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The Correlation between Body mass Index and Esophagogastroduodenoscopic Finding in Patients with Dyspepsia

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ABSTRACT

Due to its substantially increasing prevalence, obesity has become a worldwide concern. A tremendous number of studies existing nowadays highlighting the negative impacts of obesity on human health, however the potential role of obesity in gastrointestinal symptoms is unclear and, furthermore, inconsistent results reported from many studies. We, in this study, tried to answer the question whether obesity carry a higher risk for specific endoscopic diagnoses in patients with dyspepsia. This is a cross sectional study conducted at Sohar Hospital, Sultanate of Oman, on patients who complained from dyspepsia and referred as a candidate for esopagogastorduodenoscopy (EGD) due to either persistence of symptom, presence of alarming feature(s) or being more than 50 years. Clear definitions of obesity, dyspepsia, EGD findings and alarming features were made according to the general consensus. EGD was done to all patients who were included in the study for gross endoscopic abnormality as well as for *Helicobacter pylori* (H. pylori) infection. The endoscopic findings were analysed by the two the endoscopists independently. Among 118 participants, 61 were obese (51.8%) and 57 (48.2%) were non-obese. There were no statistical differences in term of patients' gender and ethnicity. Eighty four (71.2%) patients presented with one of the alarming features. Weight loss, as a presentation, was particularly more common in obese patients ($p < 0.001$). Among all patients, 14 (11.9%) had normal EGD, majority were non obese ($p < 0.033$). For obese patients, the majority, 58 (95.1%), showed abnormal EGD findings ($p < 0.001$). A significant difference was observed between obese and non-obese patients presented with respect to hiatus hernia with esophagitis ($p <0.001$), gastritis with positive H. pylori ($p = 0.046$), gastritis with negative H. pylori ($p = 0.021$) and gastritis due to bile reflux ($p = 0.002$). Obese patients in our study showed a significantly higher abnormal EGD findings compared to non-obese. Bile reflux related gastritis represented the commonest abnormality. With these findings, EGD might be considered more frequently in obese patients who presented with dyspepsia for early diagnosis and treatment.

Keywords: Esophagogastroduodenoscopic, *Helicobacter pylori*

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INTRODUCTION

Obesity is an epidemic with an increasing prevalence worldwide. According to World Health Organization (WHO) estimation, there are over 500 million obese adults defined as body mass index (BMI) equal or more than 30 kg/m^2 across the world [(1)]. According to Global prevalence of adult obesity, the prevalence of obesity in Oman is around 40.5 %.

Obesity is known to increase susceptibility to several conditions including type 2 diabetes mellitus (DM), hypertension (HTN), dyslipidaemia, coronary artery disease, some cancers, sleep apnea syndrome, gastroesophageal reflux disease (GERD) and esophageal motility disorders [(2,3)]. It is predicted that by the year 2020, 77.6% of men will be overweight and 40.2% obese; the corresponding predictions for women are 71.1% overweight and 43.3% obese [(4)].

Epidemiologic studies have shown that the prevalence of GERD and chronic dyspepsia in Western countries is approaching 20% [(5)]. This increased prevalence appears to be accelerating. A meta-analysis conducted in 2007, suggested that the prevalence of GERD has increased by 4% per year in the Western world. In North America, the incidence increased 5% annually between 1992 and 2005[(6)].

The potential role of obesity in gastrointestinal symptoms is unclear. Reports from several health surveys show inconsistent results. Most recent studies showed a relationship between obesity and the occurrence of gastrointestinal symptoms, especially of heartburn and regurgitation [(7-13)]. This relation might be explained by the fact that obese persons experience a higher intra-abdominal pressure, slower esophageal transit and decreased acid clearance from the esophagus due to hiatus hernia compared to persons with a normal body mass index (BMI) [(14-16)].

Additionally, dyspeptic symptoms decline if overweight patients lose weight [(17-18)]. A recent meta-analysis of 20 studies reported a positive association between increasing body mass index (BMI) and the presence of GERD within the USA [(19)]. However, data are scarce on a possible relation between obesity and upper gastrointestinal pathology [(20)].

Despite many studies in the literature, the relationship between obesity and gastroesophageal sphincter incompetency (GSI) and H. pylori infection has not been fully clarified [(21-24).]

By doing esophagogastroduodenoscopy (EGD), we tried in this study to find out whether obesity has a particular impact on patients who complained from dyspepsia and who either had alarming feature(s), older than 50 years or they did not respond to initial treatment.

MATERIALS AND METHOD

This is a cross sectional study conducted at Sohar Hospital, Oman. Patients who complained from dyspepsia and referred from primary health centres to the gastro outpatient clinic at

Sohar Hospital for either persistence of symptom despite initial treatment, presence of alarming feature(s) or being more than 50 years in the period from December 2015 till February 2017 were included in the study. All patients underwent measurement of their height and weight to calculate the BMI manually by one of the investigators. All patients were subjected to abdominal ultrasonography (Philips, WavePure-HD15) by the same operator prior to EGD to exclude abnormalities which might also present with dyspepsia such as gallbladder and pancreatic diseases. Those who were eligible for EGD and were using proton pump inhibiter were asked to stop the medication at least 4 weeks before the procedure.

Definitions:

1. Dyspepsia is defined as epigastric pain or discomfort, upper abdominal fullness or feeling of indigestion or fullness[25-27].
2. Obesity was defined according to WHO classification of BMI. Patients with a BMI equal to, or more than, 30.00 kg/m^2 were defined as obese. Non-obese patients were defined as those who have BMI of 18.5-24.99 kg/m^2. To avoid overlapping, those with a BMI between 25.00 and 29.99 kg/m^2 were excluded from the study as they were considered as overweight[28].
3. Endoscopic findings

   Hiatus hernia: More than 2 cm separation of the caudally displaced esophagogastric junction and diaphragmatic impression[29].

Esophagitis: Any superficial mucosal breakdown which noticed between the gasrtoesphageal junction and up to 10cm proximally[30].

Gastritis: Erythematous mucosal lining of the stomach with or without erosions[25,26]. Gastritis was subdivided as either caused by Helicobacter pylori (H. pylori) infection, identified using Biohit HealthCare H. pylori quick test for urease enzyme detection, or gastritis with no H. pylori infection, i.e. negative urease test (UT).

Ulcer (gastric or duodenal): Disruption of the mucosal integrity leading to a local defect or excavation [25,26].

Gastroduodenitis: Erythematous mucosal lining of the stomach and the duodenum, with or without erosions, with negative UT and no bile reflux [25,26].

Bile reflux related gastritis: Erythematous mucosal lining of the stomach, with or without erosions, due to presence of bile refluxing to the stomach with negative UT [31-34].

4. Alarming features were defined as the presence of one or more of the following: anemia (in men as hemoglobin of less than 13.5 gram/100 ml and in women as hemoglobin of less

than 12.0 gram/100 ml), weight loss (defined as a loss of 4.5 kg or more body weight over a period of 6 months or more), vomiting, hematemesis and melena[27].

Esophagogastroduodenoscopy:

Apprehensive informed consent was obtained from all participants. Decision whether to sedate the patient or not depended on whether the patient was at risk of sedation, presumed tolerability and patient's preference. For sedation, we used intravenous diluted midazolam 2-5 mg. Oral Lidocaine spray (0.1%) was used for patients who underwent EGD without sedation.

Throughout the procedure, patients were monitored for their vitals and SpO2.

Using Olympus EVIS, Exera-II, CLV-180 endoscope version, EGD was done to all patients who were included in the study for gross abnormality as well as for H. pylori infection using UT. A biopsy sample was taken from gastric incisora and embedded in the agar reagent for urease detection. A positive result, indicated by significant changes in agar colour (yellow to red) around the biopsy tissue, was confirmed by the two endoscopists.

During the procedure, endoscopic finding was analysed by the two the endoscopists independently. Ideally both endoscopists concomitantly confirmed the findings at the time of the procedure. However, if only one endoscopist conducted the procedure, the finding documented by printed photos to be approved or disproved by the other endoscopist. Cases which had different diagnosis were excluded.

Patients were also analysed in respect to their gender, age, race, presence or absence of alarming feature(s) and previous testing and/or treatment for H. pylori.

Exclusion criteria:

1. Those with a BMI between 25 and 29.99 kg/m^2
2. Alcoholics
3. Active smokers
4. Those who were using medications, or suffering from illness, known to cause dyspepsia
5. Those who have gallbladder or pancreatic abnormality proved by abdominal ultrasound examination
6. Disagreement of EGD finding between the two edoscopists

Statistical analysis and results

A total of 118 patients with dyspepsia were recruited in this study. Sixty one were obese (51.8%) and 57 (48.2%) were non-obese. The mean age was 42.49 ±15.7 years, range from 17 to 81 years. Among all, 56 (47.5%) were male and 62 (52.5%) were female participants *(p* = 0.985). Overall, 91 (77.1%) were white and 27 (22.9%) were black participants.

Among all study participants, 24.6% male and 27.1% females were obese. Probability associated with the chi square statistic (p = 0.985) indicating there is no relationship between gender and obesity. Similarly, no statistically significant (p = 0.982) association was observed between whether or not H. pylori tested or treated and obesity (Table 1). Among all study participants, 40.7% white and 11% black participants were obese. On the other hand, 36.4% white and 11.9% black participants were non-obese. Study results did not reveal any association between the ethnicity and obesity (p = 0.675).

Table 1: Independent variables and Obesity - n (%)

	Non-Obese	Obese	Total	P - Value
Gender				0.985
Male	27 (22.9)	30 (24.6)	57 (47.5)	
Female	29 (25.4)	32 (27.1)	61 (52.5)	
Ethnicity				0.675
White	43 (36.4)	48 (40.7)	91 (77.1)	
Black	14 (11.9)	13 (11)	27 (22.9)	
H Pylori				0.982
Not tested	24 (20.3)	25 (8.2)	49 (41.5)	
Tested & treated	15 (12.7)	17 (66)	32 (27.1)	
Tested & not treated	18 (15.3)	19 (6.6)	37 (31.4)	
Presentation				0.815
Without alarming features	17 (14.4)	17 (14.4)	34 (28.8)	
With alarming features	40 (33.9)	44 (37.3)	84 (71.2)	

Thirty four (28.8%) dyspeptic patients did not have any alarming feature. The rest, 84 (71.2%), presented with either vomiting, anemia, weight loss, hematemesis or melena. A statistically significant difference (p < 0.001) was observed in the weight loss between obese and non-obese patients. However no significant difference (p > 0.05) was observed between obese and non-obese patients with respect to vomiting, anemia, weight loss, hematemesis and melena (Figure 1).

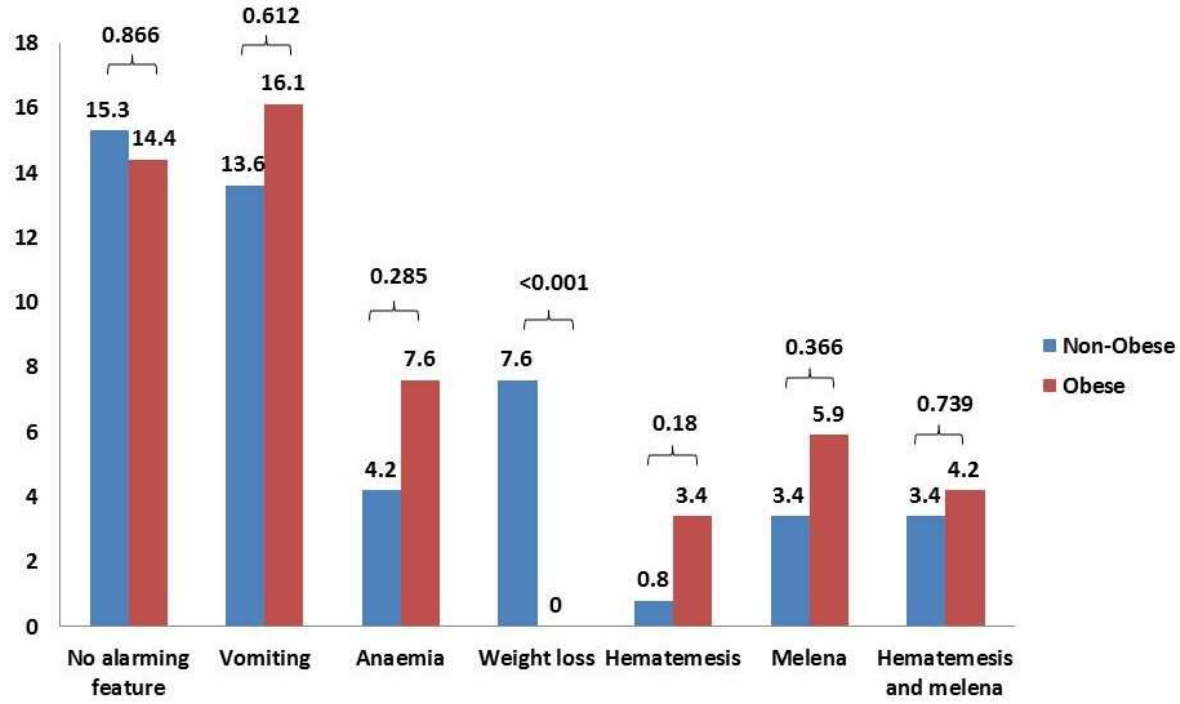

Figure 1: Alarming Features in Dyspeptic Patients

EGD showed different findings in the study patients (Table 2). Among all patients, 14 (11.9%) have normal EGD. Among those, however, only 3 (2.6%) were obese patients and 11 (9.3%) were non obese ($p < 0.033$). For obese patients, 3 (4.9%) have shown normal EGD finding versus 58 (95.1%) showed abnormal findings ($p < 0.001$). Abnormal findings include hiatus hernia with or without esophagitis, gastritis with positive or negative H. pylori testing, gastric or duodenal ulcer, gastroduodenitis and gastritis with bile reflux. A significant difference was observed among obese and non-obese patients presented with hiatus hernia with esophagitis (p <0.001), gastritis with positive H. pylori (p = 0.046), gastritis with negative H. pylori ($p = 0.021$) and gastritis due to bile reflux ($p = 0.002$).

Table 2:Endoscopic Findings - n (%)

	Non-Obese	Obese	Total	P - Value
Normal	11 (9.3)	3 (2.6)	14 (11.9)	0.033
Hiatus hernia without esophagitis	4 (3.4)	5 (4.2)	9 (7.6)	0.739
Hiatus hernia with esophagitis	1 (0.8)	17 (14.4)	18 (15.3)	<0.001
H. pylori positive gastritis	12 (10.2)	4 (3.4)	16 (13.6)	0.046
H. pylori negative gastritis	10 (8.5)	2 (1.7)	12 (10.2)	0.021
Gastric ulcer	2 (1.7)	8 (6.8)	10 (8.5)	0.058
Duodenal ulcer	8 (22.9)	4 (24.6)	12 (10.2)	0.248
Gastric and duodenal ulcers	1 (0.8)	1 (0.8)	2 (1.7)	1
Gastroduodenitis	6 (5.1)	2 (1.7)	8 (6.8)	0.157
Gastritis with bile reflux	2 (1.7)	15 (12.7)	17 (14.4)	0.002

DISCUSSION

We studied the EGD finding in obese and non-obese patients referred due to dyspepsia.

Approximately half of our patients in this study were obese, a figure which is almost similar to what has been done by Van Oijen et al[35].

Prevalence of abnormal EGD findings prior to gastric bypass surgery in obese patients, from six studies, ranged from 14 to 91% [36]. In our study, the proportion of patients (obese and non-obese) with abnormal endoscopic findings was 81.25% which was consistent with the reports in literature such as the studies by Sharaf et al. (89.7%) and Madan et al. (91%) [37,38].

Certain abnormal EGD finding (gastritis, reflux esophagitis and hiatal hernia) in our study was comparable to many literatures [21,36,39].

After exclusion of other possible causes, endoscopic gastritis associated with biliary gastroduodenal reflux was significantly higher among obese patients in our study ($p < 0.002$).

Typically, bile reflux gastropathy results from the regurgitation of bile into the stomach because of an operative stoma, an incompetent pyloric sphincter, or abnormal duodenal motility[31]. Bile salts break down the gastric mucosal barrier [40]. Gastropathy in patients with non-operated stomachs should not be attributed to exposure to bile without evidence of duodenogastric reflux. Such reflux can be demonstrated by visualization during endoscopy, bile salt analysis in gastric juice, or radionuclide scanning [31-34]. In our study we depended on EGD macroscopic feature in identifying bile reflux related gastropathy. Symptom wise, a significant proportion of obese patients in this study had dyspepsia which can only be attributed to bile reflux. Niemelä S et al has found that epigastric fullness symptoms with rising intragastric bile acid concentrations was statistically significant ($p < 0.05$) [31].

Weight loss, as a presentation, was particularly more common in obese patients ($p < 0.001$).

As many literatures pointed, such as Locke *et al.* [9] which found a significant association between reflux esophagitis and obesity ($p < 0.05$), we also notice an obvious association between obesity and esophagitis, however with much higher significance ($p < 0.001$).

Hiatus hernia with esophagitis was significantly more prevalent in the obese patients in our study ($p < 0.001$). A study by El-Serag *et al.* also showed more frequent hiatus hernia and reflux esophagitis among obese patients [7]. They suggested that obesity could be the cause of hiatus hernia, which could be followed by GERD and reflux esophagitis. The explanation of this correlation is debatable. Herbella et al. [23] reported increased lower esophageal sphincter (LES)pressure in obese persons, but Ayazi et al. [21] demonstrated that LES pressure is less pronounced in obese individuals and that LES defects increase with higher BMIs.

In our study, obesity and *H. pylori* infection rate was 3.4% which was barely statistically significant ($p = 0.046$). This is in contrary to what has been found in Ozaydin N et al which

showed a significantly higher figure (82.5 %)[41]. H. pylori prevalence in obese individuals is still disputable. Reports from different studies demonstrated that it ranged from 8.7 to 85.5% [24,42].

The variability in the prevalence of H. pylori infection in obese patient may be explained by basic variability of such infection in the general population.

CONCLUSION

Obese patients in our study showed a significantly higher abnormal EGD finding compared to non-obese. Bile reflux related gastritis represented the commonest abnormality. With these findings, EGD might be considered more frequently in obese patients for early diagnosis and treatment.

REFERENCES

1. Laffin M, Chau J, Gill RS, Birch DW, Karmali S. Sleeve gastrectomy and gastroesophageal reflux disease. J Obes 2013; 2013: 741097.
2. Guh DP, Zhang W, Bansback N, Amarsi Z, Birmingham CL, Anis AH. The incidence of co-morbidities related to obesity and overweight: a systematic review and meta-analysis. BMC Public Health 2009; 25: 88.
3. Munoz R, Ibanez L, Salinas J, et al. Importance of routine preoperative upper GI endoscopy: why all patients should be evaluated? Obes Surg 2009; 19: 427-31.
4. Ruhm CJ. "Current and future prevalence of obesity and severe obesity in the United States" (June 2007). National Bureau of Economic Research (NBER) Working Paper No. W13181
5. Dent J, El-Serag HB, Wallander MA, et al. Epidemiology of gastroesophageal reflux disease: a systematic review. Gut 2005;54:710–717. doi:10.1136/gut.2004.051821.
6. El-Serag HB. Time trends of gastroesophageal reflux disease: a systematic review. Clin Gastroenterol Hepatol 2007;5(1):17–26.
7. El-Serag HB, Graham DY, Satia JA, et al. Obesity is an independent risk factor for GERD symptoms and erosive oesophagitis. Am J astroenterol 2005;100:1243-50.
8. Murray L, Johnston B, Lane A, et al. Relationship between body mass and gastro-oesophageal reflux symptoms: The Bristol Helicobacter project. Int J Epidemiol 2003;32:645-50.
9. Locke III GR, Tally NJ, Fett SL, Zinsmeister AR, Melton LJ III. Risk factors associated with symptoms of gastroesophageal reflux. Am J Med 1999;106:642-9.
10. Lagergren J, Bergström R, Nyrén O. No relation between body mass and gastro-oesophageal reflux symptoms in a Swedish population based study. Gut 2000;47:26-9.

11. Fisher B, Pennathur A, Mutnick JLM, Little AG. Obesity correlates with gastroesophageal reflux. Dig Dis Sci 1999;44:2290-4.
12. Lundell L, Ruth M, Sandberg N, Bove-Nielsen M. Does massive obesity promote abnormal gastroesophageal reflux? Dig Dis Sci 1995;40:1632-5.
13. Crowell M, Cheskin LJ, Musial F. Prevalence of gastrointestinal symptoms in obese and normal weight binge eaters. Am J Gastroenterol 1994;89:387-91.
14. Sugerman H, Windsor A, Bessos M, Wolfe L. Intra-abdominal pressure, sagittal abdominal diameter and obesity comorbidity. J Intern Med 1997;241:71-9.
15. Mercer CD, Rue C, Hanelin L, Hill LD. Effect of obesity on esophageal transit. Am J Surg 1985;149:177-81.
16. Petersen H, Johannessen T, Sandvik AK, et al. Relationship between endoscopic hiatus hernia and gastroesophageal reflux symptoms. Scan J Gastroenterol 1991;26:921-6.
17. Fraser-Moodie CA, Norton B, Gornall C, Magnago S, Weale AR, Holmes GKT. Weight loss has an independent beneficial effect on symptoms of gastro-oesophageal reflux in patients who are overweight. Scan J Gastroenterol 1999;34:33 7-40.
18. Mathus-Vliegen EMH, Tytgat GNJ. Gastro-oesophageal reflux in obese subjects: influence of overweight, weight loss and chronic gastric balloon distension. Scan J Gastroenterol 2002;37:1246-52.
19. Corley DA, Kubo A. Body mass index and gastroesophageal reflux disease: a systematic review and meta-analysis. Am J Gastroenterol 2006;108:2619–2628.
20. Aro P, Ronkainen J, Talley NJ, et al. Body mass index and chronic unexplained gastrointestinal symptoms: an adult endoscopic population based study. Gut 2005;54:13 77-83.
21. Ayazi S, Hagen JA, Chan LS, et al. Obesity and gastroesophageal reflux: quantifying the association between body mass index, esophageal acid exposure, and lower esophageal sphincter status in a large series of patients with reflux symptoms. J Gastrointest Surg 2009; 13: 1440-7.
22. Dietz J, Ulbrich-Kulcynski JM, Souto KE, Meinhardt NG. Prevalence of upper digestive endoscopy and gastric histopathology findings in morbidly obese patients. Arq Gastroenterol 2012; 49: 52-5.
23. Herbella FA, Sweet MP, Tedesco P, Nipomnick I, Patti MG. Gastroesophageal reflux disease and obesity. Pathophysiology and implications for treatment. J Gastrointest Surg 2007; 11: 286-90.

24. Carabotti M, D'Ercole C, Iossa A, Corazziari E, Silecchia G, Severi C. Helicobacter pylori infection in obesity and its clinical outcome after bariatric surgery. World J Gastroenterol 2014; 20: 647-53.
25. Tinsley R. Harrison. Harrison's Principles of Internal Medicine (19th ed.). New York: McGraw-Hill Education. 2015.
26. **N. Franklin Adkinson. Current Medical Diagnosis & Treatment (56th ed.). New York:** McGraw-Hill Education. **2017.**
27. Nicholas J. Guidelines for the Management of Dyspepsia. Am J Gastroenterol 2005;100:2324–2337.
28. http://apps.who.int/bmi/index.jsp?introPage=intro_3.html.
29. Chandrasoma P, Makarewicz K, Wickramasinghe K, Ma Y, Demeester T. A proposal for a new validated histological definition of the gastroesophageal junction. Hum Pathol. 2006;37(1):40.
30. DeVault KR, Castell DO. Updated guidelines for the diagnosis and treatment of gastroesophageal reflux disease. The Practice Parameters Committee of the American College of Gastroenterology. *Am J Gastroenterol*. 1999 Jun. 94(6):1434-42.
31. NiemeläS. Duodenogastric reflux in patients with upper abdominal complaints or gastric ulcer with particular reference to reflux-associated gastritis. Scand J Gastroenterol Suppl. 1985;115:1.
32. Karttunen T, NiemeläS. Campylobacter pylori and duodenogastric reflux in peptic ulcer disease and gastritis. Lancet. 1988;1(8577):118.
33. Stein HJ, Smyrk TC, DeMeester TR, Rouse J. Clinical value of endoscopy and histology in the diagnosis of duodenogastric reflux disease. Hinder RA Surgery. 1992;112(4):796.
34. NiemeläS, Karttunen T, HeikkiläJ, Lehtola J. Characteristics of reflux gastritis. Scand J Gastroenterol. 1987;22(3):349.
35. M.G.H. van Oijen1, D.F.G.M. Josemanders, R.J.F. Laheij, L.G.M. van Rossum, A.C.I.T.L Tan, J.B.M.J. Jansen. Gastrointestinal disorders and symptoms: does body mass index matter?. Netherlands J Med 2006;64:45-49.
36. Peromaa-Haavisto P, Victorzon M. Is routine preoperative upper GI endoscopy needed prior to gastric bypass? Obes Surg 2013; 23: 736-9.
37. . Sharaf RN, Weinshel EH, Bini EJ, Rosenberg J, Sherman A, Ren CJ. Endoscopy plays an important preoperative role in bariatric surgery. Obes Surg 2004; 14: 1367-72.

38. Madan AK, Speck KE, Hiler ML. Routine preoperative upper endoscopy for laparoscopic gastric bypass: is it necessary? Am Surg 2004; 70: 684-6.
39. Csendes A, Burgos AM, Smok G, Beltran M. Endoscopic and histologic findings of the foregut in 426 patients with morbid obesity. Obes Surg 2007; 17: 28-34.
40. Davenport HW. Destruction of the gastric mucosal barrier by detergents and urea. Gastroenterology. 1968;54(2):175.
41. Ozaydin N, Turkyilmaz SA, Cali S. Prevalence and risk factors of Helicobacter pylori in Turkey: a nationally-representative, crosssectional, screening with the 13C-Urea breath test. BMC Public Health 2013; 13: 1215.
42. Lender N, Talley NJ, Enck P, et al. Review article: Associations between Helicobacter pylori and obesity an ecological study. Aliment Pharmacol Ther 2014; 40: 24-31.