Review paper

The impact of COVID-19 on the course and possible initiation of ulcerative colitis

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Abstract

This review presents an analysis of the available scientific research concerning the impact of COVID-19 infection on the course of ulcerative colitis (UC). There are cases suggesting that SARS-CoV-2 infection may be associated with exacerbations of UC; however, establishing a direct cause-and-effect relationship remains difficult. The exacerbations observed in UC patients during the COVID-19 pandemic may be attributable to stress-related factors. In addition, infection in UC patients may lead to complications and therapeutic challenges. The available reports indicate a potential role of COVID-19 as a triggering factor for the development of UC. Although further research is required to elucidate the role of this infection in both the progression and initiation of UC, current data suggest its potential significance in symptom exacerbation and triggering of autoimmune processes. Yet larger registry and longitudinal data suggest that COVID-19 usually does not worsen UC in the long term.

Introduction

Ulcerative colitis (UC) is a chronic, relapsing inflammatory bowel disease (IBD) characterized by inflammation of the colonic mucosa. The disease can spread proximally in a continuous manner, involving a part or the entirety of the colon. The annual incidence of UC in Europe ranges from 0.6 to 24.3 per 100,000 person-years. The first peak in incidence occurs during the second or third decade of life, while a second peak is observed between 50 and 80 years of age [1–4]. The disease may manifest with abdominal pain, diarrhoea, rectal bleeding, an urgent need to defecate and incontinence. Fatigue and weight loss may also occur [5, 6]. The clinical course of the disease typically involves repeated phases of relapse and remission, which can arise either spontaneously or as a reaction to therapeutic interventions [7, 8]. UC is considered idiopathic, meaning its exact aetiology is unknown. Its pathogenesis involves colonic epithelial cells, with contributing factors including immunological influences, genetic variations, diet, alterations in the gut microbiota, and certain medications [3, 4]. The diagnosis of UC is based on clinical symptoms, blood and stool tests, colonoscopy, and histopathological evaluation of tissue biopsies obtained from the inflamed areas of the colon [6]. The standard first-line treatment involves the use of 5-aminosalicylic acid. If this therapy proves ineffective, the addition of corticosteroids should be considered. Infliximab is utilized for both induction and maintenance of remission. In cases where pharmacological treatment fails, particularly in severe forms of the disease, surgical intervention should be considered [9].

SARS-CoV-2 (Severe Acute Respiratory Syndrome Coronavirus 2) is an enveloped RNA virus responsible for coronavirus disease 2019 (COVID-19), which was declared a pandemic in 2020. The virus, first documented in Wuhan, China, is primarily transmitted through person-to-person contact either directly via respiratory droplets or indirectly by touching contaminated surfaces. COVID-19 is predominantly characterized by respiratory symptoms, such as cough, sneezing, and dyspnoea, and less frequently by gastrointestinal symptoms, including diarrhoea, nausea, and abdominal pain [10–13]. The entry of the virus into host cells is facilitated by the angiotensin-converting enzyme 2 (ACE2). High concentrations of this enzyme have been identified in the lungs, oesophagus, small intestine and colon [14]. The entry of SARS-CoV-2 via the gastrointestinal tract triggers an immune response, an inflammatory reaction, and increased secretion of pro-inflammatory cytokines, which may, in turn, lead to an exacerbation of inflammation and intestinal damage in patients suffering from IBD [15].

Methods

A literature review was conducted using the international scientific database PubMed, employing the following key words: COVID, COVID 19, COVID-19, SARS-CoV-2, UC, and ulcerative colitis. Only articles in English were considered.

Results

COVID-19 as a trigger for UC exacerbation: case reports

Several cases have been reported suggesting that infection with SARS-CoV-2 may lead to an exacerbation of UC. However, proving a direct cause-and-effect relationship remains challenging. For example, 2 cases of patients with UC who experienced disease exacerbation following COVID-19 infection have been reported:

Case 1: A 35-year-old man with a 2-year history of UC, treated topically with 5-aminosalicylic acid (5-ASA), was admitted with bloody diarrhoea, urgency, and weakness. SARS-CoV-2 infection was confirmed by a positive nasopharyngeal swab.

Case 2: A 40-year-old woman with an 11-year history of UC, treated with infliximab and azathioprine, was admitted with bloody diarrhoea. One week earlier, she had experienced flu-like symptoms and a dry cough. A nasopharyngeal swab for SARS-CoV-2 was also positive [16].

Impact of COVID-19-related stress on UC activity

The literature further suggests that disease exacerbations in UC patients during the COVID-19 pandemic may have been associated with stress-related factors, such as social isolation [17–19]. For instance, one study from Japan compared the UC Disease Activity Index (UC-DAI) between two groups: UC patients assessed in March and April 2020, and a control group of patients evaluated during the same months in 2019. Each patient was assessed using the UC-DAI, which considered three criteria: stool frequency, rectal bleeding, and the physician's global assessment of disease activity (endoscopic evaluation was not included). The study found that the average UC-DAI score was significantly higher in UC patients during the first wave of COVID-19 than in the control group. However, it should be emphasized that during the pandemic, many people–including most patients with UC exacerbations–experienced significant stress. Based on these results, the authors concluded that, in some UC patients, the COVID-19 pandemic led to a worsening of symptoms, likely related to both psychological and physical stress [17]. The next study followed the same group of UC patients who had been included in the previously described study, with evaluations conducted in March and April 2021 and again in March and April 2022. Patients were again assessed using the UC-DAI, and an additionally endoscopic evaluation of the mucosa was performed and the Matts classification was used. The study demonstrated that the disease activity in UC patients, which had worsened during the first wave of the COVID-19 pandemic in 2020, improved in 2021 and remained stable in 2022. Although the pandemic persisted in 2021 and 2022, it is possible that during these years it ceased to be the predominant stressor (given that stress exacerbates UC symptoms) or that patients' sensitivity to stress diminished [18]. Another study conducted between August and December 2021 involved an online survey among UC patients from various countries (United States, Canada, Japan, France, and Finland), which included questions about disease activity. In 2020, compared to 2019, 57% of patients experienced a similar number of exacerbations, 25% had more exacerbations, and 18% had fewer exacerbations. It is possible that patients' perceptions of their disease during the COVID-19 pandemic were influenced by anxiety, stress, and fear related to it, with many reporting a negative impact on their emotional well-being [19].

Increased interventions for acute severe UC during COVID-19

It is also noteworthy that during the COVID-19 pandemic, patients with acute severe UC more frequently required medical or surgical interventions. This may be attributed to a more severe disease course, reduced access to healthcare services, and concerns about personal safety [20].

Complications and risk factors of COVID-19 in patients with UC

Based on the literature analysis, it can be concluded that SARS-CoV-2 infection in patients with UC may lead to various complications, therapeutic challenges, and an overall more complex disease course. This may be related to various risk factors affecting disease progression. Primary risk factors include age, comorbidity burden, and the use of medications, such as 5-ASA. Secondary risk factors include male sex, smoking, and IBD severity. The presence of UC and the use of 5-ASA have been associated with an increased risk of COVID-19–related mortality. For example, one reported case involved an 80-year-old patient with a 3-year history of UC, treated with mesalamine, who was admitted with fever and bloody diarrhoea. She was diagnosed with COVID-19–induced pneumonia and, despite hospitalization, she died after 2 weeks [21]. Another case illustrating the complexity of disease progression involved a 26-year-old woman with UC, whose disease exacerbation occurred during remission, 6 weeks prior to her COVID-19 diagnosis. After her initial discharge, her condition deteriorated again,

and an RT-PCR test confirmed SARS-CoV-2 infection. During her second hospitalization, the patient experienced a series of complications, including pleuritic pain and a spontaneous miscarriage. Although it remains unclear which factor contributed most significantly to her clinical deterioration, this case demonstrates that the infection can create new challenges in managing UC and provides valuable insights for further research [22]. Moreover, studies have shown that patients with both UC and COVID-19 have a higher risk of developing deep vein thrombosis compared with patients with COVID-19 alone. This increased risk may be attributable to the chronic inflammation associated with UC, reduced mobility (due to abdominal pain and fatigue), corticosteroid use, malnutrition, or endothelial cell damage associated with SARS-CoV-2 infection [23].

New-onset UC following COVID-19 infection

COVID-19 may serve as a potential initiating factor in the development of autoimmune diseases, including inflammatory bowel diseases. Although the mechanisms behind this phenomenon are not yet fully understood, available reports suggest that viral infection may trigger disease in genetically predisposed individuals. This may occur through interactions between the virus and the host's immune system, weakening the protective role of the intestinal mucosal barrier, and modifying the composition of the gut microbiota. SARS-CoV-2 infection may disrupt the balance of the gut microbiota by decreasing the number of beneficial bacteria (*Bifidobacterium*, *Lactobacillus*, *Eubacterium*) while increasing pathogenic strains (*Corynebacterium*, *Ruthenibacterium*). Such dysbiosis promotes inflammation and a weakened intestinal barrier [24–33]. A key factor is the expression of the ACE2 receptor, which is exploited by SARS-CoV-2 to enter cells and is highly expressed in both the respiratory and the gastrointestinal tracts. Moreover, excessive ACE2 expression may exacerbate the inflammatory process in the colon [32]. Below are case descriptions that indicate such a potential connection:

In a 50-year-old patient with no previous gastrointestinal complaints, gastrointestinal symptoms – including bloody diarrhoea – appeared 2 weeks after completing treatment for COVID-19-induced pneumonia. Colonoscopy and histopathological examination confirmed the diagnosis of UC. The patient began treatment with 5-ASA, which led to clinical improvement. This case suggests that the immune response to COVID-19 infection might have been a mechanism contributing to the initiation of UC [24].

Another article presents 2 cases. In the first one, a 37-year-old patient experienced bloody diarrhoea 8 weeks after recovering from a SARS-CoV-2 infection. Based on colonoscopy and histopathological examination, UC was diagnosed. In the second case, a 64-year-old patient developed symptoms – abdominal pain and bloody diarrhoea – 3 weeks after COVID-19 infection. Again, colonoscopic examination and histopathological evaluation led to the diagnosis of UC, and treatment was initiated [25].

In another case, new-onset UC occurred in a 21-year-old patient, 1 week after a second episode of COVID-19. The clinical presentation, sigmoidoscopy, and biopsy supported the diagnosis of UC [26].

Another report describes a series of cases involving three adolescent female patients in whom new-onset UC developed some time after COVID-19 infection. In the first one, a 16-year-old patient experienced abdominal pain and blood in the stool 3 weeks after a COVID-19 diagnosis; colonoscopy revealed diffuse inflammation with erosions and ulcerations. The second one, also a 16-year-old patient, reported abdominal pain and blood in the stool 8 weeks after COVID-19 infection; colonoscopy showed continuous inflammation extending from the rectum to the caecum. In the third case, a 12-year-old patient developed abdominal pain and blood in the stool 3 weeks after COVID-19 infection colonoscopy revealed diffuse inflammation with ulcerations in the colon [27].

A separate case involves a 13-year-old patient from Japan who, one month after a COVID-19 infection – manifesting with fever, cough, and taste disturbances that later resolved spontaneously – developed abdominal pain and bloody diarrhoea. A nasopharyngeal swab for SARS-CoV-2 was performed in the hospital, yielding a negative PCR result. Ileocolonoscopy revealed a loss of the vascular pattern in the colon with erosions, and a diagnosis of UC was made. Steroid therapy led to remission [28].

Another case describes a 28-year-old man who had a mild COVID-19 infection and was treated with hydroxychloroquine tablets, which initially improved his condition. However, on the fifth day of treatment, he began experiencing fever and bloody diarrhoea. Based on the clinical presentation, colonoscopy, and histopathological examination, an acute severe UC was diagnosed – most likely triggered by COVID-19 infection, as other known factors (infections, smoking, drug use) were excluded based on history and laboratory tests [30].

Yet another case involves an 84-year-old man who, in December 2021, presented with recurrent diarrhoea that had become bloody in recent weeks and was accompanied by abdominal pain and weight loss. Incidentally, a COVID-19 infection was detected. In January 2022, sigmoidoscopy and histopathological examination revealed changes extending from the rectum to the descending colon consistent with UC. It remains unclear whether it represented an exacerbation of a mild, previously undiagnosed UC or a new UC triggered by SARS-CoV-2. Nonetheless, the case highlights that COVID-19 infection may have

contributed to the worsening of the patient's condition [31].

Another article describes the case of a 55-year-old man with no significant medical history who developed de novo UC after a SARS-CoV-2 infection. Initially, he experienced COVID-19 with pulmonary involvement, requiring treatment with steroids, azithromycin, and heparin. One month after recovery, he was asymptomatic, but two months later he developed severe gastrointestinal symptoms, including abdominal pain and chronic, bloody diarrhoea. Imaging, endoscopic, and histopathological studies led to the diagnosis of UC [32].

A case involving a 33-year-old previously healthy patient is also described. Two months after recovering from COVID-19, he developed chronic colitis resembling UC, autoimmune pancreatitis (AIP), and suspected autoimmune hepatitis (AIH). The patient was admitted with acute abdominal pain, nausea, vomiting, and chronic, bloody diarrhoea that had persisted for two months following the COVID-19 infection. Imaging and histopathological examinations confirmed a diagnosis of UC, while computed tomography (CT) and magnetic resonance imaging (MRI) revealed features of AIP. Additionally, elevated liver transaminases and the presence of antinuclear antibodies (ANA) and anti-smooth muscle antibodies (ASMA) suggested an autoimmune basis for his hepatopathy. Steroid treatment led to rapid improvement, and after 7 months, the patient remains asymptomatic, continuing therapy with mesalazine and azathioprine [34].

Another article describes a 74-year-old patient in whom UC was diagnosed following a SARS-CoV-2 infection. The patient, with no previous history of gastrointestinal diseases, initially presented with typical COVID-19 symptoms including fever, runny nose, and diarrhoea. One month after the acute phase of the infection subsided, he developed bloody diarrhoea and fever. Laboratory tests showed anemia and elevated inflammatory markers, while stool cultures did not reveal common pathogens. Endoscopy revealed extensive inflammatory changes involving the rectum and sigmoid colon, and histopathology confirmed the characteristic features of UC. The patient responded well to mesalazine treatment, achieving clinical remission [33].

Finally, a case was presented involving two previously healthy sisters, aged 10 and 9, who in August 2021 were admitted to the emergency department with acute diarrhoea and haematochezia lasting five days. During the initial phase of their illness, they also experienced low-grade fever and cough. Both tested positive for SARS-CoV-2 by PCR, had not been vaccinated against COVID-19, and stool microbiological tests (bacterial cultures, tests for parasites, and *Clostridioides difficile* toxins) were negative for other pathogens. The 10-year-old one (Patient A) experienced short-term diarrhoea and rectal bleeding that resolved within 2 weeks. However, the 9-year-old one (Patient B) initially had a similar course, but after 4 weeks her symptoms worsened, leading to a diagnosis of UC. Endoscopy of Patient B revealed extensive, continuous inflammation of the colonic mucosa (pancolitis) with histological changes characteristic of UC. Treatment was initiated with prednisone, which brought about improvement; however, upon dose reduction, relapses occurred. Only the introduction of infliximab resulted in clinical and biochemical remission. This case suggests that SARS-CoV-2 infection may serve as an initiating factor for an acute inflammatory reaction in the intestine that, in some patients, subsides spontaneously, while in others it leads to the development of a chronic inflammatory disease such as UC [35].

These case reports underscore the importance of thoroughly examining patients who have recently recovered from COVID-19 and who present with bloody diarrhoea, so as not to confuse it with viral gastroenteritis, a gastrointestinal manifestation of COVID-19, or transient intestinal upset [25, 26, 29].

UC disease course remains stable after COVID-19

In contrast to earlier reports suggesting a detrimental relationship, several studies have found no significant adverse effect of COVID-19 on UC activity [36, 37]. In the multicentre J-COSMOS registry, COVID-19 did not alter disease activity in most patients with IBD; many individuals with UC remained in remission during and after the infection [36]. Likewise, a longitudinal cohort that followed patients for up to 6 months before and after SARS-CoV-2 infection observed no clinically meaningful changes in clinical, endoscopic, or laboratory indices of disease activity. Although new gastrointestinal symptoms were frequently reported during acute infection, they did not translate into increased hospitalizations, surgery, or sustained alterations of the gut microbiome, suggesting that COVID-19 does not drive durable changes in the UC course [37].

Pathophysiological intersections between ulcerative colitis and COVID-19

Ulcerative colitis (UC) is a chronic, idiopathic inflammatory bowel disease with a pathogenesis that remains incompletely understood. One potential point of intersection between UC and COVID-19 is the expression of receptors that facilitate SARS-CoV-2 entry into host cells. In a study by Hamamoto *et al.*, a significant increase in the proportion of ACE2-positive cells, as well as enhanced TMPRSS2 expression, was observed in colonic and rectal tissues of patients with UC compared to controls [38]. These findings suggest that receptor upregulation in the intestinal mucosa of UC patients may predispose them to

greater susceptibility to viral entry or may influence the course of both COVID-19 and the underlying disease, although the role of ACE2 as either protective or pro-inflammatory remains unclear.

Another mechanism linking UC with COVID-19 is excessive immune activation. Severe COVID-19 is characterized by a so-called cytokine storm, including markedly elevated IL-6 and TNF-α levels, both of which play a pivotal role in UC pathogenesis [39]. This excessive cytokine production may therefore act synergistically to intensify the inflammatory response and aggravate mucosal injury.

Endothelial dysfunction and thrombosis represent an additional shared pathophysiological pathway. SARS-CoV-2 infection induces generalized endothelial injury, compromising vascular integrity, reducing nitric oxide bioavailability, increasing oxidative stress, promoting leukocyte adhesion, and triggering procoagulant changes involving von Willebrand factor and factor VIII. These processes contribute to COVID-19–associated coagulopathy [40]. Importantly, patients with inflammatory bowel disease – particularly UC – demonstrate a significantly higher risk of venous thromboembolism in the context of COVID-19 compared with infected patients without IBD (OR ≈ 1.44). Moreover, UC patients appear to have a higher incidence of VTE and pulmonary embolism than those with Crohn's disease [41]. This observation points to a potential synergistic effect arising from the convergence of SARS-CoV-2–induced endothelial dysfunction with the baseline prothrombotic tendency observed in UC.

Conclusions

The available research indicates that SARS-CoV-2 infection may affect the course of ulcerative colitis in various ways. COVID-19 infection can trigger disease exacerbations (potentially related to pandemic-induced stress and a decline in emotional well-being), lead to complications (such as an increased risk of deep vein thrombosis), and act as a triggering factor for the development of UC in genetically predisposed individuals. At the same time, in most patients, COVID-19 does not appear to produce a sustained change in UC activity. Further research is necessary to better understand the pathophysiological mechanisms linking COVID-19 and UC.

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Conflict of interest

The authors declare no conflict of interest.

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