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Comparative Study between Endoscopic Biopsies and Gastropanel (Serum Pepsinogen II and Gastrin 17) for Diagnosis of Helicobacter Pylori Associated Gastritis

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Abstract

Previously, the only way to get reliable information about the stomach mucosa had been gastroscopy and histological analysis of the biopsy. Many studies have been reported on the evaluation of blood tests such as gastropanel (pepsinogen I, II and gastrin 17) to predict normal gastric mucosa and screening markers for chronic atrophic gastritis.was to compare endoscopic gastric biopsy with serum pepsinogen II (PG II) and serum gastrin 17 (G-17) to detect their association with H. pylori gastritis. This study included 75 patient more than 18 years old with recurrent abdominal pain that motive the performance of Upper Gastrointestinal Endoscopy and 90 apparently healthy individual matched for age and sex. Serum pepsinogen II and serum gastrin 17 were quantitated using Enzyme Linked ImmunoSorbent Assay (ELISA).Upper GIT endoscopy and histopathology of gastric and duodenal biopsies obtained from 75 consecutive patients undergoing upper gastrointestinal endoscopy for the investigation of recurrent abdominal pain were done.Our study histopathology of gastric ,doudenal biopsies showed that 32/75 (42.7) of H.pylori positive patients have H.pylori-associated gastritis and duodenitis with mild activity & mild H.pylori density (+).Serum Pepsinogen II (ng/ml)Mean ± SD of H.pylori +ve group (24.54 ± 8.66) was higher than H.pylori -ve group which was(4.55 ± 2.37)with p value (<0.001). serum gastrin 17 (pg/ml)Mean ± SD of H.pylori +ve group was (240.68 ± 231.77) was higher than H.pylori -ve group (29.26 ± 5.41) with p value (<0.001).Serum PG II and gastrin 17 can be a useful non-invasive marker for H.pylori gastritisn with recurrent abdominal pain with no apparent organic reason.

Keywords: Helicobacter Pylori **,** Serum Pepsinogen II and Gastrin 17**,** Upper GIT endoscopy.

1.Introduction

Helicobacter pylori (H. pylori) is one of the most common causes of bacterial infection in human beings and was first isolated and cultured by Warren and Marshall in 1983[1].It is the major cause of gastritis, that plays a key role in the etiology of peptic ulcer and is a risk factor for gastric carcinoma [2].

In 1996, Martin J. Blaser advanced the hypothesis that *H. pylori* has a beneficial effect: by regulating the acidity of the stomach contents [3] but the hypothesis is not universally accepted.

Urea breath test or invasive methods, such as gastroscopy with biopsies and/or urease tests are the "gold standard" for detection of H. pylori. These are technically advanced, time consuming methods and unsuitable for children. Serological test is also available but in children it often shows lower specificity. A major drawback of serological test is that it does not discriminate between current and past infections. The faecal monoclonal antigen test has a high sensitivity, specificity and accuracy in children [1].

The aim was to compare endoscopic gastric biopsy with serum PG II and G-17 to detect their association with H. pylori gastritis.

2.Subjects and methods

This study was conducted at Pediatric Department, Benha university hospital on 75 patient with recurrent abdominal pain attending pediatric gastroenterology and hepatology clinic of Benha University Hospital and 90 apparently healthy children as controls during the period from December 2017 to October 2019 after obtaining informed consent from parents of those children before the study.

2.1 Inclusion Criteria were

patients more than 18 years old with recurrent abdominal pain that motive the performance of Upper Gastrointestinal Endoscopy or gastroscopy Include children with :

2.1.1 Peptic-like dyspepsia

diagnosed by the presence of two or more of the following: periodic pain, pain relieved by food, pain relieved by antacid, pain before meal or while hungry, nausea and/or vomiting, and night pain.

2.1.2 Dysmotility-like dyspepsia

diagnosed by the presence of two or more of the following: abdominal bloating or distention, anorexia or weight loss, pain aggravated by food or milk, and pain relieved belching.

2.1.3 Reflux-like dyspepsia

diagnosed if the child presented with either heartburn, chest pain or acid regurgitation.

2.1.4 Children who have red flags

including (anemia, high ESR, GI bleeding, failure to thrive).

2.2 Exclusion Criteria were

Patients with gastrointestinal diseases that might explain abdominal pain e.g., inflammatory bowel disease, celiac disease and functional abdominal pain.

All subjects included in this study were subjected to:-

2.3 Full history taking

Including age, sex, residence, onset, course and duration of abdominal

pain, other associated symptoms and family history.

2.4 Full clinical examination

Including anthropometric measurement (Head circumference, weight, length and body mass index), abdominal examination for (Localized epigastric tenderness , halitosis and dehydration signs) and other systems examination.

2.5 Investigations

All subjects were investigated by

1- H.pylori stool antigen test.
2- Serum pepsinogen II using Sun Red Human Pepsinogen C (PGC) ELISA Kit.
3- Serum gastrin 17 using Sun Red Human Gastrin(GAS)ELISA Kit.
4- Upper GIT endoscopy and histopathology of gastric and doudenal biopsies using an Olympus XQ20 gastroscope.
5- Abdominal ultrasound.
6- Complete blood count.
7- Erythrocyte sedimentation rate
8- Stool analysis and culture.
9- Urine analysis and culture.

2.6 Statistical Analysis

Results were organized, tabulated and statistically analyzed using statistical package for social sciences (SPSS) software statistically computer package version 11. For quantitative data, the mean and standard deviation were calculated, the difference between two means was statistically analyzed using the student (t) test.

For qualitative data the number and percent disturbation were calculated. Chi square will be used as a test of significance and when found inappropriate fisher exact test was used significance was adopted to $P< 0.05$ for interpretation of results of tests significance.

3. Results

Our study demonstrated increase percentage of H.pylori +ve males than females on the reverse of H.pylori -ve patients (53.3% vs 46.7% but this was not statistically significant (p value 0.477), significant difference in residence between H.pylori +ve cases and H.pylori -ve controls with higher prevalence of H.pylori infection in rural areas, no significant difference in weight, weight centile, height, height centile, BMI and its centile between cases and controls.

The main clinical presentation of H. pylori positive children was the abdominal pain before meal followed by abdominal pain aggravated by food followed by nausea then heartburn then night abdominal pain then abdominal pain relieved by antacid then vomiting then anorexia &weight loss.

Histopathology of gastric, doudenal biopsies showed that 32/75 (42.7) of H.pylori positive children have H.pylori-associated gastritis and duodenitis with mild activity & mild H.pylori density (+).

Serum Pepsinogen II (ng/ml) Mean ± SD of H.pylori +ve group (24.54 ± 8.66) was higher than H.pylori -ve group which was(4.55 ± 2.37)with p value (<0.001).

And Serum gastrin 17 (pg/ml) Mean ± SD of H.pylori +ve group was (240.68 ± 231.77) was higher than H.pylori -ve group (29.26 ± 5.41) with p value (<0.001).

Table (1) Comparison between the studied groups regarding demographic data

			H.pylori +ve group	H.pylori -ve group	X^2	P. value
Sex	**Female**	**No. (%)**	35(46.7%)	47(52.2%)	0.505	0.477
	Male	**No. (%)**	40(53.3%)	43(47.8%)		
Residence	**Rural**	**No. (%)**	46(61.3%)	37(41.1%)	6.69	0.01
	Urban	**No. (%)**	29(38.7%)	53(58.9%)		
Weight (kg)	**Mean ± SD**		33.11 ± 14.76	33.439 ± 14.85		
	Rang		15.0 - 65.0	15.0 - 65.0	0.141	0.888
Height (cm)	**Mean ± SD**		131.87 ± 20.02	132.98 ± 19.77	0.357	0.721
	Rang		105 – 160	105 - 160		

Table (2) Frequency of clinical presentation of H.pylori +ve group

Presenting complaint	Percent
Abdominal pain before meal	100.00%
Abdominal pain aggravated by food	100.00%
nausea	76.00%
heart burn	72.00%
Night abdominal pain	69.30%
Abdominal Pain relieved by antacid	68.00%
Vomiting	48.00%
anorexia &weight loss	44.00%
anemia	32.00%
GIT bleeding	32.00%
Constipation	28.00%
Chest pain	28.00%
failure to thrive	28.00%
Abdominal pain relieved by bleching	24.00%
abdominal distension	16.00%
Diarrhea	12.00%
Abdominal pain relieved by food	0.00%

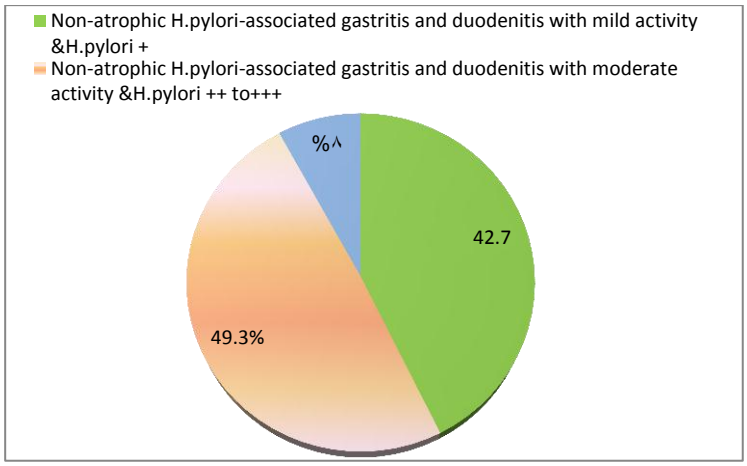

Fig (1) Histopathology of gastric, duodenal biopsies findings among cases

Table (3) Comparison between the studied groups regarding gastropanel (Serum Pepsinogen II & Serum gastrin 17).

		H.pylori +ve group	H.pylori –ve group	t.test	P.value
Serum Pepsinogen II (ng/ml)	**Mean ± SD**	24.54 ± 8.66	4.55 ± 2.37	20.978	<0.001
	Range	8.9 - 40.9	1.0 - 8.0		
Serum gastrin 17 (pg/ml)	**Mean ± SD**	240.68 ± 231.77	29.26 ± 5.41	8.657	<0.001
	Range	4.0 - 819.8	21.0 - 39.8		

4.Discussion

In Egypt, a high prevalence of H. pylori infections has been reported, ranging from 70% in the general population, 73% among school children [4].

Our results revealed that no significant difference in mean of sex between H.pylori positive cases and H.pylori negative controls. [5] in their meta-analysis on 10 studies, showed that there is no significant relationship between sex and incidence of H. pylori infection.. As regard effect of residence on the prevalence of H.pylori infection, our study revealed that there is significant difference in residence between H.pylori +ve cases and H.pylori -ve controls with higher prevalence of H.pylori infection in rural areas. This finding was in accordance with [6] who reported that living in rural areas were risk factors for the acquisition of H. pylori in Egyptian studies. our study showed that there was no significant difference in weight, weight centile, height, height centile ,BMI and its centile between cases and controls. According to

the studies by [7], there is no significant association between H. pylori infection and FTT but [8] revealed that H. pylori infection had adverse effects on growth parameters (weight and height) of pediatric patients and demonstrated that gastric H. pylori infection, growth faltering are substantially interrelated in pediatric patients.

Our study showed that the main clinical presentation of H. pylori positive children was the abdominal pain before meal followed by abdominal pain aggravated by food followed by nausea then heartburn then night abdominal pain then abdominal pain relieved by antacid then vomiting then anorexia &weight loss.

This finding was in accordance with [8] study that revealed a statistically significant ($P < 0.01$) positive correlation between H. pylori infection and gastrointestinal symptoms (recurrent abdominal pain, vomiting and chronic anorexia).

Our study histopathology of gastric ,doudenal biopsies showed that 32/75 (42.7) of H.pylori positive children have H.pylori-associated gastritis and duodenitis with mild activity & mild H.pylori density (+) 37/75(49.3%) were with moderate activity & moderate to marked H.pylori density (++ to+++) and 6/75 (8%) had severe activity & marked H.pylori density (+++) with superifial ulceration in gastric and duodenal mucosa. [9] reported that there were 64 H. pylori positives and 36 H. pylori negatives among them 61 H.pylori +ve (95.3%) and 3 H.pylori -ve (8.3%) had evidence of chronic superficial gastritis, mild (25), moderate (21) and severe (13).

Our study reported that Serum Pepsinogen II of H.pylori +ve group (24.54 ± 8.66) was higher than H.pylori -ve group .This finding is in accordance with [10] who reported that among children with abdominal pain (cases), those infected with H. pylori had significantly ($P < 0.001$) higher median levels of PGI and PGII and a lower PGI/PGII ratio than uninfected children ($P < 0.001$).

Our study reported that serum gastrin 17 of H.pylori +ve group was higher than H.pylori -ve group .This finding is in accordance with [11] found that mean gastritis scores and fasting serum gastrin levels were significantly higher in teenage subjects with H. pylori positive duodenal ulcer or gastritis than in those with H. pylori negative gastritis or normal mucosa, [9] no significant difference among the +ve,-ve groups of children was noticed in both fasting and postprandial gastrin levels while a significant reduction of basal and postprandial serum gastrin was noticed after H. pylori eradication.

5.Conclusion

Serum PG II and gastrin 17 can be a useful non-invasive marker forH.pylori gastritis, in evaluating children with recurrent abdominal pain with no apparent organic reason.

6. Recommendations

1-The use of Serum PG II and gastrin 17 can be a useful non-invasive marker for H.pylori gastritis, in evaluating children with recurrent abdominal pain with no apparent organic reason.

2- avoid unnecessary upper GIT endoscopy as an indication of recuurent abdominal pain by use of these gastropanel markers .

3- Further studies to confirm gastropanel markers significance and cutoff values .

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