Original Article

Impact of Various Diseases and Non-Gastrointestinal Medications on Digestive Disorders

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ABSTRACT

Background:
Digestive disorders, such as gastroesophageal reflux disease (GERD) and irritable bowel syndrome (IBS), are influenced by a complex interplay of genetic, environmental, and systemic factors. This study aimed to assess the associations between non-gastrointestinal comorbidities, medication use, and the prevalence of digestive disorders in the Azeri population of East Azerbaijan, Iran.

Materials and Methods:
A case-control design involving 186 patients with upper gastrointestinal diseases and 185 healthy controls was employed. Data on comorbidities, prescribed medications, and self-medications were collected through structured questionnaires. Statistical analyses were performed using Chi-square tests, with significance set at $P<0.05$. Additionally, confidence intervals (CIs) were calculated using the Wilson score method.

Results:
The results demonstrated significant associations between digestive disorders and hypertension, diabetes, cardiovascular diseases, and liver diseases ($P<0.05$). Allergies were inversely associated with digestive disorders, being more prevalent in the control group ($P<0.05$). Medication analysis revealed a higher frequency of prescribed antihypertensives, psychotropic drugs, and diabetes medications among patients compared with controls ($P<0.05$). Self-medication practices, however, were similar between groups.

Conclusion:
This study reveals significant associations between non-gastrointestinal comorbidities and altered risks of digestive disorders. Medication use, such as antihypertensives and psychotropic drugs, also correlated with gastrointestinal risk. These findings highlight the importance of integrated care and the need for further research to uncover underlying mechanisms and develop targeted strategies for prevention and treatment.

Keywords: Digestive disorders, Non-gastrointestinal comorbidities, Medication use

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INTRODUCTION

Digestive disorders encompass a wide range of chronic and acute conditions that affect the gastrointestinal (GI) tract, a vital system responsible for nutrient absorption, immune defense, and waste elimination. Common disorders such as gastroesophageal reflux disease (GERD), irritable bowel syndrome (IBS), and inflammatory bowel disease (IBD) are characterized by persistent and often debilitating symptoms, including abdominal pain, bloating, and altered bowel habits. These conditions significantly impact individuals' quality of life and frequently require long-term management strategies to alleviate symptoms and prevent complications (1). As the GI tract also plays a crucial role in maintaining immune balance, disruptions in GI health can have systemic consequences, underscoring the importance of studying these conditions in depth (2). Globally, digestive disorders have shown an increasing prevalence over the past few decades, suggesting a rising public health challenge. IBS is estimated to affect between 10% and 20% of the global population, with varying rates depending on geographic region and diagnostic criteria (3). GERD prevalence is reported to be around 18-28% in North America and 9-26% in Europe, with similar upward trends observed for IBD and other GI disorders in both developed and developing countries (4,5). The economic burden of these disorders is also substantial, as they account for a significant share of healthcare utilization, medication costs, and work absenteeism (6).A complex interplay of genetic, environmental, and host factors influences the risk of developing digestive disorders. Genetic predisposition is evident in conditions such as celiac disease (CD), where specific gene variations related to the immune response have been identified as potential risk factors (7). However, genetic predisposition alone does not fully explain the onset and progression of these conditions, as environmental and host factors are also critically involved. Environmental influences, including diet, lifestyle, medication use, and psychological stress, play a central role in the risk and severity of digestive disorders. For example, dietary patterns high in processed foods and low in fiber have been linked to alterations in gut microbiota composition, which is implicated in the pathogenesis of disorders like IBS and IBD (8). Also, host factors, such as non-GI conditions, are increasingly recognized in research exploring the complexities of digestive health. Studies indicate that digestive disorders frequently coexist with various systemic health conditions, including metabolic, cardiovascular, and immune-related diseases (9). The presence of such comorbidities has led to growing interest in understanding how broader health factors might contribute to the occurrence and characteristics of digestive disorders.

Medications prescribed for non-digestive conditions, such as non-steroidal anti-inflammatory drugs (NSAIDs), antihypertensives, and psychotropic drugs, are well-documented to have GI side effects. These side effects can range from mild discomfort to serious GI conditions, though the precise role of these medications in the development and progression of digestive disorders remains an area of ongoing investigation (10). Similarly, non-digestive conditions like cardiovascular disease (CVD) and metabolic disorders have been explored for their potential interactions with digestive health. The mechanisms through which these systemic health issues influence GI function and health are not yet fully elucidated (11). The exact role of other health conditions, such as neurological or respiratory diseases, in relation to digestive disorders is also not yet fully understood. Understanding these associations is critical, as it may reveal whether comorbidities or non-GI medications contribute to an increased susceptibility to digestive symptoms and conditions.This study investigated the associations between non-GI comorbidities, non-digestive medication use, and digestive disorders in the Azeri population of East Azerbaijan, Iran, to provide a more comprehensive understanding of potential influences on digestive health.

MATERIALS AND METHODS

186 patients with upper GI diseases and 185 healthy control individuals participated in this study, which was approved by the Ethics Committee of Tabriz University of Medical Sciences (IR.TBZMED.REC.1394.505). A demographic questionnaire was completed by both patients and controls, detailing information related to the presence of various diseases and the use of prescribed and non-prescribed medications. The control group had no GI diseases and had not undergone GI endoscopy.

Statistical analysis

Statistical analyses were conducted using the Chi-square test, considering a P value of less than 0.05 indicative of statistical significance. Confidence intervals (CIs) for proportions and differences in proportions were calculated using the Wilson score method with a 95% confidence level.

RESULTS

A total of 186 patients and 185 healthy controls were enrolled in the study. The sex distribution was similar between the two groups, with 102 men (54.8%) and 84 women (45.2%) in the patient group compared with 88 men (47.6%) and 97 women (52.4%) in the control group. The mean age of patients was higher than that of controls (44.06 ± 6.84 vs. 41.02 ± 7.88 years). Likewise, the mean body mass index (BMI) was greater in patients than in controls (25.53 ± 4.16 vs. 24.37 ± 3.92 kg/m^2). The demographic characteristics of patients and control subjects are presented in Table 1.

Table 1. Demographic characteristics of patients and healthy controls

Characteristics	Patients (%)	Healthy Controls (%)
Sex	-	-
Male	102 (54.8)	88 (47.6)
Female	84 (45.2)	97 (52.4)
Age (mean±SD)	44.06 ± 6.84	41.02 ± 7.88
Body mass index (kg/m2) (mean±SD)	25.53 ± 4.16	24.37 ± 3.92

Among the 186 patients with digestive disorders, 26 (15.1%) had co-occurring hypertension, 15 (8.1%) had diabetes, 16 (8.6%) had CVD, 13 (7.0%) had liver diseases, and 27 (14.5%) had anemia, all showing significantly higher frequencies compared with the control group (P<0.05). In contrast, allergies were significantly more common in the control group, with 37 individuals (19.9%) affected, compared with only four patients (2.2%) with digestive disorders (P<0.05, Table 2).

Table 2. Distribution of diseases in healthy controls and patients

Disease	Patients with gastrointestinal diseases (%)	Healthy controls (%)	P value	Differences (95% CI)
High Blood Pressure	28 (15.1)	1 (0.5)	0.0001	0.1451 (0.0927, 0.1976)
Diabetes	15 (8.1)	1 (0.5)	0.0009	0.0752 (0.0347, 0.1158)
Cardiovascular diseases	16 (8.6)	0 (0)	0.0001	0.0860 (0.0457, 0.1263)
Liver diseases	13 (7)	0 (0)	0.0007	0.0699 (0.0333, 0.1065)
Anemia	27 (14.5)	14 (7.5)	0.0489	0.0695 (0.0061, 0.1329)
Hypothyroidism	6 (3.2)	2 (1.1)	0.2871	0.0214 (-0.0080, 0.0509)
Kidney stones	3 (1.6)	0 (0)	0.2482	0.0161 (-0.0020, 0.0342)
Allergy	4 (2.2)	37 (19.9)	0.0001	-0.1785 (-0.2398, -0.1172)
Asthma	2 (1.1)	0 (0)	0.4807	0.0108 (-0.0041, 0.0256)
Lumbar disk disease	3 (1.6)	1 (0.5)	0.619	0.0107 (-0.0102, 0.0317)
Knee arthrosis	2 (1.1)	1 (0.5)	1	0.0053 (-0.0129, 0.0235)
Migraine	1 (0.5)	1 (0.5)	1	-0.0000 (-0.0149, 0.0149)
Depression	2 (1.1)	4 (2.2)	0.6758	-0.0109 (-0.0365, 0.0148)
Prostate hypertrophy	2 (1.1)	1 (0.5)	1	0.0053 (-0.0129, 0.0235)
Gallstones	6 (3.2)	0 (0)	0.0402	0.0323 (0.0069, 0.0577)

Additionally, the prescribed use of antihypertensive, psychiatric, and diabetic medications was significantly higher among patients with digestive disorders than in controls (P<0.05). Self-medication practices showed no significant difference between groups (P>0.05), though patients reported a slightly higher intake of painkillers, while controls reported more frequent use of antibiotics and multivitamins (Table 3).

Table 3. Non-gastrointestinal medications use in patients and healthy controls

Characteristic	Patients with gastrointestinal diseases (%)	Healthy controls (%)	P Value	Differences (95% CI)
Taking medicine without a doctor's prescription				
Yes	57 (30.6)	56 (30.3)	1	0.0037 (-0.0899, 0.0974)
No	129 (69.4)	129 (69.7)	1	-0.0037 (-0.0974, 0.0899)
Medication prescribed by a doctor				
Analgesic	29 (15.6)	19 (10.3)	0.17	0.0532 (-0.0148, 0.1213)
Antihypertensives	25 (13.4)	2 (1.1)	0.0001	0.1236 (0.0724, 0.1748)
Psychotropic medications	13 (7)	1 (0.5)	0.00282	0.0645 (0.0264, 0.1026)
Hypothyroid medication	5 (2.7)	1 (0.5)	0.2194	0.0215 (-0.0041, 0.0470)
Cortone	1 (0.5)	2 (1.1)	0.9963	-0.0054 (-0.0237, 0.0128)
Lipid-lowering medications	4 (2.2)	1 (0.5)	0.3711	0.0161 (-0.0073, 0.0395)
Diabetes medications	9 (4.8)	1 (0.5)	0.02539	0.0430 (0.0104, 0.0756)
Iron supplements	1 (0.5)	1 (0.5)	1	-0.0000 (-0.0149, 0.0149)
Self-medication				
Analgesic	42 (22.6)	32 (17.3)	0.2529	0.0528 (-0.0283, 0.1340)
Common cold medicines	7 (3.8)	8 (4.3)	0.9915	-0.0056 (-0.0457, 0.0345)
Antibiotics	1 (0.5)	5 (2.7)	0.2144	-0.0217 (-0.0473, 0.0040)
Antihistamines	1 (0.5)	3 (1.6)	0.6114	-0.0108 (-0.0319, 0.0102)
Iron supplements	2 (1.1)	4 (2.2)	0.6758	-0.0109 (-0.0365, 0.0148)
Multivitamins	1 (0.5)	4 (2.2)	0.3646	-0.0162 (-0.0397, 0.0072)
Fatty liver herbal medicine	3 (1.6)	0 (0)	0.2482	0.0161 (-0.0020, 0.0342)

DISCUSSION

The multifaceted impact of various factors on the risk of digestive diseases has driven global research efforts to better understand these associations. Studies in this area seek to identify predisposing factors and, where possible, propose strategies to mitigate the increasing prevalence of these disorders. This study was prompted by the observed variation in the incidence of digestive disorders among individuals with non-GI comorbidities and those using non-GI medications compared with healthy individuals. Analysis revealed significant associations between certain comorbidities, specific non-GI medications, and altered risk of developing digestive disorders.

Our findings demonstrate a significant association between high blood pressure and an increased risk of digestive disorders. Among patients with Gi conditions, 28 individuals (15.1%) reported high blood pressure, compared with only one individual (0.5%) in the healthy control group. Similar findings were observed in a study by Zhi-tong Li and colleagues (12), which investigated the relationship between hypertension and GERD. They reported that 44.2% of hypertensive patients also experienced GERD, highlighting a potential link between cardiovascular and GI health. Hypertension can influence digestive disorders

through various mechanisms.

High blood pressure contributes to microvascular damage in the GI tract, leading to ischemic events that impair oxygenation and nutrient flow to digestive tissues. This ischemia may weaken the gut lining, especially in areas with poor blood supply, leading to conditions such as ischemic colitis and exacerbating other GI disorders. It has been shown that reduced blood flow due to vascular complications from hypertension can damage the intestinal mucosa, thereby increasing the risk of GI bleeding and related conditions (13). Chronic hypertension also leads to a persistent inflammatory state, characterized by elevated levels of pro-inflammatory cytokines like interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), which can disrupt gut homeostasis. These cytokines are involved in gut inflammation and may contribute to the development or progression of IBDs, such as Crohn's disease and ulcerative colitis.

Additionally, patients with hypertension often experience higher oxidative stress, further damaging intestinal tissues and promoting inflammatory responses. The inflammatory cascade triggered by hypertension is thought to disrupt the balance of the gut microbiome, contributing to dysbiosis, where potentially harmful species outnumber beneficial gut bacteria. This imbalance can weaken the gut barrier, leading to leaky gut syndrome and allowing bacterial endotoxins to enter the bloodstream, which can cause systemic inflammation that further exacerbates GI symptoms and hypertension (14).

The data obtained in this study demonstrated a significant relationship between diabetes and digestive diseases ($P<0.05$). 15 patients with GI diseases (8.1%) had diabetes along with digestive diseases, while only one healthy individual (0.5%) reported diabetes (Table 2). A study by Huihui Sun and colleagues (15) yielded similar results. Conducted on 775 patients with type 2 diabetes mellitus, this study demonstrated that the prevalence of GERD was significantly higher among patients with diabetes compared with healthy individuals. Conversely, Sitara Nandyal and colleagues reported no significant association between diabetes and GERD (16). The observed differences between our study and theirs could be attributed to several factors, including the variation in the number of participants, genetic differences among the populations studied, differing environmental factors in the communities examined, and the lack of precise specification of the type of GI disease in our study. Diabetes mellitus exerts a substantial impact on GI function through a variety of mechanisms, particularly by impairing the autonomic nervous system and causing metabolic disturbances.

One of the key mechanisms of diabetes is the development of autonomic neuropathy, which affects the enteric nervous system and leads to impaired regulation of GI motility. This dysfunction manifests as delayed gastric emptying (gastroparesis), constipation, and bloating, common symptoms observed in patients with diabetes. The loss of coordinated neural control in the GI tract results in dysmotility, contributing to a range of GI disorders and discomfort (17). Metabolic disturbances, particularly insulin resistance and chronic hyperglycemia, also play a crucial role in the pathophysiology of digestive disorders in diabetes. Insulin resistance leads to elevated levels of free fatty acids and pro-inflammatory cytokines, which can disrupt gut microbiota composition. This disruption allows endotoxins and bacterial products to leak into the bloodstream, triggering a systemic inflammatory response. The resulting low-grade inflammatory state has been linked to the exacerbation of GI disorders such as IBS, IBD, and non-alcoholic fatty liver disease (NAFLD). These disorders are often more severe in patients with diabetes, highlighting the significant influence of metabolic dysregulation on gut health (18). The complex interplay between insulin resistance, altered gut microbiota, and systemic inflammation underscores the multifactorial nature of diabetes-related digestive dysfunction.

Our results showed a significantly higher frequency of CVD in patients with GI diseases than in healthy individuals ($P<0.05$). 16 patients (8.6%) reported CVD, while no healthy controls reported that (Table 2). A study by Pasini and others (19) reported findings consistent with our observations. They found that patients with chronic heart failure (CHF) exhibited intestinal overgrowth of pathogenic bacteria, including Candida species, along with increased intestinal permeability (IP). This heightened IP was proposed to play a role in various GI disorders.

CVD can substantially impact GI health through several pathophysiological mechanisms, primarily involving impaired blood flow, systemic inflammation, and endothelial dysfunction. Conditions such as atherosclerosis and chronic heart failure can lead to reduced mesenteric perfusion, resulting in ischemia within the GI tract. This compromised blood supply jeopardizes the integrity and functionality of the GI tissues, which may manifest in symptoms such as abdominal discomfort, bloating, and malabsorption (20). In more severe cases, mesenteric ischemia can induce irreversible tissue damage, necessitating surgical intervention. The systemic inflammatory response associated with CVD also plays a crucial role in the pathogenesis of GI disorders. Chronic elevations in inflammatory markers, such as C-reactive protein (CRP) and interleukins, contribute to endothelial dysfunction that not only affects vascular health but also

compromises the integrity of the intestinal barrier. This disruption increases intestinal permeability, allowing the translocation of bacterial endotoxins into the bloodstream, which can trigger systemic inflammation and exacerbate conditions such as IBD (21). Additionally, the inflammatory environment in CVD can alter the gut microbiota composition, creating a dysbiotic state that further predisposes individuals to GI disturbances, thereby amplifying cardiovascular and GI health risks (20).

A significant relationship was observed in our study between liver diseases and GI disorders ($P<0.05$). Among patients with GI diseases, 13 individuals (7%) reported liver diseases, while no one in the healthy control group reported liver diseases (Table 2). A study by Srinevas K Reddy and colleagues produced findings that align with our research (22). Their investigation revealed an association between NAFLD and digestive disorders such as diverticular and inflammatory bowel diseases. Another study by Scott McHenry and colleagues also yielded results consistent with our findings (23). They categorized patients into high, intermediate, and low NAFLD risk groups using the Dallas Steatosis Index (DSI) and examined the association between these groups and GI cancers. The study found that individuals at intermediate and high risk of NAFLD had a significantly increased risk of overall GI cancer compared with those at low risk. This positive association was observed for both liver cancer and non-liver GI cancers. Liver diseases, such as liver cirrhosis, profoundly influence GI diseases through interconnected pathophysiological mechanisms. For example, liver dysfunction impairs detoxification, accumulating systemic toxins, particularly ammonia, which compromises gut barrier integrity, increases intestinal permeability, and facilitates bacterial translocation. These disruptions promote small intestinal bacterial overgrowth (SIBO) and systemic inflammation, thereby exacerbating GI disorders like enterocolitis. Furthermore, liver diseases like liver cirrhosis can dysregulate bile acids and disturb gut microbiota composition, favoring dysbiosis and chronic low-grade inflammation (24). This inflammatory state aggravates conditions such as peptic ulcers and IBS-like symptoms (21). These mechanisms underscore the intricate relationship between liver pathology and GI diseases, highlighting the need for targeted therapeutic strategies.

We found a significant association between allergies and a decreased risk of GI disorders ($P<0.05$). Only four individuals (2.2%) in the GI diseases group reported an allergy, compared with 37 individuals (19.9%) in the healthy control group (Table 2). Yoon Jin Choi and others (25) conducted a study that aligns with our findings, revealing a significant inverse association between allergic diseases, particularly allergic rhinitis in adults, and the risk of most GI cancers, except gallbladder and biliary tract cancers. Allergic responses enhance mucosal immunity, offering a protective effect against GI disorders. The activation of mast cells and the production of IgE during allergic reactions elevate immune surveillance at mucosal surfaces, including the gut. This leads to the recruitment of immune cells, such as eosinophils, and an increased production of antimicrobial peptides, strengthening the mucosal barrier and facilitating pathogen elimination (26). While classically associated with allergies, enhanced IgE-mediated responses also provide a rapid immune defense that may prevent chronic inflammation in the gut, thereby reducing the risk of diseases like IBS and certain types of IBD (21).

The association between gallstones and other digestive disorders was statistically significant in our study ($P<0.05$). Six patients with GI diseases (3.2%) reported gallstones, while no healthy individuals reported the same (Table 2). A study by Yuanjie Pang and Collagenous found similar results (27). The study demonstrated that gallstone disease was associated with an increased risk of several GI cancers. On the other hand, the survey by Heather A Ward and others (28) yielded different results. After adjustment for BMI, diabetes, alcohol intake, and physical activity, they did not find a significant association between gallstones and colorectal cancer in women. Also, they found no significant association between gallstones and colorectal cancer in men. Gallstones, composed primarily of cholesterol, bile salts, and bilirubin, can significantly affect GI health through various mechanisms, leading to acute and chronic disorders. Obstruction of the cystic duct by gallstones often results in biliary colic and acute cholecystitis, conditions characterized by severe abdominal pain and inflammation. When gallstones migrate to the common bile duct (choledocholithiasis), they can obstruct bile flow into the intestine, leading to jaundice, steatorrhea, and malabsorption of fat-soluble vitamins, including A, D, E, and K. Additionally, in rare cases, large gallstones can erode through the gallbladder wall and enter the intestine, resulting in gallstone ileus, a mechanical bowel obstruction (29). Therefore, gallstones can impact GI health in different ways and cause disorders in the GI system.

Both groups reported taking medicine without a doctor's prescription, and there was no significant difference between the two groups ($P>0.05$). However, taking some of the prescribed medications, including antihypertensive, psychotropic, and diabetic drugs, showed a significant association with changes in GI system health.

We found a significant association between using antihypertensive drugs and GI diseases ($P<0.05$). Among

patients with GI diseases, 25 individuals (13.4%) reported using these drugs, but only two healthy individuals (1.1%) reported the same (Table 3). A study by A. Hamada and colleagues investigated the effects of calcium channel blockers (CCBs), a type of antihypertensive medication, on GI diseases and obtained similar results to our study (30). The study suggested that the risk of GI disease could be increased by long-term treatment with CCBs. On the other hand, Nagata and others yielded different results (31). They explored the link between antihypertensive medications and peptic ulcer bleeding and found no significant association between using antihypertensive drugs and peptic ulcer bleeding. The variations in results may be due to the lack of differentiation between GI diseases and antihypertensive medications in our study, as well as different genetic and environmental factors associated with the studied societies. Antihypertensive drugs can cause GI disorders through various mechanisms.

CCBs reduce smooth muscle tone by inhibiting calcium influx in smooth muscle cells, impairing intestinal motility. This often leads to constipation. Additionally, they relax the lower esophageal sphincter, predisposing patients to GERD. Chronic use may also affect intestinal blood flow, increasing susceptibility to ischemia and mucosal damage (32). Also, beta-blockers, another class of antihypertensive medications, can reduce splanchnic blood flow by decreasing cardiac output and altering sympathetic activity. A reduced blood supply to the GI mucosa can predispose individuals to ischemic conditions, including colitis, especially in those with pre-existing vascular disease (33).

Another category of drugs that showed significant results in our study was psychotropic drugs ($P<0.05$). Approximately 7% of patients, compared with only 0.5% in the control group, used psychotropic medications, indicating a strong association between these drugs and GI diseases. (Table 3). A study conducted by Yen-Po Wang and colleagues found similar results in this case (34). In this study, the impact of short-term use of selective serotonin reuptake inhibitors (SSRIs) on the risk of upper GI bleeding was examined. The study identified a significant association between short-term SRI use and an increased risk of upper GI bleeding.

In contrast, a case-control study by Alfonso Carvajal and colleagues (35) involved patients under 18 years of age from Italy and Spain and found no significant relationship between SSRI use and an increased risk of GI bleeding. The different results observed in this study can be due to the various societies in which the study was conducted. Also, this study only focuses on SSRIs and GI bleeding, while ours does not have a distinguished categorization of psychotropic medications and digestive disorders.

One possible reason for this association could be that medications like SSRIs may increase gastric acid secretion, potentially leading to GI bleeding and gastric ulcers. Also, drugs such as fluoxetine and escitalopram have antimicrobial effects, which could influence gut microbiota composition and increase ileum permeability (36).

The results for diabetic medications also showed a significant association with GI diseases ($P<0.05$). Approximately 4.5% of patients with GI diseases used diabetic drugs compared to only 0.5% in the control group, indicating a significant association between the use of these medications and GI diseases (Table 3).

A similar study by Manoj Saluja and colleagues, conducted as a retrospective cross-sectional analysis, investigated the variety of GI side effects of metformin in 120 patients in India (37). The study indicated that 51.66% of the patients exhibited GI symptoms, which included diarrhea (the most common), nausea/vomiting, abdominal pain, flatulence, retching, and dysgeusia. Diabetic medications, such as GLP-1 receptor agonists (GLP-1 RAs) and dipeptidyl peptidase-4 inhibitors (DPP-4Is), influence digestive health through various mechanisms.

GLP-1 RAs, such as liraglutide and exenatide, slow gastric emptying via vagal nerve modulation, which reduces postprandial glucose levels but can cause side effects, including nausea and vomiting, and may exacerbate conditions like gastroparesis. They also suppress gastric acid secretion, which can alter digestive processes and increase the risk of GERD (38). Furthermore, DPP-4Is, such as sitagliptin, enhance the activity of endogenous incretins, including GLP-1 and GLP-2, thereby regulating gut motility and barrier function. However, this may result in adverse effects, such as abdominal discomfort or a predisposition to pancreatitis in some patients.

Both classes of medications may also affect bile acid metabolism, contributing to symptoms like diarrhea or fat malabsorption in susceptible individuals. These mechanisms highlight the importance of assessing GI health before initiating therapy, as diabetic medications can significantly impact digestive processes, particularly in patients with pre-existing disorders (39).

This study is subject to several limitations.

The reliance on self-reported data for comorbidities and medication use introduces potential recall bias and limits the accuracy of the data. Furthermore, the lack of detailed subclassification of digestive disorders and the specific medications studied may have obscured nuanced associations. Lastly, critical factors such as lifestyle behaviors and psychological stressors, which are known to influence GI health, were not accounted for in the analysis, potentially confounding the observed associations.

CONCLUSION

This study identifies significant associations between non-GI comorbidities, the use of specific medications, and the prevalence of digestive disorders within the Azeri population of East Azerbaijan, Iran. Comorbid conditions, including hypertension, liver diseases, diabetes, gallstones, and cardiovascular diseases, as well as the use of antihypertensive, psychotropic, and diabetic medications, were significantly correlated with an elevated risk of GI disorders. Conversely, allergies demonstrated a protective association. Future research with larger cohorts, diverse populations, and more comprehensive data collection is essential to validate these findings and elucidate the underlying mechanisms driving these associations.

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CONFLICT OF INTEREST

The authors declare no conflict of interest related to this work.

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