ORIGINAL RESEARCH

Assessment of the Clinical Profile of Dyspepsia with Predominantly Abdominal Bloating Symptoms in Type 2 Diabetes Mellitus Patients: A Cross-sectional Observational Study

Nidhi Bhutra[1], Ravi Kant[2], Itish Patnaik[3], Vandana K Dhingra[4]

Received on: 10 September 2024; *Accepted on:* 11 October 2024; *Published on:* 09 December 2024

Abstract

Aim and background: Diabetes mellitus virtually affects every organ in the body. Gastrointestinal effects include small intestinal bacterial overgrowth (SIBO), gastrointestinal reflux disease, gastroparesis, neuropathy, pancreatopathy, and non-alcoholic fatty liver disease. Our study aimed to assess and identify various causes of dyspepsia in type 2 diabetes mellitus (DM) patients.

Materials and methods: After screening the patients for inclusion and exclusion criteria, enrolled participants were subjected to a urea breath test (UBT), glucose hydrogen breath test (HBT), upper gastrointestinal endoscopy (UGIE), pancreatic fecal elastase (PEF), and gastric scintigraphy.

Results: The study revealed that 42.5% of patients had positive UBT for *H. pylori* gastritis, while 37.5% had organic causes of dyspepsia. Pancreatic exocrine insufficiency (PEI) was present in 37.5% of patients, and slow gastric emptying in 12.5% suggested gastroparesis. There was significant negative correlation between HbA1c and pancreatic fecal elastase levels. In 26.8% of patients, no cause of dyspepsia could be identified, 34.2% had dyspepsia secondary to a single etiology; and 26.8% of patients had two underlying etiologies of dyspepsia. In contrast, the remaining had multiple causes of dyspepsia.

Conclusion and clinical significance: The study identified multiple causes of dyspepsia in type 2 diabetic patients in India, with a large proportion having PEI. Further studies are needed to determine if pancreatic enzyme supplementation can alleviate dyspeptic symptoms. In conclusion, dyspepsia in diabetic patients can be attributed to multiple coexisting causes, necessitating etiology-directed management.

Keywords: Cross-sectional observational study, Dyspepsia, Gastrointestinal, HbA1c, Pancreatic exocrine insufficiency, Peptic ulcer disease, Type 2 diabetes mellitus, Small intestinal bacterial overgrowth.

Bengal Physician Journal (2024): 10.5005/jp-journals-10070-8071

Introduction

Dyspepsia is defined by the clinical guidelines of the American College of Gastroenterology and the Canadian Association of Gastroenterology as a predominant epigastric pain that lasts for at least 1 month and is accompanied by any other upper gastrointestinal (GI) symptoms, such as heartburn, epigastric fullness, nausea, or vomiting.[1] Organic dyspepsia involves identifiable pathological processes, while functional dyspepsia lacks such identifiable causes. It emphasizes the importance of endoscopy and *Helicobacter pylori* testing to exclude organic causes. Diabetic patients can have various GI complications, such as pancreatopathy, small intestinal bacterial overgrowth (SIBO), gastro-esophageal reflux disease (GERD), gastroparesis, neuropathy, and non-alcoholic fatty liver disease, which can present with symptoms like pain, nausea, diarrhea, early satiety, and constipation. These GI symptoms affect 75% of the population in diabetic outpatient clinics.[2] In type 2 DM, autonomic neuropathy and microvascular damage are key factors in exocrine insufficiency. There are various invasive and non-invasive tests for the assessment of pancreatic exocrine insufficiency (PEI). Pancreatic fecal elastase, secreted by acinar pancreatic cells can be easily detected in stool by enzyme-linked immunosorbent assay (ELISA) and is a non-invasive easy-to-perform test to assess PEI. Another cause implicated in dyspepsia in type 2 diabetic patients is SIBO, defined as the presence of excessive bacteria in the small intestine. It presents as chronic diarrhea and malabsorption. Reduced stomach acid output and small intestinal dysmotility are the two factors that make bacteria more likely to proliferate. Long-standing and poorly controlled diabetes can damage the gut nervous system, leading to disordered GI motility

[1,2]Department of General Medicine, All India Institute of Medical Sciences (AIIMS), Rishikesh, Uttarakhand, India

[3]Department of Gastroenterology, All India Institute of Medical Sciences (AIIMS), Rishikesh, Uttarakhand, India

[4]Department of Nuclear Medicine, All India Institute of Medical Sciences (AIIMS), Rishikesh, Uttarakhand, India

Corresponding Author: Ravi Kant, Department of General Medicine, All India Institute of Medical Sciences (AIIMS), Rishikesh, Uttarakhand, India, Phone: +91 9870717859, e-mail: drkantr2006@gmail.com

How to cite this article: Bhutra N, Kant R, Patnaik I, *et al*. Assessment of the Clinical Profile of Dyspepsia with Predominantly Abdominal Bloating Symptoms in Type 2 Diabetes Mellitus Patients: A Cross-sectional Observational Study. Bengal Physician Journal 2024;11(3):120–124.

Source of support: Nil

Conflict of interest: None

and SIBO.[1] Small intestinal bacterial overgrowth can be assessed by jejunal aspirate culture (gold standard) and glucose hydrogen breath test (HBT). Gastroparesis is another important cause of diabetic dyspepsia. It is defined as an objective delay in gastric emptying without any mechanical obstruction. It occurs with poor glycemic control due to dysfunction of the coordination and function of the autonomic nervous system, pacemaker cells (interstitial cells of Cajal, ICC) of the stomach and intestine, and the smooth muscle cells of the gastrointestinal tract. Gastroparesis is assessed by measuring gastric emptying by gastric scintigraphy.[2] Thus, our study aims to assess the various causes of dyspepsia in type 2 diabetic patients via upper gastrointestinal endoscopy (UGIE), gastric scintigraphy, urea breath test (UBT), glucose HBT, and pancreatic fecal elastase and to assess the correlation of diabetic control and dyspepsia.

Materials and Methods

Ethics

The study complied with the 2013 revision of The Helinski Declaration of 1975 and was approved by the Institutional Ethics Committee on December 23, 2022.

Study Design

Selection and Description of Participants

The study was a cross-sectional observational study with a 41-patient sample size. The recruitment duration was from December 2022 to September 2023. Written Informed Consent was obtained from participants for participation and use of data for research and educational purposes. Patients with type 2 diabetes mellitus presenting with dyspepsia at more than 18 years of age and less than 60 years of age were included. Patients with acute and known chronic pancreatitis, patients with type 1 DM, and pregnant females were excluded. The sample size was based on the study by Osipenko MF et al.[3] who reported the prevalence of 71% of examined patients with type 2 DM, with 42.3% due to organic GI causes, and 57.7% due to non-organic pathology. The sample size for 95% confidence level, the prevalence of 71%, and relative precision of 20% were 41.

Technical information: All the participants were explained in detail about the purpose of the study. Informed consent was obtained, and participants were subjected to the following tests: HbA1c, glucose HBT, pancreatic fecal elastase test, UGIE, gastric scintigraphy, *H. pylori* UBT, and stool routine microscopy. We used ELISA for indirect testing of pancreatic fecal elastase. Pancreatic fecal elastase activity above 200 µg/g was considered normal, while activity within the range of 100–200 µg/g was indicative of a slight PEI, and below 100 µg/g was suggestive of severe insufficiency.[4] The severity of dyspepsia was assessed by the Short Form Leeds Dyspepsia Questionnaire (SF-LDQ), which consists of four components, including indigestion, heartburn, regurgitation, and nausea. SF-LDQ scores were interpreted as follows: a score of zero indicated no dyspepsia, a score of 1–8 indicated mild dyspepsia, a score of 9–15 indicated moderate dyspepsia, and a score higher than 15 represented severe dyspepsia.

Statistics: The data were coded and recorded in the MS Excel spreadsheet program. SPSS v23 (IBM Corp.) was used for data analysis.

Results

A total of 41 participants were included in the study. The mean age of participants was 53.39 ± 7.94 years. Among the total sample, 42.5% (17) of the participants were male, rest were female. We also evaluated for associated comorbidities, 57.5% (23) of the participants had no comorbidities, 37.5% (15) of the participants had associated hypertension, while 2.5% (1) of the participants had decompensated chronic liver disease. The most predominant symptom was indigestion, seen in 63.4% (26) participants, while heartburn was present in 24.3% (10) participants, and nausea and regurgitation were present in 7.3% (3) of the participants and 4.8% (2) of the participants, respectively. The mean SF-LDQ score was 7.68 ± 2.30. The mean HbA1c (%) was 8.52 ± 1.52. HbA1c was ≤7% in 22% (9) of the participants and was >7% in the remaining. Non-parametric tests (Spearman Correlation) were used to explore the correlation between HbA1c and SF-LDQ (degree of dyspepsia) as at least one of the variables was not normally distributed. There was no statistically significant correlation between HbA1c (%) and SF-LDQ (rho = 0.18, $p = 0.247$). Baseline characteristics of the study population are shown in Table 1. The stool routine was normal in all 100% of the participants. *H. pylori* UBT was conducted to rule out *H. pylori* gastritis. Urea breath test was positive in 42.5% (17) of the participants, rest was negative. Non-parametric tests (Wilcoxon–Mann–Whitney *U*-test) were used to make group comparisons (between variable HbA1c and positive and negative UBT). There was no significant difference between the groups in terms of HbA1c (%) (W = 263.000, $p = 0.119$), as depicted in Table 2. Glucose HBT was done to rule out SIBO. Of all the participants, 100% (41) had negative HBT. Upper gastrointestinal endoscopy was done to rule out organic causes, abnormal UGIE findings were present in 36.5% (15) of the participants. Among the participants with abnormal UGIE, 19.5% (8) had antral erythema, and 7.3% (3) showed mucosal atrophy in their UGIE. Hiatus hernia was present in 4.8% (2) of the participants. Esophagitis was present in 2.4% (1) of the participants. Peptic ulcer disease was present in 2.4% (1) of the participants. Non-parametric tests (Wilcoxon–Mann–Whitney *U*-test) were used to make group comparisons between variable

Table 1: Baseline characteristics of the study population

Age (Years)	*53.39 ± 7.94*
Age	
21–30 years	1 (2.4%)
31–40 years	2 (4.9%)
41–50 years	9 (22.0%)
51–60 years	29 (70.7%)
Gender	
Male	17 (41.5%)
Female	24 (58.5%)
Comorbidities	
Nil	215 (36.6%)
HTN	5 (61.0%)
DCLD	1 (2.4%)
Predominant symptom	
Indigestion	26 (63.4%)
Heartburn	10 (24.4%)
Nausea	3 (7.3%)
Regurgitation	2 (4.9%)
SF-LDQ	7.68 ± 2.30
HbA1c (%)	8.52 ± 1.52
HbA1c ≤7%	9 (22.0%)
>7%	32 (78.0%)

Table 2: Correlation between HbA1c and urea breath test

	UBT		*Wilcoxon–Mann–Whitney U-test*	
HbA1c (%)	*Positive*	*Negative*	*W*	*p-value*
Mean (SD)	8.94 (1.60)	8.22 (1.42)	263.000	0.119
Median (IQR)	8.5 (8–9.7)	8 (7–9.2)		
Min–Max	6.8–12	6.7–11.1		

Table 3: Correlation between HbA1c and UGIE findings

	UGIE impression		*Wilcoxon–Mann–Whitney U-test*	
HbA1c (%)	*Normal*	*Abnormal*	*W*	*p-value*
Mean (SD)	8.24 (1.45)	9.00 (1.58)	134.500	0.102
Median (IQR)	8 (7.03–9)	9 (8–10.1)		
Min–Max	6.7–12	6.8–12		

Table 4: Correlation between HbA1c and gastric scintigraphy

	Gastric scintigraphy		*Wilcoxon–Mann–Whitney U-test*	
HbA1c (%)	*Rapid gastric emptying*	*Slow gastric emptying*	*W*	*p-value*
Mean (SD)	8.31 (1.50)	9.98 (0.75)	28.500	0.014
Median (IQR)	8 (7.07–9)	10 (9.8–10)		
Min–Max	6.7–12	9–11.1		

Table 5: Correlation between HbA1c and pancreatic fecal elastase

Correlation	*Spearman correlation coefficient*	*p-value*
Pancreatic fecal elastase (µg) vs HbA1c (%)	–0.5	<0.001

HbA1c and UGIE findings. There was no significant difference between the groups in terms of HbA1c (%) (W = 134.500, $p = 0.102$), as depicted in Table 3. Gastric scintigraphy was done to assess gastric motility. Rapid gastric emptying was present in 87.8% (36) of the participants, rest had slow gastric emptying. Non-parametric tests (Wilcoxon–Mann–Whitney *U*-test) were used to make group comparisons between variable HbA1c and gastric emptying. There was a significant difference between the two groups in terms of HbA1c (%) (W = 28.500, $p = 0.014$), with the median HbA1c (%) being highest in the Gastric Scintigraphy: Slow gastric emptying group, as depicted in Table 4. Pancreatic fecal elastase was done to rule out PEI. Pancreatic exocrine insufficiency was present in 36.6% (15), rest had normal pancreatic fecal elastase (PFE). 22.0% (9) of the participants had mild-to-moderate PEI. And 14.6% (6) of the participants had severe PEI. Non-parametric tests (Spearman correlation) were used to explore the correlation between HbA1c% and PFE and this correlation was statistically significant (rho = –0.51, $p = < 0.001$), as depicted in Table 5.

Our study also aimed at identifying the various combinations of causes of dyspepsia. We found that 14.6% of the participants had *H. pylori* gastritis coexisting with abnormal UGIE findings. In 9.8% of the participants, we detected multiple causes of dyspepsia, including *H. pylori* gastritis, abnormal UGIE endoscopy findings, and PEI. In 9.8% of the participants, we found slow gastric emptying and PEI as causes of dyspepsia. In 2.4% of the participants, we found *H. pylori* gastritis, abnormal UGIE findings, slow gastric emptying, and PEI as causes of dyspepsia. About 2.4% of the participants had *H. pylori* gastritis along with PEI. In 26.8% of patients, no cause of dyspepsia could be identified, 34.2% had dyspepsia secondary to a single etiology; 26.8% of patients had two underlying etiologies of dyspepsia, while the remaining had multiple causes of dyspepsia.

Discussion

The study explores the impact of diabetes on the gastrointestinal tract, particularly focusing on diabetic enteropathy and dyspepsia. It highlights that dyspepsia in diabetic individuals may result from various factors, including gastroparesis, GERD, SIBO, PEI, and diabetic neuropathy. A systematic approach is crucial for managing dyspepsia in diabetes to address both organic and inorganic causes, aiming for symptom relief, medication adherence, and improved glycemic control. Our study utilized UGIE, UBT, glucose HBT, PFE test, and gastric scintigraphy to assess common causes of dyspepsia. 42.5% of patients had positive *H. Pylori* UBT suggestive of *H. pylori* gastritis. Comparative studies showed a significant association between *H. pylori* infection and diabetes. Mabeku et al.'s cross-sectional study in Cameroon found *H. pylori* infection prevalence was 73.11% in type 2 diabetic dyspeptic patients and 58.03% in non-diabetic dyspeptic patients ($p = 0.0279$).[5] Bener et al. discovered significantly higher *H. pylori* antibody titers in type 2 diabetics (IgA > 250: 50.7%, IgG > 300: 73.5%) compared with controls (IgA: 38.2%, IgG: 61.8%), ($p < 0.001$).[6] Quatrini et al. found *H. pylori* in 69% of diabetic and 46% of non-diabetic dyspeptic subjects, with 77% of symptomatic diabetics infected.[7] These findings underscore the need to consider *H. pylori* infection as a potential cause of dyspepsia in diabetic patients for improved gastrointestinal symptom management. We ruled out the organic pathology of dyspepsia in type 2 diabetic patients by screening UGIE. Our results showed that 37.5% of patients had abnormal UGIE. According to a study conducted by Osipenko MF et al., 42.3% of type 2 diabetic patients experience dyspepsia due to organic gastrointestinal diseases.[3] Bharucha Adil E et al. in their study found that erosive esophagitis is more prevalent in type 2 diabetics with peripheral neuropathy. Hence, our study and other comparative studies showed that UGIE should be routinely undertaken in type 2 diabetic patients presenting with dyspepsia to rule out organic pathology. Small intestinal bacterial overgrowth in type 2 diabetes presents with malnutrition, diarrhea, and abdominal distension. Diagnosis involves the glucose HBT. In a meta-analysis conducted by Xing Feng and Li X-Q, the prevalence of SIBO in diabetes was found to be 29%.[8] Rana SV et al.'s study found SIBO in 14.8% of type 2 diabetic patients, significantly delaying orocecal transit time.[9] Additionally, lactose intolerance was higher in diabetic patients, suggesting a link between delayed transit time, SIBO, and malabsorption in diabetes. In our study, we used glucose HBT to rule out SIBO, which has sensitivity and specificity of 62% and 83% compared with gold standard jejunal aspirate culture.[4]

In our study, all the patients tested negative, however, SIBO cannot be ruled out completely due to the low sensitivity of the test. We need larger studies and tests like jejunal aspirate culture to assess the prevalence of SIBO in diabetes. Autonomic neuropathy and microvascular damage in diabetes contribute to PEI. We assessed PEI by pancreatic fecal elastase, which showed 37.5% of type 2 diabetic patients with dyspepsia had PEI, defined as PFE <200 µg/g of stool. Studies by Shashank R Joshi et al., Hardt et al., and Shivprasad et al. reported a PEI prevalence of 22.9–31.4% in both

type 1 and type 2 DM.[10,11] Significant correlations exist between fecal elastase concentrations and blood glucose levels. These findings underscore the high prevalence of PEI in diabetic patients, particularly those with dyspepsia. Larger multicentric studies are needed to ascertain PEI prevalence in type 2 diabetic dyspeptic patients in India. We also need studies which explore the role of pancreatic enzyme replacement therapy in alleviating dyspepsia in these patients. Another important and troublesome cause of dyspepsia in diabetes is diabetic gastroparesis. Gastroparesis results from gastric motor dysfunction secondary to autonomic neuropathy and damage to the interstitial cells of Cajal. We assessed the gut motility by gastric scintigraphy, which showed that 12.5% of our type 2 diabetic dyspeptic patients had delayed gastric emptying suggestive of diabetic gastroparesis. Almogbel Rakan A et al. found a 10.8% prevalence of gastroparesis symptoms in type 2 diabetics, correlating with HbA1c and duration of diabetes.[12] Horowitz M et al.'s study showed slower gastric emptying in diabetic patients compared with controls. Choung RS et al.'s cohort study found a 5.2% risk of gastroparesis in type 1 diabetics and 1.0% in type 2 diabetics over 10 years.[13] The results of our study were close to the results of the study by Almogbel Rakan A et al., however, we need larger multicentric Indian studies to validate our results and assess the exact prevalence of gastroparesis in type 2 diabetic patients with dyspepsia. We also tried to identify the correlation between the severity of dyspepsia and glycemic control. The severity of dyspepsia was assessed by SF-LDQ, and glycemic control was assessed via HbA1c. Non-parametric tests (Spearman correlation) were used to explore the correlation between them, however, no statistically significant correlation between HbA1c (%) and SF-LDQ Score (rho = 0.18, $p = 0.247$) was found. Thus, the severity of dyspepsia could not be related to glycemic control in our study, but we need larger Indian studies to explore the relationship between glycemic control and the severity of dyspepsia. Diabetes affects the entire gastrointestinal tract; hence, various causes of dyspepsia, including gastric causes like *H. pylori* infection, delayed gastric emptying, and small intestinal causes like SIBO can coexist, our study aimed at identifying the various combinations of causes of dyspepsia. In our study, we found that 26.8% of patients had no identifiable cause of dyspepsia, while 34.2% had dyspepsia from a single cause, and 26.8% had two underlying causes. We investigated similar studies. Huang Ju in their study found a significantly higher *H. pylori* infection rate in diabetic gastroparesis patients compared with other groups.[14] Reddymasu Savio C and McCallum discovered a 60% prevalence of SIBO in gastroparesis patients.[15] El Kurdi B et al.'s meta-analysis revealed a 38.6% SIBO prevalence in chronic pancreatitis patients, with diabetes mellitus and PEI increasing SIBO risk.[16] These findings suggest dyspepsia in diabetes arises from diverse causes, potentially exacerbating each other, necessitating comprehensive management approaches.

Conclusion

Our study assessed the various causes of dyspepsia in type 2 diabetic patients by employing investigations like a UBT, glucose HBT, upper GI endoscopy, PFE, and gastric scintigraphy. It showed that a large proportion of patients had multiple causes of dyspepsia coexisting; hence, systematic evaluation of various causes should be done and etiology-directed management should be undertaken. The strengths of our study lie in the comprehensive approach we took by assessing multiple causes of dyspepsia simultaneously in type 2 diabetic patients. However, it is crucial to recognize the limitations of our study. Firstly, our study was conducted in a single center, which may introduce bias and limit the generalizability of our findings. The inclusion of a larger sample size and multicenter studies would strengthen the external validity of our results. Additionally, some of the tests we used because of their simplicity like the glucose HBT are not gold standard and hence are not 100% sensitive and specific.

Clinical Significance

Our study showed that dyspepsia in diabetes cannot be overlooked, it should be systematically evaluated and treated. Our study showed that a large proportion of patients also have exocrine pancreatic insufficiency, thus, we need further studies on therapeutic use of pancreatic enzyme supplementation in dyspeptic diabetic patients.

Orcid

Ravi Kant https://orcid.org/0000-0003-1144-4478
Itish Patnaik https://orcid.org/0000-0003-2726-3516
Vandana K Dhingra https://orcid.org/0000-0002-3006-8597

References

1. Kadaj-Lipka R, Rydzewska G. Exocrine pancreatic insufficiency and causes of abdominal symptoms in diabetes patients. Gastroenterol Rev/Przegląd Gastroenterol 2020;15(4):289–293. DOI: 10.5114/pg.2020.101556.
2. Meldgaard T, Keller J, Olesen AE, et al. Pathophysiology and management of diabetic gastroenteropathy. Therap Adv Gastroenterol 2019;12:1756284819852047. DOI: 10.1177/1756284819852047.
3. Osipenko MF, Zhuk EA, Medvedeva OV. Clinical characteristics of dyspepsia in patients with diabetes mellitus type 2. Ter Arkh 2013;85(2):43–47.
4. Corazza GR, Strocchi A, Gasbarrini G. Fasting breath hydrogen in celiac disease. Gastroenterology 1987;93(1):53–58. DOI: 10.1016/0016-5085(87)90313-1.
5. Kouitcheu Mabeku LB, Noundjeu Ngamga ML, et al. Helicobacter pylori infection, a risk factor for type 2 diabetes mellitus: A hospital-based cross-sectional study among dyspeptic 124 patients in Douala-Cameroon. Sci Rep 2020;10(1):1–11. DOI: 10.1038/s41598-020-69208-3.
6. Bener A, Ağan A, Al-human AAA, et al. Prevalence of Helicobacter pylori infection among type 2 diabetes mellitus. Adv Biomed Res 2020;9(1):27. DOI: 10.4103/abr.abr_248_19.
7. Quatrini M, Boarino V, Ghidoni A, et al. Helicobacter pylori prevalence in patients with diabetes and its relationship to dyspeptic symptoms. J Clin Gastroenterol 2001;32(3):215. DOI: 10.1097/00004836-200103000-00006.
8. Feng X, Li X-Q. The prevalence of small intestinal bacterial overgrowth in diabetes mellitus: A systematic review and meta-analysis. Aging (Albany NY) 2022;14(2):975–988. DOI: 10.18632/aging.203854.
9. Rana SV, Malik A, Bhadada SK, et al. Malabsorption, orocecal transit time and small intestinal bacterial overgrowth in type 2 diabetic patients: A connection. Indian J Clin Biochem 2017;32(1):84–89. DOI: 10.1007/s12291-016-0569-6.
10. Joshi SR, Chowdhury S, Dharmalingam M, et al. Prevalence of pancreatic exocrine insufficiency among patients with diabetes mellitus in India. J Assoc Physicians India 2023;71(7):11–12. DOI: 10.59556/japi.71.0316.
11. Hardt PD, Hauenschild A, Nalop J, et al. High prevalence of exocrine pancreatic insufficiency in diabetes mellitus. A multicenter study screening fecal elastase 1 concentrations in 1,021 diabetic patients. Pancreatology 2003;3(5):395–402. DOI: 10.1159/000073655.

12. Almogbel RA, Alhussan FA, Alnasser SA, et al. Prevalence and risk factors of gastroparesis-related symptoms among patients with type 2 diabetes. Int J Health Sci 2016;10(3):397–404. PMID: 27610063.
13. Choung RS, Locke RG, Schleck CD, et al. Risk of gastroparesis in subjects with type 1 and 2 diabetes in the general population. Am J Gastroenterol 2012;107(1):82–88. DOI: 10.1038/ajg.2011.310.
14. Huang J. Analysis of the relationship between Helicobacter pylori infection and diabetic gastroparesis. Chin Med J (Engl). 2017;130(22):2680–5. 29. 2680–2685. DOI: 10.4103/0366-6999.218012.
15. Reddymasu SC, McCallum RW. Small intestinal bacterial overgrowth in gastroparesis: Are there any predictors? J Clin Gastroenterol. 2010;44(1): e8–e13. 1163–1171. DOI: 10.14309/ajg.0000000000000200.
16. El Kurdi B, Babar S, El Iskandarani M, et al. Factors that affect the prevalence of small intestinal bacterial overgrowth in chronic pancreatitis: A systematic review, meta-analysis, and meta-regression. Clin Transl Gastroenterol. 2019;10(9):e00072. DOI: 10.14309/ctg.0000000000000072.