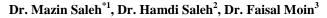
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Research Article

Open Access Journal

Diagnostic Differences between Obese and Non-Obese Patients Presented with Upper Gastro-Intestinal Symptoms



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Abstract:

Background: Obesity is associated with various diseases and conditions, particularly cardiovascular diseases, type 2 diabetes mellitus, obstructive sleep apnea, etc. The correlation between upper gastro-intestinal (GI) symptoms and obesity has yet to be completely understood in the presences of many controversial study reports.

Aim: To find the impact of obesity on patients with upper GI manifestations through doing Esophagogastroduodenoscopy (EGD).

<u>Materials and Methods</u>: A cross sectional study done at Al-Faiha General Hospital, Basra, Iraq; from January 2013 to January 2015; targeting obese patients referred for Esophagogastroduodenoscopy (EGD) clinic complained from upper gastro-intestinal symptoms (heartburn, bloating, epigastric pain, etc.) which were not responded to treatment, had alarming feature(s) or age more than 50 years. EGD was done (by two endoscopist investigators) to the included patients looking for macroscopic abnormalities, in addition to Helicobactor Pylori (H. pylori) testing using urease test (UT).

<u>Result:</u> A total of 120 patients with upper GI symptoms were included in this study. Around 51.7% were obese and 48.3% were non-obese. No statistical significances between obesity and ethnicity or gender observed in this study (p-value > 0.05). Thirty percent presented with upper GI symptoms did not have any alarming feature(s); remaining 70% were presented with vomiting, anemia, weight loss, hematemesis and/or melena. Among obese patients, only 6.5% shown normal EGD finding versus 93.5% showed abnormal findings (p < 0.001). Hiatus hernia with esophagitis, gastritis with positive or negative H. pylori and biliary gastropathy showed a statistical difference between obese and non-obese patients with p-values = <0.001, 0.046, 0.021 and 0.002 respectively. Data was analysed using SPSS version 22.

<u>Conclusion</u>: Treating physician need to consider EGD more frequently in obese patients who present with upper GI manifestations.

Keywords: Upper gastro-intestinal (GI) symptoms, Esophagogastroduodenoscopy (EGD), Obesity.

Introduction

International data indicates that the obesity epidemic is in fact a global health problem.^[1] The World Health Organization (WHO) has declared the current increase in population obesity to be an epidemic and describes obesity as one of the most blatantly visible, yet most neglected public-health problems that threaten to overwhelm both more and less developed countries.^[2]

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Department of Medicine, Oman Medical College, PO Box 391, PC 321, Al-Tareef, Suhar, Oman **Email:** <u>mazinsaleh77@yahoo.com</u> Data on overweight and obesity in Iraq is anecdotal, scarce and not representative of the community,^[3] or studying only certain variables of overweight and obesity.^[4-6]

Obesity is considered a multifactorial disease, that results from a mixture of genetic predisposition, environmental influences (e.g., sedentary lifestyle), and behavioral components (e.g., food as a reward).^[7] Being overweight and obese are well-known causes of morbidity and mortality.^[8-11]

Obesity is primary among the medical disorders composing metabolic syndrome and is associated with many

gastrointestinal diseases. Among the upper gastrointestinal diseases, obesity is an established risk factor for reflux esophagitis.^[12] As the obese population grows, so too grows the incidence of reflux esophagitis, a condition strongly linked to obesity.^[13]

Functional gastrointestinal disorders (FGIDs) such as irritable bowel syndrome (IBS) and functional dyspepsia are also extremely prevalent. Population-based data indicate that 5-10% of the US population suffers from IBS, the most common FGID.^[14,17] It is possible that FGIDs and obesity have more in common than merely high population prevalence rates. Epidemiologic data indicate that obesity is associated with a wide range of chronic gastrointestinal (GI) complaints, many of which overlap with FGIDs such as IBS or dyspepsia.^[18-23]

Although the Gastrointestinal (GI) tract is the dominant organ system associated with food intake, the relationship between GI symptoms and obesity has yet to be completely clarified. Previous studies described a greater prevalence of symptoms fulfilling criteria for irritable bowel syndrome

(IBS) and gastro-esophageal reflux disease (GERD) in morbidly obese patients compared to the general population.^[24]

Other authors showed a positive relationship between body mass index (BMI) and frequent vomiting, upper abdominal pain, bloating, and diarrhea.^[25] Fysekidis *et al.* reported a higher prevalence of functional upper and lower disorders in their obese population.^[26]

This study tries to answer the question whether obesity has an impact on patients presented with upper GI manifestations through doing Esophagogastroduodenoscopy (EGD).

Materials and Methods

We conducted this study at Al-Faiha General Hospital, Basra, Iraq. It is a cross sectional study involved Patients who complained from upper gastro-intestinal symptoms such as heartburn, epigastric pain or discomfort or indigestion and referred from primary health centres to the GI outpatient clinic at Al-Faiha Hospital for either persistence of symptoms (failure of medical therapy), presence of alarming feature(s) or those patients older than 50 years of age with upper GI manifestations in the period from January 2013 till January 2015 were included in the study. The anthropometric measurements including patients' height and weight to calculate the BMI was done by one of the investigators. Other parameters which were included: patient age, gender, race, presence or absence of alarming feature(s), previous H. pylori testing or treatment and any medication(s) the patient is currently taking like proton

pump inhibitors (PPI). Abdominal ultrasonography (Aloka, IPC-1230) by the same operator was performed to all patients prior to EGD to exclude other causes that might also predispose to upper GI symptoms such as hepatobiliary and pancreatic diseases. Those patients using proton pump inhibiter (PPI) were asked to stop the medication at least 4 weeks prior to EGD.

Definitions:

1. Functional gastrointestinal disorders (FGIDs):

Include a number of separate idiopathic which affect different parts of the gastro-intestinal tract characterised by chronic abdominal complaints without a structural or biochemical cause that could explain symptoms (e.g.: heartburn, bloating, indigestion, etc.).^[27]

2. Obesity:

Any patient with a BMI equal or more than 30 kg/m2 was defined as obese, while those with BMI range from 18.5 to 24.9 kg/m2 were classified as non-obese according to the WHO classification of obesity.^[28] Those with a BMI range from 25 and 29.9 kg/m2 (i.e. overweight) were excluded from this study.

3. Endoscopic findings:

Esophagitis: represent superficial mucosal inflammation between the gastro-esophageal junction up to 10cm proximally.^[29]

Hiatus hernia: displacement of the esophagogastric junction from the diaphragmatic impression by more than 2cm caudally.^[30]

Gastric and duodenal ulcer: represent a break in the lining of the mucosal integrity leading to a local defect. Gastroduodenitis: inflammation of the mucosal lining of the stomach and the duodenum without bile reflux or H. pylori infection.^[31,32]

Gastritis: inflammation of mucosal lining of the stomach with or without erosions.^[31,32] The gastritis sub-classified in to 2 *Categories:* Helicobacter pylori (H. pylori) infection associated gastritis and non- H. pylori infection associated gastritis using Biohit HealthCare H. pylori quick test for urease enzyme (UT).

Biliary gastritis: inflammation of the mucosal lining of the stomach due to bile refluxing to the stomach without H. pylori infection.^[33,36]

4. Alarming features: include the following red flags:

- (a) Anemia (hemoglobin less than 13.5gram/dL in male and less than 12gram/dL in female)
- (b) Weight loss (a loss of equal or more than 4.5 kg of body weight over a period of 6 months)

- (c) Vomiting
- (d) Melena and or hematemesis^[37]

Esophagogastroduodenoscopy (EGD):

For sedation, intravenous diluted midazolam 2-5 mg was given for each patient prior to EGD. Alternatively, oral Lidocaine spray (0.1%) was used for patients who underwent EGD if sedation was contra-indicated or not preferred by the patient. Consent was obtained from all patients prior to the EGD. By using Olympus STORZ 13801 NKS gastroscope, EGD was done to all patients who were included in the study as diagnostic approach as well as for H. pylori detection using urease test (UT). A biopsy specimen was taken from gastric antrum and put in the agar for urease detection (a positive UT indicated by changes in agar colour (yellow to red), the reading of the UT was done by the two endoscopists whom also responsible for given the final EGD diagnostic report separately. Any patient with discordant diagnosis was excluded. Other patients excluded from this study were smoker, alcoholic, BMI between 25 and 29.9kg/m², and those with hepatobiliary and pancreatic diseases.

Results and statistic

Data was analysed using SPSS version 22, with a P-value less than 0.05 was considered to be significant. A total of 120 patients with upper gastro-intestinal symptoms were recruited in this study. The mean age was 44.49 ± 14.7 years, range from 18 to 77 years. Around 51.7% (62 patients) were obese and 48.3% (58 patients) were non-obese. The male patients represent 47.5% (57 patients) while the female represent 52.5% (63 patients) with *p-value* = 0.985, in addition to that that no statistical relation between obesity and gender was observed in this study. Overall no statistical differences between black and white patients with *p-value* = 0.675.

Also no statistical significant relation between H. pylori testing and or treating or not and obesity with p-value = 0.982.

	Obese	Non-obese	Total	P-value
Gender				
Male	30 (25)	27 (22.5)	57 (47.5)	0.985
Female	32 (26.7)	31 (25.8)	63 (52.5)	
Ethnicity		·		
White	48 (40)	44 (36.7)	92 (76.7)	0.675
Black	14 (11.66)	14 (11.66)	28(23.3)	
H pylori				
Not tested	25 (20.83)	25 (20.83)	50 (41.7)	
Tested & treated	17 (14.16)	16 (13.33)	33 (27.5)	0.982
Tested & not treated	20 (16.7)	17 (14.1)	37 (30.8)	
Presentation				•
With alarming features	44 (36.7)	40 (33.3)	84 (70)	0.815
Without alarming features	18 (15)	18 (15)	36 (30)	

In this study 30% (36 patients) with upper gastro-intestinal symptoms did not have any alarming feature(s). The remaining 70% (84 patients) were presented with vomiting, anemia, weight loss, hematemesis and/or melena. From all

of the alarming features listed in the table-2, only weight loss showed a statistically significant difference (*p*-value = < 0.001) between obese and non-obese patients.

Table 2: Alarming Features in Patients	s with upper ga	stro-intestinal symptoms
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	Non-obese (%)	Obese (%)	P-value
Anemia	4.2	7.5	0.285
Vomiting	13.7	16	0.612
Weight loss	7.6	0	< 0.001
Melena	3.5	5.8	0.366
Hematemesis	0.8	3.4	0.18
Melena & hematemesis	3.5	4	0.739

Table-3 showed the EGD findings for patients included in this study, where 12.5% had normal EGD. Those patients with normal EGD showed some statistical difference between obese and non-obese (normal EGD significant in non-obese group) with *p*-value <0.033. Among obese patients, only 4 patients (6.5%) shown normal EGD finding versus 58 patients (93.5%) showed abnormal findings (p < 0.001).

Abnormal EGD findings were hiatus hernia with or without esophagitis, gastritis with positive or negative H. pylori testing, gastric and or duodenal ulcer, gastroduodenitis and gastritis with bile reflux. Among all of these abnormal EGD findings hiatus hernia with esophagitis, gastritis with positive or negative H. pylori and gastritis due to bile reflux showed a statistical difference between obese and non-obese patients with *p-values* = <0.001, 0.046, 0.021 and 0.002 respectively.

	Non-Obese	Obese	Total	P - Value
Normal	11 (9.2)	4 (3.3)	15 (12.5)	0.033
Hiatus hernia without esophagitis	4 (3.3)	5 (4.2)	9 (7.5)	0.739
Hiatus hernia with esophagitis	1 (0.83)	18 (15)	19 (15.8)	< 0.001
H. pylori positive gastritis	12 (10)	4 (3.3)	16 (13.3)	0.046
H. pylori negative gastritis	10 (8.3)	2 (1.7)	12 (10)	0.021
Gastric ulcer	2 (1.66)	8 (6.66)	10 (8.3)	0.058
Duodenal ulcer	8 (6.7)	4 (3.3)	12 (10)	0.248
Gastric and duodenal ulcers	1 (0.83)	1 (0.83)	2 (1.7)	1
Gastroduodenitis	6 (5)	2 (1.7)	8 (6.7)	0.157
Gastritis with bile reflux	3 (2.5)	14 (11.7)	17 (14.2)	0.002

Table 3: Endoscopic Findings - n (%)

Discussion

We studied the EGD finding in obese and non-obese patients referred due to upper gastro-intestinal symptoms.

As a comparable to other studies like Van Oijen et al.^[18] study, approximately half of the patients in this study were obese.

Literatures review showed that, among six studies, the abnormal EGD findings prior to gastric bypass surgery in obese patients ranged from 14 to 91%.^[38] In compared to our study the abnormal EGD findings in obese and non-obese patients were 87.5% which was consistent with the reports in literature such as the studies by Sharaf et al. (89.7%) and Madan *et al.* (91%).^[39,40]

Other EGD findings in our study (which were statically significant like hiatus hernia, esophagitis and gastritis), were comparable to many literatures.^[38,41,42]

Biliary gastritis was significantly higher among obese patients in our study (*p*-value < 0.002).

Bile reflux to the stomach occur because of an incompetent pyloric sphincter, or result from disturbance of duodenal motility.^[33] The presence of bile in the stomach results in disruption of the mucosal barrier.^[43] In our study the endoscopist diagnosed biliary gastritis grossly through the presence of bile in the stomach without other obvious cause of gastritis other than the presence of the bile. In our study a higher percentage of the obese patients presented with upper

bile reflux to the stomach. A similar study done by Niemelä S *et al.*,^[33] correlated the occurrence of abdominal fullness and bloating to the increase bile concentration in the stomach with *p*-*value* < 0.05. The association between obesity and hiatus hernia with

gastro-intestinal symptoms which can only be explained by

The association between obesity and hiatus hernia with esophagitis was significantly high in our study (*p*-value < 0.001), such finding also observed by Locke *et al.*^[44] study (*p*-value < 0.05) and El-Serag *et al.*^[45] study. This suggested that obesity could be the cause of hiatus hernia and esophagitis related to gastric reflux. Although such explanation has some controversy by reviewing the literatures, as the Ayazi *st al.*^[41] showed that the lower esophageal sphincter (LES) pressure in obese individual was decrease, and such finding became more prominent with increasing of the BMI. On the other hand, Herbella *et al.*^[46] showed increased lower esophageal sphincter (LES) pressure in obese persons.

In our study, the percentage of EGD findings of H. pylori positive gastritis among obese patients was 3.4%, such finding was completely differed from what found by the Ozaydin N *et al.*^[47] which showed a significantly higher figure (82.5 %). This can be explaining by the fact that H. pylori prevalence in obese individuals is still disputable, reports from different studies demonstrated that H. pylori positive gastritis among obese patients were ranged from 8.7 to 85.5%.^[48,49] In addition to that the prevalence of H. pylori infection is variable worldwide.

Conclusion

We conclude that obesity is an independent risk for abnormal EGD findings. Certain abnormalities are more prevalent in obese patients such as erosive esophagitis and biliary induced gastropathy. Subsequently, treating physician might consider EGD more frequently in obese patients who present with upper GI manifestations.

References

- Popkin BM (1998). The nutrition transition and its health implications in lower income countries. Public Health Nutr 1: 5-21.
- [2] Haslam DW, James WP (2005) Obesity. Lancet 366: 1197-1209.
- [3] Al-Tawil NG, Abdulla MM, Abdul Ameer AJ (2007). Prevalence of and factors associated with overweight and obesity among a group of Iraqi women. East Mediterr Health J 13: 420-429.
- [4] Mansour AA, Ajeel NA (2009). Parity is associated with increased waist circumference and other anthropometric indices of obesity. Eat Weight Disord 14: e50-e55.
- [5] Mansour AA, Al-Hassan AA, Al-Jazairi MI (2007). Cut-off values for waist circumference in rural Iraqi adults for the diagnosis of metabolic syndrome. Rural Remote Health 7: 765.
- [6] Mansour AA, Al-Jazairi MI (2007). Cut-off values for anthropometric variables that confer increased risk of type 2 diabetes mellitus and hypertension in Iraq. Arch Med Res 38: 253-258.
- [7] U. N. Das, "Obesity: genes, brain, gut, and environment," *Nutrition*, vol. 26, no. 5, pp. 459– 473, 2010.
- [8] G. A. Bray, "Health hazards of obesity," Endocrinology and Metabolism Clinics of North America, vol. 25, no. 4, pp. 907–919, 1996.
- [9] K. M. Flegal, B. I. Graubard, D. F. Williamson, and M.H. Gail, "Excess deaths associated with underweight, overweight, and obesity," *The Journal of the American Medical Association*, vol. 293, no. 15, pp. 1861–1867, 2005.
- [10] G.M. Reaven, "Insulin resistance: the link between obesity and cardiovascular disease," *Medical Clinics of North America*, vol. 95, no. 5, pp. 875– 892, 2011.
- [11] F. Bianchini, R. Kaaks, and H. Vainio, "Overweight, obesity, and cancer risk," *The Lancet Oncology*, vol. 3, no. 9, pp. 565–574, 2002.
- [12] F. Moki, M. Kusano, M. Mizuide et al., "Association between reflux oesophagitis and features of the metabolic syndrome in Japan," *Alimentary Pharmacology and Therapeutics*, vol. 26, no. 7, pp. 1069–1075, 2007.

- [13] N. Manabe, K. Haruma, M. Mihara et al., "The increasing incidence of reflux esophagitis during the past 20 years in Japan," *Gastroenterology*, vol. 116, no. 4, p.A244, 1999.
- [14] Saito YA, Talley NJ, Melton L, Fett S, Zinmeister AR, Locke GR. The effect of new diagnostic criteria for irritable bowel syndrome on community prevalence estimates. Neurogastroenterol Motil. 2003; 15:687-694.
- [15] Andrews EB, Eaton SC, Hollis KA, Hopkins JS, Ameen V, et al. Prevalence and demographics of irritable bowel syndrome: results from a large webbased survey. Aliment Pharmacol Ther. 2005; 22:935-942.
- [16] Hungin AP, Chang L, Locke GR, Dennis EH, Barghout V. Irritable bowel syndrome in the United States: prevalence, symptom patterns and impact. Aliment Pharmacol Ther. 2005; 21:1365-1375.
- [17] Minocha A, Johnson WD, Abell TL, Wiginton WC. Prevalence, sociodemography, and quality of life of older versus younger patients with irritable bowel syndrome: a population-based study. Dig Dis Sci. 2006; 51:446-453.
- [18] Van Oijen MG, Josemanders DF, Laheij RJ, van Rossum LG, Tan AC, Jansen JB. Gastrointestinal disorders and symptoms: does body mass index matter? Neth J Med. 2006; 64:45-49.
- [19] Delgado-Aros S, Locke GR 3rd, Camilleri M, Talley NJ, Fett S, et al. Obesity is associated with increased risk of gastrointestinal symptoms: a population-based study. Am J Gastroenterol. 2004; 99:1801-1806.
- [20] Talley NJ, Quan C, Jones MP, Horowitz M. Association of upper and lower gastrointestinal tract symptoms with body mass index in an Australian cohort. Neurogastroenterol Motil. 2004; 4:413-419.
- [21] Talley NJ, Howell S, Poulton R. Obesity and chronic gastrointestinal tract symptoms in young adults: a birth cohort study. Am J Gastroenterol. 2004; 99:1807-1814.
- [22] Svedberg P, Johansson S, Wallander MA, Hamelin B, Pedersen NL. Extra-intestinal manifestations associated with irritable bowel syndrome: a twin study. Aliment Pharmacol Ther. 2002; 16:975-983.
- [23] Aro P, Ronkainen J, Talley NJ, Storskrubb T, Bolling-Sternevald E, Agréus L. Body mass index and chronic unexplained gastrointestinal symptoms: an adult endoscopic population based study. Gut. 2005; 54:1377-1383.
- [24] R. H. Clements, Q. H. Gonzalez, A. Foster et al., "Gastrointestinal symptoms are more intense in morbidly obese patients and are improved with

laparoscopic Roux-en-Y gastric bypass," *Obesity Surgery*, vol. 13, no. 4, pp. 610–614, 2003.

- [25] S. Delgado-Aros, G. R. Locke III, M. Camilleri et al., "Obesity is associated with increased risk of gastrointestinal symptoms: a population-based study," *The American Journal of Gastroenterology*, vol. 99, no. 9, pp. 1801–1806, 2004.
- [26] M. Fysekidis, M. Bouchoucha, H. Bihan, G. Reach, R. Benamouzig, and J. Catheline, "Prevalence and co-occurrence of upper and lower functional gastrointestinal symptoms in patients eligible for bariatric surgery," *Obesity Surgery*, vol. 22, no. 3, pp. 403–410, 2012.
- [27] Drossman DA (2016). "Functional Gastrointestinal Disorders: History, Pathophysiology, Clinical Features and Rome IV". Gastroenterology. 150 (6): 1262–1279. PMID 27144617. doi:10.1053/j.gastro.2016.02.032.
- [28] B. Abed Bakhotmah, "Prevalence of Obesity among Type 2 Diabetic Patients: Non-Smokers Housewives Are the Most Affected in Jeddah, Saudi Arabia," *Open Journal of Endocrine and Metabolic Diseases*, Vol. 3 No. 1, 2013, pp. 25-30. doi: 10.4236/ojemd.2013.31004.
- [29] 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.
- [30] 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.
- [31] Tinsley R. Harrison. Harrison's Principles of Internal Medicine (19th ed.). New York: McGraw-Hill Education. 2015.
- [32] N. Franklin Adkinson. Current Medical Diagnosis
 & Treatment (56th ed.). New York: McGraw-Hill Education. 2017.
- [33] 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.
- [34] Karttunen T, NiemeläS. Campylobacter pylori and duodenogastric reflux in peptic ulcer disease and gastritis. Lancet. 1988; 1(8577):118.
- [35] 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.
- [36] NiemeläS, Karttunen T, HeikkiläJ, Lehtola J. Characteristics of reflux gastritis. Scand J Gastroenterol. 1987; 22(3):349.

- [37] Nicholas J. Guidelines for the Management of Dyspepsia. Am J Gastroenterol 2005; 100:2324-2337.
- [38] Peromaa-Haavisto P, Victorzon M. Is routine preoperative upper GI endoscopy needed prior to gastric bypass? Obes Surg 2013; 23: 736-9.
- [39] 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.
- [40] Madan AK, Speck KE, Hiler ML. Routine preoperative upper endoscopy for laparoscopic gastric bypass: is it necessary? Am Surg 2004; 70: 684-6.
- [41] 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.
- [42] 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.
- [43] Davenport HW. Destruction of the gastric mucosal barrier by detergents and urea. Gastroenterology. 1968; 54(2):175.
- [44] 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.
- [45] 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.
- [46] 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.
- [47] 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.
- [48] 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.
- [49] 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.