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Article

Gastrointestinal Manifestations of COVID-19 in a South Asian Population: Early Pandemic Insights

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Abstract

Background: COVID-19 has affected millions globally. Initially, attention focused on pulmonary disease, but gastrointestinal (GI) and hepatobiliary manifestations soon emerged as significant components. Reported GI symptom prevalence ranges from 17% to 53%, though most evidence originates from high-income countries and later variant waves. Limited data exist from South Asia during the early wild-type phase. This study aimed to determine the frequency and characteristics of GI presentations among patients with PCR-confirmed COVID-19 in a South Asian setting. Methods: This retrospective cross-sectional study was conducted at Aga Khan University Hospital, Karachi, Pakistan. Patients aged ≥18 years with PCR-confirmed COVID-19 between February and June 2020 were included. Data were extracted from medical records and independently verified by two researchers. Statistical analysis was performed using SPSS Version 24. Results: Among 534 patients, the median age was 55 years (IQR: 24), with 61.4% male. Common symptoms included fever (72.7%), cough (55.7%), and shortness of breath (55.1%). GI symptoms included nausea (24.3%), diarrhea (15.7%), vomiting (15.0%), ageusia (9.2%), and jaundice (4.5%), while abdominal pain was uncommon (4.1%). No major GI complications were reported. Elevated SGOT, GGT, and SGPT were observed in 81%, 69.4%, and 66.8% of patients, respectively. Conclusions: GI symptoms were a notable component of COVID-19 presentations in our population during the early pandemic phase. Recognizing these manifestations supports timely diagnosis and management. The findings provide regional baseline data for comparison with later variants and underscore the need for studies on long-term GI outcomes.

Keywords

COVID-19, Gastrointestinal symptoms, Pandemic, Viral infection, Extra pulmonary symptoms

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1. Introduction

Coronavirus disease 2019 (COVID-19) has impacted millions of people worldwide. One recent study estimates an excess of 14.83 million deaths, which is 2.74 times more than the fatalities reported during the pandemic period [1]. At the beginning of the pandemic in 2019-2020, most of the focus was on the pulmonary implications of the infection, such as pneumonia and acute respiratory distress syndrome. However, over time, more cases of extra pulmonary manifestations of the disease were reported with clinical observations and emerging literature [2,3]. It was noted that the virus affected various bodily systems, including hematologic, cardiovascular, renal, gastrointestinal (GI), hepatobiliary, dermatologic, among others [4].

The GI and hepatobiliary systems have shown a spectrum of presentations. Numerous publications have reported that individuals afflicted with COVID-19 frequently exhibit GI symptoms. The exact proportion of patients with such symptoms varies markedly, with numerous studies estimating that between 17% and 53% of all cases involve GI manifestations [5].

Clinical presentation can include symptoms such as loss of appetite, diarrhea, abdominal discomfort, nausea/vomiting, and more severe rarer complications such as hepatic dysfunction, intestinal perforation, GI bleeding, ischemic colitis, and even instances of pancreatitis [6,7]. Research has proposed various mechanisms to explain the possible pathophysiology behind the role of the virus in its impact on the GI system. Theories include direct viral harm, entry into host cells, inflammatory immune reactions, and impacts on gut microbe flora, all of which can potentially lead to malabsorption, disrupted intestinal secretions, or activation of the enteric nervous system, eventually causing the symptoms and complications listed. Some studies have even found SARS-CoV-2 RNA in biopsies taken from the esophagus, stomach, duodenum, and rectum [8,9].

Existing global syntheses from 2020-2024 consistently demonstrate that GI involvement in COVID-19 remains clinically significant even in the post-vaccination era. These studies reveal considerable heterogeneity in the reported prevalence of GI symptoms and growing evidence of persistent GI morbidity after acute infection, placing vulnerable populations such as the elderly, immunocompromised individuals, and those with multiple comorbidities at particular risk. Meta-analyses estimate that approximately 20% of patients experience one or more GI symptoms during the early pandemic, including diarrhea (13%), nausea or vomiting (8%), and abdominal pain (4%-6%) [10-12]. Variant-specific analyses further suggest that the prevalence of GI symptoms has declined in subsequent waves [13].

However, much of this emerging evidence originates from high-income countries, including large cohort analyses from the United States, Europe, and East Asia. South Asian and other low- and middle-income country (LMIC) populations remain underrepresented in early-wave datasets collected before vaccination rollout, and few studies have established a rigorous baseline from the wild-type SARS-CoV-2 era that can be compared with later variant waves or long-term outcomes. This gap limits global understanding of the evolution of viral pathophysiology and its implications for digestive health, particularly in LMICs where socioeconomic and healthcare disparities may influence disease presentation and outcomes. Therefore, our retrospective study addresses this evidence gap by providing an early, region-specific baseline of acute GI involvement in patients with the wild-type COVID-19 strain.

2. Subjects and Methods

This was a retrospective cross-sectional study conducted at The Aga Khan University Hospital (AKUH), Karachi, Pakistan. The study consecutively included all patients aged 18 years and older with a polymerase chain reaction (PCR)-confirmed diagnosis of COVID-19 during the period from February to June 2020. All paediatric patients were excluded from our study. This study was approved by the AKUH Ethics Review Committee with ethical code: 2020-3722-10346. All patient identifiers were removed during data entry, and complete confidentiality was maintained throughout the study.

Our study involved the review of patients' confidential medical records including physician-documented standardized comprehensive inpatient assessment forms. All collected information was independently reviewed and confirmed by two researchers to ensure consistency. The assessed parameters included epidemiological and demographic information (including age, sex, comorbidities, and addictions), symptoms at the time of admission (both respiratory, such as cough, fever, and shortness of breath, and GI, such as nausea, vomiting, jaundice, diarrhea, abdominal pain, and ageusia/dysgeusia), and lab investigations at the time of admission (complete blood count, liver function tests, serum LDH, serum ferritin, C-reactive protein, and serum electrolytes). Other notable factors included the length of hospital stay and clinical outcomes (discharged, deceased, or leaving against medical advice). For the purposes of this study, abnormal liver function was defined as at least one lab value exceeding the upper normal limit. The standard reference values were ALT/SGPT: 40 IU/L, AST/SGOT: 40 IU/L, GGT: 50 IU/L, ALP: 125 IU/L, and Total Bilirubin: 1.2 mg/dL.

SPSS Version 24 was used to perform statistical analysis. Quantitative variables were presented in two ways: if the data were normally distributed, they were reported as mean ± standard deviation; if not, they were reported as median and interquartile range. Qualitative variables were quantified as percentages and frequencies. The association between

abnormal liver function tests and variables such as sex, age group, comorbidities, smoking history, medication use, symptoms, treatment during inpatient admission, and clinical outcome was assessed using the Chi-square or Fisher's Exact test. An Independent t-test or Mann-Whitney U test was conducted to evaluate the mean differences in quantitative variables, including laboratory features, clinical outcomes, and length of hospital stay, between groups with normal and abnormal liver function tests. A two-tailed p-value of ≤ 0.05 was considered statistically significant.

3. Results

Our study, encompassing 534 patients hospitalized over a six-month period due to COVID-19, examined the distribution of GI manifestations. The median age within our cohort stood at 55 (IQR: 24) years (Figure 1). Notably, the majority of patients were male, comprising 61.4% of the total. Overall, 41.4% (n=221) patients had no comorbidities, whereas 22.1% were found with one co-morbidity and the remaining 36.5% (n=195) were found to have more than one co-morbidity. Among them, hypertension was the most common, reported in 42.1% followed by diabetes mellitus in 36.7% and ischemic heart disease in 17.0% (Figure 2). In our study, 10.5% (n=56) patients had a travel history (Table 1).

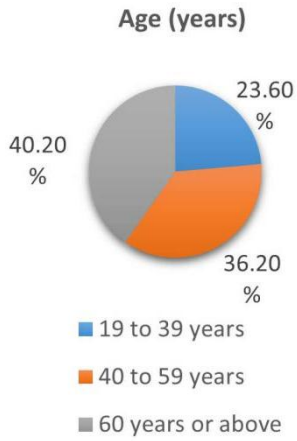


Figure 1. Distribution of age groups among hospitalized COVID-19 patients.

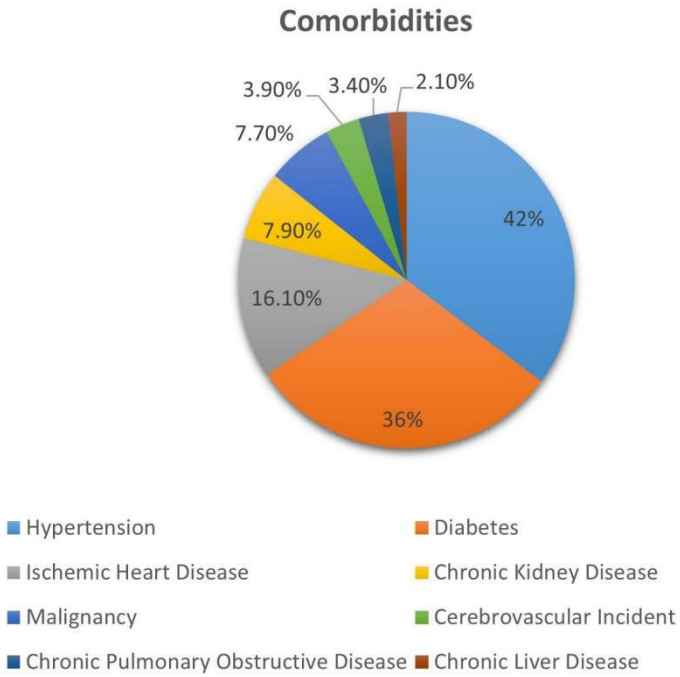


Figure 2. Distribution of comorbidities among hospitalized COVID-19 patients.

Table 1. Demographic and clinical characteristics of hospitalized COVID-19 patients.

Variable	Frequency (Percentage) N=534
Age (Years)	
Median (IQR)	55 (24)
Sex	
Male	328 (61.4%)
Female	206 (38.6%)
Comorbidities	
Hypertension	225 (42.1%)
Diabetes Mellitus	196 (36.7%)
Ischemic Heart Disease	91 (17.0%)
Chronic Kidney Disease	47 (8.8%)
Malignancy	46 (7.6%)
Cerebrovascular Incident	21 (3.9%)
Chronic Obstructive Pulmonary Disease	18 (3.4%)
Chronic Liver Disease	11 (2.0%)
Addictions	
Smoker	45 (8.4%)
Alcohol	13 (2.4%)
GI symptoms	
Nausea	130 (24.3%)
Vomiting	80 (15.0%)
Diarrhea	84 (15.7%)
Jaundice	23 (4.3%)
Abdominal Pain	22 (4.1%)
Other symptoms	
Fever	388 (72.7%)
Cough	297 (55.6%)
Shortness of Breath	294 (55.1%)
Ageusia/Dysgeusia	49 (9.2%)
Medication use	
Steroid	25 (4.7%)
Immunosuppressant	30 (5.6%)
NSAIDs (non-steroidal anti-inflammatory drugs)	93 (17.4%)
ACE inhibitors	78 (14.6%)

Fever emerged as the predominant clinical manifestation, affecting 72.7% of participants, followed by cough (55.7%), shortness of breath (55.1%) and ageusia (9.2%). The GI symptoms observed among our patients included nausea noted in 130 (24.3%), vomiting and diarrhea in 80 (15%) and 84 (15.7%), respectively. Moreover, jaundice was observed in 23 (4.3%) patients. Abdominal pain was reported in 22 (4.1%) patients (Figure 3). Overall GI symptoms were benign in nature. No major GI bleed, perforation or acute abdomen was reported among our cohort.

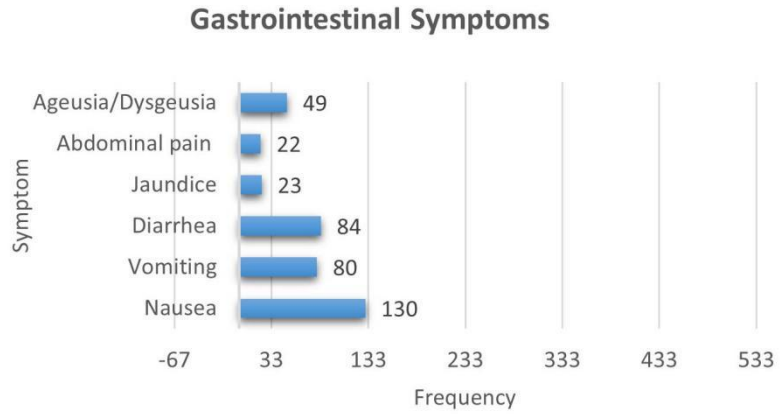


Figure 3. Frequency of GI symptoms among hospitalized COVID-19 patient.

In our patients, the baseline investigation parameters were also recorded. The median haemoglobin level was 12 (IQR: 2.9) mg/dL, the total leukocyte count was 8.6 (IQR: 6) × 10^3/µL and the median platelet count was 231 (IQR: 132) × 10^3/µL. Additionally, in the coagulation profile, the median prothrombin time (PT) was 10.9 (IQR: 1.6) seconds, and the median activated partial thromboplastin time (APTT) was 23.5 (IQR: 27) seconds. The liver function tests indicated a consistent pattern of abnormalities, with Serum glutamic oxaloacetic transaminase (SGOT), Gamma-glutamyl transpeptidase (GGT), and Serum glutamate pyruvate transaminase (SGPT) levels found to be elevated in a significant

proportion of patients, affecting approximately 81%, 69.4%, and 66.8% of the cohort, respectively.

The median length of stay was 4 (IQR: 5) days. In our study, 7.1% (n = 38) of patients required admission to the intensive care unit during hospitalization, and overall, 10.5% (n = 56) of patients died during their hospital stay.

4. Discussion

In reviewing the prevalence and incidence of GI symptoms in COVID-19 patients across various studies, it becomes evident that these manifestations represent an appreciable aspect of the disease presentation. Initially, during the onset of COVID-19 in 2019-2020, the primary focus of the pandemic centered on its pulmonary implications, with early studies indicating a low prevalence of patients exhibiting GI symptoms. However, as time progressed, subsequent research unveiled a notable shift, highlighting a rising incidence of GI manifestations among infected individuals [2,3]. Therefore, findings derived from patients infected during the early wild-type phase of the pandemic, should be interpreted in the context of subsequent SARS-CoV-2 variants, as emerging evidence suggests that the frequency and spectrum of GI manifestations may differ across variant waves, including during the Omicron era.

An international prospective observational study identified diarrhea, nausea/vomiting, abdominal pain, and anorexia as the most prevalent GI symptoms among COVID-19 patients. Furthermore, the study indicated a higher frequency of these symptoms among hospitalized individuals, with 65.6% of infected patients presenting with at least one GI symptom upon admission, while 49% exhibited two or more complaints (including anorexia) [14]. In another study conducted by Marasco et al., predominantly involving Western countries, a prevalence of 59.7% was reported for GI symptoms [15]. Additionally, Chen et al. found that up to 74% of hospitalized cases in the United States experienced GI symptoms [16]. In a study conducted in Tehran, Iran, 43% of hospitalized infected patients presented with at least one GI symptom, while another study conducted in Islamabad, Pakistan, revealed that a mere 35% of COVID-positive patients complained of GI manifestations [17,18]. The included studies highlight a notable prevalence of GI symptoms, shedding light on the diverse clinical spectrum of COVID-19. However, disparities emerge when considering different populations and geographic regions.

In examining the clinical manifestations of GI symptoms associated with COVID-19, our study corroborated existing literature indicating a wide spectrum of presentations. The most commonly reported GI symptoms include diarrhea, nausea, vomiting, abdominal pain, and, notably, jaundice. While these symptoms are relatively common, our study of 534 hospitalized COVID-19 patients found that they predominantly exhibited benign GI manifestations. Nausea was observed in 24.4% of cases, vomiting and diarrhea in 15% and 15.8%, respectively, with jaundice in 4.5% of patients. Abdominal pain was less frequent, reported in 4.1% of cases. Remarkably, our cohort did not present with major GI complications such as bleeding, perforation, or acute abdomen. Notably, liver function tests consistently revealed abnormalities, with elevated levels of SGOT, GGT, and SGPT in approximately 81%, 69.4%, and 66.8% of patients, respectively. These manifestations showcase the systemic nature of the virus and its potential to affect multiple organ systems beyond the respiratory tract. Additionally, our findings align with emerging evidence suggesting atypical presentations or symptoms unique to COVID-19, such as ageusia and jaundice, which may not typically be associated with respiratory viral infections. Some studies have indicated reports of more serious complications, including pancreatitis, ischemic enterocolitis, liver injury, and rare instances of hepatitis [5,10,19]. In a review article authored by Marcelo Augusto Fontenelle RIBEIRO-JUNIOR et al., cases of GI hemorrhages, thrombosis, perforation, and even pneumatosis intestinalis were discussed [20]. Notably, despite the prevalence of GI symptoms, our study revealed a notable absence of major complications such as GI bleeding, perforation, or acute abdomen among hospitalized COVID-19 patients. This absence of severe GI complications suggests that while GI symptoms may be prevalent, they often present as relatively benign manifestations of the disease. Furthermore, our study highlighted the consistent pattern of abnormalities in LFTs across the cohort, indicating the importance of monitoring hepatic involvement in COVID-19 patients. This comprehensive understanding of the clinical spectrum of GI symptoms and their associated findings contributes to the holistic management and surveillance of COVID-19 patients.

The GI manifestations observed in COVID-19 patients have led to several proposed hypotheses regarding the underlying pathophysiological mechanisms to explain the involvement of the GI tract and the reported symptoms. One proposed mechanism is the direct viral invasion of GI epithelial cells by SARS-CoV-2 [21]. Studies have reported the presence of viral RNA in stool samples of infected individuals, suggesting active viral replication within the GI tract [22]. This direct invasion may lead to local inflammation and tissue damage, contributing to the development of GI symptoms such as diarrhea, nausea, and abdominal pain. It is known that SARS-CoV-2 has the ability to directly infect specific cells through its binding to the angiotensin-converting enzyme 2 (ACE2) receptor [20]. Consequently, pulmonary alveolar epithelial cells, small intestine enterocytes, cholangiocytes, and vascular endothelial cells are the tissues exhibiting higher levels of this receptor and thus, are more prone to infection. ACE2 plays a role in modulating the inflammatory response, and its presence in abdominal organs may account for the extra pulmonary symptoms observed in certain patients. Cellular changes lead to inflammation in the intestines and liver, as well as modifications in the intestinal microbiota [20].

Additionally, immune-mediated damage has been implicated in the pathogenesis of GI involvement in COVID-19. Cytokine storms, triggered by COVID-19, lead to excessive release of inflammatory cytokines like IL-6 and TNF-α.

These cytokines cause widespread inflammation in the GI tract, despite limited presence of the virus. The immune response, including apoptosis and danger signal release, attracts immune cells, amplifying tissue damage. Additionally, IL-1 produced by macrophages and mast cells contributes to GI diseases by promoting inflammation and toxic effects. Dysregulated immune responses, including cytokine storms and systemic inflammation, may result in tissue injury and dysfunction within the GI tract. This immune-mediated damage could exacerbate pre-existing GI conditions or precipitate new-onset GI symptoms in COVID-19 patients [23].

Another critical aspect of the pathophysiology of COVID-19 GI involvement is high levels of D-dimer and fibrinogen in COVID-19 patients. 47% of hospitalized COVID-19 patients have elevated levels of D-dimer. This increase is unique to COVID-19 compared to other clotting disorders. It plays a key role in intestinal hypercoagulability and ischemic events. IL-6 induces mononuclear cells to express tissue factor, initiating coagulation activation and thrombin generation. Additionally, TNF-a and IL-1 suppress the endogenous anticoagulation pathway [23]. Therefore, monitoring D-dimer levels along with IL-6, TNF-a, and IL-1 is crucial for both preventing and predicting intestinal coagulation diseases in COVID-19 patients.

Furthermore, alterations in the gut microbiota composition, known as dysbiosis, have been proposed as a potential mechanism underlying GI manifestations in COVID-19. Disruption of the gut microbiome by SARS-CoV-2 infection may perturb the delicate balance of microbial communities, leading to GI dysfunction and symptoms [24]. In summary, the pathophysiological mechanisms underlying GI involvement in COVID-19 are multifaceted and likely involve a combination of direct viral invasion, immune-mediated damage, dysbiosis, and the role of ACE2 receptors. Understanding these mechanisms is crucial for developing targeted therapeutic strategies and mitigating the GI complications associated with COVID-19.

In addressing the diagnostic challenges associated with GI manifestations of COVID-19, particularly when respiratory symptoms are absent, several hurdles emerge. The overlapping symptoms between GI issues and other common illnesses make it challenging to promptly identify COVID-19-related GI complications. Additionally, the reliance on respiratory symptoms as primary indicators may lead to under-recognition of GI involvement, delaying appropriate management and potentially increasing transmission risk. GI symptoms like loss of appetite and diarrhea are frequently seen in COVID-19, but they can also happen in people without the virus. However, GI symptoms along with loss of smell or taste and a fever, highly specific (99%) for COVID-19 infection [16]. Diagnostic tests such as PCR assays, while effective in detecting viral presence, may not specifically target GI manifestations. It is crucial to recognize GI symptoms early to initiate prompt treatment, aiming to prevent severe complications and fatalities [20]. As such, there is a pressing need to develop and refine diagnostic strategies tailored to identify GI manifestations of COVID-19, independent of respiratory symptoms, to optimize patient care.

Our findings should also be interpreted within the broader context of COVID-19 as a multisystem disease. Emerging evidence suggests that disease severity is influenced not only by pulmonary involvement but also by the extent of extrapulmonary organ dysfunction and host-related factors [25,26]. GI manifestations have been associated with an increased risk of severe disease in some studies, emphasizing the importance of their early recognition and appropriate supportive management. Healthcare professionals should remain vigilant regarding the GI manifestations of COVID-19 and consider testing for SARS-CoV-2 in patients exhibiting digestive alterations, particularly if they also have respiratory symptoms [27,28].

This study has some limitations. Owing to its retrospective design, it was not possible to determine whether newly reported GI symptoms or liver enzyme abnormalities were directly attributable to SARS-CoV-2 infection or influenced by pre-existing GI or liver disease, concurrent hepatotoxic medications, or other comorbidities. Consequently, the potential effects of these confounding factors could not be fully assessed. In addition, this was a single-centre study conducted at a tertiary care hospital and predominantly included hospitalized patients with moderate-to-severe COVID-19 and a higher burden of comorbidities, which may have introduced selection bias. Therefore, the observed prevalence ofGI and hepatic manifestations may not be representative of community-managed patients or the broader Pakistani and South Asian populations. Nevertheless, our findings provide valuable baseline data on GI involvement during the early phase of the COVID-19 pandemic.

5. Conclusion

Understanding GI manifestations in COVID-19 is critical not only for managing acute illness but also for anticipating potential long-term sequelae. Our study provides comprehensive assessments of GI symptoms in a South Asian population during the early wild-type phase of the pandemic, addressing a notable gap in global evidence predominantly derived from high-income countries. By characterizing the prevalence and spectrum of GI involvement, our findings contribute towards the regional baseline data that can inform comparisons with later variant waves and guide future research into post-COVID GI complications. However, these findings should be interpreted in the context of the study's retrospective, single-center design and may not be generalizable to broader South Asian populations, vaccinated individuals, or infections caused by later SARS-CoV-2 variants. Future multicenter prospective studies across diverse populations and pandemic phases are needed to further explore predictors of persistent GI morbidity, refine diagnostic strategies, and develop targeted interventions, particularly in LMICs.

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Ethics Statement

This study was approved by the Institutional Ethical Review Committee (2020-3722-10346). The requirement for informed consent was waived due to the retrospective nature of the study, as it involved no direct patient interaction and utilized previously collected data.

Availability of Data and Materials

The datasets generated and/or analyzed during the current study are not publicly available but may be obtained from the corresponding author upon reasonable request and with appropriate justification.

Author Contributions

Abdul Hadi Shahid conceptualized the study, contributed to project design and execution, conducted a comprehensive literature review, and structured the manuscript. Masood Muhammad Karim assisted in project design and execution, interpreted findings, provided insights on data analysis, and contributed to manuscript structuring and editing. Zahabia Sohail supported study conception, design, and execution, conducted literature review, contributed to data curation, and assisted in manuscript structuring. Amna Rashid Hanfee contributed to project design, data collection and management, performed literature review, and drafted the manuscript. Zaibunnisa Kamran assisted in project design, data collection and analysis, and contributed to manuscript drafting. Om Parkash provided critical review, perform final edits, and proofread the manuscript with his expertise, ensuring its accuracy and clarity. The final draft of this work has been endorsed by all authors, who also agree to take responsibility for every aspect of the work to ensure accuracy and integrity.

Conflict of Interest

The authors have no conflicts of interest to declare.

Generative Artificial Intelligence

Generative artificial intelligence tools were not used in the design of the study, data collection, data analysis, or interpretation of results. Any language refinement, where applicable, did not alter the scientific content or conclusions of the manuscript.

Abbreviations

ACE2: Angiotensin-converting enzyme 2
AKUH: Aga Khan University Hospital
APTT: Activated partial thromboplastin time
GGT: Gamma-glutamyl transpeptidase
GI: Gastrointestinal
LMIC: Low- and middle-income country
PCR: Polymerase chain reaction
PT: Prothrombin time
SGOT: Serum glutamic oxaloacetic transaminase
SGPT: Serum glutamate pyruvate transaminase

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