

# The Assessment of Gastric Antral Wall Thickness in *Helicobacter pylori* Gastritis by Abdominal Ultrasonography, Case-Control Study (2016-2018)

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**Background and Aim:** Abdominal ultrasonography is effective in the visualization of gastric wall layers and measuring its thickness. The study aimed to assess gastric antral wall thickness in patients with *H. pylori* gastritis by abdominal ultrasonography and to study its predictive value in detecting *H. pylori* gastritis.

**Materials and Methods:** The study included ninety adult individuals, sixty of them had dyspepsia and/or upper abdominal pain and histologically confirmed gastritis, were distributed equally according to the *H. pylori* infection status into group A (*H. pylori* gastritis) or B (non-*H. pylori* gastritis), while, group C included thirty asymptomatic participants with negative *H. pylori* screening. The participants were subjected to abdominal sonography for measuring the antral wall thickness (AWT), mucosal wall thickness

(MLT) and mucosal-to-antral wall thickness ratio (MLT/AWT ratio).

**Results:** The AWT, MLT and MLT/AWT ratio were significantly greater in *H. pylori* gastritis group ( $5.65 \pm 0.58$ ,  $3.02 \pm 0.43$ ,  $0.53 \pm 0.04$  respectively) than non-*H. pylori* gastritis group ( $4.57 \pm 0.82$ ,  $2.07 \pm 0.41$ ,  $0.45 \pm 0.02$  respectively) and control group ( $3.93 \pm 0.52$ ,  $1.49 \pm 0.2$ ,  $20.37 \pm 0.03$  respectively). The optimal cut off values of AWT, MLT and MLT/AWT ratio for detecting the of *H. pylori* infection among symptomatic gastritis patients were  $> 4.94$  mm,  $> 2.46$  mm and  $> 0.48$  mm respectively.

**Conclusion:** Ultrasonography of the gastric antrum is considered beneficial in evaluating patients with presumed gastritis. The increase of AWT  $> 4.94$  mm, MLT  $> 2.46$  mm, and MLT/AWT ratio  $> 0.48$  mm in patients with gastritis is suggestive of *H. pylori* infection.

## INTRODUCTION

Gastritis is a mucosal inflammation, which could results in various clinical symptoms as nausea, vomiting, pain in the upper abdomen and feeling of fullness [1]. *H. pylori* infection (HPI) is the commonest cause of gastritis, other causes may be non-steroidal anti-inflammatory drugs, autoimmune, bile reflux and alcohol intake [2]. Gastritis could be assessed by clinical, endoscopic, and histopathological examination [3,4,5]. The endoscopy is an invasive procedure and needs patient sedation; therefore, less

invasive tests are favorable to decrease the number of endoscopic surveys [6].

*H. Pylori* infection is a major public health problem that affects around 50% of the world's population [7,8] and its prevalence ranged from  $< 15\%$  to  $100\%$ , depending on socioeconomic status and country development [9,10,11]. The clinical outcome of HPI depends on both bacterial and patient factors. HPI has a main role in the pathogenesis of many gastrointestinal diseases such as chronic gastritis, peptic-ulcer disease, gastric adenocarcinoma, and gastric

lymphoma. The antral-predominant gastritis is the most common form of *H. pylori* gastritis. Many diagnostic tools may be used for diagnosis of *H. pylori* infection as; blood antibody test, fecal antigen test, urea breath test and upper gastrointestinal endoscopy [12]. HPI leads to an increase in the thickness of mucosal, submucosal and muscularis mucosa layers, with the resultant increase in the gastric wall thickness, this could be detected by using abdominal ultrasonography [13, 14].

Abdominal ultrasonography (AUS) is a non-invasive available tool for evaluating the gastric and duodenal wall layers and measuring its thickness [14] and used in the evaluation of nonspecific abdominal symptoms and acute abdominal pain [15]. By using a high-frequency linear ultrasound transducer, five layers of the stomach wall could be assessed; (1) a hyperechoic inner layer (the border between the gastric fluid and the mucosa); (2) a hypoechoic layer, the mucosa; (3) a hyperechoic layer, the submucosa; (4) a hypoechoic layer, the muscular layer; (5) an outer hyperechoic layer, the serosa [16,17].

The studies on the radiological scanning of the gastric antral wall are few, and the majority of those studies used computerized tomography (CT) and endoscopic ultrasound methods. The studies used the ultrasonography examination were not recent and focused on the gastric lumen rather than the wall [18]. The ability of ultrasonography to assess the transmural inflammatory disorders is a major advantage over contrast radiography; also, it gives more details about layers of bowel wall than CT, which contributes to the diagnosis and monitoring [17].

Abdominal ultrasonography is an effective method for visualization and diagnosis of the antral wall thickness when applied by an experienced radiologist, and can be used as an initial screening method for diagnosing gastritis and in determining who needs a definitive histopathological diagnosis [18]. This study aimed at assessing the role of abdominal ultrasonography in the detection of *H. pylori* gastritis through evaluating antral wall thickness.

## PATIENTS AND METHOD

A case-control study, included ninety randomly selected Egyptian patients from Tropical Medicine Department, Zagazig University

Hospitals during the period from October 2016 to March 2018. The subjects were classified into three groups; each group consisted of 30 participants as follows: Group A: Patients with histologically confirmed gastritis and *H. pylori* infection diagnosed by *H. pylori* fecal antigen and/or histopathological examination. Group B: Patients with histologically confirmed gastritis and no evident *H. pylori* infection (negative results in both examinations). Group C: Control group of asymptomatic individuals with negative *H. pylori* screening. The patients of the group A and B were selected from symptomatic patients (dyspepsia and/or upper abdominal pain) who attended endoscopy unit for elective diagnostic upper endoscopy and then they were allocated in either group A or B according to the *H. pylori* infection status. All the participants fulfilled the exclusion criteria; patients with history of hepatic congestive gastropathy, gastric cancer, MALTomas, varices, gastric surgery, radiotherapy, coagulopathy, alcohol intake, Crohn's disease, end-organ failure, immunocompromised, acute pancreatitis, any cause of anasarca, peptic ulcer disease, *H. pylori* eradication, GIT bleeding, use of non steroidal anti-inflammatory drugs (NSAIDs) and acid-suppressing drugs or antibiotics use within 1 month.

All They were subjected to:

1- History taking, clinical assessment, complete blood picture, kidney function tests, and liver function tests.

2- *H. pylori* Fecal antigen test (the Spectrum *H. pylori* Ag Test device, Medical Device Safety Service, Germany); a rapid stool antigen test is based on monoclonal antibody immunochromatography to detect feces antigen that indicates active *H. pylori* infection, with 98% specificity and 94% sensitivity. Its accuracy is influenced by the antibiotic, proton pump inhibitors, histamine receptor 2 blockers N-acetyl cysteine and upper gastrointestinal bleeding. A negative result can occur if the amount of *H. pylori* antigen is below the detection limits or the collected fecal sample does not contain the antigen; therefore, a negative result does not exclude the probability of *H. pylori* infection [19].

3-Abdominal Ultrasonography (AUS); The same expert radiologist did the abdominal ultrasound scan by using the superficial probe with a high frequency (11 MHz, Philips HD 11 XE ultrasound, USA). For a good sonographic image,

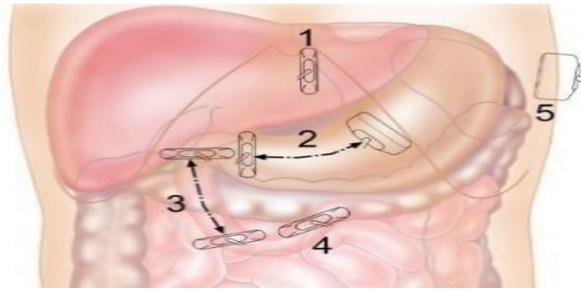
the patient ingested 500-800 ml plain water, after that, the patient had waited 10-15 minutes to allow the air bubbles to get out [13]. At the same time, 20 mg of Hyoscine-N-butyl bromide (Buscopan) was injected intravenously to induce a hypotonia [20]. The radiologist applied the transducer probe on the epigastric region, while the patient in the supine position and advanced the scanning in a sagittal plane from the cardia to the duodenum, meanwhile, he moved the probe in longitudinal and transverse sections to get a qualitative scan of the stomach as illustrated in figure 1 [21]. The stomach air usually masks the posterior wall and it could hinder cross-section scanning of the gastric body [22]. Many studies stated that the antrum is the most amenable to sonographic examination and precise identification (98–100% of cases) [23, 24]. It is found superficially in a sagittal or parasagittal scanning plane in the epigastrium, just right to the midline, between the left and caudate lobes of the liver anteriorly and the head or neck of the pancreas posteriorly [25]. The ultrasound scans will be analyzed in terms of wall thickness and stratification. The normal gastric wall stratification is demonstrated as a five-layer structure on transabdominal ultrasound image (figure 2). The gastric wall thickness is better measured at the cross-section of the antrum, especially the anterior antral wall with a longitudinal section of the superior mesenteric artery in the image (landmark to standardize a scanning plane through the antrum) [16]. The radiologist recorded the antral wall thickness (AWT), the mucosal layer thickness (MLT). Since the previously measured variables (AWT and MLT) may be affected by personal variations, and the degree of *H. pylori* infection may vary from one patient to another, the mucosal layer to antral wall thickness ratio (MLT/AWT ratio) was also taken into account and calculated.

3- Upper GIT endoscopy for group A and B patients (Pentax EPM-3500, Tokyo, Japan) was done after the patients fasted for six hours by night and the same gastroenterologist did the endoscopic examination under light intravenous sedation and local anesthetic spray to the oropharynx. The biopsies were taken according to Sydney-Houston system; one from the greater and lesser curvature within the peripyloric region, two from greater and lesser curvature within eight centimeters from the cardia and one from the incisor angularis and from any suspicious area.

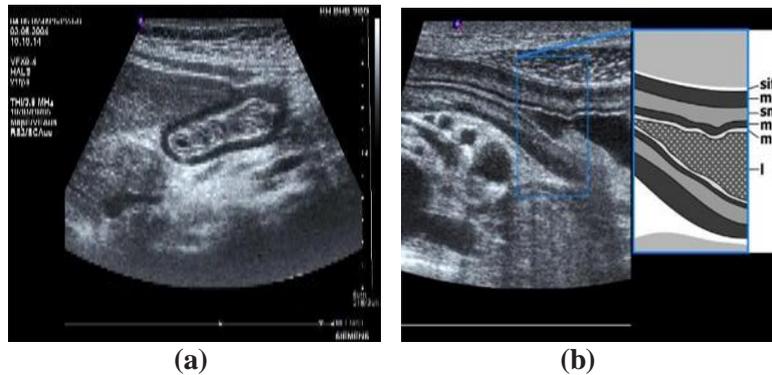
4- The same experienced pathologist inspected all the specimens: The specimens were stained with hematoxylin and eosin (H&E) and with Giemsa to confirm the presence of gastritis and to determine the *H. Pylori* infection status.

### Statistical Analysis

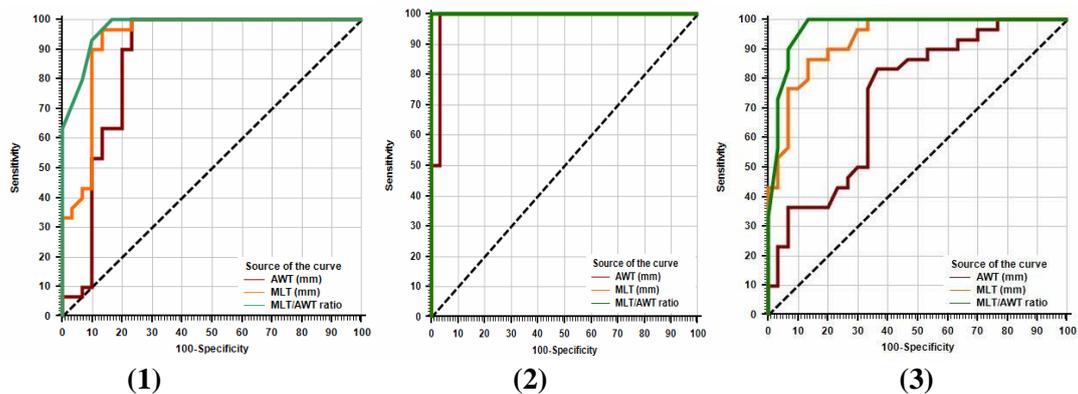
All the collected data were coded analyzed using SPSS 20.0 for Windows (SPSS Inc., Chicago, IL, USA) & MedCalc13 for windows (Med Calc Software, Ostend, Belgium). Continuous quantitative variables were expressed as the mean  $\pm$ SD & median (range), and categorical qualitative variables were expressed as absolute frequencies (number) and relative frequencies (percentage). Continuous data were analyzed by using the Shapiro Walk test. ANOVA test was used to compare more than two groups of normally distributed data while Kraskall Wallis H test was used for non-normally distributed data. Independent samples Student's t-test was used to compare two groups of normally distributed data while Mann-Whitney U test was used for non-normally distributed data. Categorical results were compared by Chi-square test or Fisher's exact test once applicable. Spearman's rank correlation coefficient was calculated to assess the correlations between gastric wall thickness and study parameters. The (+) sign indicated direct correlation and (-) sign indicated inverse correlation, also we consider values near to 1 as strong correlation and values near 0 as weak correlation. Receiver operating characteristic (ROC) curve analysis was used to identify optimal cut-off values of gastric wall thickness with maximum sensitivity and specificity for early detection of gastritis and *H. pylori* infection. The criteria for Area Under Curve (AUROC) were: 0.90 – 1 = excellent, 0.80-0.90 = good, 0.70-0.80 = fair; 0.60-0.70 = poor; and 0.50-0.6 = fail. The optimal cutoff point was established at the point of maximum accuracy. All tests were two-sided. P-value < 0.05 represented statistical significance and, p-value < 0.001 represented high statistical significance.



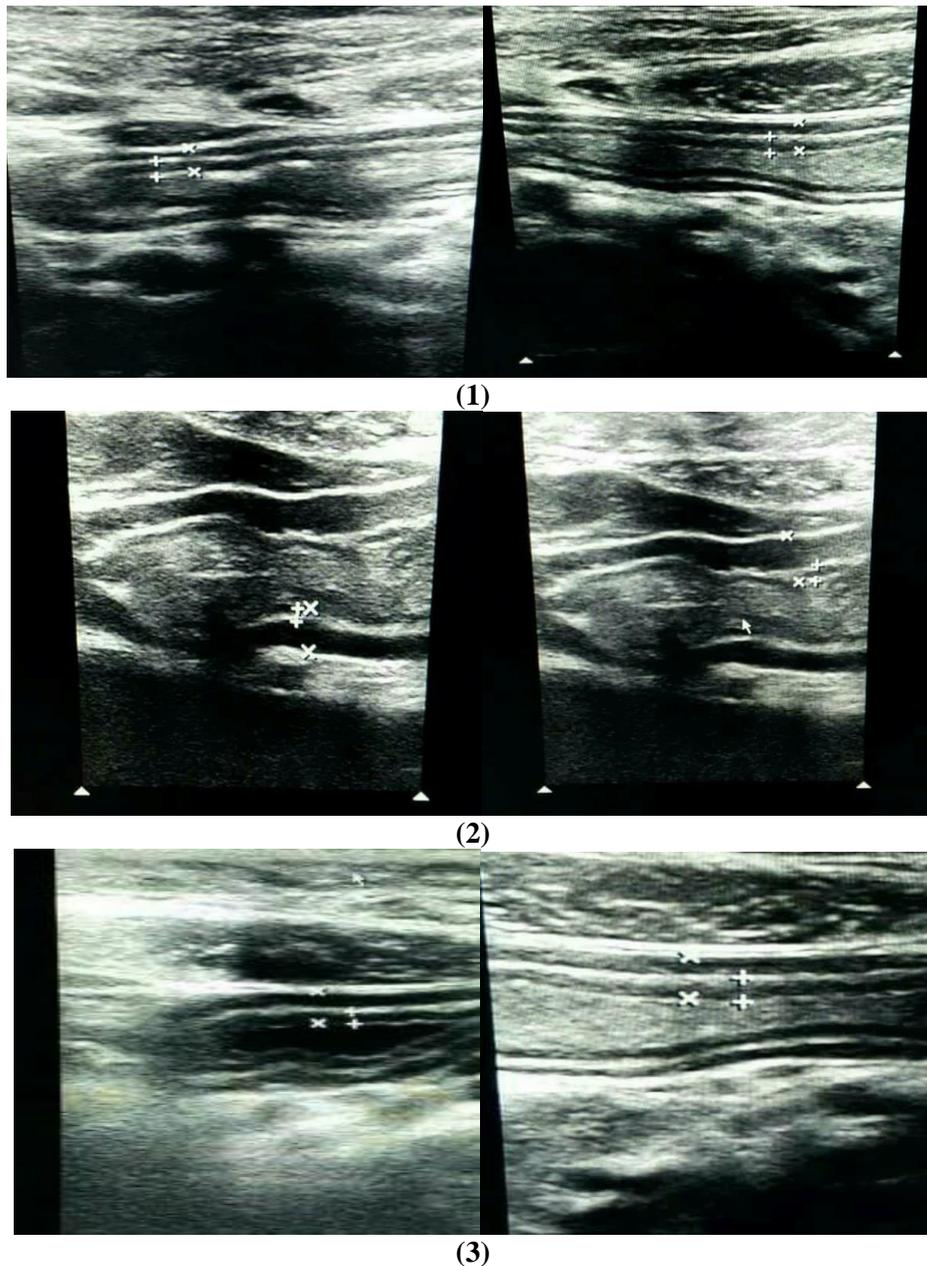
**Fig. (1):** Method of Ultrasonography examination for the stomach and the duodenum [13].



**Fig. (2):** Ultrasonography of the antrum, in cross-section (a) and zoomed longitudinal section (b). (sif: serosal interface; mp: muscularis propria; sm: submucosa; m: mucosa; mif: mucosal interface; l: lumen) [17].



**Fig. (3):** (1) Receiver operating characteristic (ROC) curve of gastric wall thickness (*H. pylori* gastritis group versus non-*H. pylori* gastritis group). (2) ROC curve of gastric wall thickness (*H. pylori* gastritis group versus control group). (3) ROC curve of gastric wall thickness (non-*H. pylori* gastritis group versus control group).



**Fig. (4):** (1) Abdominal sonography from two participants in group C without any stomach symptoms shows normal MLT and AWT (MLT =1.45 mm, 1.65 mm and AWT = 3.76 mm, 3.81 mm respectively). (2) Abdominal sonography from two participants in group B with gastric symptoms but no *H. pylori* infection after endoscopic biopsy shows increasing in MLT and AWT of the stomach( MLT=2.30 mm, 2.95 mm and AWT =5.30 mm,6.51 mmrespectively). (3)Abdominal sonography from participants in group A with gastric symptoms and a diagnosis of *H. pylori* infection after endoscopic biopsy shows increasing in MLT and AWT of the stomach (MLT=2.84 mm, 2.61 mm and AWT = 5.41 mm,5.20mmrespectively).

## RESULTS

Table 1, shows that there are no significant differences between the studied groups as regards demographic data (age, sex, body mass index, smoking, and residence). The mean age was 29.70 years in group A, 30.66 in group B and 30.32 in group C. The mean body mass index (BMI) was 26.93 in group A, 26.42 in group B, and 26.80 in group C.

The comparison between the studied groups regarding the gastric wall thickness as shown in table 2, revealed a highly significant correlation between the gastric wall thickness and *H. pylori* gastritis. The AWT, MLT and MLT/AWT ratio were greater in group A (*H. pylori* gastritis) than in group B (non-*H. pylori* gastritis) and group C (control group) with p-value < 0.001, furthermore, there was a significant increase in AWT, MLT and MLT/AWT ratio in *H. pylori* gastritis group when compared only with non-*H. pylori* gastritis group (table 3).

ROC curve analysis of gastric wall thickness in *H. pylori* gastritis group showed that the optimal cut

off values of AWT, MLT and MLT/AWT with the maximum sensitivity and specificity for detecting *H. pylori* infection among patients with symptomatic gastritis were > 4.94, > 2.64, > 0.48 respectively when it was compared with non-*H. Pylori* gastritis group (table 4) and the cut-off values for predicting gastritis among asymptomatic individuals were > 4.95 mm, > 1.95 mm > 0.45 mm, respectively, when it was compared with the control group (table 5), while, ROC curve Analysis for early detection of gastritis in non-*H. Pylori* gastritis group showed a lower significant cut off points of AWT (> 3.99 mm), MLT (> 1.71 mm), and MLT/AWT (> 0.4 mm) when it was compared the control group (table 6).

The charts of the Receiver operating characteristic (ROC) curves (figure 3) showed the largest AUROC for AWT, MLT and MLT/AWT was in *H. Pylori* gastritis group versus the control group (0.983, 1.000, and 1.000 respectively). Figure 4 showed abdominal sonography images from two participants in each group.

**Table (1):** Comparison between the studied groups regarding basic characteristics.

Basic characteristics	Group A	Group B	Group C	P-value
Gender				
Male n (%)	17 (56.7%)	22 (73.3%)	15 (50%)	0.164
Female n (%)	13 (43.3%)	8 (26.7%)	15 (50%)	
Age (years)				0.671
Mean ± SD	29.70±9.55	30.66±7.77	30.32±8.20	
Median (Range)	29 (18 – 55)	30 (1.8 – 45)	30 (18 – 55)	
BMI (kg/m <sup>2</sup> )				0.767
Mean ± SD	26.93±3.20	26.42±2.62	26.80±2.60	
Median (Range)	27.40 (20.10 – 31.60)	27.15 (21.60 – 32.10)	27.55 (22.60 – 31.20)	
Smoking(packs/year)				
Mean ± SD	1.35±3.69	1.65±4.60	1.74±5.18	
Median (Range)	0 (0 – 13.50)	0 (0 – 18)	0 (0 – 22.50)	
Residence				0.056
Rural n (%)	19 63.3%	14 (46.7%)	23 (76.7%)	
Urban n (%)	11 36.7%	16 (53.3%)	7 (23.3%)	

\* P < 0.05 is significant.

**Table (2):** Comparison between the studied groups regarding gastric wall thickness in abdominal ultrasonography.

Gastric wall thickness	Group A (n=30)	Group B (n=30)	Group C (n=30)	P-value
AWT (mm):				
Mean ± SD	5.65±0.58	4.57±0.82	3.93 ± 0.52	* < 0.001
Median (Range)	5.45(5 – 7.12)	4.23(3.55 – 6.68)	3.94(3.02 – 5.44)	
MLT (mm):				
Mean ± SD	3.02 ± 0.43	2.07±0.41	1.49 ± 0.22	* < 0.001
Median(Range)	2.88(2.39 – 3.95)	1.89(1.60 – 3.12)	1.49(0.94-1.95)	
MLT/AWT ratio:				
Mean ± SD	0.53 ± 0.04	0.45 ± 0.02	0.37± 0.03	* < 0.001
Median (Range)	0.52(0.49 – 0.68)	0.45 (0.41 – 0.51)	0.38 (0.28 – 0.45)	

\* P < 0.05 is significant.

**Table (3):** Comparison between group A and group B regarding gastric wall thickness in abdominal ultrasonography.

Gastric wall thickness	Group A(n=30)	Group B(n=30)	P-value
AWT (mm):			
Mean ± SD	5.65±0.58	4.57±0.82	* < 0.001
Median(Range)	5.45(5 – 7.12)	4.23(3.55 – 6.68)	
MLT (mm):			
Mean ± SD	3.02±0.43	2.07±0.41	* < 0.001
Median(Range)	2.88(2.39 – 3.95)	1.89(1.60 – 3.12)	
MLT/AWT ratio:			
Mean ± SD	0.53±0.04	0.45±0.02	* < 0.001
Median(Range)	0.52(0.49 – 0.68)	0.45(0.41 – 0.51)	

\* P < 0.05 is significant.

**Table (4):** ROC curve Analysis of Gastric wall thickness (*H. pylori* gastritis group versus non-*H. pylori* gastritis group).

Cut-off Values	SN % (95%CI)	SP % (95%CI)	PPV % (95% CI)	NPV % (95% CI)	Accuracy (95%CI)	AUROC (95% CI)	P-value
AWT > 4.94 mm	100%	76.7%	81.1%	100%	88.4%	0.864	* <0.001
MLT > 2.46 mm	96.7%	86.7%	87.9%	96.3%	93.4%	0.932	* <0.001
MLT/AWT > 0.48	100%	83.3%	85.7%	100%	91.7%	0.974	* <0.001

**Table (5):** ROC curve Analysis of Gastric wall thickness (*H. pylori* gastritis group versus control group).

Cut-off Values	SN % (95%CI)	SP % (95% CI)	PPV% (95% CI)	NPV% (95% CI)	Accuracy (95%CI)	AUROC (95% CI)	P-value
AWT > 4.95 mm	100%	96.7%	96.8%	100%	98.4%	0.983	* <0.001
MLT > 1.95 mm	100%	100%	100%	100%	100%	1.000	* <0.001
MLT/AWT > 0.45	100%	100%	100%	100%	100%	1.000	* <0.001

**Table (6):** ROC curve Analysis of Gastric wall thickness (Non-*H. pylori* group versus control group).

Cut-off Values	SN % (95% CI)	SP % (95% CI)	PPV % (95% CI)	NPV % (95% CI)	Accuracy (95% CI)	AUROC (95% CI)	P-value
AWT > 3.99 mm	83.3%	63.3%	69.4%	79.2%	73.3%	0.739	* <0.001
MLT > 1.71 mm	86.7%	86.7%	86.7%	86.7%	86.7%	0.932	* <0.001
MLT/AWT > 0.4	100%	86.7%	88.2%	100%	93.4%	0.971	* <0.001

ROC curve: Receiver Operating Characteristic curve; SN: Sensitivity; SP: Specificity; PPV: Positive Predictive Value; NPV: Negative Predictive Value; AUROC: Area Under Receiver Operating Characteristic curve; CI: Confidence Interval; mm: millimeter. \* P < 0.05 is significant.

## DISCUSSION

Recently In Egypt, the prevalence of HPI in the general population is about 70% [26], in addition it is prevalent in about 73% and 80% among school children and chronic active hepatitis C virus patients respectively [27,28], and the prevalence of metronidazole-resistant ranged from 25% to 100% [29,30]. Positive HPI in the Egyptian was associated with gastritis [31] and gastric cancer [32]. The less invasive tests for gastritis diagnosis are favorable to decrease the number of endoscopy surveys (cost and risks). AUS is a cheap noninvasive tool for evaluation of the gastric wall layers and thickness by using high-frequency linear transducers [6]. Therefore, this study aimed to assess the gastric antral wall thickness in patients with *H. pylori* gastritis by abdominal ultrasonography and to determine its predictive role for the detection of *H. pylori* gastritis.

In the present study, the sonographic findings showed an increase in AWT and MLT in patients with antral gastritis (group A and B) than in the control group. Moreover, antral gastritis caused by HPI had a significant thicker AWT and MLT than gastritis without HPI, which highlights the important correlation between *H. pylori* infection and the increase of gastric wall thickness in patients with gastritis. These findings were in agreement with Cakmakci et al [13], who studied 108 adult patients classified into three groups control; *H. pylori* gastritis and *H. pylori*-negative gastritis. They suggested that the thickening of the antral wall and mucosal layers on sonography were characteristic features; correlating well with *H. pylori* antral gastritis. In addition, it may be useful for both, early detection of patients with gastritis who require further investigation and for avoiding some unnecessary interventions. The significant increase of AWT in the patients with *H. pylori* gastritis compared to the patients who were *H. pylori*-negative gastritis ( $5.65 \pm 0.58$  and  $4.57 \pm 0.82$  respectively) was in agreement with studies by Mazaher et al [6] and Yazar et al [18]. Yazar and his colleagues concluded that the gastritis diagnosis could be made comfortably from the wall thickness measurements, while, study of Mazaher and his colleagues was carried out on one-hundred symptomatic children and they concluded that the US could predict some results of endoscopy and decrease the number of endoscopic evaluations in children with signs or symptoms of gastritis.

On the other hand, our results did not agree with a study carried out by Kul et al [33], who included 99 individuals without gastric symptoms and reported that AWT was higher in *H. pylori*-negative gastritis than *H. pylori*-positive gastritis ( $5.45 \pm 2.09$ ,  $4.80 \pm 1.81$  respectively). This could be explained by the difference in the study aim, they evaluated gastric wall thickness in subclinical *H. pylori* infection and included asymptomatic cases, while in our study, we included symptomatic patients. The *H. pylori* infection with clinical outcomes is expected to be more virulent than subclinical *H. pylori* infection and produce more inflammation and thickness in the stomach wall, another explanation; they had used CT scans of the abdomen to check the thickness of the gastric wall and not ultrasonography which is superior to contrast radiography in evaluating the transmural inflammatory disorders; furthermore, ultrasonography provides more information about layers of the bowel wall [17]. Cakmakci and his colleagues [21] studied, in another recent study, the predictive role of ultrasonography for detection of antral gastritis and *H. pylori* infection but in pediatric age group and stated no significant difference in total gastric wall thickness between *H. pylori* and non-*H. pylori* gastritis groups. This difference could be attributed to the different studied age groups; our cases were adult individuals with chronic gastritis, while they were pediatric patients with acute or recent infection and the longer the *H. pylori* infection lasts, the more likely the pathological outcomes and antral wall thickening will be.

In the present study, there was no significant correlation between gastric wall thickness and BMI; this was in agreement with a study by Larsen and his colleagues [34], although they used endoscopic ultrasound not transabdominal.

The parameters of antral wall thickness (AWT, MLT, and MLT/AWT) in *H. pylori* gastritis group showed a significant diagnostic and predictive value through ROC analysis when compared to either of non-*H. pylori* gastritis and control groups. The cut-off values for detection of the *H. pylori* infection among patients with symptomatic gastritis were  $> 4.94 > 2.64 > 0.48$  respectively, while, The cut-off values for predicting gastritis among asymptomatic individuals are  $> 4.95\text{mm}$ ,  $> 1.95\text{ mm} > 0.45\text{ mm}$ , respectively. Swenson and Wallach [35] reported that the marked transmural gastric wall thickening is a typical sign of gastritis and the antral wall thickness greater than 4 mm is considered suggestive of gastritis, this cut-off

value is lower than the cut-off value of AWT (> 4.95 mm) in our study for predicting gastritis, when *H. pylori* gastritis group was compared to the control group, on the other hand, it is in concordance with the cut-off value of AWT (> 3.99 mm) for predicting gastritis, when non-*H. pylori* gastritis group was compared to the control group. This could be explained by the effect of *H. pylori* infection on the wall thickness through its multiple virulent factors that result in more inflammation and pathogenicity to the stomach wall and hence more wall thickening.

## CONCLUSION

Ultrasonography of the gastric antrum is considered a beneficial tool in screening and evaluating patients with presumed gastritis. The increase of AWT > 4.94 mm, MLT > 2.46 mm, and MLT/AWT ratio > 0.48 mm in patients with gastritis is suggestive of *H. pylori* infection.

**Recommendation;** A comparison of the role of AUS in other isolates of *H. pylori* from different geographical locations can be reported. Further research is needed to explore variables that may affect the AUS role in gastritis and to study its role in the diagnosis and prognosis of cases with or without treatment. This approach might facilitate to gain insight into the profile of Egyptian isolates of *H. pylori* and different types of gastritis.

**Ethical considerations:** This study was applied in concordance with the Medical Association Code of Ethics (Declaration of Helsinki), and written informed consent for endoscopy, biopsy, and for laboratory tests was obtained from all participants. A review board of Tropical Medicine Department of Zagazig University approved the protocol.

**Source of support:** None

**Conflict of interest:** None

## REFERENCES

1. Mihály E, Micsik T, Juhász M, Herszényi L, Tulassay Z. Gastritis and gastropathy. *Orvosi Hetilap* 2014; 155(2): 43–61. doi: 10.1556/oh.2014.
2. Nordenstedt H, Graham DY, Kramer JR, Rugge M, Verstovsek G, Fitzgerald S, El-Serag HB. Helicobacter pylori-Negative Gastritis: Prevalence and Risk Factors. *American Journal of*

- Gastroenterology* 2013; 108(1): 65–71. doi: 10.1038/ajg.2012.372.
3. Rugge M, Meggio A, Pennelli G, Pisciole F, Giacomelli L, Pretis GD, Graham DY. Gastritis staging in clinical practice: the OLGA staging system. *Gut* 2007; 56(5): 631–636. doi: 10.1136/gut.2006.106666.
4. Graham DY, Rugge M. Clinical Practice: Diagnosis and Evaluation of Dyspepsia. *Journal of Clinical Gastroenterology* 2010; 44(3): 167–172. doi: 10.1097/mcg.0b013e3181c64c69.
5. Lauwers GY, Fujita H, Nagata K, Shimizu M. Pathology of non-Helicobacter pylori gastritis: extending the histopathologic horizons. *Journal of Gastroenterology* 2009; 45(2): 131–145. doi: 10.1007/s00535-009-0146-3.
6. Mazaher H, Farahmand F, Khanali F, Bozorg S, Esfe A, Mahjoub F, et al. Ultrasonographic evaluation of gastroduodenal wall thickness for prediction of gastritis and Helicobacter pylori infection in children. *Iranian Journal of Radiology* 2010; 7(1): 31–36.
7. Malaty HM. Epidemiology of Helicobacter pylori infection. *Best Practice & Research Clinical Gastroenterology* 2007; 21(2): 205–214. doi: 10.1016/j.bpg.2006.10.005
8. Carrasco G, Corvalan AH. Helicobacter pylori-Induced Chronic Gastritis and Assessing Risks for Gastric Cancer. *Gastroenterology Research and Practice* 2013; 1–8. doi: 10.1155/2013/393015
9. Rotimi O. Histological identification of Helicobacter pylori: comparison of staining methods. *Journal of Clinical Pathology* 2000; 53(10):756–759. doi: 10.1136/jcp.53.10.756.
10. Rugge M, Pennelli G, Pillozzi E, Fassan M, Ingravalle G, Russo VM, Mario FD. Gastritis: The histology report. *Digestive and Liver Disease* 2011; 43. doi: 10.1016/s1590-8658(11)60593-8.
11. Sokwala A, Shah MV, Devani S, Yonga G. Helicobacter pylori eradication: A randomised comparative trial of 7-day versus 14-day triple therapy. *South African Medical Journal* 2012; 102(6): 368. doi: 10.7196/samj.5302.
12. Miftahussurur M, Yamaoka Y. Diagnostic Methods of Helicobacter pylori Infection for Epidemiological Studies: Critical Importance of Indirect Test Validation. *BioMed Research International* 2016; 1–14. doi: 10.1155/2016/4819423.
13. Cakmakci E, Ucan B, Colak B, Cinar HG. Novel Sonographic Clues for Diagnosis of Antral Gastritis and Helicobacter pylori Infection. *Journal of Ultrasound in Medicine* 2014; 33(9): 1605–1610. doi: 10.7863/ultra.33.9.1605.
14. Swenson DW, Wallach M. Helicobacter pylori-Associated Antral Gastritis and Ulcer Disease. *Ultrasound Quarterly* 2012; 28(3): 185–187. doi: 10.1097/ruq.0b013e318262cb5f.
15. Puylaert JB, Zant FMVD, Rijke AM. Sonography and the acute abdomen: practical

- considerations. *American Journal of Roentgenology* 1997; 168(1): 179–186. doi: 10.2214/ajr.168.1.8976943.
16. Sporea I, Popescu A. Ultrasound examination of the normal gastrointestinal tract. *Med Ultrason* 2010; 12: 349–52.
  17. Hollerweger A, Dirks K, Szopinski K. Transabdominal ultrasound of the gastrointestinal tract. EFSUMB- European Course Book; chapter 8:1-5, 2014.
  18. Yazar FM, Baykara M, Karaağaç M, Bülbüloğlu E. The Role of Conventional Ultrasonography in the Evaluation of Antrum Wall Thickness in Obese Patients. *Obesity Surgery* 2016; 26(12), 2995–3000. doi: 10.1007/s11695-016-2221-1.
  19. Asfeldt AM, Løchen ML, Straume B, Steigen SE, Florholmen J, Goll R, Paulssen EJ. Accuracy of a monoclonal antibody-based stool antigen test in the diagnosis of *Helicobacter pylori* infection. *Scandinavian Journal of Gastroenterology* 2004; 39(11): 1073–1077. doi: 10.1080/00365520410007944.
  20. Worlicek H, Dunz D, Engelhard K. Ultrasonic examination of the wall of the fluid-filled stomach. *Journal of Clinical Ultrasound* 1989; 17(1): 5–14. doi: 10.1002/jcu.1870170103.
  21. Cakmakci E, Sahin GE, Hosnut FO, Cinar HG, Ucanl B, Pala M, Yildiz YT. Antral gastritis caused by *Helicobacter pylori* infection in the pediatric age group is associated with increased mesenteric lymph node dimension observed by ultrasonography. *Quant Imaging Med Surg* 2015; 5(6): 829–834.
  22. Putte PVD, Perlas A. Ultrasound assessment of gastric content and volume. *British Journal of Anaesthesia* 2014; 113(1): 12–22. doi: 10.1093/bja/aeu151.
  23. Cubillos J, Tse C, Chan VWS, Perlas A. Bedside ultrasound assessment of gastric content: an observational study. *Canadian Journal of Anesthesia/Journal Canadien Danesthésie* 2012; 59(4): 416–423. doi: 10.1007/s12630-011-9661-9.
  24. Perlas A, Chan VWS, Lupu CM, Mitsakakis N, Hanbidge A. Ultrasound Assessment of Gastric Content and Volume. *Anesthesiology* 2009; 111(1): 82–89. doi: 10.1097/aln.0b013e3181a97250.
  25. Jacoby J, Smith G, Eberhardt M, Heller M. Bedside ultrasound to determine prandial status. *The American Journal of Emergency Medicine* 2003; 21(3): 216–219. doi: 10.1016/s0735-6757(02)42243-7.
  26. Ghaith D, Elzahry M, Mostafa G, Mostafa S, Elsherif R, Ramzy I. Mutations affecting domain V of the 23S rRNA gene in *Helicobacter pylori* from Cairo, Egypt. *Journal of Chemotherapy* 2016; 28(5): 367–370. doi: 10.1179/1973947815y.0000000067.
  27. Mohammad MA, Hussein L, Coward A, Jackson SJ. Prevalence of *Helicobacter pylori* infection among Egyptian children: impact of social background and effect on growth. *Public Health Nutrition* 2008; 11(3): 230–236. doi: 10.1017/s1368980007000481.
  28. Hanafy AS, Hawary ATE, Hamed EF, Hassaneen AM. Impact of *Helicobacter pylori* eradication on refractory thrombocytopenia in patients with chronic HCV awaiting antiviral therapy. *European Journal of Clinical Microbiology & Infectious Diseases* 2016; 35(7): 1171–1176. doi: 10.1007/s10096-016-2650-8
  29. Diab M, El-Shenawy A, El-Ghannam M, Salem D, Abdelnasser M, Shaheen M, Saber M. Detection of antimicrobial resistance genes of *Helicobacter pylori* strains to clarithromycin, metronidazole, amoxicillin and tetracycline among Egyptian patients. *Egyptian Journal of Medical Human Genetics* 2018; 19(4): 417–423. doi: 10.1016/j.ejmhg.2018.01.004.
  30. Fathi MS, El-Folly RF, Hassan RA, El-Arab ME. Genotypic and phenotypic patterns of antimicrobial susceptibility of *Helicobacter pylori* strains among Egyptian patients. *Egyptian Journal of Medical Human Genetics* 2013; 14(3): 235–246. doi: 10.1016/j.ejmhg.2013.03.004.
  31. Al-Eraky DM, Helmy OM, Ragab YM, Abdul-Khalek Z, El-Seidi EA, Ramadan MA. Prevalence of CagA and antimicrobial sensitivity of *H. pylori* isolates of patients with gastric cancer in Egypt. *Infectious Agents and Cancer* 2018; 13: 24. <https://doi.org/10.1186/s13027-018-0198-1>.
  32. Essa AS, Nouh MAE, Ghaniam NM, Graham DY, Sabry HS. Prevalence of cagA in relation to clinical presentation of *Helicobacter pylori* infection in Egypt. *Scandinavian Journal of Infectious Diseases* 2008; 40(9): 730–733. doi: 10.1080/00365540802023725.
  33. Kul S, Sert B, Sarı A, et al. Subklinik *Helicobacter pylori* enfeksiyonunun mide duvarı ve kalınlığı üzerine etkisi: çok kesitli BT değerlendirmesi. *Diagnostic and Interventional Radiology* 2008; 14: 138–142.
  34. Larsen MC, Yan BM, Morton J, Dam JV. Determination of the Relationship Between Gastric Wall Thickness and Body Mass Index with Endoscopic Ultrasound. *Obesity Surgery* 2009; 21(3): 300–304. doi: 10.1007/s11695-009-9839-1.
  35. Swenson DW, Wallach M. *Helicobacter pylori*–Associated Antral Gastritis and Ulcer Disease. *Ultrasound Quarterly* 2012; 28(3): 185–187. doi: 10.1097/ruq.0b013e318262cb5f.