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Ultrasonographic Evaluation of Gastroduodenal Wall Thickness for Prediction of Gastritis and Helicobacter Pylori Infection in Children

Background/Objective: There is no report about the relationship between the thickness of gastric wall layers measured with transabdominal ultrasonography and the presence of gastritis or Helicobacter pylori infection. The aim of this study was to assess the accuracy of sonography in diagnosis of gastritis and Helicobacter pylori infection.

Patients and Methods: One-hundred children aged 1 to 15 years who needed upper gastrointestinal endoscopy and biopsy because of suspected gastritis underwent transabdominal ultrasonography to measure thickness of different layers of the stomach antrum and duodenal bulb wall. The wall layer thickness was compared with the results of endoscopy for gastritis and the presence of Helicobacter pylori infection.

Results: The mean thickness of muscularis mucosa and the sum of muscularis mucosa and submucosa in both gastric antrum and duodenal bulb were significantly higher in patients with Helicobacter pylori infection than those without infection (mean thickness in gastric antrum: 0.65±0.25mm vs. 0.53±0.19mm [p-value=0.03] and 1.21±0.35mm vs. 1.07±0.26mm [p-value=0.03], respectively; mean thickness in duodenal bulb: 0.69±0.32mm vs. 0.48±0.20mm [p-value=0.001] and 1.25±0.35mm vs. 0.99±0.28mm [p-value=0.002], respectively). The mean thickness of muscularis mucosa plus submucosa in the duodenal bulb was also more in patients with gastritis (1.09±0.35mm vs. 0.95±0.20mm [p-value=0.02]). Several cut points were determined to predict the results of endoscopy.

Conclusion: Transabdominal ultrasonography is a noninvasive and easily available method in evaluating children with suspected gastritis and predicting some findings of endoscopic evaluations.

Keywords: Ultrasonography, Gastritis, Helicobacter Pylori, Pediatrics

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Introduction

Dyspepsia and chronic abdominal pain due to gastroduodenitis is one of the common pediatric problems which may lead to malnutrition, failure to thrive and anemia. Diagnosis of this condition requires endoscopic evaluation, which is an expensive and invasive procedure and needs patient sedation. Therefore, less invasive tests as alternatives to endoscopy or at least as a patient selection tool are favorable to decrease the number of endoscopic surveys.

Transabdominal ultrasonography (TAUS) is an available and easy tool for evaluation of gastric and duodenal wall thickness using high-frequency linear transducers. It is especially applicable in children because of their thin abdominal wall. Five layers in the gut wall may be assessed sonographically: 1. Mucosa (the innermost echogenic layer) 2. Muscularis mucosa (the next hypoechoic layer) 3. Submucosa (echogenic) 4. Muscularis propria (hypoechoic) 5. Serosa (echogenic)[1] (Fig. 1). There are only limited articles about sonographic assessment of gastric wall thickness in different diseases; such as mucosal hypertrophy in Menetrier's disease,[2] gastric mucosal thickening and hyperemia in protein-

losing gastropathy,[3] gastric wall thickening in patients with varioliform gastritis, gastric ulcer and lymphoid hyperplasia,[4] gastric wall evaluation in neoplastic disease[5] and evaluation of gastric motility in Helicobacter pylori infected patients.[6] Many of the previous studies include very limited cases.

The purpose of this study was to evaluate the role of TAUS in diagnosis or prediction of gastritis or Helicobacter pylori (H. pylori) infection as one of the major precipitating factors of gastroduodenitis.

Patients and Methods

One-hundred children referred for upper gastrointestinal endoscopy underwent sonography between April, 2008 and April, 2009 to determine the thickness of gastric and duodenal bulb layers before endoscopy. Patients were 1 to 15 years old (mean, 6.6 years), 45 of which were boys and 55 were girls. The main reason for endoscopy was abdominal pain (73%) with or without failure to thrive. We decided to exclude patients with conditions, which could possibly lead to gut wall thickening, such as severe edema or ascitis, gastric wall varice or mass lesion and infiltrative diseases on the pathological exam. None of the patients had severe edema or ascitis.

Sonographic studies were performed by two experienced radiologists using 10 MHz linear transducer (Ultrasonix, Sonix OP, Canada) after obtaining oral informed consent. Images were magnified as much as possible to include the whole anterior wall of the duodenal bulb or gastric antrum without blurring of interfaces between wall layers. The maximum depth of images was 3.5 cm. The near wall of the semi-filled gastric antrum and duodenal bulb were captured in three different sites. Thickness of the inner four layers (mucosa, muscularis mucosa, submucosa and muscularis propria) and also thickness of the whole wall were measured in three different sites, and then the average thickness of each layer in the stomach and the duodenal bulb were documented separately. The radiologists were unaware of the patients' symptoms. The antral wall thickness was measured in all patients but the duodenal bulb in 87 patients. Measurements in the first 10 patients were performed by both radiologists to assess interobserver variability, but the difference between them was not significant ($0.47<p\text{-value}<0.64$).

Endoscopy was performed on the same day after sonography in all cases by one pediatric gastroenterologist using Pentax (EG2731, Japan) endoscope. None of the patients had gastric wall varice or mass lesion on endoscopy. Results of direct mucosal inspection were documented. Biopsy specimen from the antral wall was obtained in all patients and biopsy from the duodenal bulb in 40 patients. One pathologist examined specimens to determine the presence of gastritis and its type. Pathologic findings were used as the gold standard to evaluate the sonographic findings. None of the patients had diseases other than gastritis on pathologic exam.

Data were analyzed by SPSS ver. 11.5 for windows. One sample Kolmogorov-Smirnov test was used to evaluate the pattern of distribution of the wall layer thickness. Independent samples t-test was used for evaluation of the parameters with normal distribution and Mann-Whitney test was used for the parameters without normal distribution. P-value less than 0.05 was considered statistically significant. Area under the receiver operative characteristics (ROC) curve (AUC) was used to measure the accuracy of sonography compared to GI endoscopy. Cut off points for statistically significant parameters were determined with ROC curve analysis.

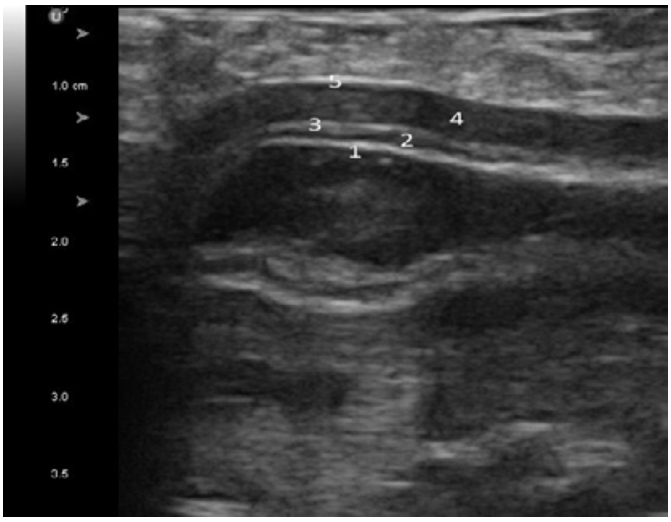

Fig. 1. Transabdominal sonogram of the duodenal bulb wall showing the different layers
1. Mucosa (the innermost echogenic layer). 2. Muscularis mucosa (the next hypoechoic layer). 3. Submucosa (echogenic). 4. Muscularis propria (hypoechoic). 5. Serosa (echogenic).

Results

Biopsy proven gastritis was found in 65 (65%) patients, in several types; mild chronic (34%), moderate chronic (3%), follicular (1%), active (1%), active mild chronic (1%), active moderate chronic (3%), active severe chronic (1%), active follicular (1%) and ulcerative (1%). We categorized them into mild gastritis (those with mild chronic gastritis) and severe gastritis (all other types), so 31% of the patients assumed to have severe gastritis. H. pylori infection was shown in gastric mucosa of 19 patients; all of them had gastritis, 94.7% of them (18 patients) had severe gastritis and 84.2% (16 patients) had active gastritis. 58% of cases with severe gastritis were H. pylori infected.

We compared thickness of stomach and duodenal wall layers individually and in combination in four categories of patients: 1. Patients with gastritis versus without gastritis (Table 1). 2. Patients with severe gastritis versus mild or no gastritis (Table 2). 3. Patients with active gastritis versus inactive or no gastritis (Table 3). 4. Patients with positive H. pylori test result versus negative results (Table 4). For easier expression of the results and discussion we have used abbreviations G1, G2, G3 and G4 for mucosa, muscularis mucosa, submucosa and muscularis propria in the antral wall of the stomach, respectively. G5 stands for the whole wall thickness in the stomach antrum. B1, B2, B3, B4 and B5 are used for the above mentioned layers in the duodenal bulb.

In patients with gastritis and cases without gastritis, no single layer showed significant difference but the mean thickness of B2 plus B3 layers was significantly increased in patients with gastritis (p-value=0.02). The same results were found during comparison of patients with active gastritis and patients with inactive gastritis or without gastritis. In cases with severe gastritis, only the mean thickness of B2 layer was significantly more than cases with mild gastritis or without gastritis (p-value=0.02).

H. pylori infected patients had significantly thicker B2 and G2 layers (p-value=0.001 and 0.03, respectively). The mean thickness of B2 plus B3 and G2 plus G3 layers were also significantly different from patients without H. pylori infection.

Using ROC curve analysis, several cut points with different sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) were determined for some of the statistically significant values. Cut point of 1.1mm for B2+B3 had 39% sensitivity, 93% specificity, 42% NPV and 92% PPV for detection of gastritis. For prediction of H. pylori infection, different cut points were found (Table 5).

Table 1. Thickness of Stomach and Duodenal Wall Layers Individually and in Combination in Patients with Gastritis versus Patients without Gastritis.

Layer	With Gastritis (mean thickness in mm ±SD)	Without Gastritis (mean thickness in mm ±SD)	P-Value
	Duodenum (n=65)	*Duodenum (n=35)*	
B1	0.30 ± 0.09	0.27 ± 0.08	0.66
B2	0.55 ± 0.27	0.45 ± 0.16	0.52
B3	0.53 ± 0.17	0.50 ± 0.13	0.44
B4	1.03 ± 0.53	1.21 ± 0.56	0.14
B5	2.51 ± 0.67	2.52 ± 0.73	0.94
B2+B3	1.09 ± 0.35	0.95 ± 0.20	**0.02**
B2+B3+B4	2.11 ± 0.66	2.16 ± 0.66	0.75
	Stomach (n=59)	*Stomach (n=28)*	
G1	0.35 ± 0.12	0.34 ± 0.12	0.97
G2	0.58 ± 0.21	0.50 ± 0.19	0.09
G3	0.55 ± 0.18	0.56 ± 0.16	0.52
G4	1.30 ± 0.62	1.33 ± 0.56	0.83
G5	2.91 ± 0.77	2.86 ± 0.76	0.76
G2+G3	1.12 ± 0.30	1.06 ± 0.25	0.35
G2+G3+G4	2.43 ± 0.71	2.40 ± 0.72	0.85

(B1, B2, B3, B4: mucosa, muscularis mucosa, submucosa and muscularis propria in the duodenal bulb wall, respectively. B5: the whole wall thickness in duodenal bulb. G1, G2, G3, G4: mucosa, muscularis mucosa, submucosa and muscularis propria in the antral wall of the stomach respectively. G5: the whole wall thickness in the stomach antrum)

Table 2. Thickness of Stomach and Duodenal Wall Layers Individually and in Combination in Patients with Severe Gastritis versus Mild or No Gastritis

Layer	Severe Gastritis (mean thickness in mm ±SD)	Mild or No Gastritis (mean thickness in mm ±SD)	P-Value
	Duodenum (n=27)	*Duodenum (n=60)*	
B1	0.31 ± 0.11	0.29 ± 0.08	0.46
B2	0.63 ± 0.32	0.47 ± 0.18	**0.02**
B3	0.52 ± 0.16	0.52 ± 0.16	0.98
B4	0.96 ± 0.59	1.14 ± 0.51	0.15
B5	2.51 ± 0.73	2.52 ± 0.68	0.95
B2+B3	1.16 ± 0.40	0.99 ± 0.25	0.06
B2+B3+B4	2.12 ± 0.73	2.13 ± 0.63	0.90
	Stomach (n=31)	*Stomach (n=69)*	
G1	0.37 ± 0.14	0.33 ± 0.11	0.49
G2	0.59 ± 0.22	0.53 ± 0.20	0.16
G3	0.56 ± 0.19	0.55 ± 0.17	0.83
G4	1.28 ± 0.74	1.33 ± 0.53	0.74
G5	2.97 ± 0.87	2.86 ± 0.71	0.54
G2+G3	1.15 ± 0.30	1.08 ± 0.27	0.25
G2+G3+G4	2.44 ± 0.80	2.40 ± 0.66	0.85

(B1, B2, B3, B4: mucosa, muscularis mucosa, submucosa and muscularis propria in duodenal bulb wall respectively. B5: the whole wall thickness in duodenal bulb. G1, G2, G3, G4: mucosa, muscularis mucosa, submucosa and muscularis propria in the antral wall of the stomach respectively. G5: the whole wall thickness in the stomach antrum)

Table 3. Thickness of Stomach and Duodenal Wall Layers Individually and in Combination in Patients with Active Gastritis versus Inactive or No Gastritis

Layer	Active Gastritis (mean thickness in mm ±SD)	Inactive or No Gastritis (mean thickness in mm ±SD)	P-Value
	Duodenum (n=16)	*Duodenum (n=71)*	
B1	0.29 ± 0.09	0.30 ± 0.09	0.84
B2	0.67 ± 0.34	0.49 ± 0.20	0.05
B3	0.54 ± 0.14	0.52 ± 0.17	0.69
B4	0.96 ± 0.47	1.11 ± 0.56	0.32
B5	2.58 ± 0.69	2.50 ± 0.69	0.67
B2+B3	1.21 ± 0.37	1.01 ± 0.29	**0.02**
B2+B3+B4	2.17 ± 0.72	2.12 ± 0.65	0.78
	Stomach (n=17)	*Stomach (n=83)*	
G1	0.35 ± 0.12	0.34 ± 0.12	0.99
G2	0.63 ± 0.26	0.53 ± 0.20	0.14
G3	0.56 ± 0.17	0.50 ± 0.20	0.60
G4	1.28 ± 0.72	1.32 ± 0.58	0.78
G5	2.91 ± 0.87	2.89 ± 0.74	0.92
G2+G3	1.13 ± 0.34	1.10 ± 0.27	0.39
G2+G3+G4	2.41 ± 0.81	2.42 ± 0.69	0.95

(B1, B2, B3, B4: mucosa, muscularis mucosa, submucosa and muscularis propria in duodenal bulb wall respectively. B5: the whole wall thickness in duodenal bulb. G1, G2, G3, G4: mucosa, muscularis mucosa, submucosa and muscularis propria in the antral wall of the stomach respectively. G5: the whole wall thickness in the stomach antrum)

Discussion

The results of the study revealed a significantly higher mean thickness of muscularis mucosa in both gastric antrum and duodenal bulb in patients with H. pylori infection than in those without infection (0.65±0.25mm vs. 0.53±0.19mm [p-value=0.03] and 0.69±0.32mm vs. 0.48±0.20mm [p-value=0.001], respectively). The mean thickness of the sum of muscu-

Table 4. Thickness of Stomach and Duodenal Wall Layers Individually and in Combination in Patients with Positive H. Pylori Test Result versus Negative Results

Layer	H.P Positive (mean thickness in mm ±SD)	H.P Negative (mean thickness in mm ±SD)	P-Value
	Duodenum (n=17)	*Duodenum (n=70)*	
B1	0.31 ± 0.11	0.29 ± 0.08	0.77
B2	0.69 ± 0.32	0.48 ± 0.20	**0.001**
B3	0.57 ± 0.14	0.51 ± 0.16	0.23
B4	0.92 ± 0.48	1.12 ± 0.55	0.17
B5	2.60 ± 0.67	2.49 ± 0.70	0.57
B2+B3	1.25 ± 0.35	0.99 ± 0.28	**0.002**
B2+B3+B4	2.18 ± 0.69	2.12 ± 0.65	0.73
	Stomach (n=19)	*Stomach (n=81)*	
G1	0.37 ± 0.15	0.34 ± 0.10	0.71
G2	0.65 ± 0.25	0.53 ± 0.19	**0.03**
G3	0.56 ± 0.22	0.55 ± 0.16	0.72
G4	1.25 ± 0.76	1.33 ± 0.56	0.59
G5	2.99 ± 0.89	2.87 ± 0.73	0.52
G2+G3	1.21 ± 0.35	1.07 ± 0.26	**0.03**
G2+G3+G4	2.46 ± 0.87	2.40 ± 0.67	0.74

(B1, B2, B3, B4: mucosa, muscularis mucosa, submucosa and muscularis propria in the duodenal bulb wall, respectively. B5: the whole wall thickness in the duodenal bulb. G1, G2, G3, G4: mucosa, muscularis mucosa, submucosa and muscularis propria in the antral wall of the stomach, respectively. G5: the whole wall thickness in the stomach antrum)

Table 5. Several Cut Points with Different Sensitivity, Specificity, Positive Predictive Value and Negative Predictive Values were Determined for Prediction of H. Pylori Infection

	AUC	Standard. Error	P-Value	Cut Point (mm)	Sensitivity	Specificity	Negative Predictive Value	Positive Predictive Value
B2	0.733	0.074	0.003	0.7	35%	96%	86%	67%
				0.4	88%	47%	94%	29%
B2+B3	0.739	0.072	0.002	1.4	29%	96%	85%	63%
				0.9	88%	44%	94%	28%
G2	0.663	0.080	0.027	0.8	21%	96%	84%	57%
				0.4	79%	36%	88%	22%
G2+G3	0.669	0.083	0.022	1.4	21%	94%	84%	44%
				1.0	79%	48%	91%	26%

(B2, B3: muscularis mucosa, submucosa in the duodenal bulb wall, respectively. G2, G3: muscularis mucosa, submucosa in the antral wall of the stomach, respectively)

laris mucosa and submucosa was also higher in H. pylori infected patients in the antrum (1.21±0.35mm vs. 1.07±0.26mm [p-value=0.03]) and the bulb (1.25±0.35mm vs. 0.99±0.28mm [p-value=0.002]).

H. pylori colonization in the stomach can lead to gastric wall inflammation and increase in gastrin secretion and gastric acid production. Increased acid per se or with induction of gastric metaplasia in the duodenum can result in duodenal ulceration. Inflammation in stomach may lead to some atrophic changes.[7] This may explain why thickening of the duodenal wall was more prominent than the gastric wall in our study.

For prediction of the status of H. pylori by sonographic measurement of the gastric and duodenal wall, several cut points may be used. A cut point of 0.7mm for B2 has a 96% specificity, a 67% PPV, and a sensitivity of 35%, which is low compared to a cut point of 0.4mm, which has an 88% sensitivity, and a 94% NPV. If measurement of B2 alone is difficult for the sonographist, combination of B2+B3 can be measured with cut points of 1.4mm and 0.9mm, and the results will be almost similar. Using G2 and G2+G3, achieves an almost similar specificity and NPV, but the sensitivity and PPV will be lower (Table 5).

In patients with gastritis, regardless of their H. pylo-

ri test result, the mean thickness of muscularis mucosa plus submucosa in the duodenal bulb (B2+B3) was also more than the cases without gastritis (1.09±0.35mm vs. 0.95±0.20mm [p-value=0.02]). A cut point of 1.1mm for B2+B3 had a very high specificity (93%) and PPV (92%) for the detection of gastritis.

These findings indicate that in children with signs or symptoms of gastritis, TAUS can predict some results of endoscopy and decrease the number of endoscopic evaluations. Unfortunately, the number of duodenal biopsies in our study was not sufficient for assessment of correlation of duodenal wall thickness with duodenitis, which may be evaluated in future studies. However, the thickness of different gut wall layers is low, resulting in technical problems for accurate measurement leading to undesirable interobserver or intraobserver variability. Larger studies are required to confirm the results and evaluate the possible inaccuracy, generalizability and modifications in measurement if necessary.

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