Non-Ulcer Dyspepsia: Abnormal Myoelectrical Activity and Gastric Emptying, Fact Or Fiction?.

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Abstract: Background/Aim: Functional or non-ulcer dyspepsia is the presence of bothersome postprandial fullness, early satiation, epigastric pain and or epigastric burning sensation with no evidence of organic disease,systemic disease and/ or metabolic disease which explain the symptoms. The pathophysiology of non-ulcer dyspepsia is complex and includes many theories. This work was conducted to study the gastric myoelectrical activity and gastric emptying in Egyptian patients with non-ulcer dyspepsia. **Study design and methods:** This study was carried out on 150 subjects classified into three groups: **Group I:** included 50 patients with non-ulcer dyspepsia and *H.pylori* infection. **Group II:** included 50 patients with non-ulcer dyspepsia and without *H.pylori* infection. **Group III:** included 50 age and sex matched "healthy" asymptomatic volunteers; 25 positive and (IIIa) and 25 negative (IIIb) for *H.pylori*. All participants were subjected to abdominal ultrasonography for assessment of gastric emptying, Elecrogastrography for Gastric electrical activity recording and *H.pylori* infection was detected by histopathology of antral gastric biopsies for patient groups and by detection of antigen in stool for the control subjects. **Results:** Dominant frequency, percent of normal rhythm both fasting and after test meal, power of dominant frequency both fasting and after the test meal, and power ratio showed statistically non-significant difference between patients with non-ulcer dyspepsia and *H-pylori* infection and healthy subjects with *H-pylori* infection [2.8±0.56 vs. 2.79±0.39, 67.8±27.6 vs. 76.1±19.1, 75.57±23.18 vs. 71.9±18.53, 3280±2223.1 vs. 2309.8±1543.0, 5068.3±3037.8 vs. 5498.4±2751.3, and 2.44±3.68 vs. 2.94±1.92, respectively, with $p > 0.05$ in all comparisons]. Dominant frequency, percent of normal rhythm both fasting and postprandial, power of dominant frequency both fasting and postprandial, and power ratio was statistically non-significantly different on comparing patients with non-ulcer dyspepsia and without *H-pylori* infection and healthy subjects without *H-pylori* infection [3.01±0.26 vs. 2.95±0.35, 65.57±26.58 vs. 69.5± 31.36, 73.04±28.2 vs. 68.8±34.8, 2952.1±1765.19 vs. 2142.8±763.5, 3185.1±1859.9 vs. 4441.5±2124.3, and 1.48± 1.52 vs. 2.1±0.7 and $p > 0.05$ for all comparisons]. On comparing patients with non-ulcer dyspepsia and H-pylori infection to healthy subjects with *H-pylori* infection; Full emptying time was significantly shorter in patients with non-ulcer dyspepsia[12.85±3.17 vs. 15.4± 3.34, $p <0.05$], while ½ emptying time, antral dimension both postprandial and fasting were statistically non-significantly different[5.06±2.02 vs. 6.39±2.43, 7.3±1.69 vs. 8.44± 3.8, and 4.14±1.37 vs. 4.3± 1.58, respectively, with $p > 0.05$ for all comparisons]. On comparing patients with non-ulcer dyspepsia without *H-pylori* infection and healthy subjects without *H-pylori* infection ; there was no significant statistical difference regarding the full emptying time, ½ emptying time, full antrum dimension, and fasting antrum dimension [15.45±2.79 vs. 15.7±2.75, 7.05±3.52 vs. 6.7±1.39, 7.71±1.39 vs. 8.85± 2.44, and 3.68±1.13 vs. 3.68±0.73 with $p > 0.05$ for all comparisons]. There was no significant statistical difference between the patients with H-pylori infection and non-ulcer dyspepsia before and after *H-pylori* eradication therapy regarding the dominant frequency, percent of normal rhythm both fasting and postprandial, power of the dominant frequency both fasting and postprandial, and power ratio [2.8±o.56 vs. 2.93±0.44, 67.8±27.6 vs. 80.67±23.0, 75.57±23.18 vs. 79.9±23.09, 3280±2223.1 vs. 3073.25±1465.96, 5068.3±3037.78 vs. 6167.15±2339.94, and 2.44±3.68 vs. 2.22±0.85, and $p > 0.05$ for all comparisons]. On comparing patients with non-ulcer dyspepsia and H-pylori infection before and after eradication therapy there was a non-significant statistical difference in full emptying time[12.85±3.17 vs. 14.45±1.85 and $p > 0.05$], but ½ emptying time was statistically significantly longer after eradication than that before [5.06±2.02 vs. 6.05±109 and $p <0.05$], but, full antrum dimension and fasting antrum dimension was statistically non-significantly different before and after eradication therapy[7.3±1.69 vs. 7.37± 1.8 and 4.14± 1.37 vs. 4.12±1.19 with $p > 0.05$ for both comparisons. **Conclusion:** gastric myoelectrical activity is the same in patients with non-ulcer dyspepsia patients and healthy subjects regardless of the *H-pylori* status. *H-pylori* infection in non-ulcer dyspepsia patients may accelerate gastric emptying and *H-pylori* eradication therapy improves that abnormal gastric emptying. Non-ulcer dyspepsia patients without *H-pylori* infection may have different pathophysiological mechanisms than those with *H-pylori* infection.

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Key words: non-ulcer dyspepsia, abnormal myoelectrical activity and gastric emptying.

1. Introduction

Functional or non-ulcer dyspepsia [NUD] is the presence of bothersome postprandial fullness, early satiation, epigastric pain and or epigastric burning sensation with no evidence of organic disease [including at upper endoscopy], systemic disease and/ or metabolic disease which explain the symptoms. These symptoms should be present in the last 3 months and symptoms onset at least 6 months prior to the diagnosis. There are two subcategories, the postprandial distress syndrome and the epigastric pain syndrome [1].

The pathophysiology of NUD includes many theories; dysmotility has been the prim focus of interest in functional dyspepsia including delayed gastric emptying, accelerated gastric emptying, impaired fundic accommodation, unsuppressed postprandial fundic contractility, antral distension, and duodenal dysmotility [2].Visceral hypersensitivity is present in 30-40% of patients with functional dyspepsia based on the barostat technique and water load test [3].Aberrant cerebral processing of visceral stimuli in the form of lack of activation of the medial pain system and activation of the lateral pain system components at lower distending pressures occurs in NUD patients with visceral hypersensitivity [4]. Gastric tone and motility are influenced by duodeno-gastric reflexes, whose dysfunction has been proposed to play a role in the pathogenesis of symptoms in NUD. Receptors involved in this reflex include those for CCKA, dopamine, 5-HT1A, and gastrin releasing peptide [5]**.** Gastric myoelectrical abnormality such as tachygastria, bradygastria and flat line pattern has been reported in patients with NUD [6].

Gastric colonization by *H.pylori* has been found in 33-79% of patients with non-ulcer dyspepsia [7]. The possible effect of *H.pylori* infection on gastric motor function may be mediated by alternation of gastrin levels, gastric acid hypersecretion and release of cytokines (IL-1B, IL-6, IL-8, and TNF) [8]. Interaction between polymorphisms of genes responsible for components of the immune response and *H.pylori* infection among some patients with functional dyspepsia is suggested by a Japanese study[9].

Aim of the work:

This work was conducted to study the gastric myoelectrical activity and gastric emptying in Egyptian patients with non-ulcer dyspepsia.

2. Study design and methods:

This study was carried out on 150 subjects classified into three groups: Group I: included 50 patients with non-ulcer dyspepsia and *H.pylori* infection,20 females and 30 males with mean age 44±15.64. years.Group II: included 50 patients with non-ulcer dyspepsia and without *H.pylori* infection, 20 females and 30 males with mean age 42±14.42 years. Group III: included 50 age and sex matched "healthy" asymptomatic volunteers; 25 positive (IIIa) and 25 negative (IIIb) for *H.pylori.* The following were excluded from the study; patients with diabetes mellitus, patients with end-organ failure [e.g. renal and liver cell failure],patients with calcular and non-calcular cholecystitis,pregnant females, females taking contraceptive pills, patients taking drugs affecting gastric motility [e.g. prokinetics, anticholinergics, antibiotics like macrolides, dopaminergic agonists, adrenergic agents],patients with previous abdominal surgery and patients with systemic illness. All participants were subjected to the following:1. Full history taking; Subjects were asked about dyspeptic symptoms (e.g. nausea, vomiting, belching, fullness, bloating, epigastric pain,).2. Thourgh clinical examination**.**3. Laboratory investigations; Complete blood count, ESR, fasting and 2- hour postprandial blood sugar, liver function tests [serum albumin, total proteins, total and direct bilirubin, transaminases, Prothrombin time], kidney function tests [BUN, creatinine, Serum electrolytes],Urinalysis, stool analysis. 4.Chest X-ray,abdominal ultrasonography [U/S]**.**5. Upper gastrointestinal endoscopy for patients with NUD and antral biopsies were taken**. 6.**Histopathological examination of antral biopsied tissue for *H.pylori* infection determination. 7. Abdominal ultrasonography for assessment of gastric emptying: All subjects were fasting for at least 6 hours before examination, abdominal U/S was performed with 3.5 array transducer. With the subject sitting in a chair and slightly inclined backwards, the sonographic probe was positioned at the epigastrium to measure the pre-spinal antral area at the level of antrum-body junction in a single section. The superior mesenteric vein and the aorta were used as a reference points to standardize the scan position. Both patient's sitting position and small volume of liquid meal (<300ml water) are safe factors against floating of gastric contents in the corpus and fundus. The following markers of gastric motility were measured: A. Basal antral area: was the mean of two measurements taken 5 and 0 min. before the meal. B. Maximal postprandial antral area: was measured after maximal widening of the antrum had occurred, usually within 2 min. postprandial**.** C. Minimal postprandial antral area: was the smallest area measured at any time postprandial. D. Half emptying time: was the time in minutes to observe a 50% decrease in maximal antral area [t ½ time], Calculated by linear regression analysis from the linear part of antral emptying curve. Antral emptying curves were obtained by plotting antral area vs. time [10]. 8. Elecrogastrography*:* Gastric electrical activity was recorded from five disposal pre-gelled silver chloride surface electrodes placed on the upper abdomen. This was done after the skin has been carefully abraded to

decrease resistance to obtain a good signal to noise ratio.The patient was kept in a reclining position to minimize motion artifacts. Four EGG signals were recorded bipolar from these five electrodes as the potential differences between each of the four electrodes, and one central electrode. A reference electrode was placed at the left clavicle. The electrical signals are recorded with appropriate amplification and filtering. One hour recording while the patient is fasting was done, then given a standardized test meal [pastes and 250ml milk] and postprandial recording for one hour was done.After the recording session The EGG signals were subjected to spectral analysis [Fast Fourier Transform]. The dominant frequency [The frequency with the highest power], the percent of normal rhythm both fasting and postprandial, the power of the dominant frequency [The dominant power] both fasting and postprandial, and the power ratio [the ratio of the power of the mean spectrum of the postprandial state to the power of the mean spectrum of the fasting state, indicative of the postprandial increase in gastric motor activity] were determined for each subject. Dysrhythmia was defined as follows: A tachygastria was considered to be present when the power spectrum contained a sharp-peaked component with a frequency >3.7 cpm and <10.8 cpm, which was not of respiratory origin. For a definite diagnosis of tachygastria it was required that at the same time the normal gastric signal (2.6-3.7 cpm) was absent in all four EGG signals and that the abnormal rhythm was present for at least 2min.A so-called bradygastria was defined as presence of a sharp peak at a frequency less than 2.6 cpm, in the absence of a normal 3 cpm component in all four EGG leads.9. *Helicobacter pylori* eradication therapy: An eradication triple regimen for *H.pylori* [omeprazole 20mg bid, amoxicillin 1g bid and clarithromycin 500mg bid]was given for 10 days for every *H.pylori* positive patient, followed by 20 days of omeprazole. 10. Endoscopy, electrogastrography, and measurement of gastric emptying time using ultrasonography, were repeated one month after completion of therapy.10. Detection of *H.pylori* status in the control group: *H.pylori* status in the control group V was assessed by detection of *H.pylori* antigen in stool.11. Statistical analysis of results: student *t* test was used for comparison between different groups and a p value < 0.05 was considered statistically significant.

Informed consent was obtained from all participants before enrollment in the study. The study was approved by the local ethical committee.

Statistical analysis:

Data were analyzed using Statistical Program for Social Science (SPSS) version 17.0. Quantitative data were expressed as mean ± stander deviation (SD). comparison between two independent mean groups for parametric data using student T test, the probability of error at or less than 0.05 was considered significant.

3. Results:

Dominant frequency, percent of normal rhythm both fasting and after test meal, power of dominant frequency both fasting and after the test meal, and power ratio showed statistically non-significant difference between patients with non-ulcer dyspepsia and *H-pylori* infection and healthy subjects with *H-pylori* infection [2.8±0.56 vs. 2.79±0.39, 67.8±27.6 vs. 76.1±19.1, 75.57±23.18 vs. 71.9±18.53, 3280±2223.1 vs. 2309.8±1543.0, 5068.3±3037.8 vs. 5498.4±2751.3, and 2.44±3.68 vs. 2.94±1.92, respectively, with p > 0.05 in all comparisons [Table 1].

Dominant frequency, percent of normal rhythm both fasting and postprandial, power of dominant frequency both fasting and postprandial, and power ratio was statistically non-significantly different on comparing patients with non-ulcer dyspepsia and without *H-pylori* infection and healthy subjects without H-pylori infection [3.01±0.26 vs. 2.95±0.35, 65.57±26.58 vs. 69.5± 31.36, 73.04±28.2 vs. 68.8±34.8, 2952.1±1765.19 vs. 2142.8±763.5, 3185.1±1859.9 vs. 4441.5±2124.3, and 1.48± 1.52 vs. 2.1±0.7, respectively, and p > 0.05 for all comparisons.] [Table 2].

On comparing patients with non-ulcer dyspepsia and *H-pylori* infection to healthy subjects with *H-pylori* infection; Full emptying time was significantly shorter in patients with non-ulcer dyspepsia [12.85±3.17 vs. 15.4± 3.34, p <0.05], while ½ emptying time, antral dimension both postprandial and fasting were statistically non-significantly different [5.06±2.02 vs. 6.39±2.43, 7.3±1.69 vs. 8.44± 3.8, and 4.14±1.37 vs. 4.3± 1.58, respectively, with p > 0.05 for all comparisons] [Table3].

On comparing patients with non-ulcer dyspepsia without *H-pylori* infection and healthy subjects without *H-pylori* infection; there was no significant statistical difference regarding the full emptying time,½ emptying time, full antrum dimension, and fasting antrum dimension [15.45±2.79 vs. 15.7±2.75, 7.05±3.52 vs. 6.7±1.39, 7.71±1.39 vs. 8.85± 2.44, and 3.68±1.13 vs. 3.68±0.73, respectively, with p > 0.05 for all comparisons][Table 4].

There was no significant statistical difference between the patients with *H-pylori* infection and non-ulcer dyspepsia before and after *H-pylori* eradication therapy regarding the dominant frequency, percent of normal rhythm both fasting and postprandial, power of the dominant frequency both fasting and postprandial, and power ratio [2.8±0.56 vs. 2.93±0.44, 67.8±27.6 vs. 80.67±23.0, 75.57±23.18 vs. 79.9±23.09, 3280±2223.1 vs. 3073.25±1465.96, 5068.3±3037.78 vs. 6167.15±2339.94, and 2.44±3.68 vs. 2.22±0.85,

respectively, with $p > 0.05$ for all comparisons][Table 5].

On comparing patients with non-ulcer dyspepsia and *H-pylori* infection before and after eradication therapy there was a non-significant statistical difference in full emptying time[12.85±3.17 vs. 14.45±1.85 and $p > 0.05$], but ½ emptying time was statistically significantly longer after eradication than that before[5.06±2.02 vs. 6.05±109 and $p < 0.05$], but, full antrum dimension and fasting antrum dimension was statistically non-significantly different before and after eradication therapy[7.3±1.69 vs. 7.37± 1.8 and 4.14± 1.37 vs. 4.12±1.19, respectively, with $p > 0.05$ for both comparisons][Table 6].

Table 1: Comparison between Group I and control [group III a] regarding EGG parameters expressed as mean ± SD:

Group / EGG	Group I n=50	Group IIIa n=25	t value	*P*
DF (cpm)	2.8 ±0.56	2.79 ±0.39	0.07	>0.05
% normal rhythm [fasting]	67.8±27.6	76.1±19.1	0.84	>0.05
% normal rhythm [post prandial]	75.57±23.18	71.9 ±18.53	0.43	>0.05
Power- DF[fasting]	3280±2223.1	2309.8±1543.0	1.23	>0.05
Power- DF[post prandial]	5068.3±3037.8	5498.4 ±2751.3	0.38	>0.05
Power ratio	2.44 ±3.68	2.94 ±1.92	0.4	>0.05

DF; dominant frequency. Cpm; cycle per minute.

Table 2: Comparison between Group II and control [Group IIIb] regarding EGG parameters expressed in mean ± SD:

Group / EGG	Group II n=50	Group IIIb n=25	t- value	*P*
DF (cpm)	3.01 ±0.26	2.95±0.35	0.49	>0.05
% normal rhythm [fasting]	65.57±26.58	69.5±31.36	0.36	>0.05
% normal rhythm [postprandial]	73.04 ±28.2	68.8 ±34.8	0.35	>0.05
Power –DF [fasting]	2952.1±1765.19	2142.8±763.5	1.37	>0.05
Power-DF [postprandial]	3185.1 ±1859.9	4441.5±2124.3	1.66	>0.05
Power ratio	1.48 ±1.52	2.1±0.7	1.2	>0.05

Table 3: Comparison between Group I and control [group IIIa] regarding gastric emptying parameters expressed in mean ± SD:

Group / Emptying	Group I n=50	Group IIIa n=25	t -value	*P*
Full emptying time(min.)	12.85 ±3.17	15.4±3.34	2.04	<0.05
1/2 emptying time(min.)	5.06±2.02	6.39±2.43	1.59	>0.05
Full antrum (cm2)	7.3±1.69	8.44±3.8	1.13	>0.05
Fasting antrum(cm2)	4.14±1.37	4.3±1.58	0.3	>0.05

Table 4: Comparison between Group II and control [group IIIb] regarding gastric emptying parameters expressed in mean ± SD:

Group / Emptying	Group II N=50	Group IIIb n=25	t- value	*P*
Full emptying time (min.)	15.45±2.79	15.7±2.75	0.23	>0.05
1/2 emptying time(min.)	7.05±3.52	6.7±1.39	0.26	>0.05
Full antrum(cm2)	7.71±1.39	8.85±2.44	1.64	>0.05
Fasting antrum(cm2)	3.68±1.13	3.87±0.73	0.48	>0.05

Table 5: Comparison among Group I patients before and after eradication therapy as regards EGG parameters expressed in mean ± SD:

Group / EGG	Before Eradication therapy	After Eradication therapy	t- value	*P*
DF (cpm)	2.8±0.56	2.93±0.44	1.05	>0.05
% normal rhythm [fasting]	67.8±27.6	80.67±23.0	1.38	>0.05
% normal rhythm Postprandial]	75.57±23.18	79.9±23.09	0.6	>0.05
Power-DF [fasting]	3280.0±2223.1	3073.25±1465.96	0.31	>0.05
Power-DF [postprandial]	5068.3±3037.78	6167.15±2339.94	1.27	>0.05
Power ratio	2.44±3.68	2.22±0.85	0.27	>0.05

Table 6: Comparison between group I patients regarding gastric emptying parameters before and after eradication therapy expressed in mean ± SD:

Group / Emptying	Before eradication therapy	After eradication therapy	t- value	*P*
Full emptying time (min.)	12.85±3.17	14.45± 1.85	2.22	>0.05
1/2 emptying time (min.)	5.06±2.02	6.05±1.9	1.74	<0.05
Full antrum (cm2)	7.3± 1.69	7.37± 1.8	0.13	>0.05
Fasting antrum(cm2)	4.14± 1.37	4.12±1.19	0.06	>0.05

4. Discussion:

The absence of statistically significant difference in EGG parameters between patients with non-ulcer dyspepsia with *H-pylori* infection and healthy subjects with *H-pylori* colonization.Also, the absence of significant difference in EGG parameters before and after *H-pylori* eradication in non-ulcer dyspepsia patients. Furthermore, the absence of significant difference in EGG parameters between non-ulcer dyspepsia patients without *H-pylori* infection and healthy subjects without *H-pylori* infection suggest the EGG as a non-useful test in the assessment of non-ulcer dyspepsia patients and question the contribution of abnormal gastric myoelectrical activity to non-ulcer dyspepsia with or without *H. pylori* infection. That results are in accordance with the results reported by **Holmvall and Lindberge** who concluded that severe functional dyspepsia is not associated with abnormal EGG parameters. Also, that conclusion agree with the conclusion reported by **Oba-Kuniyoshi and his colleagues**, who suggested that postprandial symptoms in dysmotility-like functional dyspepsia are not related to disturbance of gastric myoelectrical activity [11,12]

Furthermore, **Abid and Lindberg** reported poor correlation between EGG parameters and antro-duodenal manometry and suggested doubtful clinical usefulness in adults [13].

In addition, some studies reported that *H-pylori* infection in very common among non-ulcer dyspepsia patients and its role in pathogenesis is debatable [14-16].

But, this is not in agreement with some studies confirming abnormal EGG parameters in non-ulcer dyspepsia patients, although they are not necessary to show the similar abnormalities in various patient groups [17- 19].

Also, our results do not agree with the results obtained by some studies demonstrating that *Helicobacter pylori* infection causes both inflammation and the co-existing motor disorders in functional dyspepsia patients with improvement of both symptoms and correction of the abnormal rhythm and power after eradication therapy of *H-pylori* [20, 21].

The great differences between different studies may be explained by the fact that EGG is still an investigational, yet, non-standardized tool in medicine with building of its standards which needs accumulation of tremendous number of studies with consequent systematic review and meta-analysis of that studies for conclusion of its rules to apply on human diseases.

The significantly shorter gastric full emptying time in patients with non-ulcer dyspepsia and *H. pylori* infection than healthy subjects with *H. pylori* colonization and the significant prolongation of the gastric half emptying time after *H. pylori* eradication in that group of patients suggests that accelerated gastric emptying rather than delayed gastric emptying is an associated finding in non-ulcer dyspepsia patients with

H. pylori infection and eradication therapy of *H. pylori* may improve that enhanced gastric emptying. The absence of that change in non-ulcer dyspepsia patients without *H. pylori* infection suggests the disease a different pathophysiological entity than *H. pylori* associated non-ulcer dyspepsia.

That results are in agreement with results reported by **Miyaji and his colleagues**, who found that *H. pylori* infected non-ulcer dyspepsia patients may have delayed gastric emptying, normal gastric emptying or accelerated gastric emptying and *H. pylori* eradication corrects the disturbed gastric emptying [22]. Also, accelerated rather than delayed gastric emptying associated with *H. pylori* infection was reported by Caldwell and his colleagues and **Minocha and his colleagues** [23,24]. However, our results are in contradiction to results obtained by some studies reporting lack of association of *H. pylori* infection or eradication therapy with gastric emptying abnormality in functional dyspepsia patients[25- 27].

Also, our results contradicts with the results of a study reporting delayed gastric emptying in *H. pylori* infected non-ulcer dyspepsia patients [28].

The accelerated gastric emptying in *H. pylori* infected functional dyspepsia patients may be explained by enhanced postprandial gastric sensation due to abnormal afferent function and irritable stomach syndrome [29].

The normal gastric emptying in *H. pylori* negative functional dyspepsia patients in our study is not in agreement with the results obtained by some studies reporting delayed gastric emptying in 24-78% of patients with functional dyspepsia [30, 31].

Conclusion:

Gastric myoelectrical activity is the same in patients with non-ulcer dyspepsia patients and healthy subjects regardless of the H.pylori status. H.pylori infection in non-ulcer dyspepsia patients may accelerate gastric emptying and H.pylori eradication therapy improves that abnormal gastric emptying. Non-ulcer dyspepsia patients without *H. pylori* infection may have different pathophysiological mechanisms than those with *H. pylori* infection.

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