Diagn Interv Radiol 2008; 14:138-142

ORIGINAL ARTICLE

Effect of subclinical *Helicobacter pylori* infection on gastric wall thickness: multislice CT evaluation

Sibel Kul, Burak Sert, Ahmet Sarı, Mehmet Arslan, Polat Koşucu, Ali Ahmetoğlu, Hasan Dinç

PURPOSE
To evaluate the effect of subclinical *Helicobacter pylori* infection on the gastric wall thickness with multislice computed tomography (MSCT).

MATERIALS AND METHODS
In 99 subjects without gastric disease, CT scans of the abdomen were obtained after water ingestion and intravenous contrast administration. CT images were evaluated for degree of luminal distention and the thickness of the walls of the gastric antrum and body. We also looked for other radiological signs of gastritis such as the presence of fold thickening, mucosal enhancement, submucosal hypodensity, focal gastric mass-like lesion, and focal wall thickening. All subjects were tested with rapid urease test or stool antigen test and grouped as *H. pylori* positive or negative according to the results.

RESULTS
The average gastric body and antrum wall thicknesses did not show statistically significant difference between *H. pylori* positive and negative groups. The average antral wall thickness was greater than the gastric body wall thickness in 68.5% of cases, independent of *H. pylori* positivity; and antral wall thickness was more than 5 mm in more than 50% of cases. There were no significant differences between the groups in terms of other signs of gastritis.

CONCLUSION
Wall thickening of gastric antrum relative to gastric body is a common finding even in the use of MSCT, and antral thickness commonly exceeds 5 mm. Subclinical *H. pylori* infection has no effect on gastric wall thickness.

Key words: • *Helicobacter pylori* • *gastritis* • *gastric wall* • *multidedector CT*

Gastric wall thickening is one of the most important signs of gastrointestinal diseases. Results of many studies suggest that normal gastric wall thickness ≤5 mm on computed tomography (CT). However, the wall of the gastric antrum is often thickened on CT, and 5 mm may not be an appropriate cutoff.

The antrum is the most common site of involvement for *Helicobacter pylori*, one of the most prevalent human pathogens worldwide. More than 90% of the population in developing countries and 50% of the population in developed countries are infected with *H. pylori*. The bacterium colonizes and infects the stomach. It is now well known that infection commonly shows a subclinical course and causes histological gastritis (1). We investigated the effect of subclinical *H. pylori* infection on gastric wall thickness.

Materials and methods

This study included patients referred to the department of radiology at our institution for abdominal CT examination during a 5-month period. Exclusion criteria were symptoms of dyspepsia or pain, history of prior gastric surgery, known or suspected diagnosis of abdominal malignancy, known or suspected diagnosis of acute pancreatitis or inflammatory bowel disease, previous history of abdominal surgery and/or abdominal radiotherapy, use of nonsteroidal antiinflammatory drugs, previous treatment for *H. pylori* infection, and contraindications for use of intravenous iodinated contrast agent.

Ninety-nine patients (52 males and 47 females; age range, 18–82 years; mean age, 51 years) constituted the study population. After the multislice computed tomography (MSCT) scan, rapid urease test of the endoscopic biopsy material or stool antigen test was used to detect *H. pylori*. Forty-three patients who consented to endoscopy underwent upper gastrointestinal endoscopy and biopsy at the gastroenterology department. Endoscopic features of increased vascularity, edema, rugal hypertrophy or atrophy, erythema, and erosions were considered signs of gastritis. Rapid urease test (CLO test, Delta West Ltd., Perth, Australia) was applied to the biopsy materials; the change of the color indicator from red to yellow was considered positive for *H. pylori*. Stool antigen test (Rapid HpSA Test, Linear Chemicals, Spain) was performed at the microbiology department for the 56 patients who did not consent to the endoscopic procedure. The appearance of pink-blue color in addition to blue control line on reaction stripe was considered a positive result. The institutional review board approved the study, and informed consent was obtained from all participating patients.

From the Departments of Radiology (S.K. ✉ *sibel_ozy@yahoo.com*, B.S., A.S., P.K., A.A., H.D.) and Gastroenterology (M.A.), Karadeniz Technical University School of Medicine, Trabzon, Turkey.

Received 11 December 2007; revision requested 21 March 2008; revision received 4 April 2008; accepted 8 May 2008.

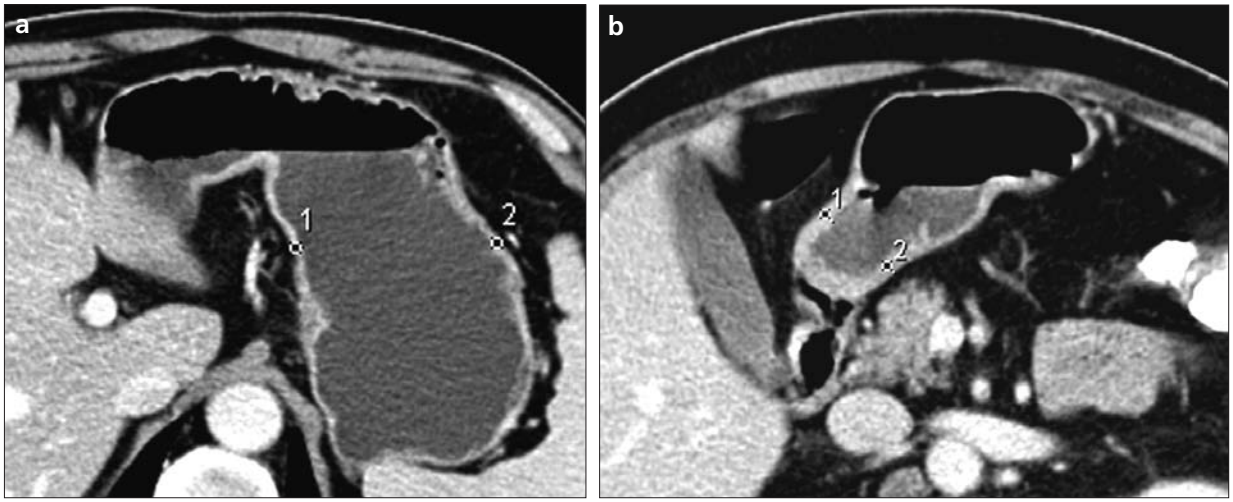

Figure 1. a, b. CT images show the sites of measurements for lesser (1) and greater curvature (2) wall thicknesses of the gastric body **(a)** and the gastric antrum **(b)**.

MSCT technique

MSCT scanning of the abdomen was performed with a Somatom Volume Zoom scanner (Siemens, Germany). Standard scanning parameters were 120 kVp, 165 mAs, 4 × 2.5 mm collimation, and 0.5-s tube rotation. Images were reconstructed with 5-mm sections for routine review on film and workstation. Patients fasted ≥6 hours before scanning. Patients were given 750 mL of water approximately 15 minutes before scanning and an additional 250 mL immediately before the scanning. Images were obtained at portal venous phase with 60-s delay after the administration of 120 mL of non-ionic intravenous contrast material iohexol (Amersham Health, Ireland) or iopromide (Schering, Berlin, Germany) via the antecubital vein with automatic injector (Medrad Vistron CT, Pittsburgh, Pennsylvania, USA) at a rate of 3 mL/s. Dual-phase imaging was performed in some patients according to scanning indications, but the arterial phase images were not evaluated in this study. Scanning was done from dome of the diaphragm to the iliac crest for the upper abdomen and to the symphysis pubis for the whole abdomen at prone position.

Evaluation of CT images

The obtained CT images were transferred to the work station (Virtuosa, Siemens, Germany). Two reviewers together evaluated the images for gastric distention, wall morphology, and thickness on computer without prior knowledge of the results of *H. pylori* tests. Luminal distension was scored according to the diameter of the gastric lumen. A gastric body luminal diameter <5 cm was interpreted as grade 0; 5–8 cm as grade 1; and >8 cm as grade 2. A gastric antrum luminal diameter <2.5 cm was interpreted as grade 0; 2.5–4 cm as grade 1; and >4 cm as grade 2. Grade 0 luminal distension was interpreted as insufficient, and grade 2 luminal distension was considered good.

The wall thicknesses at the sides of greater and lesser curvatures of the gastric body and antrum were measured on axial images using electronic calipers. For the gastric body, the lesser and the greater curvature wall thicknesses were measured on the medial and the lateral walls, respectively (Fig. 1a). For the antrum, the lesser and the greater curvature wall thicknesses were measured on the anterior and the posterior walls, respectively (Fig. 1b). For the gastric body, the midline slice between the esophagogastric junction and incisura angularis was selected; for the antrum, the midline slice between the incisura angularis and antropyloric junction was selected. The measurements were taken perpendicular to the gastric wall, and the folds were not included.

The CT scans were also evaluated for the presence of known CT criteria of gastritis. Gastric wall >5 mm was assumed to be thickened. The thickened wall was evaluated for symmetry as circumferential or asymmetric, and for focal vs. diffuse distribution. Gastric folds >5 mm were considered to be thickened; greater enhancement of the mucosa than the gastric wall was considered significant. Thickened folds, mucosal enhancement, presence of low attenuating stripe of the submucosal layer, and enhancing focal gastric masses were also noted for all CT scans.

Statistical analyses

For the purpose of this study, patients were grouped as *H. pylori* positive and negative according to the results of *H. pylori* tests. The wall thicknesses at the greater and lesser curvature of the gastric body and the antrum were expressed as the mean and standard deviation with 95% confidence intervals. The statistical analysis was performed with SPSS statistical package (SPSS Inc. Chicago, Illinois, USA). The differences in wall thicknesses between *H. pylori* positive and negative groups were evaluated by using Student t test at the antrum and by using Mann-Whitney U test at the gastric body. $P < 0.05$ was considered statistically significant. The relationships of the luminal distention and age to gastric wall thickness were evaluated by Pearson correlation test.

The presence of the other CT findings of gastritis such as the circumferential antral wall thickening, fold thickening, mucosal enhancement, submucosal hypodense stripe, focal gastric mass, and focal thickening of the posterior gastric wall along the greater curvature were evaluated for both groups; and the results were analyzed statistically

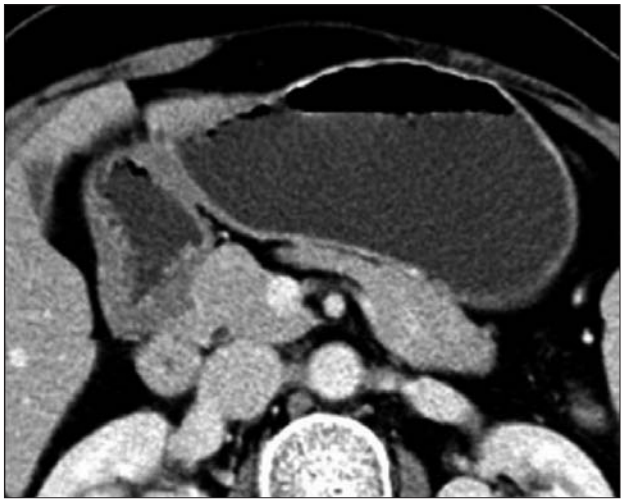

Figure 2. Contrast enhanced abdominal CT image of a 41-year-old woman with breast cancer demonstrates marked circumferential wall thickening of grade 1 distended antrum without gastric body wall thickening. Her stool antigen test was negative.

by chi-square test. $P < 0.05$ was considered statistically significant.

Results

The gastric body and the antrum were easily identified in all patients. According to the results of rapid urease and stool antigen tests, of 99 patients, 43 (43.4%) were *H. pylori* positive and 56 (56.6%) were negative. *H. pylori* positivity was detected in 20 of 43 (46%) patients tested with the rapid urease test and 23 of 56 (41%) patients tested with the stool antigen test.

The luminal distension of the gastric body was grade 1 in 13 (13.1%) and grade 2 in 86 (86.9%) patients; luminal distension of the gastric antrum was grade 0 in 2 (2%), grade 1 in 38 (38.4%), and grade 2 in 59 (59.6%). Insufficient distension was observed at the gastric antrum in two cases, one *H. pylori* positive and the other *H. pylori* negative, with mean antral wall thicknesses of 6.3 mm and 5.3 mm, respectively. The distribution of the distension scores did not show significant difference between *H. pylori* positive and negative groups. The correlation between the gastric distention and the wall thickness was evaluated for gastric body and antrum revealed no correlation ($P = 0.491$ and $P = 0.578$, respectively). Also, no correlation was detected between age and wall thickness ($P > 0.05$).

The mean antral wall thicknesses (mean ± SD) at the greater curvature of *H. pylori* positive and negative groups were 4.80 ± 1.81 mm and 5.45 ± 2.09 mm; at the lesser curvature, mean antral wall thicknesses were 4.72 ± 1.74 mm and 4.96 ± 1.90 mm, respectively. For antral wall thickness, there was no significant difference between *H. pylori* positive and negative groups at the greater ($P = 0.099$) or lesser curvature ($P = 0.523$). The average antral wall thicknesses were 4.77 ± 1.72 mm and 5.21 ± 1.88 mm for *H. pylori* positive and negative groups, respectively.

The mean gastric body wall thicknesses at the greater curvature were 3.80 ± 1.28 mm and 3.43 ± 0.70 mm and at the lesser curvature were 4.38 ± 1.63 mm and 3.83 ± 0.85 mm for *H. pylori* positive and negative groups, respectively. The wall thickness of the greater curvature of gastric body did not demonstrate significant difference between *H. pylori* positive and negative groups ($P = 0.095$), whereas for the lesser curvature, borderline significance ($P = 0.047$) was detected. Averages of the wall thicknesses of two curvatures were 4.09 ± 1.40 mm and 3.63 ± 0.74 mm for *H. pylori* positive and negative groups, respectively. However, the difference between the groups was not statistically significant. Table summarizes the mean wall thicknesses for *H. pylori* positive and negative groups.

Circumferential antral wall thickening was found in 13 (30.2%) of 43 *H. pylori* positive cases and in 21 (37.5%) of 56 *H. pylori* negative cases (Fig. 2). The difference between the two groups was not statistically significant ($P = 0.588$). Thickened folds were found in six (14%) of 43 *H. pylori* positive cases and in 10 (17.9%) of 56 *H. pylori* negative cases; the difference between two groups was not significant ($P = 0.579$). Mucosal enhancement was found in nine (20.9%) of 43 *H. pylori* positive cases and in 17 (30.4%) of 56 *H. pylori* negative cases (Fig. 3). The difference between two groups was not significant ($P = 0.409$).

Submucosal hypodense stripes were found in four (9.3%) of 43 *H. pylori* positive cases and 13 (23.2%) of 56 *H.*

Table. Mean and minimum–maximum wall thicknesses at greater and lesser curvatures of the stomach for *H. pylori* positive and negative groups

			Mean wall thickness (mm)	Minimum–maximum values (mm)
H. pylori (+) (n = 43)	Gastric body	GC	3.80 ± 1.28	2.4–7.5
		LC	4.38 ± 1.63	2.4–10.2
	Antrum	GC	4.80 ± 1.81	2.4–8.4
		LC	4.72 ± 1.74	2.0–8.9
H. pylori (–) (n = 56)	Gastric body	GC	3.43 ± 0.70	2.2–5.9
		LC	3.83 ± 0.85	2.1–6.2
	Antrum	GC	5.45 ± 2.09	3.0–10.2
		LC	4.96 ± 1.90	2.0–8.5

GC: greater curvature; LC: lesser curvature

Figure 3. Contrast-enhanced abdominal CT image of a 72-year-old woman with negative stool antigen test demonstrates marked mucosal enhancement of the stomach.

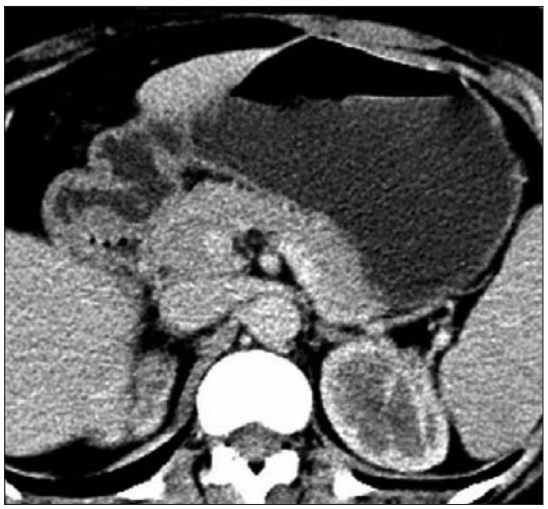

Figure 4. Contrast-enhanced abdominal CT image of a 33-year-old woman with hydatid cyst of the liver, shows a non-contrast enhancing focal mass-like lesion of 15–19 mm, located on the posterior wall of the grade 1 distended antrum and relative thickening of the antrum compared with gastric body. Endoscopic examination did not demonstrate any mass lesion, and *H. pylori* test was negative.

pylori negative cases. The difference between two groups was not significant ($P = 0.121$). The focal gastric mass-like lesion was found in five (11.6%) of 43 *H. pylori* positive cases and two (3.6%) of 56 *H. pylori* negative cases (Fig. 4). The number of cases was not sufficient for statistical comparison. Focal thickening of the posterior gastric wall along the great curvature was found in eight (18.6%) of 43 *H. pylori* positive cases and in nine (16.1%) of 56 *H. pylori* negative cases; the difference between two groups was not significant ($P = 0.950$).

Discussion

Gastric wall thickening is one of the most important signs of gastrointestinal disease. CT, particularly using multislice technology, is a very effective imaging modality in evaluation of the gastric wall, assessing its thickness and contour. For gastric evaluation, adequate distension of stomach is essential. Water is a commonly used oral contrast agent for gastric distention; it is well tolerated, inexpensive, and results in good gastric distension as well as excellent visualization of the enhancing gastric wall. It also allows accurate measurement of gastric wall thickness. Intravenous contrast material is also necessary for the complete evaluation of neoplastic and inflammatory diseases of the stomach (2).

Most sources report that a normal gastric wall thickness is 5 mm or less in an adequately distended stomach at CT (3–6). However, the antral wall is usually thicker than the other parts of the stomach wall. Using MSCT, Pickhardt and Asher (7) evaluated the gastric antrum wall thickness in 153 patients without gastric disease. They concluded that the smooth wall thickening of the gastric antrum relative to the proximal stomach was a normal finding; they found that antral wall thickness commonly exceeded 5 mm, and thickness up to 12 mm was seen in the absence of disease. *H. pylori* is the most common cause of antral thickening, and it commonly has a subclinical course. However, it is not known if there is a relationship between subclinical *H. pylori* infection and antral wall thickening.

H. pylori is a motile, gram-negative bacterium which has the ability to colonize and infect the stomach. It can infect the gastric antrum and/or body, but inflammation tends to be more severe at gastric antrum. It causes chronic gastritis, peptic ulcer, and lymphoproliferative diseases, and it is a major risk factor for gastric cancer. It is the most frequent cause of gastritis in the adult population throughout the world, present in nearly 50% of symptomatic patients undergoing endoscopic examination (1, 8–11). Infection can also be seen in asymptomatic persons. Doolley et al. (1) reported a 32% prevalence of *H. pylori* infection in asymptomatic persons. In our study population, the prevalence of asymptomatic infection was 43.3%, which was higher than that reported in the literature; this was thought to be the result of regional differences.

H. pylori infection can be diagnosed by invasive and noninvasive techniques such as the histological examination, the urea breath test, several serum tests, and the stool antigen test. According to European *H. pylori* Study Group, diagnosis of infection should be by urea breath test or stool antigen test (12). The stool antigen test is both an economical and reliable test for *H. pylori*. Endoscopy allows direct visualization of gastritis, and the rapid urease test allows diagnosis of *H. pylori* with high sensitivity (13).

The most common CT findings of *H. pylori* gastritis are thickening of the gastric folds and wall (14). Urban et al.

reviewed CT scans of 61 biopsy-proven cases of *H. pylori* gastritis retrospectively and found gastric abnormality in 31% of patients (14). Of the gastric abnormalities, 68% were circumferential antral wall thickening and 42% were posterior gastric wall thickening along the greater curvature. In severe cases, the gastric wall may also demonstrate low attenuation or the mucosa may enhance. Infection sometimes can simulate an infiltrating carcinoma or focal gastric mass (14–17).

In our study group of patients without gastric complaints or known gastric disease, the gastric body and the antrum average wall thicknesses did not show statistically significant difference between *H. pylori* positive and negative groups. At the lesser curvature, the gastric body wall was found to be slightly thicker (4.39 mm) for *H. pylori* positive cases than the control group, but this was not more than 5 mm, which is the commonly accepted cutoff point for abnormal thickening. In the literature, there is no reported association between the lesser curvature wall thickening and *H. pylori* infection. Thus this difference might be incidental.

Although, Pickhardt and Asher reported smooth wall thickening of the distal gastric antrum relative to the gastric body in 99% of 153 cases (7), we found the average antral wall thickness was greater than the gastric body wall thickness in 29 (67.4%) of 43 *H. pylori* positive cases and in 39 (69.6%) of 56 *H. pylori* negative cases. In 41.8% of the *H. pylori* positive cases and in 57.0% of the *H. pylori* negative cases, at least one antral wall thickness was >5 mm (range, 5.1–9.3 mm). Our findings show that the greater thickness of the gastric antrum wall than the gastric body wall is a common finding, even using MSCT; it is independent of *H. pylori* positivity. Because antral thickness commonly exceeded 5 mm, we concluded that a 5-mm point may not be appropriate in evaluating the gastric antrum.

Thickened gastric folds, low attenuation of the gastric wall, mucosal enhancement, and focal or circumferential wall thickening are the well-known CT features of the gastritis (14–17). We demonstrated these findings in both *H. pylori* positive and negative cases, and there was no significant difference between groups.

Gastritis was demonstrated in 20 of the 43 cases undergoing endoscopic examination; all were *H. pylori* positive. Gastritis was found at the gastric body in two (10%), at the gastric antrum in seven (35%), and at both antrum and body in 11 (55%) cases. We could not provide endoscopic correlation in all patients because of its invasive nature. Endoscopic correlation is important to exclude causes of antral inflammation and gastric wall thickening other than *H. pylori*. However, the results obtained from whole study population were consistent with the results of 43 endoscopically evaluated patients.

In conclusion, the wall thickening of the gastric antrum relative to the gastric body is a common finding when using MSCT; antral thickness commonly exceeds 5 mm. However, subclinical *H. pylori* infection does not affect wall thicknesses of either the gastric antrum or the gastric body. The gastric body wall thickening at the lesser curvature found in *H. pylori* positive cases is of borderline significance and thought to be incidental. Gastric mucosal enhancement is a common finding, but like other less commonly observed CT findings of gastritis, may be seen in both subclinical *H. pylori* infection and normal stomach and is not a differentiating feature.

References

1. Dooley CP, Cohen H, Fitzgibbons PL, et al. Prevalence of Helicobacter pylori infection and histologic gastritis in asymptomatic persons. N Engl J Med 1989; 321:1562–1566.
2. Horton MK, Fishman EK. Current role of CT in imaging of stomach. Radiographics 2003; 23:75–87.
3. Lee JK, Sagel SS, Stanley RJ, Heiken JP. Computed body tomography with MRI correlation, vol 1. 3rd ed. New York: Lippincott; 1988:646–656.
4. Balfe DM, Koehler RE, Karstaedt N, Stanley RJ, Sagel SS. Computed tomography of gastric neoplasms. Radiology 1981; 140:431–436.
5. Scatarige JC, DiSantis DJ. CT of the stomach and duodenum. Radiol Clin North Am 1989; 27:687–706.
6. Desai RK, Tagliabe JR, Wegryn SA, Einstein DM. CT evaluation of wall thickening in the alimentary tract. Radiographics 1991; 11:771–783.
7. Pickhardt PJ, Asher DB. Wall thickening of the gastric antrum as a normal finding: multidetector CT with cadaveric comparison. AJR Am J Roentgenol 2003; 181:973–979.
8. Graham DY, Go MF. Helicobacter pylori: current status. Gastroenterology 1993; 105:279–282.
9. Gelfand DW, Ott DJ. Helicobacter pylori and gastroduodenal diseases: a minor revolution for radiologists. AJR Am J Roentgenol 1997; 168:1421–1422.
10. Pattison CP, Combs MJ, Marshall BJ. Helicobacter pylori and peptic ulcer disease: evolution to revolution to resolution. AJR Am J Roentgenol 1997; 168:1415–1420.
11. Correa P, Houghton JM. Carcinogenesis of Helicobacter pylori. Gastroenterology 2007; 133:659–672.
12. Malfertheiner P, Megraud F, O'Morain C, et al. The European Helicobacter pylori Study Group (EHPSG). Current concepts in the management of Helicobacter pylori infection-The Maastricht 2-2000 Consensus Report. Aliment Pharmacol Ther 2002; 16:167–180.
13. Cirak MY, Akyon Y, Megraud F. Diagnosis of Helicobacter pylori. Helicobacter 2007; 12(suppl 1):4–9.
14. Urban BA, Fishman EK, Hruban RH. Helicobacter pylori gastritis mimicking gastric carcinoma at CT evaluation. Radiology 1991; 179:689–691.
15. Fishman EK, Urban BA, Hruban RH. CT of the stomach: spectrum of disease. Radiographics 1996; 16:1035–1054.
16. Morrison S, Dahms BB, Hoffenberg E, Czinn SJ. Enlarged gastric folds in association with Campylobacter pylori gastritis. Radiology 1989; 171:819–821.
17. Hazell SL, Carrick J, Edwards P. Frommer DL, Lee A. Acute infection with Campylobacter pylori can mimic gastric cancer. Gastroenterology 1988; 94:A178.