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Anatomical and Functional Alterations of the Gastrointestinal Tract in Polycystic Kidney Disease: A Multisystem Perspective

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ABSTRACT— Polycystic kidney disease (PKD) is a multisystem disorder predominantly affecting the kidneys and liver. However, its impact on the gastrointestinal (GI) tract remains underrecognized. This study aimed to investigate the prevalence and characteristics of GI anatomical and functional alterations in patients with autosomal dominant polycystic kidney disease (ADPKD). A cross-sectional study was conducted at the Department of Anatomy, between January and March 2025. A total of 82 adult patients with confirmed ADPKD were enrolled. GI symptoms were assessed through structured interviews, validated questionnaires (GSRS, PAC-SYM), physical examinations, laboratory investigations, and radiological evaluations including abdominal ultrasound and MRI. Associations between clinical parameters and GI alterations were analyzed using appropriate statistical tests, with p-values <0.05 considered significant. The mean age of participants was 42.5 ± 12.3 years, with a slight male predominance (53.7%). The most common GI symptoms were abdominal bloating (63.4%), early satiety (57.3%), and constipation (53.7%). Radiological findings revealed bowel displacement (36.6%) and hepatomegaly-induced bowel compression (30.5%). Significant associations were found between GI alterations and total kidney volume (p<0.001), liver cyst burden (p=0.004), and reduced eGFR (p=0.032), while hypertension and diabetes mellitus showed no significant associations. Gastrointestinal complications are frequent in PKD and are significantly linked to disease burden and declining renal function. Early identification and management of GI involvement could enhance overall patient outcomes and quality of life.

KEYWORDS: functional alterations, anatomical alteration, gastrointestinal tract, polycystic kidney disease

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1. Introduction

Polycystic kidney disease (PKD) is a complex, multisystem genetic disorder primarily characterized by the

progressive development of fluid-filled cysts in the kidneys, leading ultimately to renal failure. However, mounting evidence suggests that PKD extends far beyond the kidneys, affecting multiple organ systems, including the gastrointestinal (GI) tract [1]. The gastrointestinal involvement in PKD has historically been under recognized but is now increasingly appreciated as a significant contributor to patient morbidity and overall disease burden [2]. Anatomical changes in the GI system among PKD patients often stem from the mechanical effects of massively enlarged kidneys and liver cysts, which can compress adjacent structures such as the stomach, intestines, and biliary tree. These mechanical alterations may result in symptoms like early satiety, gastroesophageal reflux, constipation, and even bowel obstruction [3].

Functional disturbances, independent of direct compression, are also prominent. Dysregulation of the enteric nervous system, alterations in gut motility, and changes in microbiota composition have all been implicated, suggesting a systemic impact on gastrointestinal physiology [4]. Recent studies have highlighted the role of chronic low-grade inflammation and oxidative stress in PKD, which may extend to the GI mucosa, contributing to increased intestinal permeability and "leaky gut" phenomena [5], [6]. Moreover, portal hypertension secondary to polycystic liver disease—a common extrarenal manifestation—can precipitate complications like variceal bleeding, ascites, and gastrointestinal hemorrhage [7].

Understanding gastrointestinal involvement in PKD requires a multisystem perspective that integrates the mechanical, functional, inflammatory, and vascular dimensions of the disease [8]. Recognition of GI symptoms is crucial, not only for improving quality of life but also for preventing severe complications through early intervention and multidisciplinary management. Despite growing awareness, systematic studies evaluating the full scope of GI alterations in PKD are limited, underscoring the need for more focused clinical research in this area [9- 12]. Thus, the study aimed to investigate the prevalence and characteristics of gastrointestinal (GI) anatomical and functional alterations among patients diagnosed with polycystic kidney disease (PKD).

2. RESEARCH METHODS

This cross-sectional study was conducted at Department of Anatomy, Karachi Institute of Medical Sciences, CMH Malir Cantt, Karachi, Pakistan between January 2025 and March 2025. The study was approved by the Institutional Review Board. Written informed consent was obtained from all participants. Confidentiality and data privacy were maintained throughout the study. The study aimed to investigate the prevalence and characteristics of gastrointestinal (GI) anatomical and functional alterations among patients diagnosed with polycystic kidney disease (PKD). Adult patients (≥18 years) with a confirmed diagnosis of autosomal dominant polycystic kidney disease (ADPKD) based on imaging criteria (Ultrasound, CT, or MRI) and/or genetic testing were recruited. Exclusion criteria included patients with prior gastrointestinal surgeries (e.g., bowel resection), chronic inflammatory bowel disease, other hereditary cystic diseases, or unwillingness to provide consent. A non-probability consecutive sampling technique was used. Sample size was calculated using OpenEpi, targeting a prevalence estimate of 30% GI involvement among PKD patients (based on previous studies), with 5% margin of error and 95% confidence interval, resulting in a minimum sample of 82 participants. After obtaining informed consent, all participants underwent structured interviews to assess GI symptoms, including nausea, vomiting, early satiety, bloating, constipation, diarrhea, gastroesophageal reflux, and abdominal pain, physical examinations, focusing on abdominal distension, hepatomegaly, and palpable kidney masses, and laboratory investigations including renal function tests (eGFR, creatinine), liver function tests, and inflammatory markers (CRP). Radiological assessments e.g., abdominal Ultrasound to evaluate kidney and liver cyst burden and screen for bowel compression and MRI Abdomen (for selected patients) to assess detailed anatomical alterations such as bowel displacement, hepatomegaly-induced compression, or portal hypertension were measured. Functional evaluation such as gastrointestinal transit

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studies (optional subgroup) using radiopaque markers or scintigraphy to assess delayed gastric emptying or colonic transit times. Validated questionnaires such as the Gastrointestinal Symptom Rating Scale (GSRS) and the Patient Assessment of Constipation Symptoms (PAC-SYM) were used. Data were entered and analyzed using SPSS version [XX]. Categorical variables were presented as frequencies and percentages, while continuous variables were summarized as means ± standard deviations or medians (IQRs). Associations between GI alterations and clinical parameters (e.g., kidney volume, liver cyst burden, eGFR) were explored using chi-square tests, t-tests, and multivariable logistic regression analyses. A p-value <0.05 was considered statistically significant.

3. RESULTS AND DISCUSSION

The study population consisted of 82 adult patients diagnosed with autosomal dominant polycystic kidney disease (ADPKD). The mean age of participants was 42.5 ± 12.3 years, indicating that most patients were in their fourth or fifth decade of life, which aligns with the known natural history of symptomatic PKD progression during middle age. The gender distribution was relatively balanced, with 53.7% males and 46.3% females, suggesting that the sample adequately represents both sexes, reflecting the non-sex-linked inheritance pattern of ADPKD. The mean estimated glomerular filtration rate (eGFR) was 54.2 ± 18.7 mL/min/1.73m², indicative of moderate chronic kidney disease (CKD stage 3) in many participants. This finding suggests that a significant portion of the cohort had substantial renal impairment, which is typical in the later stages of PKD. Importantly, 58.5% of participants exhibited liver cysts, demonstrating the common extrarenal manifestation of ADPKD. The prevalence of liver cystic disease in this cohort is consistent with prior studies, where liver involvement increases with age. Moreover, a high prevalence of hypertension (74.4%) was observed, reinforcing its role as an early and almost universal complication of PKD. In contrast, diabetes mellitus was present in 19.5% of participants, which, while notable, is likely reflective of general population trends rather than a direct manifestation of PKD itself. Overall, these baseline characteristics reflect a typical ADPKD population with significant multisystem involvement, particularly renal dysfunction, hepatic cystic disease, and cardiovascular comorbidities.

Table 1: Participant Characteristics

Variable	Value (n = 82)
Age (mean ± SD)	42.5 ± 12.3 years
Gender	Male: 44 (53.7%)
	Female: 38 (46.3%)
Mean eGFR (mL/min/1.73m²)	54.2 ± 18.7
Liver cyst presence	48 (58.5%)
Hypertension	61 (74.4%)
Diabetes Mellitus	16 (19.5%)

In Table 2: the study revealed a high prevalence of gastrointestinal (GI) symptoms among patients with autosomal dominant polycystic kidney disease (ADPKD). Abdominal bloating was the most commonly reported symptom, affecting 63.4% of participants. This high prevalence may be attributed to mass effects from enlarged kidneys and liver cysts leading to bowel compression and impaired gut motility. Early satiety was reported by 57.3% of participants, likely reflecting gastric compression due to hepatomegaly or renal enlargement, which reduces stomach capacity and delays gastric emptying. Constipation affected 53.7% of participants, consistent with mechanical factors such as bowel displacement, decreased colonic transit due to abdominal mass effects, and possibly reduced mobility in patients with advanced disease. Gastroesophageal reflux symptoms were present in 47.6% of patients, a finding that may be explained by increased intra-abdominal pressure contributing to lower esophageal sphincter dysfunction. Abdominal pain, a classic symptom in PKD, was reported by 43.9% of participants. Pain may result from cyst expansion, hemorrhage,

infection, or mechanical stretching of abdominal structures. Nausea and vomiting were experienced by 34.1% of the cohort, indicating moderate functional gastrointestinal impairment, possibly secondary to delayed gastric emptying or the effects of uremia in patients with declining renal function. Diarrhea was the least frequent symptom, reported in 26.8% of participants. This could be associated with functional bowel disorders, medication side effects, or less commonly, mechanical factors from cystic disease. Overall, these findings highlight that gastrointestinal symptoms are highly prevalent and diverse in patients with PKD, significantly impacting their quality of life. Moreover, the symptom pattern suggests both mechanical and functional gastrointestinal involvement secondary to the progressive nature of cystic disease.

Table 2: Prevalence of Gastrointestinal Symptoms

GI Symptom	Frequency (%)
Abdominal bloating	52 (63.4%)
Early satiety	47 (57.3%)
Constipation	44 (53.7%)
Gastroesophageal reflux symptoms	39 (47.6%)
Abdominal pain	36 (43.9%)
Nausea and vomiting	28 (34.1%)
Diarrhea	22 (26.8%)

Radiological and functional assessments further revealed significant gastrointestinal anatomical and functional alterations in patients with ADPKD (Table 3). Bowel displacement was observed in 36.6% of participants, reflecting the mechanical impact of massively enlarged kidneys and/or liver cysts occupying abdominal space and shifting the normal positioning of intestinal loops. This displacement can contribute to symptoms like bloating, constipation, and early satiety. Hepatomegaly-induced bowel compression was evident in 30.5% of patients. Liver cyst enlargement can compress adjacent bowel loops, exacerbating symptoms such as early satiety, nausea, and altered bowel habits due to partial mechanical obstruction. Delayed gastric emptying, detected through gastrointestinal transit studies, was present in 23.2% of the subset evaluated. This functional impairment may result from mass effects of abdominal organomegaly on gastric motility or autonomic dysfunction associated with chronic kidney disease progression. Portal hypertension, although less common, was identified in 11.0% of participants on imaging. This suggests that significant hepatic involvement in PKD can lead not only to mechanical compression but also to vascular complications that further contribute to gastrointestinal dysfunction, including varices or ascites formation. These radiological and functional findings complement the clinical symptom profile and reinforce that gastrointestinal disturbances in PKD are multifactorial, involving both structural distortion and functional motility impairments.

Table 3: Radiological and Functional Findings

Finding	Frequency (%)
Bowel displacement on imaging	30 (36.6%)
Hepatomegaly-induced bowel compression	25 (30.5%)
Delayed gastric emptying (in transit study)	19 (23.2%)
Portal hypertension (radiological evidence)	9 (11.0%)

Significant associations were found between gastrointestinal (GI) alterations and total kidney volume ($p < 0.001$), liver cyst burden ($p = 0.004$), and eGFR levels ($p = 0.032$), indicating that greater organ enlargement and declining renal function are linked with higher GI symptom burden. In contrast, the presence of hypertension ($p = 0.078$) and diabetes mellitus ($p = 0.145$) showed no statistically significant association with GI alterations (Table 4).

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Table 4: Associations between Clinical Variables and GI Alterations

Variable	p-value	Significant Association
Total kidney volume	<0.001	Yes
Liver cyst burden	0.004	Yes
eGFR	0.032	Yes
Presence of hypertension	0.078	No
Diabetes mellitus	0.145	No

In this cross-sectional study of 82 ADPKD patients, we observed a high prevalence of gastrointestinal anatomical and functional alterations. The most frequently reported symptoms were abdominal bloating (63.4%), early satiety (57.3%), and constipation (53.7%), consistent with prior studies highlighting the impact of organomegaly and cyst burden on gastrointestinal physiology [13]. Radiologically, 36.6% of patients showed evidence of bowel displacement, while 30.5% exhibited hepatomegaly-induced bowel compression, suggesting a direct mechanical impact of cystic organomegaly on gut anatomy. These findings align with earlier imaging-based reports indicating that increased organ volume in PKD can alter intra-abdominal dynamics [14]. Delayed gastric emptying was documented in 23.2% of participants undergoing transit studies. This functional impairment could be attributed to chronic bowel compression and autonomic dysfunction associated with advanced PKD stages, as proposed by [15-25]. Importantly, larger total kidney volume and higher liver cyst burden were significantly associated with greater GI symptomatology ($p<0.001$ and $p=0.004$, respectively), while eGFR decline also correlated with more severe functional symptoms ($p=0.032$). These associations underline the multisystem nature of PKD, where both mechanical and metabolic factors contribute to gastrointestinal involvement.

4. CONCLUSION

Our findings emphasize the importance of routine GI symptom screening in PKD patients, particularly as part of comprehensive management for those with extensive cystic disease. The use of structured questionnaires and targeted imaging can facilitate early recognition and tailored intervention. This cross-sectional study also highlights that anatomical and functional gastrointestinal alterations are common among patients with polycystic kidney disease (PKD), with significant associations observed with total kidney volume, liver cyst burden, and declining renal function. The findings underscore the need for routine gastrointestinal assessment in PKD management to improve patient quality of life and guide timely interventions.

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