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ORIGINAL ARTICLE

The Specificity of Endoscopic Criteria in the Diagnosis of Biliary Reflux Gastritis

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Abstract

Background: **Bile reflux gastritis can be triggered by an excess of bile in the duodenum, the absence of a pylorus as a barrier to retrograde flow, and/or decreased stomach and duodenum retrograde peristalsis. This can occur as a result of gastric or biliary surgery or due to biliary reflux disease.**

The purpose of this research was to assess the specificity of endoscopic criteria in the diagnosis of biliary gastritis by comparing endoscopic findings with histopathological examination.

Patients and methods: **In this prospective research, cases with upper gastrointestinal symptoms were subjected to esophagogastroduodenoscopy. In all, 113 patients with intragastric bile were involved in the research. All cases were subjected to endoscopic examination for biliary reflux gastritis (BRG) and histopathological examination.**

Results: **In this prospective study, 113 individuals were involved, who were divided into two groups, BRG (62 patients) and nonbiliary reflux gastritis (NBRG) (51 patients). Endoscopic findings of the patients in the BRG group showed that all patients had intragastric bile and most of the patients had erythema (83.9%) followed by erosions (54.8%). Histological findings in the BRG group showed that most patients had foveolar hyperplasia (83.9%) followed by chronic active inflammation (75.8%) and congestion (64.5%). The positive predictive value of endoscopic diagnosis of biliary gastritis was 54.86%.**

Conclusion: **In BRG, the endoscopic findings are not specific but can aid in diagnosis. Histopathological features can characterize BRG from NBRG.**

Keywords: Biliary, Endoscopy, Gastritis, Reflux

1. Introduction

Endoscopic examination became standard practice at the start of the 20th century, allowing for the first time the investigation of biliary gastritis (BG) and other forms of gastritis.[1] The backward flow of duodenal fluid, which contains bile, pancreatic juices, and secretions of the intestinal mucosa into the stomach and esophagus,[2] results in mucosal lesions, which is a pathological disease known as bile reflux gastropathy.[3] Heartburn, regurgitation, epigastric discomfort, and other symptoms of bile reflux gastropathy are thought to be triggered by bile acids reacting with gastric acid.[4]

Bile reflux gastropathy is common following gastric surgeries that injure the pyloric sphincter and biliary surgeries and procedures such as cholecystectomy, endoscopic sphincterotomy, endoscopic stenting, or choledochoduodenostomy that trigger malfunction of the sphincter of Oddi.[5] In cases of extended fasting, bile gastropathy manifests as a natural physiological process.[6]

Primary and secondary bile reflux are the two main categories. Bile reflux in individuals without a

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history of gastroduodenal surgery is referred to as primary bile reflux, whereas bile reflux following gastric surgery is referred to as secondary bile reflux.[7] Regurgitation and inflammation are the defining features of bile reflux gastritis (BRG), a kind of gastrointestinal illness in clinical practice.[8] Li et al.[7] also reported that the detection rate of biliary reflux and the degree of reflux increased with the progression of mucosal lesions. Clinicians' basic understanding or even ignorance of the condition is due to absence of guidelines.[8]

Duodenogastroesophageal reflux is characterized by the presence of intragastric bile and different degrees of inflammation in the esophagus.[8] Basnayake et al.[9] demonstrated that patients with gastroesophageal reflux disease have a higher prevalence of duodenogastroesophageal reflux.

Changes in endoscopy and histology are possible after prolonged and repeated exposure of the stomach mucosa to bile reflux. Abdominal discomfort, dyspepsia, nausea, vomiting, weight loss, and heartburn are just some of the symptoms that may or may not be present[10]. Therefore, established endoscopic findings are required for the diagnosis of BRG.

We aimed to assess the specificity of endoscopic criteria in the diagnosis of BG by comparing the endoscopic findings with histopathological examination.

2. Patients and methods

In this prospective research, 113 patients were involved.

2.1. Inclusion criteria

The article is ethically approved by Faculty of Medicine, Al-Azhar University, Cairo, Egypt.

Individuals over the age of 18 years with upper gastrointestinal symptoms (e.g. nausea, epigastric discomfort, vomiting, heartburn, or abdominal pain) who visited the inpatient or outpatient clinics of the gastroenterology and hepatology unit at Al-Hussein University Hospital, Al-Azhar University's Internal Medicine Department were eligible for inclusion. All participants gave their informed consent before enrolment.

2.2. Exclusion criteria

Exclusion criteria included the presence of hiatal hernia, a history of upper gastrointestinal surgery that altered normal stomach emptying processes, current use of NSAIDs, and elevated blood bilirubin levels.

2.3. All patients were subjected to the following

2.3.1. Full clinical assessment

Full clinical assessment included clinical symptoms such as abdominal pain, dyspepsia, nausea, dysphagia, bilious vomiting, heartburn, weight loss, history of *Helicobacter pylori* eradication, gallbladder dysfunction, cholecystectomy, biliary sphincterotomy, NSAIDs and opioid medications, type II diabetes mellitus, and BMI.

2.3.2. Endoscopic examination of biliary gastritis

Endoscopic findings of BRG included intragastric bile, erythema, erosions, ulcers, gastric atrophy, as well as determination of the site of gastropathy (antrum, body, and fundus).[3]

2.3.3. Histopathological examination of the gastric biopsy

A tissue biopsy specimen was collected from the antral region's lesser curvature (a), from the antral region's greater curvature (b), from the antral region's lesser curvature (c), from the antral region's greater curvature (d), and from the antral region's incisura angularis (e).[11] The tissue was fixed immediately in a 10% formaline solution. One experienced gastrointestinal pathologist examined the specimens to assess the presence and grade of gastritis as well as the cause of gastritis. Histological findings included foveolar hyperplasia. The foveolar cells showed regenerative changes with mucin depletion, erosions, edema, smooth muscle fibers in the lamina propria, chronic or acute inflammation, congestion of superficial capillaries in lamina propria, gastric atrophy, the status of *H. pylori* infection, and intestinal metaplasia or dysplasia.[12]

In the study of Dixon et al.,[13] they developed a 'reflux score' for BRG. Foveolar hyperplasia, edema, and smooth muscle fibers in the lamina propria, as well as vasodilatation and congestion of the lamina propria, were each given a score between 0 (normal or nonexistent) and 3 (severe) based on their severity. Both acute and chronic inflammatory cells were scored on a scale from 0 (severe increase) to 3 (no polymorphs present; chronic inflammatory cell count is normal or decreased). Patients' total reflux gastritis scores (which varied from 0 to 15) were determined by aggregating the points they received on each of the individual factors. Scores more than 10 are considered suggestive of reflux gastropathy out of a possible 15.

According to histopathology results, we divided patients into two groups: BRG group (which the endoscopy showed intragastric bile with positive histopathology for BG) and the nonbiliary reflux gastritis (NBRG) group: which the endoscopy showed intragastric bile with negative histopathology for BG.

2.4. Statistical analysis of data

IBM's statistical program, SPSS, version 20.0, was used for analyzing the data provided into the computer (IBM Corp., Armonk, New York, USA). Quantitative and percentage descriptions were used for qualitative information. To ensure a normally distributed sample, we used the Kolmogorov–Smirnov test. Quantitative information was summarized using mean, SD, and range (minimum and maximum). The acquired findings were deemed significant at the 5% level. A statistically significant *P* value was defined as less than 0.05.

2.5. The used tests

The χ^2 test (which is used to compare categorical variables across groups), Student's *t*-test (which is used to compare continuous variables across groups), and the Mann–Whitney *U* test (which is used to compare continuous variables across groups) are all available.

3. Results

Regarding the age of the patients in the BRG group, the age ranged between 20 and 40 years for almost half of the patients (48.4%) with a mean ± SD of 41.47 ± 11.255 years, while in the NBRG group more than half of the patients were aged ranged between 20 and 40 years (60.8%) and it ranged between 18 and 81 years with mean ± SD of 37.45 ± 14.346 years. As regards mean ± SD, there were significant variations among the two groups ($P = 0.030$, Table 1).

As regards CBC and BMI, there were no significant distinctions among two groups (Table 2).

Patients with BRG had many complaints, there were significant variations among BRG and NBRG groups regarding heartburn, which was 16.1 and 58.8%, respectively ($P = 0.001$). Instead, abdominal pain and dyspepsia were significantly higher in the BRG group than the NBRG group (Table 3).

Endoscopic findings of the patients in the BRG group showed that all patients had intragastric bile with most patients exhibiting erythema (83.9%) followed by erosions (54.8%). Regarding the extent of findings in the stomach, about half of the patients had antral gastritis 27 (43.5%). In the NBRG group also all patients had intragastric bile and most of the patients had erythema (94.1%) followed by erosion (33.3%), as regards the extent of findings in the stomach. More than half of the patients had antral gastritis 26 (51.0%). The erosions in the stomach were more in BRG than NBRG (54.8 and 33.3%, respectively), which is statistically significant ($P = 0.024$). According to the site of lesions in the stomach, pangastritis was significantly higher in NBRG (45.1%) than BRG (21%) ($P = 0.008$), while both antrum and body were affected more in the BRG group ($P \leq 0.001$, Table 4).

Histological findings in the BRG group showed that most patients had foveolar hyperplasia (83.9%) followed by chronic active inflammation (75.8%) and congestion (64.5%); in the NBRG group the majority of the patients had chronic active inflammation (82.4%) followed by congestion (66.7%). Foveolar hyperplasia, smooth muscle fibers in lamina propria, and mucin depletion were significantly higher in the BRG group, while *H. pylori* positive was significantly higher in the NBRG group than the BRG group (54.9 and 21% respectively, Tables 5 and 6 and Figs. 1–6).

Table 1. Comparison between bile reflux gastritis group and nonbile reflux gastritis group as regards patient's demographic data.

Demographic data	BRG group ($N = 62$) [*n* (%)]	NBRG group ($N = 51$) [*n* (%)]	Test of significance	*P* value
Age				
<20	2 (3.2)	5 (9.8)	$\chi^2 = 15.281$	0.152
20-40	30 (48.4)	31 (60.8)		
40-60	24 (38.7)	12 (23.5)		
>60	6 (9.7)	3 (5.9)		
Minimum–maximum	18–70	18–81	$U = 1204.50$	0.030[a]
Mean ± SD	41.47 ± 11.255	37.45 ± 14.346		
Sex				
Male	36 (58.1)	26 (51.0)	–	0.569[FE]
Female	26 (41.9)	25 (49.0)		

BRG, biliary reflux gastritis; FE, Fisher's exact test; NBRG, nonbiliary reflux gastritis; *U*, Mann–Whitney test; χ^2, Chi-square test.
P: *P* value for comparing the two examined groups.
[a] Statistically significant at *P* value less than 0.05.

Table 2. Comparison between bile reflux gastritis group and nonbile reflux gastritis group as regards patient's clinical presentation.

Clinical presentation	BRG group ($N = 62$) [n (%)]	NBRG group ($N = 51$) [n (%)]	Test of significance	P value
BMI				
Minimum–maximum	19–28	21–29	$U = 1391.00$	0.268
Mean ± SD	25.15 ± 1.924	24.78 ± 2.043		
Comorbidity				
DM	17 (27.4)	12 (23.5)	–	0.671[FE]
HTN	26 (41.9)	15 (29.4)	–	0.238[FE]
Hemoglobin				
Minimum–maximum	8.7–15.7	9.8–15.7	$U = 1403.00$	0.303
Mean ± SD	12.16 ± 1.322	12.43 ± 1.310		
White blood cells				
Minimum–maximum	3.95–13.50	4.24–12.50	$U = 1308.00$	0.115
Mean ± SD	7.96 ± 1.923	8.36 ± 1.822		
N/L ratio				
Minimum–maximum	0.45–3.43	0.77–6.06	$U = 1385.00$	0.258
Mean ± SD	1.47 ± 0.518	1.47 ± 0.752		
Platelets				
Minimum–maximum	140–403	80–340	$U = 1461.00$	0.488
Mean ± SD	230.24 ± 60.058	215.33 ± 51.204		

BRG, biliary reflux gastritis; DM < diabetes mellitus; FE, Fisher's exact test; HTN, hypertension; NBRG, nonbiliary reflux gastritis; U, Mann–Whitney test.

Table 3. Comparison between the bile reflux gastritis group and nonbile reflux gastritis groups as regards patients' complaints.

Complains	BRG group ($N = 62$) [n (%)]	NBRG group ($N = 51$) [n (%)]	P value
Abdominal pain	17 (27.4)	18 (35.3)	0.417[FE]
Dyspepsia	10 (16.1)	2 (3.9)	0.062[FE]
Heartburn	10 (16.1)	30 (58.8)	<0.001*[FE]
Bilious vomiting	2 (3.2)	0	0.500[FE]
Dyspepsia and weight loss	1 (1.6)	0	1.000[FE]
Dyspepsia and heartburn	6 (9.7)	0	0.031*[FE]
Abdominal pain and weight loss	1 (1.6)	0	1.000[FE]
Abdominal pain and heart burn	8 (12.9)	1 (2.0)	0.039*[FE]
Abdominal pain and dyspepsia	6 (9.7)	0	0.031*[FE]
Abdominal pain, dyspepsia and heartburn	1 (1.6)	0	1.000[FE]
Total	62 (100)	51 (100)	

BRG, biliary reflux gastritis; FE, Fisher's exact test; NBRG, nonbiliary reflux gastritis.
*:Statistically significant at $P < 0.05$.

Table 4. Comparison between bile reflux gastritis group and nonbile reflux gastritis groups as regards patient's endoscopic findings.

Endoscopic findings	BRG group ($N = 62$) [n (%)]	NBRG group ($N = 51$) [n (%)]	P value
Intragastric bile	62 (100)	51 (100)	–
Erythema	52 (83.9)	48 (94.1)	0.138[FE]
Erosions	34 (54.8)	17 (33.3)	0.024[a,FE]
Ulceration	6 (9.7)	2 (3.9)	0.291[FE]
Polyp	3 (4.8)	0	0.250[FE]
Gastric atrophy	0	1 (2.0)	0.451[FE]
Site of lesions			
Pan gastritis	13 (21.0)	23 (45.1)	0.008[a,FE]
Antral	27 (43.5)	26 (51.0)	0.454[FE]
Antral + Body	20 (32.3)	1 (2.0)	<0.001[a,FE]
Prepyloric	2 (3.2)	0	0.500[FE]
Body	0	1 (2.0)	0.451[FE]

BRG, biliary reflux gastritis; FE, Fisher's exact test; NBRG, nonbiliary reflux gastritis.
P: *P* value for comparing the two examined groups.
[a] Statistically significant at *P* value less than 0.05.

Table 5. Comparison between biliary reflux gastritis and nonbiliary reflux gastritis groups as regards patient's histological findings.

Histopathological findings	BRG group ($N = 62$) [n (%)]	NBRG group ($N = 51$) [n (%)]	P value
Foveolar hyperplasia	52 (83.9)	18 (35.3)	<0.001[aFE]
Chronic active inflammation	47 (75.8)	42 (82.4)	0.490[FE]
Congestion and vasodilatation	40 (64.5)	34 (66.7)	0.845[FE]
Smooth muscle fibers in the lamina propria	49 (79.0)	3 (5.9)	<0.001[aFE]
Mucin depletion	41 (66.1)	12 (23.5)	<0.001[aFE]
Erosions	26 (41.9)	14 (27.5)	0.119[FE]
Edema	21 (33.9)	24 (47.1)	0.179[FE]
H. pylori positive	13 (21.0)	28 (54.9)	<0.001[aFE]
Ulcers	6 (9.7)	1 (2.0)	0.126 [FE]
Gastric atrophy	0	1 (2.0)	0.451[FE]
OLGA staging	0	1 (2.0)	0.451[FE]
OLGIM	1 (1.6)	2 (3.9)	0.588[FE]
Intestinal metaplasia	1 (1.6)	2 (3.9)	0.588[FE]

BRG, biliary reflux gastritis; FE, Fisher's exact test; NBRG, nonbiliary reflux gastritis.
P: *P* value for comparing the two examined groups.
[a] Statistically significant at *P* value less than 0.05.

Table 6. Positive predictive value of endoscopic diagnosis of biliary reflux gastritis.

	Histopathology findings		
	Positive	Negative	PPV
Positive endoscopic findings of BRG	62 (true positive for endoscopy)	51 (false positive for endoscopy)	54.86%

BRG, biliary reflux gastritis; PPV, positive predictive value.

$$\text{The positive predictive value} = \frac{\text{True positive}}{\text{True positive} + \text{False positive}} = 54.86\%$$

4. Discussion

Bile reflux gastritis, often referred to as alkaline reflux gastritis, is characterized by persistent stomach mucosal inflammation, erosion, and ulceration.[14] In terms of prevalence, an endoscopic evaluation for stomach discomfort revealed that 23.9% of the participants had bile reflux.[15] Foveolar hyperplasia, mucin depletion, and vascular congestion in the superficial layer of the stomach mucosa are the most prominent histological alterations in BRG..[8] The diagnosis was based on clinical findings, pH monitoring of the aspirated gastric juice on the assumption that the bile reflux would cause an increase of pH over 7 because of the alkaline nature of duodenal juice[16] along with the aid of endoscopic and pathologic findings.

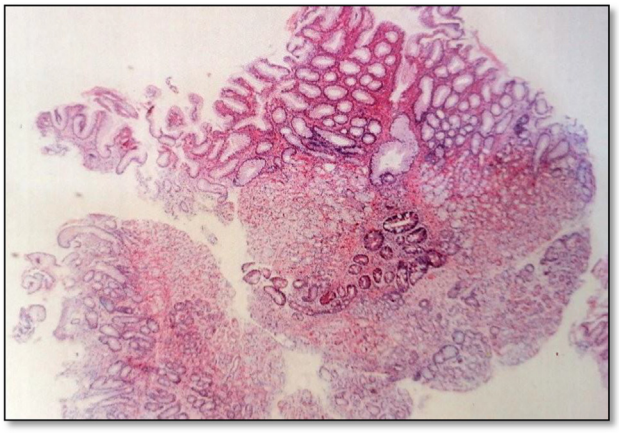

Fig. 1. Antral gastric mucosa in a case of BRG showing foveolar hyperplasia and complete intestinal metaplasia with minimal inflammatory reaction (hematoxylin and eosin, × 40). BRG, biliary reflux gastritis.

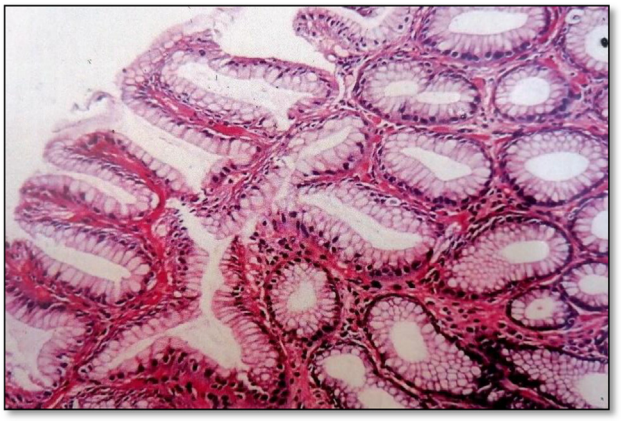

Fig. 2. High power field of the previous figure showing prominent foveolar hyperplasia and incomplete intestinal metaplasia (hematoxylin and eosin, × 400).

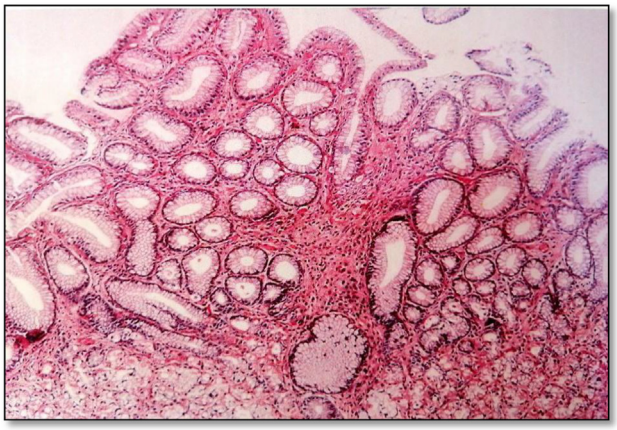

Fig. 3. BRG showing foveolar hyperplasia and smooth muscle proliferation in the lamina propria with minimal inflammatory reaction (hematoxylin and eosin, × 100). BRG, biliary reflux gastritis.

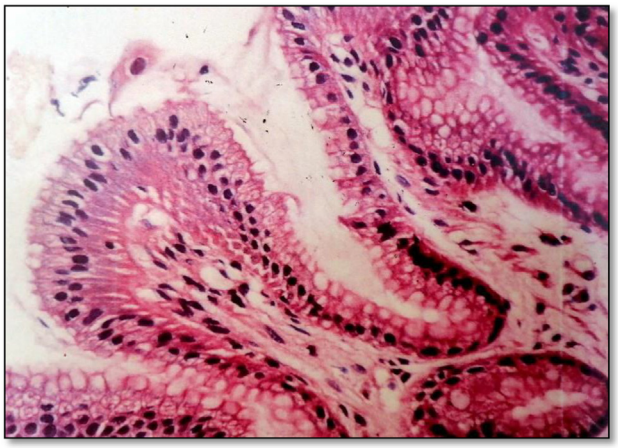

Fig. 4. BRG showing smooth muscle proliferation in the lamina propria (hematoxylin and eosin, × 400).BRG, biliary reflux gastritis.

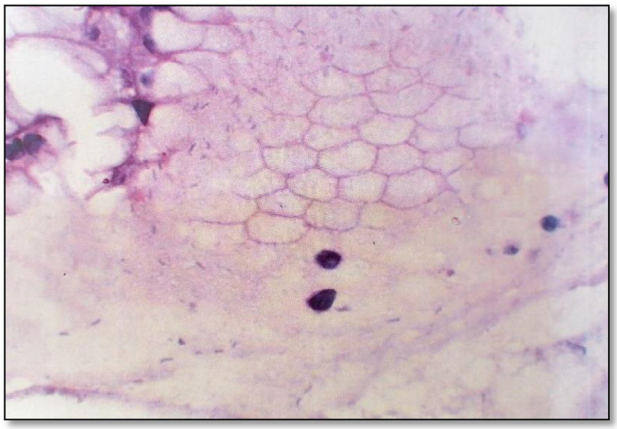

Fig. 5. Many H. pylori overlying the surface mucosa in a case of NBRG (hematoxylin and eosin, × 1000). NBRG, nonbiliary reflux gastritis.

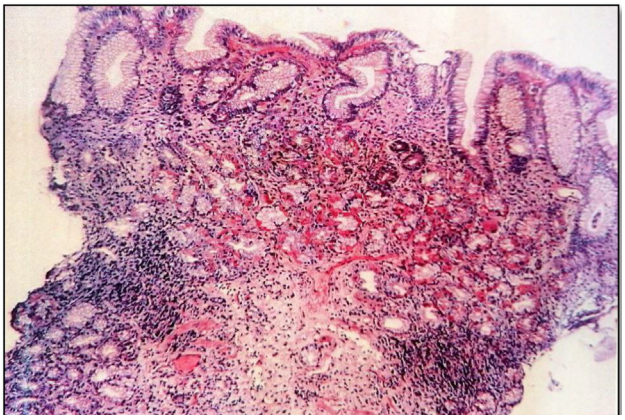

Fig. 6. Body gastric mucosa showing infiltration of the deeper glands by heavy lymphocytic aggregates consistent with mild gastric atrophy in NBRG (hematoxylin and eosin, × X100). NBRG, nonbiliary reflux gastritis.

[17]. So, our study aimed to assess the specificity of endoscopic criteria in the diagnosis of BG by comparing the endoscopic findings with histopathological examination.

Analysis of our findings showed that in BRG, almost half of patients were in the age range of between 20 and 40 years (48.4%). In agreement with our findings, Barakat et al.[6] and Al-Bayati and Alnajjar[17] showed that the age in BRG was 45.03 + 14.4 years and under 50 years, respectively. Taking all results together, the age distribution in BRG was more common among the younger age group.

Vere et al.[3] reported that 60% of BRG cases were in males, with a male/female ratio of 1.5/1. This is in a line with our cohort, where 58.1% were male, and the male/female ratio was 1.3/1. In contrast to our findings, Barakat et al.[6] found that in the BRG group, female patients exceeded male patients by a factor of 1.5 to 1. These results highlight the disease is common in both males and females. As regards BMI our results were supported by Barakat et al.[6] and Lake et al.[18] There is no statistical difference between BRG and NBRG. BMI is not a risk factor for BRG.

Diabetes gastroparesis was characterized by delayed gastric emptying without mechanical obstruction[19] and was correlated with gastroduodenal dysmotility. This condition could be brought on by persistent hyperglycemia in either type 1 or type 2 diabetes, which was responsible for proper gastric movement.[10] Also, Barakat et al.[6] and Lake et al.[18] reported that diabetes mellitus presented in 26.8 and 16.7% in the BRG group, respectively, which agrees with our result. Although there is no statistically significant difference between BRG and

NBRG, the number of diabetic patients was more in the BRG group which highlights that DM could be a risk factor for BRG.

Almost half of BRG patients in our cohort had epigastric pain (53.2%) and only two patients had bilious vomiting and this is in agreement with Chen et al.[20] Also, Al-Bayati and Alnajjar[17] reported that the most common symptoms were epigastric pain (46%). In contrast to our findings, Barakat et al.[6] concluded that nausea was the most reported symptom in patients with BRG followed by epigastric pain/discomfort (69.6 and 58.9%, respectively). However, heartburn was a common presentation in NBRG in our cohort.

As regards complete blood count indices, hemoglobin, white blood cells, and platelets in the BRG group were almost within normal ranges, which gives an idea about the effect of BRG on gastric mucosa that does not cause a severe bleeding gastric ulcer. Our result is in agreement with Turk et al.[21]

In our study, all patients had intragastric bile; erosion was significantly higher in BRG, and erythema of gastric mucosa was present in 83.9%. Moreover, Vere et al.[3] Al-Bayati and Alnajjar[17] and Lake et al.[18] were in agreement with our study as they describe erythema of the gastric mucosa in 64.43, 50, and 88.9% of cases, respectively. In contrast to our findings, Lake et al.[18] reported that NBRG patients had a significantly increased prevalence of superficial erosions than BRG patients (22.3 vs. 8.9%). This difference might be related to the small sample size of our cohort, and most of their patients had postcholecystectomy in BRG. Considering all these results, erythema, which is commonly seen in people with BRG, can aid in the identification of the condition by endoscopic examination. However, its usefulness is limited by a lack of specificity in terms of endoscopic symptoms. Most of our patients (43.5%) had antral gastritis in BRG which is in contrast to Barakat et al.[6] who reported 41.1% have pangastritis in the BRG group. These findings highlight the BRG could range from mildI involvment of the antrum or severe cause pangastritis.

Histopathological findings in the BRG group showed that the majority of patients had foveolar hyperplasia (83.9%) followed by chronic active inflammation (75.8%) and congestion (64.5%). There were highly statistically significant distinctions among the two groups as regards the foveolar hyperplasia, smooth muscle fibers proliferation in the lamina propria, mucin depletion, and *H. pylori* status.

As regards histological diagnosis of BRG, foveolar hyperplasia, smooth muscle fibers in the lamina propria, and mucin depletion were the main findings in our cohort that support BRG, and this is in agreement with Vere et al.[3] Mino-Kenudson et al.[22] and Lake et al.[18] These data suggest that bile reflux into the stomach is associated with more severe gastric mucosal alterations than NBRG.

In our study, these endoscopic lesions (such as intragastric bile) found in the stomach are not specific for the BRG, and they could be found in any other disease. The proportion of patients who have intragastric bile with other gastric mucosal lesions giving positive test results for BRG after confirmation by histopathology diagnosis was 54.86%.

The limitations of our study include a small sample size, single-center design and the absence of another group with negative endoscopic findings for BRG for comparison with histopathology findings of BRG.

We conclude that the endoscopic findings of BRG are not specific but can aid diagnosis. There were important histopathological features that can characterize BRG from NBRG. We recommend more multicenter studies, for the diagnosis of BRG, that rely on a scoring module that incorporates endoscopic (intragastric bile) and histopathological findings.

Conflicts of interest

There are no conflicts of interest.

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