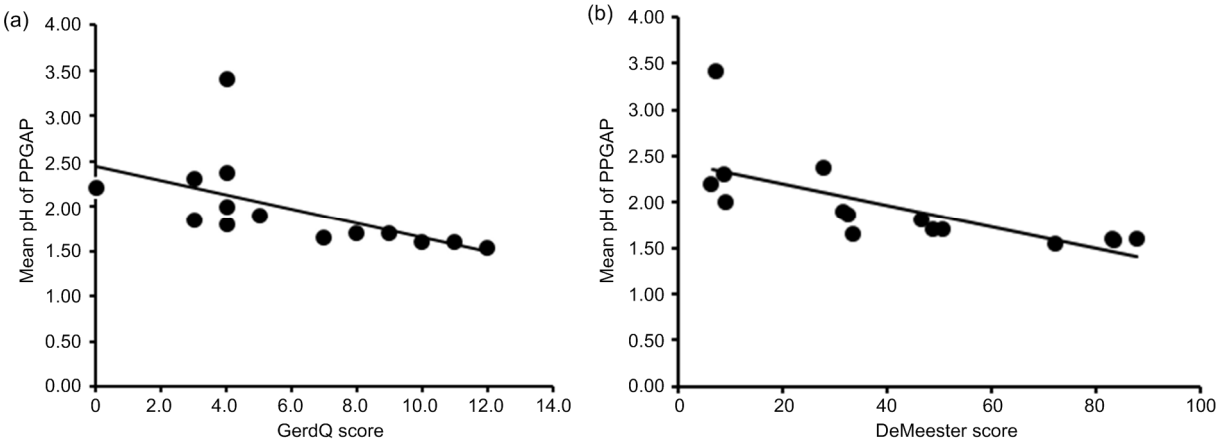

Fig. 4 Associations of the mean pH of postprandial proximal gastric acid pocket (PPGAP) with the GRED questionnaire (GerdQ) (a) and DeMeester scores (b) in patients with short-segment Barrett's esophagus (BE)

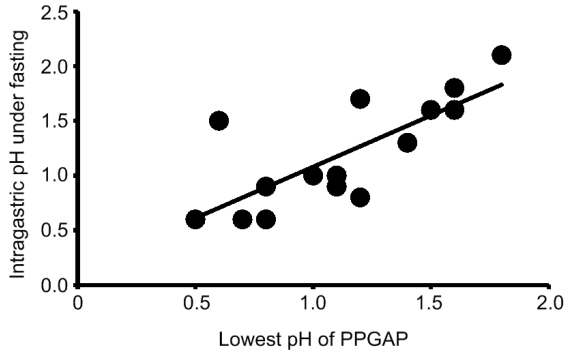

Fig. 5 Association between the lowest pH of postprandial proximal gastric acid pocket (PPGAP) and the fasting intragastric pH in patients with short-segment Barrett's esophagus (BE)

4 Discussion

In this study, a PPGAP was present in all healthy individuals and patients with short-segment BE. The PPGAP was significantly longer and its pH was significantly lower in patients with short-segment BE than in healthy individuals. Furthermore, in patients with short-segment BE, the lowest pH of the PPGAP was associated with its appearance time and length. The length and pH of the PPGAP were significantly correlated with gastroesophageal acid reflux episodes, and the pH of the PPGAP was associated with the DeMeester scores, GerdQ scores, and the fasting intragastric pH. These findings indicate that PPGAPs

may play important roles in the pathology of short-segment BE, although further investigation with a larger number of patients is required to draw a firm conclusion.

A PPGAP has been reported to appear 10–15 min after a meal in patients with GERD and 19 min after a meal in healthy individuals (Clarke et al., 2008; Beaumont et al., 2010; Wu et al., 2018). In our study, PPGAPs were detected in patients with short-segment BE 10 min after a standard meal, and the median appearance time was 14 min. The acidified part of the PPGAP was already present at the start of PPGAP detection in some patients, suggesting that it may have appeared just a few minutes after the standard meal. In healthy individuals, PPGAPs occurred 19 min after a meal. This suggests that PPGAP detection should be started immediately following a standard meal in patients with short-segment BE.

In this study, PPGAPs extended proximally from the gastroesophageal pH step-up point, and a higher level of acidity (lower pH) was generally found in the distal segment of the PPGAP both in patients with short-segment BE and in healthy individuals. After the PPGAP had extended to its maximum range, its acidity continued to increase until a peak was reached. The PPGAP then gradually reduced from the proximal segment with a decline in both its length and acidity. A decline in the esophageal pH was also detected above the PPGAP with a weak acidity (high pH). This process of change in the PPGAP has not been reported before.

Reflux episodes are conventionally measured 5 cm proximal to the proximal margin of the lower esophageal sphincter. However, mucosal pathology is usually limited to the distal esophageal mucosa, which is consistent with the location of BE (Grigolon et al., 2009). Fletcher et al. (2004) found that 24-h esophageal acid exposure was about six times greater at 0.5 cm than at 5.5 cm proximal to the squamocolumnar junction, and acid exposure adjacent to the squamocolumnar junction is less likely to be symptomatic than conventionally measured reflux. Long-term exposure to acid and weak acidity (high pH) due to a PPGAP may contribute to mucosal inflammation, intestinal metaplasia, and BE. This phenomenon is pathophysiologically important, especially for patients with short-segment BE or BE without reflux symptoms.

It remains unclear how PPGAPs develop in patients with short-segment BE. Previous studies have reported that several factors, such as the escape of gastric juice from neutralization by food in the proximal stomach, gastric motility, gastric anatomical structure, hiatus hernia, and types of food, may be involved in the formation of the acid pockets (Hila et al., 2006; McColl et al., 2010; Boecxstaens et al., 2011; Curcic et al., 2012; Herbella et al., 2012). The persistence of acid in the proximal stomach would be facilitated by the relative quiescence of the most proximal part of the stomach after a meal, with motor contractions arising distal to this point and then moving toward the antrum (Fletcher et al., 2001). Hiatus hernia impairs gastroesophageal motility by lowering both the esophageal sphincter function and esophageal clearance. Thus, the presence of hiatus hernia may influence the size and position of a PPGAP (McColl et al., 2010). Conversely, other study has indicated that PPGAPs may result from postprandial gastroesophageal acid reflux (Rohof et al., 2014). To rule out the confounding influence of hiatus hernia on the association between BE and PPGAPs, we excluded patients with hiatus hernia from the present study. The acid pockets in the patients studied were remarkably long. This may be because although hiatal hernia is associated with acid pockets, other factors as mentioned above may contribute to long acid pockets.

In the present study, no postprandial food buffering was found within the region 5 cm beneath the gastroesophageal pH step-up point in patients with short-segment BE, although all patients had a PPGAP. This suggests that postprandial food buffering or neutralization is not an essential factor for the formation of a PPGAP in patients with short-segment BE. However, the theory that PPGAPs can be caused by escape from neutralization by food needs to be verified in further studies.

The present study showed that a low gastric pH under fasting correlated with a low pH of PPGAPs in patients with short-segment BE, which partly explained the therapeutic effect of PPIs and other drugs on PPGAPs (Sweis et al., 2013; Rohof et al., 2014). A recent study reported that patients with *Helicobacter pylori* infection had a reduced range and intensity of PPGAP, which was considered to be associated with the decreased acidity (increased pH) caused by the

infection (Mitchell et al., 2017). Herbella et al. (2010, 2011a, 2011b) showed that PPGAPs were uncommon after Nissen fundoplication, distal gastrectomy, and Roux-en-Y gastric bypass (RYGB). This can be explained by the small size of the gastric pouch and the lack of stimulus to the antrum after surgery, resulting in a reduction or absence of acid production in the stomach. Therefore, we assume that, in patients with short-segment BE, the level of gastric acid may directly affect the development and severity of PPGAPs.

The present study revealed positive correlations between the acidity, length of PPGAPs and the gastroesophageal acid reflux, DeMeester score, GerdQ score in patients with short-segment BE. It is accepted that DeMeester scores are an objective indicator of the severity of gastroesophageal acid reflux and that GerdQ scores are correlated with the degree of subjective symptoms of gastroesophageal reflux. Therefore, consistent with our previous observation (Wu et al., 2018), the findings of this study indicate that PPGAPs play an important role in esophageal acid reflux and gastroesophageal symptoms in short-segment BE.

There were several limitations in the present study. First, the number of subjects, including those with short-segment BE and the healthy individuals, was relatively small. This was mainly because the procedure of PPGAP detection is complicated and time-consuming (duration: 3–4 h). For PPGAP detection, multiple pull-throughs (5–7) are required for each subject, which is associated with poor tolerance. Therefore, it is difficult to recruit subjects, especially healthy individuals who are age-, gender- and BMI-matched to the patients with BE. Second, patients with long-segment BE were not included in this study. This was because we encountered very few patients with long-segment BE. Thus, the findings in the present study may reflect the characteristics of PPGAP only in patients with short-segment BE, and further investigation with long-segment BE patients is required in future studies. Third, the total duration of each PPGAP was not determined in the present study. Previous studies have indicated that this parameter was 90–120 min (Simonian et al., 2005; Clarke et al., 2009; Wu et al., 2018). Finally, due to the lack of the required instrument, we did not detect whether a "bile pocket" existed in the gastroesophageal junction.

In conclusion, PPGAPs are common, and their length and acidity AP are associated with gastroesophageal acid reflux, DeMeester score, and GerdQ score in patients with short-segment BE. These findings indicate that PPGAPs may play an important role in the development of esophageal acid reflux and its symptoms in patients with short-segment BE.

Contributors

Yuan-yuan NIAN performed the experimental research, writing and editing of the manuscript. Jing WU performed the data analysis. Xian-mei MENG and Jun ZHANG performed the study design, data analysis, and editing of the manuscript. Fu-chu JING, Xue-qin WANG, and Tong DANG contributed to the study design. All authors have read and approved the final manuscript and, therefore, have full access to all the data in the study and take responsibility for the integrity and security of the data.

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Compliance with ethics guidelines

Yuan-yuan NIAN, Xian-mei MENG, Jing WU, Fu-chu JING, Xue-qin WANG, Tong DANG, and Jun ZHANG declare that they have no conflict of interest.

This study was approved by the Medical Ethics Committee of the Second Affiliated Hospital of Xi'an Jiaotong University (Xi'an, China). All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2008 (5). Informed consent was obtained from all patients for being included in the study.

References

Beaumont H, Bennink RJ, de Jong J, et al., 2010. The position of the acid pocket as a major risk factor for acidic reflux in healthy subjects and patients with GORD. *Gut*, 59(4): 441-451. https://doi.org/10.1136/gut.2009.178061

Boecxstaens V, Bisschops R, Blondeau K, et al., 2011. Modulation of the postprandial acid and bile pockets at the gastro-oesophageal junction by drugs that affect gastric motility. *Aliment Pharmacol Ther*, 33(12):1370-1377. https://doi.org/10.1111/j.1365-2036.2011.04664.x

Clarke AT, Wirz AA, Manning JJ, et al., 2008. Severe reflux disease is associated with an enlarged unbuffered proximal gastric acid pocket. *Gut*, 57(3):292-297. https://doi.org/10.1136/gut.2006.109421

Clarke AT, Wirz AA, Seenan JP, et al., 2009. Paradox of gastric cardia: it becomes more acidic following meals while the rest of stomach becomes less acidic. *Gut*, 58(7): 904-909. https://doi.org/10.1136/gut.2008.161927

Curcic J, Schwizer A, Kaufman E, et al., 2012. Effect of baclofen on the mechanism of reflux protection in healthy volunteers and patients with gastro-esophageal reflux disease (GERD) assessed by magnetic resonance imaging (MRI) and high resolution manometry (HRM): a randomized controlled study. *Gastroenterology*, 142(5):S-92-S-93. https://doi.org/10.1016/S0016-5085(12)60354-0

Falk GW, 2016. Updated guidelines for diagnosing and managing Barrett esophagus. *Gastroenterol Hepatol (NY)*, 12(7):449-451.

Fitzgerald RC, di Pietro M, Ragunath K, et al., 2014. British Society of Gastroenterology guidelines on the diagnosis and management of Barrett's oesophagus. *Gut*, 63(1):7-42. https://doi.org/10.1136/gutjnl-2013-305372

Fletcher J, Wirz A, Young J, et al., 2001. Unbuffered highly acidic gastric juice exists at the gastroesophageal junction after a meal. *Gastroenterology*, 121(4):775-783. https://doi.org/10.1053/gast.2001.27997

Fletcher J, Wirz A, Henry E, et al., 2004. Studies of acid exposure immediately above the gastro-oesophageal squamocolumnar junction: evidence of short segment reflux. *Gut*, 53(2):168-173. https://doi.org/10.1136/gut.2003.022160

Grigolon A, Cantú P, Bravi I, et al., 2009. Subcardial 24-h wireless pH monitoring in gastroesophageal reflux disease patients with and without hiatal hernia compared with healthy subjects. *Am J Gastroenterol*, 104(11):2714-2720. https://doi.org/10.1038/ajg.2009.443

Herbella FAM, Vicentine FPP, del Grande JC, et al., 2010. Postprandial proximal gastric acid pocket in patients after Roux-en-Y gastric bypass. *J Gastrointest Surg*, 14(11):1742-1745. https://doi.org/10.1007/s11605-010-1309-5

Herbella FAM, Vicentine FPP, Silva LC, et al., 2011a. Postprandial proximal gastric acid pocket in patients after distal gastrectomy. *Neurogastroenterol Motil*, 23(12):1081-1083. https://doi.org/10.1111/j.1365-2982.2011.01790.x

Herbella FAM, Vicentine FPP, del Grande JC, et al., 2011b. Postprandial proximal gastric acid pocket in patients after laparoscopic Nissen fundoplication. *Surg Endosc*, 25(10):3198-3201. https://doi.org/10.1007/s00464-011-1688-z

Herbella FAM, Vicentine FPP, Silva LC, et al., 2012. Postprandial proximal gastric acid pocket and gastroesophageal reflux disease. *Dis Esophagus*, 25(7):652-655. https://doi.org/10.1111/j.1442-2050.2011.01293.x

Hila A, Bouali H, Xue SW, et al., 2006. Postprandial stomach contents have multiple acid layers. *J Clin Gastroenterol*, 40(7):612-617. https://doi.org/10.1097/00004836-200608000-00010

Jankowski JA, Odze RD, 2009. Biomarkers in gastroenterology: between hope and hype comes histopathology. *Am J Gastroenterol*, 104(5):1093-1096. https://doi.org/10.1038/ajg.2008.172.

Jones R, Junghard O, Dent J, et al., 2009. Development of the GerdQ, a tool for the diagnosis and management of gastro-oesophageal reflux disease in primary care. *Aliment Pharmacol Ther*, 30(10):1030-1038. https://doi.org/10.1111/j.1365-2036.2009.04142.x

Kahrilas PJ, McColl K, Fox M, et al., 2013. The acid pocket: a target for treatment in reflux disease? *Am J Gastroenterol*, 108(7):1058-1064. https://doi.org/10.1038/ajg.2013.132

McColl KEL, Clarke A, Seenan J, 2010. Acid pocket, hiatus hernia and acid reflux. *Gut*, 59(4):430-431. https://doi.org/10.1136/gut.2009.192922

Mitchell DR, Derakhshan MH, Robertson EV, et al., 2016. The role of the acid pocket in gastroesophageal reflux disease. *J Clin Gastroenterol*, 50(2):111-119. https://doi.org/10.1097/MCG.0000000000000439

Mitchell DR, Derakhshan MH, Wirz AA, et al., 2017. The gastric acid pocket is attenuated in *H. pylori* infected subjects. *Gut*, 66(9):1555-1562. https://doi.org/10.1136/gutjnl-2016-312638

Rohof WO, Bennink RJ, Boeckxstaens GE, 2014. Proton pump inhibitors reduce the size and acidity of the acid pocket in the stomach. *Clin Gastroenterol Hepatol*, 12(7):1101-1107.e1. https://doi.org/10.1016/j.cgh.2014.04.003

Simonian HP, Vo L, Doma S, et al., 2005. Regional postprandial differences in pH within the stomach and gastroesophageal junction. *Dig Dis Sci*, 50(12):2276-2285. https://doi.org/10.1007/s10620-005-3048-0

Sweis R, Kaufman E, Anggiansah A, et al., 2013. Post-prandial reflux suppression by a raft-forming alginate (Gaviscon Advance) compared to a simple antacid documented by magnetic resonance imaging and pH-impedance monitoring: mechanistic assessment in healthy volunteers and randomised, controlled, double-blind study in reflux patients. *Aliment Pharmacol Ther*, 37(11):1093-1102. https://doi.org/10.1111/apt.12318

Wu J, Liu D, Feng C, et al., 2018. The characteristics of postprandial proximal gastric acid pocket in gastroesophageal reflux disease. *Med Sci Monit*, 24:170-176. https://doi.org/10.12659/MSM.904964

中文概要

题　目：短节段 Barrett's 食管的胃食管酸反流与餐后近端胃酸袋的相关性研究

目　的：检测并分析短节段 Barrett's 食管（BE）患者的餐后近端胃酸袋（PPGAP）的特点，并分析其与胃食管酸反流的相关性。

创新点：PPGAP 可能是胃食管反流病的病理因素之一，引起广泛关注，而 BE 患者的 PPGAP 特点及与反流

的相关性尚无报道。

方　法：15 名短节段 BE 患者和 15 名健康志愿者分别以牵拉法检测 PPGAP 的发生时间、长度、最低 pH 值和平均 pH 值。BE 患者完成 GerdQ 问卷调查和 24 h 食管 pH 监测。

结　论：短节段 BE 患者普遍存在 PPGAP，同时 PPGAP 的长度和酸度与其胃食管酸反流周期数、DeMeester 评分和 GerdQ 评分具有相关性。

关键词：短节段 Barrett's 食管；餐后近端胃酸袋（PPGAP）；胃食管酸反流