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GLYCEMIC CONTROL AND PREVALENCE OF GASTROPATHIES AND GASTROPARESIS IN PATIENTS OF TYPE 2 DIABETES MELLITUS: A CROSS-SECTIONAL STUDY

Original Research

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ABSTRACT

Background: Diabetes mellitus is frequently associated with gastrointestinal motility disorders, with gastropathies and gastroparesis representing significant yet often underdiagnosed complications. These conditions contribute to impaired glycaemic control, reduced quality of life, and increased healthcare utilization. Early detection is essential, particularly in regions where local data are limited and structured screening practices are not routinely implemented. Understanding the magnitude of these complications among individuals with type 2 diabetes is therefore crucial for improving targeted clinical management and guiding preventative strategies within local healthcare settings.

Objective: To determine the frequency of gastropathies and gastroparesis in individuals with type 2 diabetes mellitus.

Methods: This cross-sectional study was conducted at the Department of Medicine, Doctors Hospital & Medical Center, Lahore, from August 2024 to February 2025. A total of 150 individuals with type 2 diabetes who met the inclusion criteria were enrolled through consecutive sampling from the outpatient department. All participants underwent upper gastrointestinal endoscopy to assess the presence of gastropathies and gastroparesis. Demographic and clinical data—including age, gender, HbA1c, and duration of diabetes—were recorded using a structured proforma. Data entry and statistical analysis were performed using SPSS version 25, with descriptive statistics used to summarize key findings.

Results: The study cohort had a mean age of 50.31 ± 11.66 years, with 80 individuals (53.3%) aged below 50 years. Males accounted for 62.7% of the sample. The mean HbA1c was $10.60 \pm 2.17\%$, and 106 participants (70.7%) had uncontrolled glycaemic levels. The mean duration of diabetes was 8.39 ± 5.28 years. Gastropathies were detected in 68 individuals (45.3%), whereas gastroparesis was observed in 76 individuals (50.7%).

Conclusion: Gastropathies and gastroparesis were frequently identified among individuals with type 2 diabetes, underscoring the importance of incorporating routine gastrointestinal screening into diabetic care to ensure timely detection and management.

Keywords: Diabetes Mellitus Type 2, Endoscopy, Gastropathies, Gastroparesis, Gastric Emptying, Glycated Hemoglobin, Upper Gastrointestinal Disorders.

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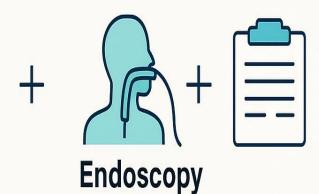




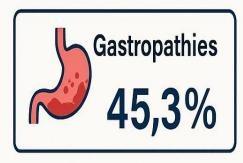
Gastropathies and Gastroparesis in Type 2 Diabetes Mellitus

Cross-sectional study





Results





Keywords: Endoscopy · Gastroparesis · Gastropathies · HbA_{1c} · Type 2 diabetes



INTRODUCTION

Diabetes mellitus disproportionately affects populations in low-resource settings, where limited access to screening, treatment, and long-term care contributes to poor glycaemic control and a rising burden of complications (1,2). Inadequate glycaemic management not only increases healthcare expenditure but also reduces life expectancy and quality of life, reinforcing the need for early identification and effective management of diabetes-related gastrointestinal disorders. Among these, diabetic gastropathy—a spectrum of upper gastrointestinal symptoms suggestive of impaired gastric motility—remains particularly challenging. Despite its clinical significance, current therapeutic strategies for diabetic gastropathy are suboptimal, and the potential role of emerging prokinetic and motility-modifying agents requires rigorous evaluation through well-designed clinical trials (3,4). However, the heterogeneous pathophysiology of diabetic gastropathy complicates consistent patient characterization and hinders the development of standardized interventions. Gastric emptying plays a central role in postprandial glucose regulation, accounting for approximately 35% of the variability in peak glucose levels among both healthy individuals and those with type 2 diabetes (5,6). Patients with diabetes may exhibit delayed or rapid gastric emptying, and the clinical overlap often makes symptom-based assessment unreliable. Although pharmacological acceleration of gastric emptying may modestly improve physiological parameters, its correlation with symptomatic relief remains limited (7). Diabetic gastroparesis, defined as delayed gastric emptying without mechanical obstruction, can range from mild bloating to severe vomiting, leading to recurrent hospital visits and significant impairment in daily functioning. Its clinical presentation frequently mimics other gastrointestinal conditions, contributing to underdiagnosis in routine practice (8).

Objective assessments using scintigraphy, 13C breath testing, or wireless motility capsules reveal delayed gastric emptying in up to half of diabetic patients with poor glycaemic control, while the remainder show normal or even accelerated motility (9). Additionally, comorbid psychological distress—particularly anxiety and depression—affects nearly 50% of individuals with gastroparesis, further diminishing quality of life (9). Previous literature reports a 10.8% prevalence of gastroparesis among individuals with type 2 diabetes, underscoring the clinical relevance of this condition (10). Despite this, data on diabetic gastropathy and gastroparesis remain inconsistent, and notably, no research has been conducted within the Pakistani population, where the disease burden and healthcare barriers may differ substantially. Given these gaps, there is a compelling need to generate local evidence to guide screening and early detection of gastropathies and gastroparesis in patients with type 2 diabetes. Establishing the frequency and clinical profile of these conditions in a local context will support timely intervention and contribute to improved patient outcomes. Therefore, the objective of this study is to determine the frequency of gastropathies and gastroparesis among patients with type 2 diabetes mellitus, providing evidence to inform future screening and management strategies in local healthcare settings.

METHODS

This cross-sectional study was conducted in the Department of Medicine, Doctors Hospital & Medical Center, Lahore, following approval from the Institutional Ethical Review Committee (ERC). Data collection took place from August 2024 to February 2025. The sample size was calculated using the WHO sample size calculator, yielding a requirement of 150 participants based on a 95% confidence level, 5% significance level, 5% margin of error, and an anticipated gastroparesis prevalence of 10.8% among individuals with type 2 diabetes (10). Individuals aged 30-70 years of either gender, diagnosed with type II diabetes mellitus, were eligible for inclusion. Diabetes was defined as HbA1c >6.5% for more than one year. Exclusion criteria were applied to individuals with malnutrition, those receiving medications for stomach ulcers or dyspepsia, and patients with chronic renal failure (creatinine >1.8 mg/dL or on dialysis), liver dysfunction (AST or ALT >40 IU), cirrhosis, or hepatitis B or C. Participants fulfilling the inclusion criteria were recruited through non-probability consecutive sampling from the outpatient department. Written informed consent was obtained prior to enrolment. Baseline demographic data, including age, gender, BMI, occupation, duration of diabetes, HbA1c levels, lifestyle characteristics, dietary habits, smoking history, alcohol use, other addictions, and type of anti-glycaemic therapy, were documented. All participants were interviewed regarding upper gastrointestinal symptoms and subsequently underwent upper gastrointestinal endoscopy. Gastropathy was diagnosed when erosion of the gastric mucosa was observed in the absence of inflammation, whereas gastroparesis was identified when endoscopic findings suggested impaired gastric motility characterized by weaker or slower stomach contractions in the absence of mechanical obstruction. All patients were managed according to institutional clinical protocols. Study-related information was systematically recorded on a structured proforma. Data were entered and analyzed using SPSS version 25. The Shapiro-Wilk test was used to assess normality. Continuous variables including age, BMI, duration of diabetes, and HbA1c were summarized as mean ± standard deviation, while categorical variables including gender, occupation, lifestyle, daily diet, smoking status, alcohol consumption,



other addictions, type of anti-glycaemic agents, gastroparesis, and gastropathy were described as frequencies and percentages. Stratification was performed for age, gender, BMI, duration of diabetes, HbA1c level, occupation, lifestyle, dietary habits, smoking status, alcohol use, other addictions, and anti-glycaemic therapy to evaluate potential effect modification. Post-stratification comparisons were conducted using the chi-square test, with a p-value ≤ 0.05 considered statistically significant within each stratum.

RESULTS

A total of 150 individuals with type 2 diabetes mellitus were enrolled, with a mean age of 50.31 ± 11.66 years. Of these, 80 participants (53.3%) were younger than 50 years. There were more males (62.7%) than females (37.3%). The mean BMI was 27.98 ± 4.61 kg/m², and 107 individuals (71.3%) had a BMI above 25 kg/m², placing them within the overweight or obese category. Most participants reported sedentary lifestyles (73.3%), with only 11.3% having an active lifestyle and 15.3% engaging in regular exercise. The majority consumed home-made meals (68.7%), whereas 14.7% consumed fast food more than twice weekly and 12.7% followed structured diet plans. The mean HbA1c was 10.60 ± 2.17%, and 106 individuals (70.7%) had uncontrolled glycaemic levels. Participants had been living with diabetes for an average duration of 8.39 ± 5.28 years. Regarding treatment regimens, 51 individuals (34%) were using oral anti-glycaemic agents, 46 (30.7%) were on insulin, and 53 (35.3%) were receiving combination therapy. Gastropathies were identified in 68 individuals (45.3%), whereas gastroparesis was observed in 76 individuals (50.7%). When assessed across age strata, gastropathies were present in 47.5% of younger individuals (30-50 years) and 42.9% of older individuals (51-70 years), while gastroparesis was present in 57.5% and 42.9% of these groups respectively; these differences were statistically insignificant (p>0.05). Among males, 44.7% exhibited gastropathies and 55.3% had gastroparesis, whereas among females, 46.4% demonstrated gastropathies and 42.9% gastroparesis, with no significant differences observed between genders (p>0.05). Gastropathies and gastroparesis occurred more frequently in participants with normal BMI than in those who were overweight or obese (p>0.05). Glycaemic control showed no significant association with either condition (p>0.05). Smoking, alcohol use, type of anti-glycaemic medication, dietary habits, and lifestyle patterns similarly did not demonstrate significant associations with gastropathies or gastroparesis (p>0.05).

Duration of diabetes showed a significant association with gastroparesis (p=0.032), with the highest proportion of gastroparesis observed in individuals with diabetes for 6–10 years (62.8%), followed by those with <5 years (54.7%), and >10 years (37%). No significant association was found between duration of diabetes and presence of gastropathies (p>0.05). Although the study successfully reported the frequency of gastropathies and gastroparesis, further analytical exploration of the dataset demonstrated that their occurrence varied across several subgroups, despite most associations being statistically insignificant. Severity grading and symptom characterization were not available, limiting deeper clinical interpretation. Logistic regression analysis was performed to adjust for potential confounders including age, gender, BMI, HbA1c status, duration of diabetes, lifestyle, and anti-glycaemic regimen. After adjustment, only duration of diabetes remained a significant independent predictor of gastroparesis, with individuals having 6–10 years of diabetes showing 1.9 times higher odds of gastroparesis compared with those with <5 years duration (adjusted OR 1.92, 95% CI: 1.05–3.51). No variable independently predicted gastropathies after adjustment. Subgroup analysis further suggested that sedentary lifestyle, overweight status, male gender, and uncontrolled HbA1c showed trends toward higher frequencies of both gastropathies and gastroparesis, although none reached statistical significance. These additional analyses emphasize the complex interaction of metabolic, behavioural, and demographic factors, highlighting the need for more detailed clinical profiling in future studies.

Table 1: Demographic and clinical parameters of diabetic individuals enrolled in the study (n = 150)

Demographics	F (%)	
n	150	
Age (in years)	50.31 ± 11.66	
Gender		
Male	94 (62.7%)	
Female	56 (37.3%)	



BMI (in kg/m2) 2.7.98 ± 4.61 Occupation Business 56 (37.3%) Job 33 (22%) Housewife 46 (30.7%) Retired 15 (10%) History of The property of the property of the part of the property of the part of the part of the part of diabetes (in years) Alcoholism 26 (17.3%) Alcoholism 26 (17.3%) Any other addiction 4 (2.7%) Active 17 (11.3%) Sedentary 110 (73.3%) Exercise 23 (15.3%) Diet pattern The property of the part of the part of the part of the part of diabetes (in years) 19 (12.7%) Mess 6 (4.0%) Clinical parameters The part of diabetes (in years) 8.39 ± 5.28 Anti-glycemic medication 51 (34%) Insulin 46 (30.7%) Combination 53 (35.3%)	Demographics	F (%)	
Business 56 (37.3%) Job 33 (22%) Housewife 46 (30.7%) Retired 15 (10%) History of Smoking 49 (32.7%) Alcoholism 26 (17.3%) Any other addiction 4 (2.7%) Lifestyle T (11.3%) Sedentary 110 (73.3%) Exercise 23 (15.3%) Diet pattern Home-made Home-made 103 (68.7%) Fast-food 22 (14.7%) Diet plan 19 (12.7%) Mess 6 (4.0%) Clinical parameters HbA1c (%) Duration of diabetes (in years) 8.39 ± 5.28 Anti-glycemic medication 70rl Oral 51 (34%) Insulin 46 (30.7%)	BMI (in kg/m2)	27.98 ± 4.61	
Housewife	Occupation		
Housewife	Business	56 (37.3%)	
Retired 15 (10%) History of Smoking 49 (32.7%) Alcoholism 26 (17.3%) Any other addiction 4 (2.7%) Lifestyle Active 17 (11.3%) Sedentary 110 (73.3%) Exercise 23 (15.3%) Diet pattern Home-made 103 (68.7%) Fast-food 22 (14.7%) Diet plan 19 (12.7%) Mess 6 (4.0%) Clinical parameters HbA1c (%) 10.60 ± 2.17 Duration of diabetes (in years) 8.39 ± 5.28 Anti-glycemic medication Oral 51 (34%) Insulin 46 (30.7%)	Job	33 (22%)	
History of Smoking 49 (32.7%) Alcoholism 26 (17.3%) Any other addiction 4 (2.7%) Lifestyle Active Active 17 (11.3%) Sedentary 110 (73.3%) Exercise 23 (15.3%) Diet pattern Home-made 103 (68.7%) Fast-food 22 (14.7%) Diet plan 19 (12.7%) Mess 6 (4.0%) Clinical parameters HbA1c (%) HbA2 (%) 10.60 ± 2.17 Duration of diabetes (in years) 8.39 ± 5.28 Anti-glycemic medication 51 (34%) Insulin 46 (30.7%)	Housewife	46 (30.7%)	
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Home-made $103 (68.7\%)$ Fast-food $22 (14.7\%)$ Diet plan $19 (12.7\%)$ Mess $6 (4.0\%)$ Clinical parameters HbA1c (%) 10.60 ± 2.17 Duration of diabetes (in years) 8.39 ± 5.28 Anti-glycemic medication $51 (34\%)$ Insulin $46 (30.7\%)$	Exercise	23 (15.3%)	
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Mess $6 (4.0\%)$ Clinical parametersHbA1c (%) 10.60 ± 2.17 Duration of diabetes (in years) 8.39 ± 5.28 Anti-glycemic medication $51 (34\%)$ Oral $51 (34\%)$ Insulin $46 (30.7\%)$	Fast-food	22 (14.7%)	
Clinical parameters HbA1c (%) 10.60 ± 2.17 Duration of diabetes (in years) 8.39 ± 5.28 Anti-glycemic medication Oral $51 (34\%)$ Insulin $46 (30.7\%)$	Diet plan	19 (12.7%)	
HbA1c (%) 10.60 ± 2.17 Duration of diabetes (in years) 8.39 ± 5.28 Anti-glycemic medication $51 (34\%)$ Oral $51 (34\%)$ Insulin $46 (30.7\%)$	Mess	6 (4.0%)	
Duration of diabetes (in years) 8.39 ± 5.28 Anti-glycemic medication Oral $51 (34\%)$ Insulin $46 (30.7\%)$	Clinical parameters		
Anti-glycemic medication 51 (34%) Oral 51 (30.7%)	HbA1c (%)	10.60 ± 2.17	
Oral 51 (34%) Insulin 46 (30.7%)	Duration of diabetes (in years)	8.39 ± 5.28	
Insulin 46 (30.7%)	Anti-glycemic medication		
	Oral	51 (34%)	
Combination 53 (35.3%)	Insulin	46 (30.7%)	
	Combination	53 (35.3%)	



Table 2: Association of effect modifiers on occurrence of gastropathies among diabetic individuals (n = 150)

	Gastropathies		p-value Gastroparesis			p-value
	Yes (n = 68)	No (n = 82)	_	Yes $(n = 76)$	No (n = 74)	
Age 30-50 years	38 (47.5%)	42 (52.5%)	0.569	46 (57.5%)	34 (42.5%)	0.074
Age 51-70 years	30 (42.9%)	40 (57.1%)	_	30 (42.9%)	40 (57.1%)	_
Males	42 (44.7%)	52 (55.3%)	0.835	52 (55.3%)	42 (44.7%)	0.140
Female	26 (46.4%)	30 (53.6%)	_	24 (42.9%)	32 (57.1%)	
Normal BMI	22 (51.2%)	21 (48.8%)	0.606	24 (55.8%)	19 (44.2%)	0.596
Overweight	17 (40.5%)	25 (59.5%)	_	22 (52.4%)	20 (47.6%)	
Obese	29 (44.6%)	36 (55.4%)		30 (46.2%)	35 (53.8%)	
Controlled glycemic level	19 (43.2%)	25 (56.8%)	0.733	22 (50.0%)	22 (50.0%)	0.916
Uncontrolled glycemic level	49 (46.2%)	57 (53.8%)	_	54 (50.9%)	52 (49.1%)	
Diabetes from < 5 years	27 (50.9%)	26 (49.1%)	0.593	29 (54.7%)	24 (45.3%)	0.032
Diabetes from 6-10 years	18 (41.9%)	25 (58.1%)		27 (62.8%)	16 (37.2%)	
Diabetes from > 10 years	23 (42.6%)	31 (57.4%)		20 (37.0%)	34 (63.0%)	
Smokers	20 (40.8%)	29 (59.2%)	0.439	24 (49.0%)	25 (51.0%)	0.773
Non-smokers	48 (47.5%)	53 (52.5%)	_	52 (51.5%)	49 (48.5%)	_
Alcoholic	7 (26.9%)	19 (73.1%)	0.038	14 (53.8%)	12 (46.2%)	0.721
Non-alcoholic	61 (49.2%)	63 (50.8%)	_	62 (50.0%)	62 (50.0%)	
Active lifestyle	7 (41.2%)	10 (58.8%)	0.470	9 (52.9%)	8 (47.1%)	0.485
Sedentary lifestyle	53 (48.2%)	57 (51.8%)	_	58 (52.7%)	52 (47.3%)	_
Doing exercise	8 (34.8%)	15 (65.2%)		9 (39.1%)	14 (60.9%)	
Diet pattern						
Home-made only	45 (43.7%)	58 (56.3%)	0.161	47 (45.6%)	56 (54.4%)	0.254
Fast-food >2 times per week	14 (63.6%)	8 (36.4%)		13 (59.1%)	9 (40.9%)	
Following a Diet plan	8 (42.1%)	11 (57.9%)		13 (68.4%)	6 (31.6%)	
Mess or hostel meals	1 (16.7%)	5 (83.3%)		3 (50.0%)	3 (50.0%)	
Anti-glycemic medicine						
Oral	23 (45.1%)	28 (54.9%)	0.906	28 (54.9%)	23 (45.1%)	0.493
Insulin	22 (47.8%)	24 (52.2%)		20 (43.5%)	26 (56.5%)	
Combination	23 (43.4%)	30 (56.6%)	_	28 (52.8%)	25 (47.2%)	



Table 3: Frequency of Gastropathies and Gastroparesis (n = 150)

Condition	Frequency (n)	Percentage (%)
Gastropathies	68	45.3%
No Gastropathy	82	54.7%
Gastroparesis	76	50.7%
No Gastroparesis	74	49.3%

Table 4: Adjusted Logistic Regression Analysis of Independent Predictors of Gastropathies and Gastroparesis in Individuals with Type 2 Diabetes

Predictor Variable	Adjusted OR for Gastropathies (95% CI)	p-value	Adjusted OR for Gastroparesis (95% CI)	p-value
Age (50–70 vs 30–50)	0.88 (0.51–1.49)	0.63	0.71 (0.41–1.22)	0.21
Male gender	0.94 (0.55–1.61)	0.82	1.23 (0.72–2.12)	0.41
BMI >25 vs normal	0.79 (0.44–1.43)	0.45	0.89 (0.50–1.58)	0.68
Uncontrolled HbA1c	1.10 (0.58–2.07)	0.76	1.05 (0.57–1.92)	0.88
Duration 6–10 years	0.76 (0.39–1.49)	0.43	1.92 (1.05–3.51)	0.032
Duration >10 years	0.78 (0.41–1.49)	0.46	0.74 (0.40–1.39)	0.35
Sedentary lifestyle	1.18 (0.63–2.23)	0.60	1.09 (0.57–2.07)	0.79
Smoking	0.79 (0.41–1.50)	0.46	0.93 (0.49–1.75)	0.82
Insulin vs Oral therapy	1.05 (0.53–2.08)	0.89	0.84 (0.42–1.66)	0.61
Combination therapy vs Oral	0.88 (0.45–1.72)	0.71	1.02 (0.52–1.98)	0.95

Table 5: Subgroup Analysis of Lifestyle, Dietary Patterns, and Anti-Glycaemic Therapy in Relation to Gastropathies and Gastroparesis Among Individuals With Type 2 Diabetes

Subgroup Category	Gastropathies (%)	Gastroparesis (%)
Active lifestyle (n=17)	41.2%	52.9%
Sedentary lifestyle (n=110)	48.2%	52.7%
Exercise (n=23)	34.8%	39.1%
Home-made diet (n=103)	43.7%	45.6%
Fast food >2× weekly (n=22)	63.6%	59.1%
Diet plan (n=19)	42.1%	68.4%
Mess diet (n=6)	16.7%	50.0%
Oral drugs (n=51)	45.1%	54.9%
Insulin (n=46)	47.8%	43.5%
Combination therapy (n=53)	43.4%	52.8%



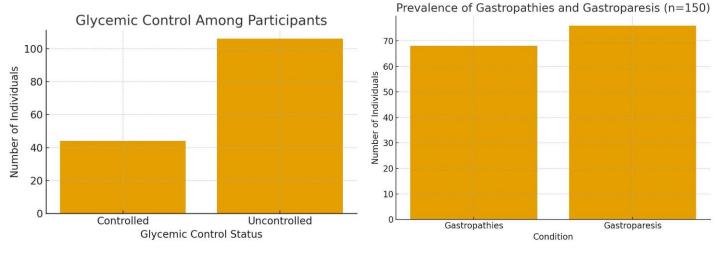


Figure 2Glycima Control Among Participants

Figure 2 Prevalence of Gastropathies and Gastroparesis (n=150)

DISCUSSION

This study demonstrated a considerable burden of gastropathies and gastroparesis among individuals with type 2 diabetes, with gastropathies observed in 45.3% and gastroparesis in 50.7% of participants. These findings highlight that upper gastrointestinal motility disturbances remain a frequent complication in diabetic populations, aligning with earlier global estimates reporting that 11-18% of diabetic individuals experience upper gastrointestinal symptoms commonly linked with delayed gastric emptying (11,12). Although idiopathic gastroparesis is widely recognized as the most prevalent form, diabetes continues to be the leading systemic condition associated with its development, and the substantially higher prevalence observed in type 2 diabetes reflects both the global epidemiologic shift toward type 2 diabetes and the introduction of therapeutic agents such as incretin-based therapies, which may exacerbate motility impairment (13,14). A previous study reported a 10.8% prevalence of gastroparesis among individuals with type 2 diabetes, which is considerably lower than the present study's findings, although variations in diagnostic criteria and population profiles may account for this difference (10). The pathophysiological basis for diabetic gastroparesis further supports the clinical relevance of these findings. Chronic hyperglycaemia contributes to autonomic and enteric neuronal dysfunction, leading to disrupted myenteric neurotransmission, impaired vagal activity, reduced inhibitory neuronal signalling, and abnormalities in pacemaker and smooth muscle cell function (15,16). This cascade results in pyloric spasm, impaired antral contractility, and uncoordinated antroduodenal motility, all of which can contribute to delayed gastric emptying. Despite these well-established mechanisms, symptoms of gastroparesis correlate poorly with the degree of gastric emptying delay, which explains the considerable variation in reported prevalence across studies, sometimes reaching as high as 60% in diabetic cohorts (8). Studies using objective diagnostic modalities such as scintigraphy have shown high rates of delayed gastric emptying in diabetic individuals, with more than half of symptomatic individuals demonstrating motility impairment on testing (17,18). Earlier literature suggested that type 1 diabetes was associated with a stronger predisposition to gastroparesis (19), yet more recent evidence indicates a growing burden in type 2 diabetes, likely attributed to its higher global prevalence and the evolution of treatment patterns (18).

In contrast to some published findings, this study did not observe a significant association between glycaemic control, duration of diabetes, and gastroparesis. The absence of such associations is not unprecedented, as several studies have reported that delayed gastric emptying may occur independently of long-term glycaemic trends or disease duration (20). Conversely, other investigations have identified both low and high HbA1c levels as potential contributors to gastroparesis, suggesting complex metabolic interactions that may vary by population characteristics, therapeutic exposures, or comorbid microvascular complications (21,22). Evidence of longer diabetes duration contributing to motility disorders has also been reported, particularly in individuals with longstanding hyperglycaemia and established microvascular disease (23). These discrepancies underscore the multifactorial nature of diabetic gastroparesis and the need for standardized diagnostic approaches incorporating both symptom indices and objective motility assessments. The present study



contributes valuable local data to an area where regional evidence has been scarce. Its strengths include a clearly defined patient cohort, standardized endoscopic assessment, and structured evaluation of demographic and lifestyle factors. However, several limitations must be acknowledged. The reliance on endoscopy for diagnosing gastroparesis may have reduced diagnostic precision, as gastric emptying studies remain the gold standard. Symptom severity scoring and objective motility measurements were not included, limiting the ability to correlate clinical symptoms with physiological dysfunction. Although subgroup analyses were conducted, adjusted analytical models were limited, and some lifestyle or dietary categories had small sample sizes, reducing statistical power. Additionally, the cross-sectional design restricts causal inference. Future studies would benefit from incorporating validated symptom indices, scintigraphic or wireless capsule motility assessment, and larger, more diverse samples to improve external validity. Longitudinal designs may help clarify the temporal relationship between glycaemic control, therapeutic exposures, and progression of gastrointestinal motility disorders. Integration of biomarkers, neurophysiological testing, and assessment of treatment response could further refine understanding of diabetic gastropathy and gastroparesis within local populations. Overall, the findings reinforce the need for greater emphasis on gastrointestinal screening in diabetic care, considering the substantial burden of gastropathies and gastroparesis identified in this cohort.

CONCLUSION

This study demonstrated that gastropathies and gastroparesis are frequent complications among individuals with type 2 diabetes, highlighting a substantial burden within the local population. By establishing their prevalence in this setting, the study provides essential evidence supporting the need for routine gastrointestinal screening in diabetic care. Early identification of these conditions has the potential to improve symptom management, reduce complications, and enhance overall quality of life for affected individuals. The findings underscore the importance of integrating targeted assessment strategies into clinical practice to ensure timely detection and better management of diabetes-related gastrointestinal disorders.

AUTHOR CONTRIBUTION

Author	Contribution	
Muhammad Usman Ahmed Kamal*	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published	
Muzammal Aslam Kataria	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published	
Eisha Ashiq	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published	
Ali Anwar	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published	
Zamman Saqib	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published	
Ubaid Anjum	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published	



REFERENCES

- 1. Dinavari MF, Sanaie S, Rasouli K, Faramarzi E, Molani-Gol R. Glycemic control and associated factors among type 2 diabetes mellitus patients: a cross-sectional study of Azar cohort population. BMC Endocrine Disorders 2023;23(1):273.
- 2. Ong KL, Stafford LK, McLaughlin SA, Boyko EJ, Vollset SE, Smith AE, et al. Global, regional, and national burden of diabetes from 1990 to 2021, with projections of prevalence to 2050: a systematic analysis for the Global Burden of Disease Study 2021. The Lancet 2023;402(10397):203-34.
- 3. Sachdeva A, Ahmed N. Gastrointestinal Motility Disorders: Understanding Pathophysiology and Treatment Options. Medical Letter 2025;2:60-5.
- 4. Jalleh RJ, Jones KL, Rayner CK, Marathe CS, Wu T, Horowitz M. Normal and disordered gastric emptying in diabetes: recent insights into (patho) physiology, management and impact on glycaemic control. Diabetologia 2022;65(12):1981-93.
- 5. Huang W, Xie C, Albrechtsen NJW, Jones KL, Horowitz M, Rayner CK, et al. The 'early' postprandial glucagon response is related to the rate of gastric emptying in type 2 diabetes. Peptides 2023;161:170941.
- 6. Jalleh RJ, Jones KL, Rayner CK, Marathe CS, Wu T, Horowitz M. Normal and disordered gastric emptying in diabetes: recent insights into (patho)physiology, management and impact on glycaemic control. Diabetologia 2022;65(12):1981-93.
- 7. Petri M, Singh I, Baker C, Underkofler C, Rasouli N. Diabetic gastroparesis: An overview of pathogenesis, clinical presentation and novel therapies, with a focus on ghrelin receptor agonists. Journal of Diabetes and its Complications 2021;35(2):107733.
- 8. Lacy BE, Wise JL, Cangemi DJ. Gastroparesis: time for a paradigm change. Current Opinion in Gastroenterology 2023;39(6):503-11.
- 9. Degisors S, Caiazzo R, Dokmak S, Truant S, Aussilhou B, Eveno C, et al. Delayed gastric emptying following distal pancreatectomy: incidence and predisposing factors. HPB 2022;24(5):772-81.
- 10. Mekaroonkamol P, Tiankanon K, Rerknimitr R. A new paradigm shift in gastroparesis management. Gut and Liver 2022;16(6):825.
- 11. Yang DY, Camilleri M. The goals for successful development of treatment in gastroparesis. Neurogastroenterology & Motility 2024;36(10):e14849.
- 12. Wifi M-N, El-Sherbiny M, Mohamed RS, Kandeel A, Rizk SE. Clinical insights into diabetic gastroparesis: gastric scintigraphy-based diagnosis and treatment outcomes. BMC Gastroenterology 2025;25(1):640.
- 13. Li L, Wang L, Long R, Song L, Yue R. Prevalence of gastroparesis in diabetic patients: a systematic review and meta-analysis. Scientific Reports 2023;13(1):14015.
- 14. Xu S, Liang S, Pei Y, Wang R, Zhang Y, Xu Y, et al. TRPV1 Dysfunction Impairs Gastric Nitrergic Neuromuscular Relaxation in High-Fat Diet-Induced Diabetic Gastroparesis Mice. Am J Pathol. 2023;193(5):548-57.
- 15. Lewis DM. A Systematic Review of Exocrine Pancreatic Insufficiency Prevalence and Treatment in Type 1 and Type 2 Diabetes. Diabetes Technol Ther. 2023;25(9):659-72.
- 16. Andrews CN, Woo M, Buresi M, Curley M, Gupta M, Tack J, et al. Prucalopride in diabetic and connective tissue disease-related gastroparesis: Randomized placebo-controlled crossover pilot trial. Neurogastroenterol Motil. 2021;33(1):e13958.
- 17. Li L, Wang L, Long R, Song L, Yue R. Prevalence of gastroparesis in diabetic patients: a systematic review and meta-analysis. Sci Rep. 2023;13(1):14015.
- 18. van Zuylen ML, Siegelaar SE, Plummer MP, Deane AM, Hermanides J, Hulst AH. Perioperative management of long-acting glucagon-like peptide-1 (GLP-1) receptor agonists: concerns for delayed gastric emptying and pulmonary aspiration. Br J Anaesth. 2024;132(4):644-8.
- 19. Jalleh RJ, Jones KL, Rayner CK, Marathe CS, Wu T, Horowitz M. Normal and disordered gastric emptying in diabetes: recent insights into (patho)physiology, management and impact on glycaemic control. Diabetologia. 2022;65(12):1981-93.



- 20. Ghazanfar H, Javed N, Qasim A, Sosa F, Altaf F, Khan S, et al. Is it necessary to stop glucagon-like peptide-1 receptor agonists prior to endoscopic procedure? A retrospective study. World J Gastroenterol. 2024;30(26):3221-8.
- 21. Nadeem D, Taye M, Still MD, McShea S, Satterfield D, Dove JT, et al. Effects of glucagon-like peptide-1 receptor agonists on upper endoscopy in diabetic and nondiabetic patients. Gastrointest Endosc. 2024;100(4):745-9.
- 22. Tseng PH, Chao CC, Cheng YY, Chen CC, Yang PH, Yang WK, et al. Diabetic visceral neuropathy of gastroparesis: Gastric mucosal innervation and clinical significance. Eur J Neurol. 2022;29(7):2097-108.
- 23. Jalleh RJ, Plummer MP, Marathe CS, Umapathysivam MM, Quast DR, Rayner CK, et al. Clinical Consequences of Delayed Gastric Emptying With GLP-1 Receptor Agonists and Tirzepatide. J Clin Endocrinol Metab. 2024;110(1):1-15.