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A Study of the Concordance between Endoscopic Gastritis and Histological Gastritis in Nonulcer Dyspeptic Patients with and Without *Helicobacter Pylori* Infection

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ABSTRACT:

Background/Aim: Dyspepsia significantly impairs quality of life and is a major burden on healthcare funding. *Helicobacter pylori* (*H. pylori*) infection is the main cause of active chronic gastritis and peptic ulcer disease in both adults and children.

Patients &Methods: A total of 90 consecutive patients with dyspepsia attending Tanta University Hospitals for diagnostic upper gastrointestinal endoscopy and found to have nonulcer dyspepsia were enrolled in this study. Eight basic endoscopic and histological types of gastritis (superficial, hemorrhagic, erosive, verrucous, atrophic, metaplastic, hyperplastic and special types) were defined. Four or more biopsy specimens were obtained from the lesser and the greater curvatures of the antrum and the corpus of each patient, and the histological findings of gastritis and *Helicobacter pylori* infection were assessed. The endoscopic and the histological diagnoses were then compared in a blinded fashion.

Results: The sensitivity of endoscopic diagnosis in comparison to histological diagnosis of superficial gastritis, hemorrhagic gastritis, erosive gastritis, verrucous gastritis, atrophic gastritis, hyperplastic gastritis, and congestive gastropathy were 86%, 100%, 67%, 50%, 90%, 25%, and 50% respectively.

Conclusion: The following conclusions were drawn: (i) it is better to use six basic types: superficial, hemorrhagic, erosive, verrucous, atrophic and special types; (ii) it is appropriate to exclude metaplastic and hyperplastic types from the fundamental types; however, they might be additionally described if they are significant; (iii) The significant endoscopic features in the patients infected with *H. pylori* were erythema, edema, exudate, friability, nodularity, vascular pattern, flat erosion, atrophy, and hypertrophy.

Key words: Gastritis, Histological Gastritis, Nonulcer Dyspeptic, *Helicobacter Pylori*.

INTRODUCTION

Dyspepsia is not a diagnosis, but merely a cluster of symptoms believed to be referable to the upper gastrointestinal tract.¹ It is very

common in the adult population with prevalence rates ranging from 19% up to 41% in several epidemiological studies.² About

one out of every four subjects with dyspepsia consults his general practitioner³, and these visits account for 1%–4% of all consultations in primary care.⁴ Twenty five per cent of these patients are referred for further investigations (that is, endoscopy, ultrasonography, etc) or to a secondary care physician (about 10%) but the majority of patients are managed empirically by their general practitioner.⁵ Altogether, **dyspepsia** is an important health issue and constitutes a significant clinical problem in primary care.

The isolation of *Helicobacter pylori* (*H. pylori*) from clinical specimens by Marshall and Warren 23 years ago⁶ launched revolutions in gastroenterology and microbiology.⁷

The Gram-negative, spiral-shaped bacterium *Helicobacter pylori* is a common human pathogen and public health problem that causes gastritis and peptic ulcers.⁸ Infection with *H. pylori* is also associated with severe gastric pathologies, including peptic ulcer, chronic active gastritis and gastric cancer. This microorganism is able to invade and colonize human stomach, directly interacting with gastric epithelial cells. Several histological changes are linked with different clinical outcomes, as infiltration of the mucosa by inflammatory cells (chronic gastritis), loss of lining mucosa (ulcer, atrophy), inflammation with infiltrating lymphocytes and development of masses (hyperplastic polyps, adenomas, carcinomas, lymphomas).⁹

It is common practice for endoscopists to make judgements on the presence or absence of gastritis on the basis of the endoscopic appearance of the gastric mucosa. The

concept of “endoscopic gastritis” was given further credence by the acknowledgement of its existence by the working party that formulated the Sydney System of classifying gastritis.¹⁰ A number of studies have shown that the concordance between endoscopy and histology with regard to the diagnosis of gastritis is abysmal¹¹ while the results of others have suggested that the concordance is not unreasonable.¹² The more enthusiastic viewpoint is that concordance may be good in the severe forms of gastritis¹⁰ and that a normal endoscopy excludes active gastritis.¹³

PATIENTS & METHODS

Patient Selection: From Oct 2005 till Jan 2007, 90 patients who suffered from functional dyspepsia were recruited and included in this study.

Functional dyspepsia (synonymous with “non-ulcer” dyspepsia) is defined by the Rome II consensus as persistent or recurrent pain or discomfort (persistent for 12 weeks in the past 12 months) centred in the upper abdomen, without evidence of organic disease. The upper abdominal pain or discomfort is often described as indigestion, fullness, bloating or early satiety accompanied by belching, nausea and vomiting. The definition has been further refined by excluding patients with the above symptoms when these are related to their bowel movements, who are then considered to have the irritable bowel syndrome, and those with predominant heartburn which favours a diagnosis of gastro-oesophageal reflux.¹⁴

All patients in this study were subjected to:

History: full history taken with special emphasis on the following; special habits (smoking, alcohol), upper gastrointestinal symptoms, medications, and any history of chronic medical condition or cancers

Clinical examination: all patients underwent clinical examination.

Laboratory investigations; urine, stool, complete blood picture, ESR, blood urea and serum creatinine.

Abdominal ultrasonography: any abnormalities in the liver, gall bladder, spleen, and kidneys were recorded. Any abdominal lymphadenopathy, ascites, stomach wall abnormality, portal hypertension, abdominal masses, and midline structure were also recorded. Patients were examined after 6 hours fasting.

Measurement of IgG antibodies against *H. pylori*.

Upper GIT endoscope with taking many biopsies for urease studies and histological examination.

Exclusion criteria: Patients who gave history of the following medications were excluded:

NSAIDS treatment on regular basis.

Recent history of NSAIDS, proton pump inhibitors or H₂ blockers, and/or antibiotics intake within a month or less.

Alcoholic.

H. pylori treatment.

Gastro-oesophageal reflux disease.

Peptic ulcer disease.

Cholelithiasis or choledocholithiasis.

Any chronic disease or end organ failure other than those included in our groups (i.e. diabetes mellitus and cirrhotic liver).

Abdominal malignancy (especially pancreatic and gastric cancer).

Gastrectomy.

Coagulopathy.

Written informed consent to a biopsy of the gastric mucosa & blood sample for diagnosis of *H. pylori* infection was obtained from each study participant.

H. PYLORI ASSESSMENT

H. pylori was said to be present if results of at least two of three tests (urease, histological examination, and IgG) were positive. Patients were considered not to be infected if results for all three tests were negative. Retesting was planned if only one test result had been positive; but, in fact, there were no such results.

UPPER ENDOSCOPY

Was done by the conventional method after 6 hours fasting prior to the procedure, with a fibre-optic flexible endoscope attached to a cold source of light with flash, under throat anesthesia with 10% xylocaine spray. Intra venous sedation (midazolam) was used. The endoscopic findings were photographed whenever needed.

Standard biopsy forceps were used to take biopsy specimens from the following sites as described by the Sydney system: two each from the anterior and posterior antrum, 2-5 cm from the pylorus; two each from the anterior and posterior body, 10 cm from the cardia; and two from any additional area of

abnormalities. A further antral biopsy specimen was taken for assessment for *H. pylori* by the urease test.¹⁰

The Sydney classification of endoscopic gastritis aims to standardize reporting by classifying endoscopic gastritis based on many endoscopic mucosal features: edema, punctuate and confluent erythema, friability, punctuate and confluent exudate, flat and raised erosion, rugal hyperplasia and atrophy, visibility of vascular pattern, punctuate and confluent intramural bleeding spots, and coarse nodularity.¹⁰

All endoscopies were reported including a subjective assessment of severity as mild, moderate or severe, and then classified into one of the following eight categories: superficial gastritis, hemorrhagic gastritis, erosive gastritis, verrucous gastritis, atrophic gastritis, metaplastic gastritis, hyperplastic gastritis, and special gastritis.¹⁵

HISTOPATHOLOGICAL EXAMINATION

Biopsy samples from the antrum and body were formalin-fixed and paraffin-embedded and cut into 2-3 μm sections, which were stained using hematoxylin and eosin (H&E). Five H&E sections were examined for each case. Morphology was based on 16 histological characteristics and their severity were assessed. Based on these findings, the histological diagnoses of gastritis were grouped into eight types of gastritis (Table 2), and their names were used for endoscopic diagnosis (Table 1).

STATISTICS

Statistical analysis was done with MINITAB STATISTICAL SOFTWARE™ (MINITAB Release 13.1). Clinical data were analyzed by the unpaired *t* test (for age) and Fisher's exact test (for sex). Differences with *p* values < 0.05 were considered to be statistically significant. Odds ratios for endoscopic gastritis were derived from multiple logistic regression analysis.

RESULTS

A total of 90 consecutive patients with dyspepsia attending Tanta University Hospitals for diagnostic upper gastrointestinal endoscopy between Oct 2005 and Jan 2007 and found to have nonulcer dyspepsia were enrolled in this study.

According to history, examination, and investigations, the patients were divided into the two following groups:

Group I: Dyspeptic patients infected with *H. pylori*.

Group II: Dyspeptic patients not-infected with *H. pylori*.

The study included 51 (57%) males and 39 (43%) females. Their ages ranged from 25 to 65 years. Comparison of mean age and sex ratios by the unpaired *t* test or Fisher exact test, were tabulated & figurate in table 3, & figure 1.

Odds Ratio for cigarette smoking, and main dyspepsia complaint associated with *H. pylori* Infection were shown in table 4. Odds ratio for cigarette smoking was 0.63. Odds ratio for pain was 0.77. Odds ratio for discomfort was 2.61. Odds ratio for bloating was 2.39. Odds ratio for early satiety was 1.08. Odds ratio for indigestion was 0.26.

Endoscopic findings in all patients, categorized as infected and non-infected by *H. pylori* were tabulated in table 5. Edema was present in forty-one patients (58% of infected patients) and two patients (11% of non-infected patients). Erythema was present in fifty-two patients (73% of infected patients) and three patients (16% of non-infected patients). Reddish streaks were present in one patient (1% of infected patients) and two patients (11% of non-infected patients). Friability was present in ten patients (14% of infected patients) and one patient (5% of non-infected patients). Exudate was present in eighteen patients (25% of infected patients) and one patient (5% of non-infected patients). Flat erosion was present in five patients (7% of infected patients) and one patient (5% of non-infected patients). Raised erosion was present in two patients (3% of infected patients) and one patient (5% of non-infected patients). Rugal hypertrophy was present in two patients (3% of infected patients) and one patient (5% of non-infected patients). Rugal atrophy was present in eight patients (11% of infected patients) and two patients (11% of non-infected patients). Vascular pattern was present in eleven patients (15% of infected patients) and two patients (11% of non-infected patients). Bleeding spots was present in two patients (3% of infected patients) and

one patient (5% of non-infected patients). Nodularity was present in seven patients (10% of infected patients) and one patient (5% of non-infected patients). Endoscopic diagnosis for both groups was shown in table 6. Odds ratio for findings of *H. pylori* infection associated with endoscopic features by logistic regression analysis were tabulated in table 7.

Table (8) shows histopathological diagnosis of all patients. There were three inappropriate specimens, from two infected and one non-infected patients. Normal mucosa was reported for nine patients with *H. pylori* (12.7%), and ten patients without *H. pylori* (53%). Superficial gastritis was reported for nineteen patients with *H. pylori* (27%), and two patients without *H. pylori* (11%). Hemorrhagic gastritis was reported for one patient with *H. pylori* (1%), and none in patients without *H. pylori*. Erosive gastritis was reported for three patients with *H. pylori* (4.2%), and none in patients without *H. pylori*. Verrucous gastritis was reported for one patient with *H. pylori* (1%), and one patient without *H. pylori* (5%). Atrophic gastritis was reported for eight patients with *H. pylori* (11.3%), and two patients without *H. pylori* (11%). Metaplastic gastritis was reported for four patients with *H. pylori* (5.6%), and one patient without *H. pylori* (5%). Hyperplastic gastritis was reported for three patients with *H. pylori* (4.2%), and one patient without *H. pylori* (5%). Congestive gastropathy was reported for twenty-one patients with *H. pylori* (30%), and one patient without *H. pylori* (5%).

Endoscopic diagnosis compared to histopathological diagnosis was shown in table 9. The highest sensitivity of endoscope was achieved in hemorrhagic gastritis, while the lowest one was achieved in hyperplastic gastritis. The highest specificity of endoscope was achieved in hemorrhagic gastritis and verrucous gastritis, while the lowest one was

achieved in superficial gastritis. The highest positive predictive value of endoscope was achieved in normal endoscope, while the lowest one was achieved in hyperplastic gastritis. The highest negative predictive value of endoscope was achieved in hemorrhagic gastritis, while the lowest one was achieved in congestive gastropathy.

Table (1): Fundamental types of endoscopic findings and their diagnostic criteria. ¹⁵

Fundamental types	Definition according to endoscopic findings
Superficial gastritis	Findings including edema and redness (spotted, patchy, linear), friability, and/or exudate are observed
Hemorrhagic gastritis	Hemorrhage is evidenced
Erosive gastritis	Erosive changes including flat or depressed types
Verrucous gastritis	Erosive changes including elevated type
Atrophic gastritis	Findings such as color change of mucosa, visible vascular pattern and thinning are observed
Metaplastic gastritis	Intestinal metaplasia is noted *
Hyperplastic gastritis	Remarkable irregularity of mucosa or rugal hypertrophy of greater curvature in corpus
Special gastritis	Congestive gastropathy: The term 'portal hypertensive gastropathy' refers to the mosaic-like pattern, congestion and edema of the mucosa with or without red spots seen endoscopically in patients with portal hypertension. 16

*The intestinal metaplasia was defined as the lesion visualized as an ash-colored nodular change by conventional endoscopy alone without dyeing.¹⁷

Table (2): Histological classification of gastritis and its diagnostic criteria. ¹⁵

Fundamental types	Definition according to histological findings
Superficial gastritis	Atrophy and inflammation are hardly observed in glands with observation of inflammatory cell infiltration only at the surface of mucosa
Hemorrhagic gastritis	Hemorrhage, hemosiderin sedimentation, hemosiderin phagocytic macrophage are observed
Erosive gastritis	Defect of superficial mucosa is observed, with relevant bioresponse (fibrin precipitation, hemorrhage, edema, neutrophil infiltration and growth of capillary) being evidenced
Verrucous gastritis	This is in the state of hyper-regeneration after erosion, with irregular running of muscle fibers of muscularis mucosae and hyperplasia of pyloric glands surrounded by myofibers in the area of pyloric glands, as well as replacement of pseudopyloric glands and alterations in regeneration of foveolar epithelium
Atrophic gastritis	Atrophy of glands is observed
Metaplastic gastritis	In the biopsy specimens, intestinal metaplastic tube is observed in more than 1/3 of mucosal tissues
Hypertrophic gastritis	Hypertrophy of glands is observed while foveolar epithelium is almost normal or hypertrophic
Congestive gastropathy	Is characterized by submucous vessel dilatation and twist, and no obvious inflammation. ¹⁸

Table (3): Age and sex of all patients.

		Group I (Infected)	Group II (Non-Infected)
Age*	Range	25-63	36-65
	Mean	44.30	48.20
	SD \pm	10.24	6.79
Sex*	Males	40 (56%)	11 (58%)
	Females	31 (44%)	8 (42%)

* $p > 0.1$ for comparison of mean age and sex ratios by the unpaired t test or Fisher exact test, respectively. Percentages are approximated to nearest integer.

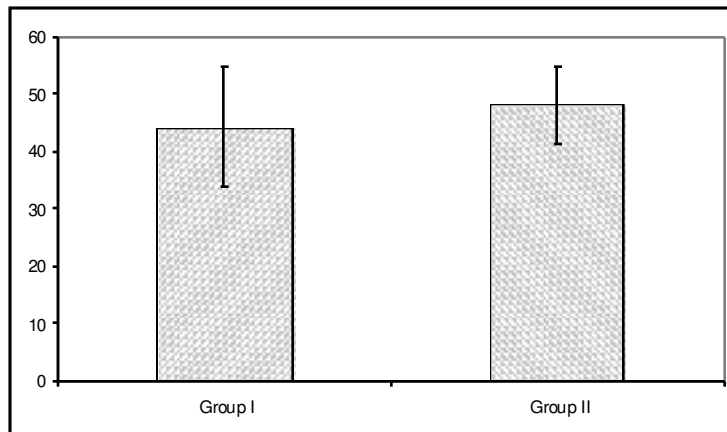


Figure [1]: Age of all patients.

Table (4): Odds ratio for cigarette smoking, and main dyspepsia complaint associated with *H. pylori* infection by logistic regression analysis.

Factor		Odds Ratio	95% CI	
			Lower	Upper
Predisposing:	Cigarette	0.63	0.19	2.05
Symptoms:	Pain	0.77	0.27	2.22
	Discomfort	2.61	0.31	22.02
	Bloating	2.39	0.63	9.07
	Early satiety	1.08	0.27	4.31
	Indigestion	0.26	0.07	0.97

Table (5): Endoscopic findings.

Findings n (%)	Infected (n =71)	Not infected (n =19)
Edema	41 (58%)	2 (11%)
Erythema	52 (73%)	3 (16%)
Reddish streaks	1 (1%)	2 (11%)
Friability	10 (14%)	1 (5%)
Exudate	18 (25%)	1 (5%)
Flat erosions	5 (7%)	1 (5%)
Raised erosions	2 (3%)	1 (5%)
Rugal hypertrophy	2 (3%)	1 (5%)
Rugal atrophy	8 (11%)	2 (11%)
Vascular pattern	11 (15%)	2 (11%)
Bleeding spots	2 (3%)	1 (5%)
Nodularity	7 (10%)	1 (5%)

Percentages are approximated to nearest integer.

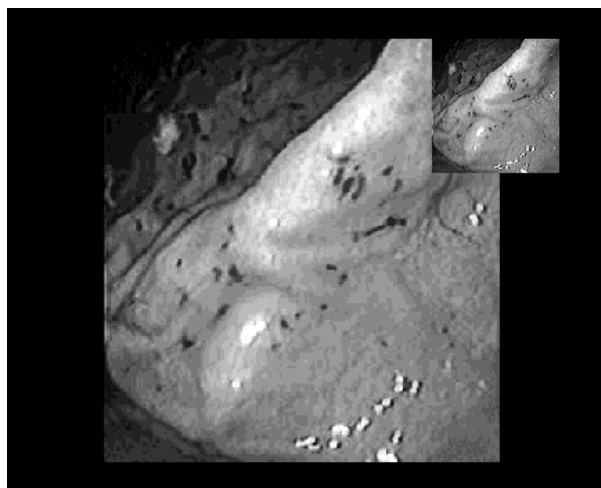


Figure (2): hemorrhagic gastritis

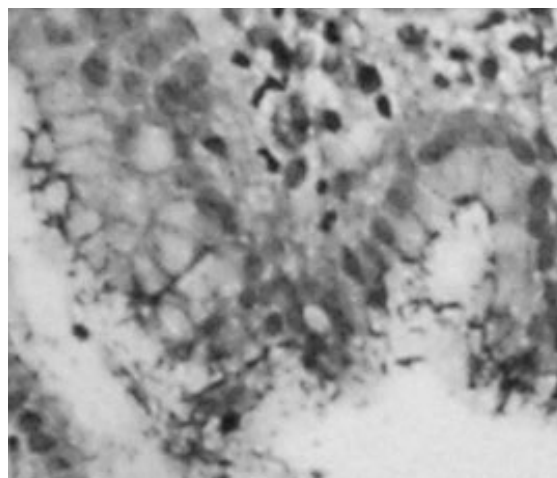


Figure (3): *H. pylori* (Van Gieson stain X400)

Table (6): Endoscopic diagnosis.

Diagnosis N (%)	Infected (n =71)	Not infected (n =19)
Normal	7 (10%)	10(52%)
Superficial gastritis	29 (41%)	3 (16%)
Hemorrhagic gastritis	2 (3%)	0 (0%)
Erosive gastritis	5 (7%)	0 (0%)
Verrucous gastritis	2 (3%)	0 (0%)
Atrophic gastritis	11 (15%)	3 (16%)
Metaplastic gastritis	0 (0%)	0 (0%)
Hyperplastic gastritis	2 (3%)	1 (5%)
Congestive gastropathy	13 (18%)	2 (11%)

Percentages are approximated to nearest integer.

Table (7): Odds Ratio for Findings of H. pylori Infection Associated With Endoscopic Features by Logistic Regression Analysis.

Endoscopic Findings	Odds Ratio	95% CI	
		Lower	Upper
Edema	11.62	2.49	54.13
Erythema	14.60	3.82	55.77
Reddish streaks	0.12	0.01	1.42
Friability	2.95	0.35	24.63
Exudate	6.11	0.76	49.10
Flat erosions	1.36	0.15	12.42
Raised erosions	0.79	0.08	8.10
Rugal hypertrophy	1.07	0.11	10.22
Rugal atrophy	1.08	0.21	5.56
Vascular pattern	1.56	0.31	7.72
Bleeding spots	0.52	0.04	6.08
Nodularity	1.97	0.23	17.06

Table (8): Histological diagnosis.

Diagnosis N (%)	Infected (n =71)	Not infected (n =19)
Normal	9 (12.7%)	10(53%)
Inappropriate specimens	2 (3%)	1(5%)
Superficial gastritis	19 (27%)	2 (11%)
Hemorrhagic gastritis	1 (1%)	0 (0%)
Erosive gastritis	3 (4.2%)	0 (0%)
Verrucous gastritis	1 (1%)	1 (5%)
Atrophic gastritis	8 (11.3%)	2 (11%)
Metaplastic gastritis	4 (5.6%)	1 (5%)
Hyperplastic gastritis	3 (4.2%)	1 (5%)
Congestive gastropathy	21 (30%)	1 (5%)

Percentages are approximated to nearest integer.

Table (9): Endoscopic diagnosis compared to histological diagnosis.

	Sensitivity	Specificity	Positive predictive value	Negative predictive value
Normal	74 %	96 %	82 %	93 %
Superficial gastritis	86 %	80 %	56 %	95 %
Hemorrhagic gastritis	100 %	99 %	50 %	100 %
Erosive gastritis	67 %	97 %	40 %	99 %
Verrucous gastritis	50 %	99 %	50 %	99 %
Atrophic gastritis	90 %	94 %	64 %	99 %
Hyperplastic gastritis	25 %	98 %	33 %	97%
Congestive gastropathy	50 %	94 %	73 %	85 %

Percentages are approximated to nearest integer.

DISCUSSION

Dyspepsia describes a symptom complex, which can be caused by several underlying conditions such as peptic ulceration, esophagitis, or gastric carcinoma. The cardinal feature of dyspepsia is a pain or discomfort in the upper abdomen, with or

without reflux or dysmotility-like symptoms. There is no discernable biochemical or structural explanation for the symptoms in around 60% of patients with dyspepsia, who are classified as having functional (or nonulcer) dyspepsia. ¹⁹

Our aim was to identify endoscopic features associated with *H. pylori* infection in patients with nonulcer dyspepsia and to determine the correlation between endoscopic and histologic diagnoses of gastritis.

An Egyptian serological study on a group of asymptomatic persons, all aged below 30 years, revealed an overall prevalence of *H. pylori* was 87.6% with a prevalence of 53% in those below 10 years, 100% in those between 11-20 years and 95% of those between 21-30 years.²⁰

Epidemiologic studies show that between 30% and 60% of all patients with dyspeptic symptoms are infected with *H. pylori*.²¹

In this study, the overall prevalence of *H. pylori* positive patients was 78.89% (71 patients) that is to a certain extent related to that obtained by El-Zayadi et al.²² where the prevalence of *H. pylori* among 92 non-ulcer dyspepsia Egyptian patients using the rapid urease test was 86.4%. The differences may be attributed to different methods of *H. pylori* assessment.

The kind and frequency of endoscopic changes associated with gastritis in subjects infected with *H. pylori* are not known in detail and this disease could not be diagnosed from the endoscopic appearance alone.²³ Additionally, no consensus has been reached about whether gastritis related to *H. pylori* can be diagnosed from macroscopic changes in the gastric mucosa.^{24,25} Most of these studies have been prospective, evaluating the endoscopic appearance as a diagnostic predictor of gastritis related to *H. pylori*. Here we identified endoscopic changes associated with gastritis in patients infected with *H. pylori*, making comparisons with patients who were not infected with the bacterium. Hypothetically, endoscopic inspection without the need for biopsies would be a convenient way to diagnose such gastritis, if possible. It is uncertain what endoscopic changes are characteristic of gastritis in subjects infected

with *H. pylori*. In fact, one more recent case-control study on 281 patients in Tokyo Medical and Dental University that focuses on identifying endoscopic features associated with *H. pylori* infection in patients with non-ulcer dyspepsia.²⁶

Our endoscopic findings in all patients were categorized as infected and non-infected as regard *H. pylori*. In infected-patients group our results revealed endoscopic finding in the following order, erythema (73% of infected patients), edema (58% of infected patients), exudate (25% of infected patients), vascular pattern (15% of infected patients), friability (14% of infected patients), rugal atrophy (11% of infected patients), nodularity (10% of infected patients), flat erosion (7% of infected patients), raised erosion (3% of infected patients), rugal hypertrophy (3% of infected patients), bleeding spots (3% of infected patients), and reddish streaks (1% of infected patients).

While between *H. pylori*-positive patients, Toshifumi et al.²⁶ had reported endoscopic finding in the following order, vascular pattern (94% of infected patients), exudate (66% of infected patients), erythema (62% of infected patients), edema (52% of infected patients), nodularity (30% of infected patients), rugal atrophy (28% of infected patients), flat erosion (26% of infected patients), raised erosion (22% of infected patients), friability (14% of infected patients), rugal hypertrophy (14% of infected patients), bleeding spots (2% of infected patients), and reddish streaks (0% of infected patients).

For *H. pylori* non-infected patients, our endoscopic findings were ordered as following, erythema (16% of non-infected patients), vascular pattern (11% of non-infected patients), reddish streaks (11% of non-infected patients), edema (11% of non-infected patients), rugal atrophy (11% of non-infected patients), exudate (5% of non-infected patients), nodularity (5% of non-infected patients), flat erosion (5% of non-infected

patients), raised erosion (5% of non-infected patients), friability (5% of non-infected patients), rugal hypertrophy (5% of non-infected patients), and bleeding spots (5% of non-infected patients).

Toshifumi et al.²⁶ had reported endoscopic finding between *H. pylori*-negative patients, in the following order, reddish streaks (26% of non-infected patients), vascular pattern (14% of non-infected patients), bleeding spots (14% of non-infected patients), exudate (12% of non-infected patients), raised erosion (10% of non-infected patients), erythema (8% of non-infected patients), edema (2% of non-infected patients), rugal atrophy (2% of non-infected patients), nodularity (2% of non-infected patients), flat erosion (2% of non-infected patients), friability (0% of non-infected patients), and rugal hypertrophy (0% of non-infected patients).

Comparison of those data and our study is statistically difficult, so we will use odds ratio for such it. Our study demonstrated that, odds ratios were > one for the following endoscopic findings, erythema, edema, exudate, friability, nodularity, vascular pattern, flat erosion, rugal atrophy, and rugal hypertrophy which were (14.60, 11.62, 6.11, 2.95, 1.97, 1.56, 1.36, 1.08, and 1.07) respectively.

On the other hand, Toshifumi et al.²⁶ found odds ratios which > one for the following endoscopic finding, vascular pattern, edema, rugal hypertrophy, nodularity, rugal atrophy, erythema, friability, flat erosion, exudate, and raised erosion which were (96.2, 53.1, 40.1, 21.0, 19.1, 18.8, 17.4, 17.2, 14.2, and 2.54) respectively.

So, the only difference was raised erosion, but by considering its 95% confidence intervals in Toshifumi et al.²⁶ study, included 1.00 (0.81-7.94), it could be eliminated.

So, the significant endoscopic features in the patients infected with *H. pylori* were an erythema, edema, exudate, friability, nodularity, vascular

pattern, flat erosion, rugal atrophy, and rugal hypertrophy.

The odds ratios for reddish streaks and bleeding spots were <1.00 in this study. These results suggest that the endoscopic findings of reddish streaks and bleeding spots were not associated with *H. pylori* induced-inflammation of the gastric mucosa.

In our study, the odds ratio for the erythema was the highest for the infected patients. Erythema is the most easily seen endoscopic abnormality, and chronic gastritis judged histologically has been found in 75% or more of the patients in large studies.²⁷

In the study of Toshifumi et al.²⁶, the odds ratio for the visibility of a vascular pattern was the highest for the infected patients. This endoscopic finding was generally found in such patients when the histological results showed that atrophy was present for both parts of the stomach considered separately. Type B chronic gastritis is chronic atrophic gastritis that extends from the antrum to the corpus²⁸; the high odds ratios for a vascular pattern being visible in the dyspeptic patients infected with *H. pylori* confirmed that this bacterium is the main cause of type B gastritis. This agreement of endoscopic and histological findings is compatible with the report of Sauerbruch et al.²⁹ that the macroscopic criterion of visibility of submucosal vessels is significantly related to the histological diagnosis of atrophic gastritis, and with other earlier reports.¹²

The reasons for such a difference could be ascribed to the rate of progression of *H. pylori* pangastritis to atrophy and subsequent cancer varies in different regions of the world, being rapid in regions with high rate of gastric cancer and slow in regions where duodenal ulcer is common, as well-known as, gastric

adenocarcinoma remains the most common malignancy in Japan and China.³⁰

As far as isolated endoscopic findings concerns, no overall association was found between the endoscopic abnormalities described by the Sydney classification and histological gastritis. The endoscopic abnormalities described in the Sydney classification are also not specific for the diagnosis of gastritis, and such abnormalities were often found in the absence of histological changes.²³

We therefore used a new endoscopic classification of gastritis that devised by Michio Kaminishi et al.¹⁵. It is easy to use, be based on the histopathology, employ expressions that are universally understood, and the endoscopic and histological diagnoses were divided into the same eight items making the comparison more easy and reliable.

It has been well established that gastric histology becoming the "gold standard" for detecting *H. pylori* infection and its associated lesions.³¹

Comparing our consistency of endoscopic diagnosis to histological diagnosis we found that, for normal endoscopic appearance sensitivity was 74%, specificity was 96%, positive predictive value was 82% and negative predictive value was 93%.

The sensitivity of endoscopic diagnosis of superficial gastritis, hemorrhagic gastritis, erosive gastritis, verrucous gastritis, atrophic gastritis, hyperplastic gastritis, and congestive gastropathy were 86%, 100%, 67%, 50%, 90%, 25%, and 50% respectively. Consequently, endoscope is a good negative for hemorrhagic gastritis, atrophic gastritis, and superficial gastritis. It is relatively good negative for erosive gastritis, verrucous gastritis, and congestive gastropathy but weak negative for hyperplastic gastritis.

The specificity of endoscopic diagnosis of superficial gastritis, hemorrhagic gastritis, erosive gastritis, verrucous gastritis, atrophic gastritis,

hyperplastic gastritis, and congestive gastropathy were 80%, 99%, 97%, 99%, 94%, 98%, and 94% respectively. Consequently, endoscope is a good positive for all types of gastritis except metaplasia.

The sensitivity of endoscopic diagnosis of hyperplastic gastritis was low compared to the histopathological diagnosis, but its specificity was high. Thus, ordinary endoscopic examinations are unsuitable for diagnosing hyperplastic gastritis, and the magnification endoscopy, high-resolution endoscopy, and chromoscopy methods are needed to make the diagnosis.

The intestinal metaplasia was defined as the lesion visualized as an ash-colored nodular change by conventional endoscopy alone without dyeing.¹⁷

Contrary to a previous study, by using conventional endoscopy alone without dyeing we missed four cases with metaplasia that proved by histopathology. Thus, apart from subjective endoscopist error, we recommend using of staining techniques (endoscopy with methylene blue) as it carry a great diagnostic potential and should be further put to the test for use in daily clinical practice.

Supporting our observation, Bruno³², claimed that, endoscopic intravital staining techniques using absorptive and contrast stains can be used to enhance the visual characteristics of both normal and abnormal (that is, metaplasia, dysplasia and carcinoma) tissue creating a visual distinction for early detection and targeted biopsy.

Therefore, hyperplastic gastritis, and intestinal metaplasia should be additionally described only in notable occasions and should not be incorporated into the fundamental representing endoscopic diagnosis.

The results of the comparison between endoscopic and histopathological diagnoses in regards to

atrophic gastritis revealed that sensitivity and specificity are high (90% and 94%) respectively. The endoscopic findings (i.e. color changes in the mucosa, mucosal thinning and visibility of vascular pattern) proved to be valid.

The Sydney System classified atrophy and atrophic gastritis as independent wings.³³ Patients with gastric mucosal atrophy but no gastritis (histologically mild to severe atrophy of the gastric glands with less than 'slight' lymphocyte infiltration and no neutrophil infiltration) considered atrophy rather than atrophic gastritis. We investigated the incidence of these cases that was 0%, suggesting that evidence of atrophy observed by endoscopy might permit a pathological diagnosis of atrophic gastritis in these cases. However, they are closely correlated with each other and can be diagnosed as 'atrophic gastritis' both endoscopically and histopathologically. This point needs a further study on large number of patients to clarify that conflict.

Overall, as endoscopic examinations are 2-Dimensions and performed on in situ tissue whereas histopathological observations are pinpoint and performed on in vitro specimens, differences might be responsible for discrepancies in diagnosis.

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