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Original article

The effect of *Helicobacter pylori* eradication on dyspeptic symptoms, acid reflux and quality of life in patients with functional dyspepsia

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ABSTRACT

Background: The aims of this study were to investigate the effect of *Helicobacter pylori* eradication on gastroesophageal reflux, gastrointestinal symptoms and quality of life in patients with functional dyspepsia.
Methods: 20 *H. pylori* positive patients diagnosed as having functional dyspepsia according to Roma-II criteria completed the study period. Esophageal motility testing and pH recordings were obtained from each patient before and at the end of the study period. Each patient's gastrointestinal symptoms were evaluated according to Glasgow dyspepsia score. 36-Item short-form health survey (SF-36) and EDQ5 health survey were obtained form each patient.
Results: H. pylori eradication was accomplished in 13 patients (65%). Glasgow dyspepsia symptom score improved in *H. pylori* eradicated patients (10.3 vs 7.5, $p<0.05$) compared to baseline. Fasting lower esophageal sphincter pressures increased (21.6 vs 25.4 mmHg, $p<0.05$) after *H. pylori* eradication Neither the amplitude of peristaltic contractions in the esophageal body (59.4 vs 57.7 mmHg, $p=$ ns) nor the velocity of peristaltic contractions changed before and after eradication. The percent time of esophageal pH<4 (0.7 vs 2.6, $p<0.001$), reflux events longer than 5 min (0 vs 0.7, $p<0.005$) and total reflux number (10.3 vs 19.3, $p<0.005$) significantly increased after eradication therapy. In the SF-36 health-related quality of life survey, general health score was 3.5 vs. 3.4, physical activity 25.2 vs. 26.4, physical role 5.9 vs. 6.3, emotional pain 4.6 vs. 4.5, social activity 2.0 vs. 1.9, physical pain 3.4 vs. 3.0, vitality 32.3 vs. 34.6, and mental health 11.9 vs. 11.5, before and at the end of treatment ($p=$ ns), respectively.
Conclusions: H. pylori eradication did not influence quality of life in patients with functional dyspepsia. Majority of the patients experienced a significant change in esophageal acid exposure after *H. pylori* eradication. *H. pylori* eradication significantly decreases gastrointestinal symptoms, however has no effect on quality of life in patients with functional dyspepsia.

1. Introduction

Helicobacter pylori (*H. pylori*) is a Gram-negative bacteria that colonizes gastric mucosa and is the important etiologic agent for peptic ulcer disease, gastric carcinoma and mucosa-associated lymphoid tissue lymphoma [1]. Benefits of the eradication of *H. pylori* have been reported from patients presenting with these clinical conditions. However, the improvement in symptoms in patients with functional dyspeptic disorders after *H. pylori* eradication remains controversial. The prevalence rate of *H. pylori* in patients without gastroesophageal reflux disease (GERD) is slightly higher than in those with the disorder, suggesting a protective role of *H. pylori* in GERD [2]. Although a number of clinical trials have assessed the clinical and endoscopic manifestations of GERD after the eradication of *H. pylori* in patients with peptic ulcer disease [3–5], only a few studies included patients with functional dyspepsia (FD) [6]. However, the studies drew different conclusions. These conflicting results bring into mind the question of whether *H. pylori* eradication in patients with FD causes reflux disease and thus could be an argument against *H. pylori* eradication treatment in patients with FD.

In view of the above, the aims of this study were to identify whether *H. pylori* eradication 1) causes acid reflux into the esophagus and 2) has any effect on esophageal motility, dyspeptic symptoms and quality of life in patients with FD infected with *H. pylori*.

2. Patients and methods

Thirty-five patients diagnosed as having FD according to Rome II criteria and infected with *H. pylori* were enrolled into the study [7]. Exclusion criteria were as follows: peptic ulcer disease; previous gastric surgery; gallbladder disease; heartburn or regurgitation as the single or main symptom of dyspepsia; symptoms accompanied with irritable bowel syndrome; illnesses such as heart failure, diabetes, asthma, and kidney insufficiency; history of *H. pylori* eradication; antibiotic usage in the last month; and pregnancy.

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Age, gender, tobacco and alcohol consumption, medications in the time span of dyspepsia symptoms and body mass index (BMI) were recorded. Glasgow dyspepsia symptom score, EuroQol health survey (EQ-D5) and a 36-item short-form health-related quality of life survey (SF-36), were applied in all patients [8,9]. On the same day, esophageal motility study and 24-hour pH monitoring were performed. For each patient, Glasgow dyspepsia symptom score was determined in order to diagnose symptom severity in the beginning and at the end of the 4th month [10]. Dyspepsia symptoms in 7 categories over 6 months were taken into consideration (min. score: 0, max. score: 20): frequency of dyspepsia symptoms, effects of symptoms on daily activities, workforce loss, frequency of consulting doctor, requirement of emergency service, diagnosis of dyspepsia and treatment for dyspepsia. The EQ-D5 health survey evaluation consists of 5 questions in the domains of mobility, self-care, usual activities, pain, and anxiety and despondency and 3 different response categories (no/medium/intensive) [8]. The SF-36 evaluation form was used to determine quality of life, and consists of 36 subjects and 8 basic headings, as health, physical activity, taking physical role, emotional activity, social activity, physical pain, vitality and mental health [9].

Esophageal motility was evaluated with pull through technique by manometer apparatus that runs microperfusion process using brand MMS. Average lower esophageal sphincter (LES) relaxation pressure (reference 6–25 mmHg), percentage of wet swallowing over peristaltic waves (reference >80%) and average esophagus corpus amplitude (reference 30–160 mmHg) were determined. Localization of LES was defined by manometric observation, and pH measurement was made by using double sensor antimony catheter which was placed 5 cm above the LES.

We used the following pH monitoring parameters with corresponding reference values for quantification of GERD: total pH<4 reflux period (<4.2 total %), while standing (<6.3 total %), while supine (<1.2 total %), number of reflux episodes (<50), longest episode (<9 min), number of long reflux periods >5 min (<3), and the Johnson–DeMeester score (>14.7). The measurements of 24-hour pH meter and manometer were analyzed by using MMS software program.

Upper gastrointestinal endoscopy was performed at inclusion and at the end of the study period. The patients were evaluated in terms of erosive esophagitis, and,three antral biopsy samples were taken from each patient during the endoscopic examination. In these biopsies, rapid urease test, culture and cytological examination were performed in order to detect the presence of *H. pylori*. Reflux esophagitis was defined according to the Los Angeles classification [11]. As an eradication treatment, lansoprazole 30 mg b.i.d., clarithromycin 500 mg b.i.d., and amoxicillin 1000 mg b.i.d. before meals were introduced for 14 days. *H. pylori* eradication was considered when negative results were obtained in the histological examination, culture and rapid urease test at the 4th month. The use of liquid antacids was permitted during the trial in order to relieve dyspeptic symptoms.

A written informed consent was obtained from each patient before the initiation of the study and the local ethics committee approved the study protocol.

2.1. Statistical analysis

The data were analyzed using the Wilcoxon, chi-square and Mann–Whitney *U* tests. A *p* value of less than 0.05 was considered to be significant.

3. Results

In the beginning of the study, 35 FD patients were enrolled. At the end of the 4th month, after the eradication treatment, 26 patients were available for re-evaluation. Of the 9 unavailable patients, 4 did not wish to attend as they had no complaints, 3 could not be reached, 1 was in another health center due to dysphonia, and 1 patient was in her second month of pregnancy. Two patients of the 26 who presented for evaluation did not accept repeat manometric/pH investigation; in an additional 4 patients, pathological acid reflux was determined on pH monitoring despite no explicit symptoms of reflux in the beginning and their results were not evaluated. Hence, the final evaluation included the results of 20 patients.

Average age of the 20 patients was 44.6 years (range: 24–66) and 15 were female. The average duration of disease was 22.1 months (range: 3–96 months). Only 4 patients were smokers and only 1 regularly consumed alcohol. None of these patients had any preponderant illnesses for which they were receiving medications. *H. pylori* eradication was successfully achieved in 13 of the 20 (65%) patients. After the treatment, Glasgow dyspepsia score decreased from 10.5 ± 3.1 to 8.1 ± 3.1 for the whole group ($p<0.01$). As for *H. pylori* eradicated patients, Glasgow dyspepsia symptom score decreased from 10.3 to 7.5 compared to baseline ($p<0.05$). However, Glasgow dyspepsia symptom score did not improve in non-eradicated patients (10.7 vs. 9, $p=$ ns).

In the SF-36 health-related quality of life survey, general health score was 3.5 vs. 3.4, physical activity 25.2 vs. 26.4, physical role 5.9 vs. 6.3, emotional pain 4.6 vs. 4.5, social activity 2.0 vs. 1.9, physical pain 3.4 vs. 3.0, vitality 32.3 vs. 34.6, and mental health 11.9 vs. 11.5, before and at the end of treatment, respectively. In the EQ-D5 survey, the respective domain scores were moving 0.4 vs. 0.5, self-care 0.2 vs. 0.2, ordinary activities 0.4 vs. 0.5, pain 1.2 vs. 0.9, and anxiety/bad mood 0.9 vs. 0.6. Only the decline in the EQ-D5 pain complaint score was statistically significant ($p<0.05$).

No effect of eradication treatment on esophageal motility was observed. An increase was detected in pH<4 total reflux duration while standing, pH<4 reflux duration, number of reflux periods>5 min, longest reflux duration, and in the Johnson–DeMeester score. In Table 1, results of esophageal motility and pH monitorization have been summarized for whole group.

In the 4-month observation, in the 13 patients in whom *H. pylori* eradication was successful, Glasgow dyspepsia score was significantly improved compared to pre-treatment (10.3 vs. 7.5, $p<0.05$). No change was observed in BMI from the beginning to the end of the treatment (27.23 ± 4.41 vs. 27.43 ± 3.98). No statistically significant difference was determined in the SF-36 health survey, measuring the quality of life, after the treatment. In the EQ-D5 health survey, a distinctive decline was determined only in the pain score (Table 2).

Two of the 13 patients in whom *H. pylori* eradication was successful reported heartburn. Prior to treatment in these 2 patients, there was

Table 1
Esophageal manometry and twenty-four hour ambulatory pH data at entry and after 4-month of treatment of the study population.

	Before treatment ($n=20$)	After treatment ($n=20$)	*p* values
Manometric results			
LES pressure (mmHg)	20.9 ± 7.6	25.6 ± 6.7	$p<0.005$
LES relaxation (%)	96.2 ± 5.2	94.4 ± 4.2	$p<0.05$
Esophageal body contraction amplitude (mmHg)	54.9 ± 22.4	56.7 ± 23.0	NS
Peak velocity (sec)	3.7 ± 1.6	3.5 ± 2.1	NS
Esophageal pH monitoring (reference)			
Total reflux time (<4.2 total %)	0.8 ± 0.7	$2.2 + 2.2$	$p<0.005$
Upright total time spent in reflux (<6.3 total %)	1.2 ± 1.5	2.6 ± 3.0	$p<0.05$
Supine total time spent in reflux (<1.2 total %)	0.6 ± 0.6	$1.1 + 1.6$	NS
Number of reflux periods >5 min (<3)	0 ± 0.2	0.7 ± 0.8	$p<0.01$
Longest reflux duration (<9.2 min)	2.9 ± 2.9	6.0 ± 4.8	$p<0.005$
Total reflux number (<50)	10.7 ± 8.3	16.1 ± 10.2	NS
Johnson-DeMeester score (<14.72)	3.4 ± 2.4	7.3 ± 5.8	$p<0.01$
Gastric pH (% total time pH<4)	68.6 ± 16.2	79.1 ± 10.8	$p<0.05$

Table 2
SF-36, EQ-D5 and Glasgow dyspepsia symptom scores of the patients according to *Helicobacter pylori* status.

	H pylori eradication successful ($n=13$)			*H pylori* eradication not successful ($n=7$)		
SF-36 health survey	Before treatment	After treatment	p values	Before treatment	After treatment	p values
General health	3.6	3.5	NS	3.3	3.0	NS
Physical activity	25.8	26.9	NS	24.3	25.6	NS
Physical role	6.0	6.6	NS	5.7	5.8	NS
Emotional pain	4.4	4.6	NS	4.7	4.0	NS
Social activity	1.5	1.8	NS	3.2	3.1	NS
Body pain	3.3	2.7	NS	3.6	3.5	NS
Vitality	32.2	34.0	NS	32.4	35.9	NS
Mental health	11.9	11.2	NS	11.9	12.0	NS
EQ-D5 health survey						
Mobility	0.3	0.4	NS	0.3	0.3	NS
Self-care	0.2	0.1	NS	0.3	0.3	NS
Usual activities	0.3	0.4	NS	0.6	0.7	NS
Pain	1.0	0.9	$p<0.05$	1.7	1.0	$p<0.05$
Anxiety-despondency	0.8	0.7	NS	1.1	0.4	NS
Glasgow dyspepsia score	10.3	7.5	$p<0.05$	10.7	9.0	NS

no dominant heartburn complaint or esophageal motility disorder. In the endoscopic examination, both patients had esophagitis grade A and in the esophageal pH monitoring, pathological acid reflux was determined. In the manometric examination, LES pressure was higher when compared to pre-eradication treatment (19 vs. 22; 25 vs. 27 mmHg), but body contraction amplitudes were normal. While there were no changes in peak velocity or esophageal body contraction amplitude after the eradication treatment, a statistically significant increase was detected in pH<4 total reflux duration while standing, pH<4 reflux duration, number of reflux periods >5 min, longest reflux duration, total reflux number and Johnson–DeMeester score (Table 3).

4. Discussion

In our study, successful *H. pylori* eradication was achieved in 13 of 20 patients. In the 4-month check-up in endoscopic examination and in pH monitoring, newly presenting GERD was detected in 2 of the 13 patients in this group. Those 2 patients did not suffer from heartburn or esophageal motility disorder. No gastroesophageal reflux was detected in endoscopic examination and pH monitoring in those patients in whom *H. pylori* eradication was unsuccessful.

The 13 patients with successful eradication were determined as having increase in LES pressure (21.6 vs. 25.4 mmHg), but there was no change in esophageal body motility. In the 2 patients with abnormal pH parameters, there was an increase in LES pressure, but there was no change in contraction amplitudes.

Verma et al. conducted a prospective study with 20 patients and reported that only 1 of 11 patients, who did not have GERD in the beginning, suffered from GERD after a year. They reported that *H. pylori* eradication treatment had no effect on LES pressure or esophageal body motility [12].

Tanaka et al. studied 9 *H. pylori* positive patients before and 6 months after successful *H. pylori* eradication by means of gastric emptying, esophageal manometry, gastric and esophageal pH monitoring. They observed that in the patients without esophageal and gastric motility disorder, there was no significant change in fasting or postprandial LES pressure, in esophagus primary peristaltic contractions or in frequency of temporary LES relaxation. They reported only 1 patient with erosive GERD, who had irregularities in esophageal functions [13].

In a similar study, 20 FD patients were observed with esophageal manometric study and with 24-hour pH monitoring before eradication and in the 3rd month after eradication. After eradication, no new GERD presentation was detected on endoscopic examination or pH monitoring. In manometric observation, while LES and UES pressure showed significant changes, there was no change in gastric emptying time and in pH measurements [14].

In our study, in 8 of 13 patients with successful *H. pylori* eradication, we observed an increase in total pH<4 time and pH<4 time while standing. In 10 of them, there was an increase in the number of reflux periods and in 6 patients there was an increase in number of reflux periods >5 min and the longest reflux duration ($p=0.002$, $p=0.036$, $p=0.025$, $p=0.008$, $p=0.024$). However, in the patients in whom *H. pylori* eradication was unsuccessful, there was no significant increase in these five parameters ($p>0.05$).

In a study in which only FD patients were considered, no significant change was observed in pH monitoring in the 3rd month [14]. In another study, 9 of 20 patients with GERD before eradication treatment were determined to have an increase in reflux episode number; however, there were no significant changes in reflux number >5 min, Johnson–DeMeester score and longest reflux time. An increase was detected in the number of reflux episodes, but the difference was not statistically significant [12].

In our study group, there was no change in esophageal body motility or velocity after *H. pylori* eradication. We also observed increase in LES pressure and decrease in % LES relaxation. One study including FD patients reported a decrease in both LES and UES

Table 3
Esophageal manometry and twenty-four hour ambulatory pH data of the patients in which *Helicobacter pylori* eradication achieved and not achieved.

	H pylori eradication successful ($n=13$)			*H pylori* eradication unsuccessful ($n=7$)		
	Before treatment	After treatment	p values	Before treatment	After treatment	p values
Esophageal manometry						
LES pressure (mmHg)	21.6 ± 5.9	25.4 + 4.7	$p<0.05$	19.7 ± 10.3	26.0 ± 9.9	NS
LES relaxation (%)	97.1 ± 3.9	94.5 ± 3.4	$p<0.05$	94.6 ± 6.9	94.1 ± 5.8	NS
Esophageal body contraction amplitude (mmHg)	59.4 ± 22.7	57.7 ± 23.8	NS	46.6 ± 20.9	54.9 ± 23.2	NS
Peak velocity (sec)	3.6 ± 1.6	3.0 ± 1.6	NS	3.7 ± 1.8	4.3 ± 2.9	NS
Esophageal pH monitoring (reference)						
Total reflux time (<4.2 total %)	0.7 ± 0.7	2.6 ± 2.6	$p<0.001$	0.9 ± 0.6	1.4 ± 0.8	NS
Upright total time spent in reflux (<6.3 total %)	1.2 + 1.8	3.4 ± 3.3	$p<0.03$	0.9 ± 0.6	0.8 ± 1.1	NS
Supine total time spent in reflux (<1.2 total %)	0.5 ± 0.5	0.8 ± 1.2	NS	0.4 ± 0.6	0.5 ± 0.6	NS
Number of reflux periods >5 min (<3)	00 ± 00	0.7 ± 0.9	$p<0.05$	0.1 ± 0.3	0.6 ± 0.8	NS
Longest reflux duration (<9.2 min)	2.3 ± 1.5	6.5 ± 5.1	$p<0.001$	3.9 ± 4.6	5.1 ± 4.6	NS
Number of reflux periods (<50)	10.3 ± 9.7	19.2 ± 11.1	$p<0.05$	11.4 ± 5.4	10.3 ± 4.8	NS
Johnson–DeMeester score (<14.72)	2.9 ± 2.4	8.1 ± 6.7	$p<0.05$	4.2 ± 2.3	5.7 ± 3.5	NS
Gastric pH (% total time pH<4)	68.7 ± 19.4	78.9 ± 12.6	NS	68.3 ± 8.7	79.3 ± 8.1	$p<0.05$

pressure after eradication treatment (respectively 20.9 vs. 13.5, $p=0.06$; 62 vs. 44.2, $p=0.01$) [14]. Wu et al. investigated *H. pylori*-negative and -positive patients with GERD, and reported LES pressure as 12.2 mmHg in the *H. pylori*-negative group and as 15.3 mmHg in the *H. pylori*-positive group. Distal esophageal contraction amplitude values in these groups were 56 mmHg and 68 mmHg, respectively [15]. Other studies have also reported increases in some values in manometric observation after *H. pylori* treatment when compared to baseline, but the differences were not statistically significant [12,13,16].

In our study, there was an improvement in Glasgow dyspepsia score in 13 patients with successful eradication in the 4th month, but no improvement was noted in the group without eradication. Glasgow dyspepsia severity score is a reliable and validated tool for assessing the severity of dyspepsia. It is highly reproducible and high validity and responsiveness. In addition, it is simple and rapid to perform, and provides a valuable tool for assessing the response to treatment in patients with dyspepsia. The questionnaire is concerned with assessing the severity of the symptoms rather than assessing the type of the symptom. It can be used for any symptom of combination of symptoms thought to be related to the upper gastrointestinal tract [10].

In a study of Beşisik et al., dyspepsia symptom score decreased from 9.7 ± 2.9 to 4.1 ± 2.0 in FD patients ($p<0.0001$) [14]. Gisbert et al. randomized 50 *H. pylori*-positive FD patients, and used *H. pylori* eradication treatment in 34 patients and ranitidine in 16 patients. After 12 months, they observed no differences between the eradication treatment group and the ranitidine group [17]. In two placebo-controlled studies, no differences in symptom scores were determined when the successful and non-successful *H. pylori* eradication groups were compared [18,19]. Our study could be criticized due to small sample size and only two-thirds of patients attended for a repeat pH/manometry study.

The quality of life declines in FD patients, but there have been controversial results on the effect of *H. pylori* eradication in this group. In a study of 100 patients with *H. pylori*-positive FD, an 82.7% cure rate was achieved with triple eradication treatment. However, improvements in dyspepsia symptom score and in quality of life were determined in only 11.7% [20]. In our study there was no improvement in SF-36 health survey in the group with successful eradication. In contrast, a significant decrease in health score in the EQ-5D health survey, which is a mobility capability survey that provides partial information about quality of life, was considered meaningful.

In *H. pylori*-related corpus gastritis, gastric acid secretion is suppressed. Gastric pH measurement is lower than in patients with antral gastritis. In our study, there was an increase in gastric pH (% total time pH<4) duration percentage in both the successful and unsuccessful eradication treatment groups. The decrease in gastric pH is explained with increasing acid secretion after *H. pylori* eradication and healing of corpus gastritis. In one study, a decline was documented in night gastric pH>4 time period in *H. pylori*- positive patients in the 6th month after the treatment [13].

One of the factors for GERD is obesity. GERD has been found 1.82 times more in overweight and 2.91 times more in obese individuals compared to those with normal weight [21]. Azuma et al. used eradication treatment in 302 people with *H. pylori* infection out of 932 industry workers. There was no difference between *H. pylori*-positive and *H. pylori*-negative subjects with respect to BMI. BMI and weight gain were more increased in the group with eradication than in the non-eradicated group in the 12th month [22]. In our study, which had a shorter observation period, there was no documented change in BMI or weight gain after *H. pylori* eradication treatment.

The one primary mechanism by which *H. pylori* might influence the pathogenesis of GERD is by modifying the gastric refluxate. *H. pylori* infection has a major effect on somatostatin secreting D cells in the gastric antrum such that feedback inhibition by luminal acid on gastrin release is interrupted. As a result, gastrin levels are higher in *H. pylori* infected individuals and these levels do not exhibit normal feedback inhibition [23]. Lack of feedback inhibition is hypothesized to be ultimately responsible for the increased acid secretion found in patients with duodenal ulcers who are *H. pylori* positive and have antral-predominant gastritis. *H. pylori* infection associated with corpus-predominant gastritis is also associated with increased gastrin levels. However, despite increased serum gastrin levels, such patients have decreased acid secretion. Corpus-predominant gastritis reduces acid secretion as a result of local inflammation and increased levels of cytokines, such as tumor necrosis factor alpha and IL-1Beta. This can eventually lead to hypochlorhydria and gastric atrophy. Reversal of *H. pylori*-induced corpus gastritis (and associated hypochlorhydria) has the potential to increase gastric acid secretion, which could render caustic what was previously asymptomatic reflux [24]. Return of acid secretion shortly after successful eradication is associated with increased H/K ATPase mRNA expression [25]. Another hypothesized mechanism accounting for worsening reflux disease in patients with *H. pylori* is that urease-mediated ammonia production may exert a buffering effect on gastric juice. As a result, eradicating *H. pylori* in patients with corpus-predominant gastritis could worsen GERD.

There is increasing evidence as to the lack of impact of endoscopy – based management in younger patients, relative to its high cost. *H. pylori* test and treat appears to be more cost effective than endoscopy-based management, and more effective than acid suppression alone. Early investigation by endoscopy or *H. pylori* testing may benefit some patients with dyspepsia but is not cost effective as part of an overall management strategy [26].

In conclusion, eradication treatment led twenty-four hour ambulatory pH parameters became abnormal in FD patients after H. pylori eradication. In twenty-four hour ambulatory pH, eradication treatment caused an increase in the percentage of time under pH 4, the percentage of time under pH 4 while standing. Furthermore an increase was observed in the total reflux episode number, reflux period more than 5 min and Johnson–DeMeester score; eradication had no effects on esophageal motility. Although there was an improvement in dyspepsia symptom score after *H. pylori* eradication treatment, there was no change in quality of life. We suggest that, a randomised, placebo-controlled trial is urgently needed in patients with FD utilizing twenty-four hour ambulatory pH and esophageal manometry to verify results obtained from this study.

5. Learning points

- Majority of the patients with functional dyspepsia experienced a significant change in esophageal acid exposure after *H. pylori* eradication.
- *H. pylori* eradication significantly decreases gastrointestinal symptoms, however has no effect on quality of life in patients with functional dyspepsia.

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