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Post Covid –19 induced Autoimmune Colitis: A Case Report

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Abstract

Statement of the Problem: At the time of writing, the novel coronavirus SARS-CoV-2-caused coronavirus disease-2019 (COVID-19) has resulted in more than 6.4 million fatalities globally. Angiotensin-converting enzyme 2 receptor (ACE-2 receptor), which is also present in the intestinal epithelium, primarily in the terminal ileum and colon, is how the SARS-CoV-2 virus enters hosts. When there is intestinal inflammation, the expression of the ACE-2 receptor is enhanced. The risk and/or progress of COVID-19 disease has therefore been documented to be altered by coexisting inflammatory bowel disease (IBD). When the related previous literature was reviewed, there was little evidence that either IBS increases the risk of COVID-19 cell entry or that COVID-19 induces cellular inflammation, which leads to the development of IBS.

The purpose of this study: in this report, we will present a case of autoimmune colitis in a child. A 13-year-old boy with typical Covid 19 symptoms and a positive PCR was admitted to the hospital with gastrointestinal symptoms of intestinal obstruction. Colonoscopy confirmed the patient's IBD diagnosis, which was supported by laboratory tests. The patient was given a treatment protocol that included steroids, mesalazine, and ursodeoxycholic acid, and he recovered well and was discharged with further follow-up in the pediatric GI clinic.

Keywords: *COVID-19, autoimmune colitis.*

Introduction

The clinical course of Coronavirus disease-2019 (COVID-19), caused by the novel coronavirus SARS-CoV-2, was highly variable (1). Several studies have found that the gastrointestinal tract is involved in COVID-19 infection (2). The SARS-CoV-2 virus enters hosts by binding to the angiotensin-converting enzyme 2 receptor, which is also expressed in the intestinal epithelium, primarily in the terminal ileum and colon, and even more so when inflamed. Thus, it has been proposed that comorbid inflammatory bowel disease (IBD), particularly in the context of immunosuppression, alters the risk and/or disease course of COVID-19 (3).

Case Presentation

A 13-year-old Saudi male had a history of mild COVID-19 infection 5 months prior that resolved spontaneously. A month later, the patient began to experience infrequent non-bloody non-bilious vomiting 2-3 times per day, along with abdominal pain and poor appetite, as well as a history of diarrhea. The diarrhea was watery three times per day and did not contain any blood or mucosa. In the last two weeks, the diarrhea subsided, and he developed constipation. His vomiting frequency increased (about 10 times per day), and it was greenish in color, accompanied by epigastric abdominal pain. There was a history of weight loss of about 20 kg over two months.

The patient was physically ill, moderately dehydrated, consciously alert, not in distress, and had no skin manifestations. The abdomen was soft and lax, with mild epigastric tenderness, no organomegaly or palpable masses, and no ascites. The rest of the systemic examination was unremarkable.

Except for an elevated C-reactive protein level, laboratory tests for hematological parameters were unremarkable. Liver enzymes were slightly elevated, but serum amylase and lipase were within normal limits. The results of viral markers, stool microscopy, and culture were unremarkable, and the blood coagulation profile was within normal limits.

The patient was admitted to the pediatrics' ward and began receiving IV fluids, omeprazole 40mg, and ondansetron. A computed tomography of the abdomen was performed on the second day of admission and revealed thickening of the gastric mucosa. On the third day of admission, the patient was started on an empirical antibiotic regimen of intravenous cefotaxime and metronidazole and scheduled for a GI endoscopy.

On the fifth day, an upper endoscopy and lower colonoscopy revealed diffuse hemorrhagic gastritis with bile secretion in the stomach, multiple hyperemic rounded patches scattered throughout the colon, and marked hyperemia. Following the endoscopy, the patient was started on 4 mg/hr. intravenous esomeprazole.

An endoscopy histopathological examination revealed reactive gastropathy in gastric samples. Small bowel mucosa with lymphoid hyperplasia was found in terminal ileum samples, and colonic biopsy revealed mild active colitis with minimal architectural changes and no dysplasia. Biopsy results indicated inflammatory bowel disease (IBD).

A repeat abdominal ultrasound revealed a gallbladder filled with unmovable sludge and tiny stones, as well as thickening of the gallbladder walls with a double wall sign. As a result, calculous cholecystitis was diagnosed and oral ursodeoxycholic acid was started.

The patient was started on peripheral parenteral nutrition (PPN) and intravenous methylprednisolone 60 mg IV OD on the eighth day. The patient's abdominal pain subsided over the next two days, and his vomiting improved as it became less frequent and non-bilious and non-bloody, and he began to take oral juice and his PPN was discontinued. The IV antibiotics were stopped the following week, and the patient was put on oral ursodeoxycholic acid, omeprazole, and prednisolone. Prednisolone 60 mg PO OD for 1 week, then 40 mg PO OD for 1 week, then 20 mg PO OD for 1 week, Omeprazole 20 mg PO BID for 5 weeks, and ursodeoxycholic acid 250 mg BID for 2 weeks were prescribed. The patient returns to the Pediatrics gastroenterology clinic without complications.

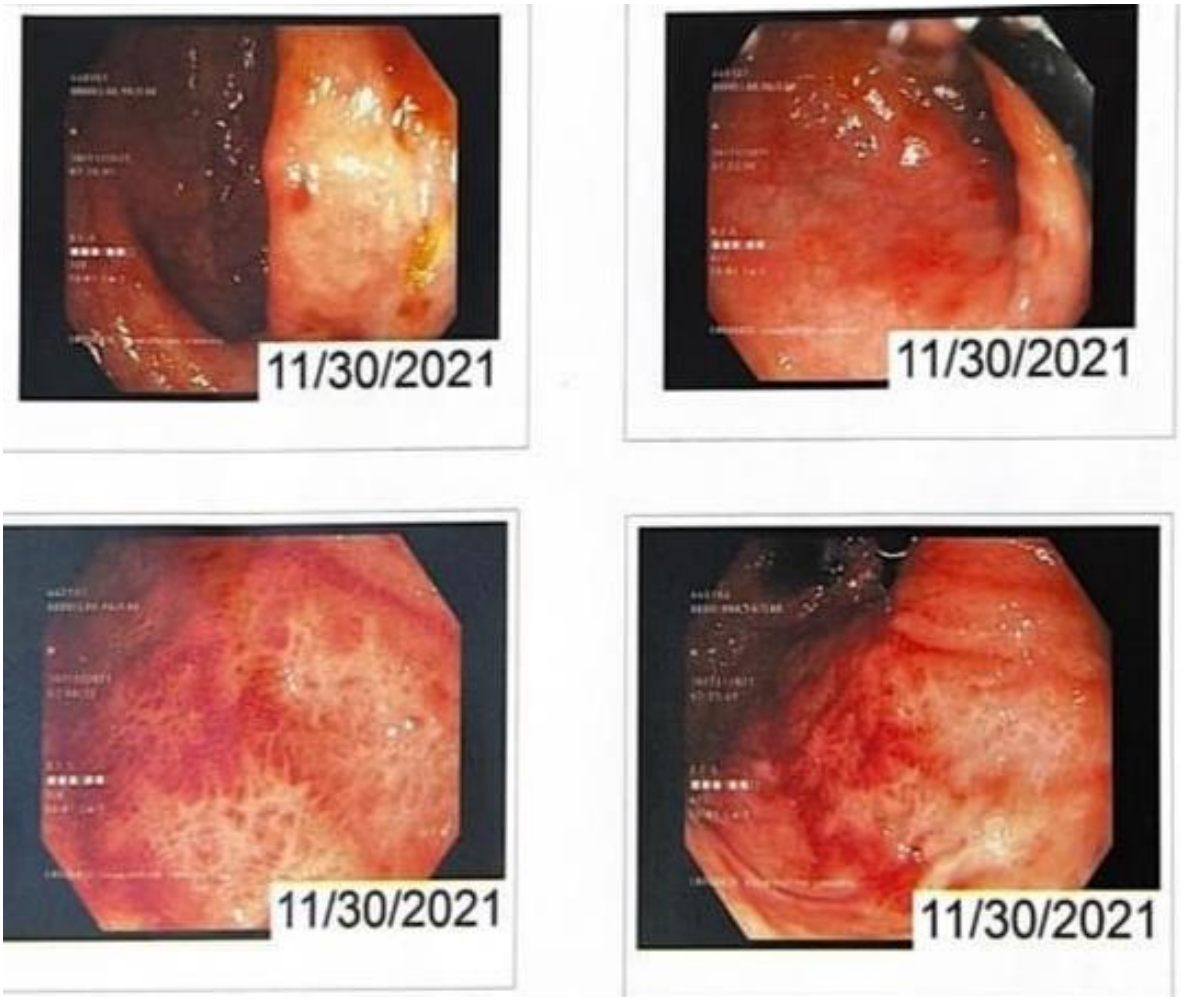

Figure (1): Endoscopy demonstrating diffuse hemorrhagic gastritis with bile secretion

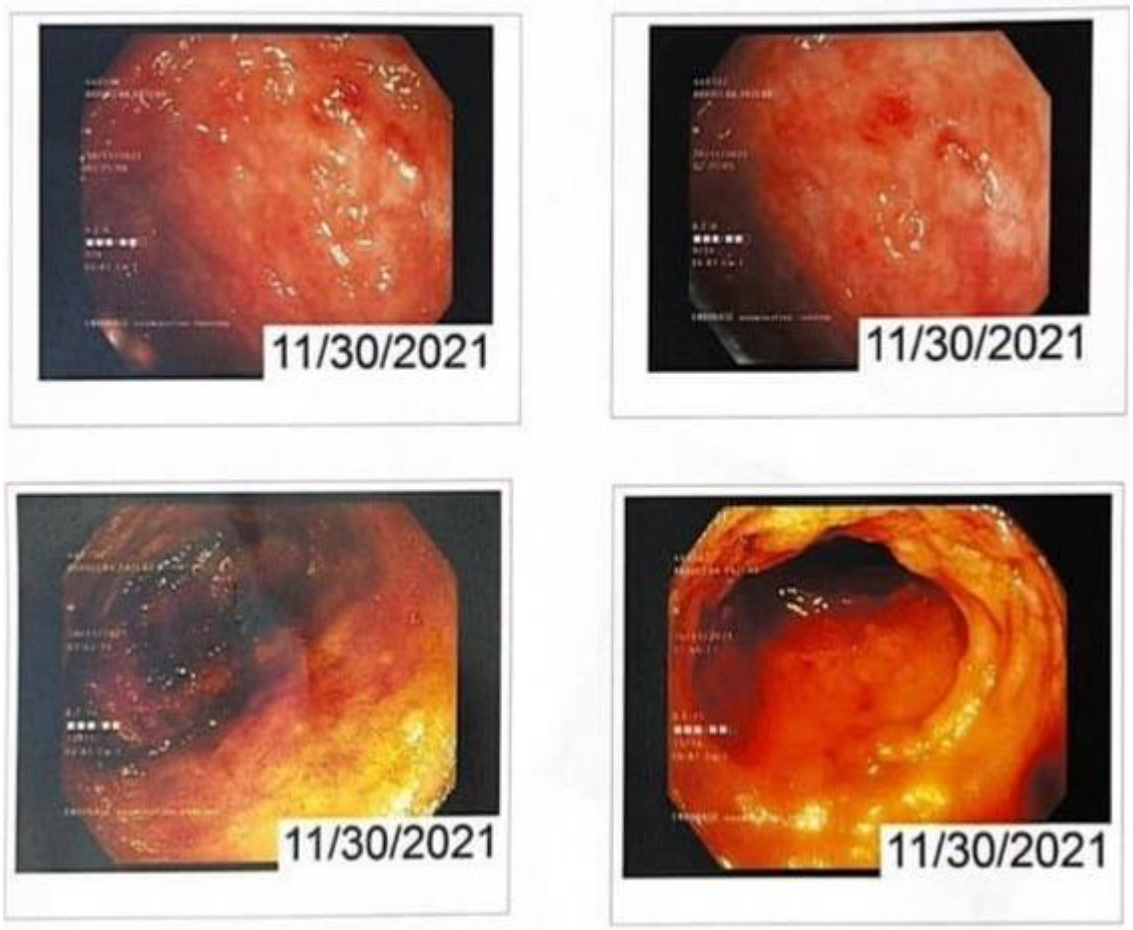

Figure (2): Colonoscopy Demonstrating Multiple Hyperemic Rounded Patches Scattered Throughout the Colon

Discussion

Gastrointestinal manifestations secondary to SARS-CoV-2 infection are now considered one of the most frequent complications of this disease, both during the active infection and up to five months after treatment of COVID-19 pneumonia (2). As mentioned before, elevated ACE-2 activity in the terminal ileum, colonic mucosa, and lungs, explaining the frequent association between COVID-19 pneumonia and diarrhea. Up to our knowledge, the current case is the first reported IBD secondary to COVID-19 infection in pediatrics.

Since the outbreak of the COVID-19 pandemic in 2019, four cases of IBD were reported secondary to COVID-19 infection. However, only one case was reported without prior pneumonia. Calabrese et al. reported a case of a 19-year-old female who presented with fever, vomiting, bloody diarrhea, and loss of taste. CT was revealed only enhanced ileum and colon without pulmonary affection. Gastrointestinal

symptoms persisted despite treatment with hydroxychloroquine; therefore, biopsies were done which were suggestive of UC (4).

The remaining three cases were preceded by COVID-19 pneumonia in the absence of known UC. Cases were two males and one female with mean age 52.5 years (50-55 years). Clinical presentation in three cases was obscure and unsuggestive of IBD at first, failure to improve raised the suspicion of underlying gastrointestinal pathology. Diagnosis of IBD was confirmed in all of the cases with colonoscopy and tissue biopsy (5).

The exact underlying mechanisms of co-morbidity between COVID-19 infection and IBD is still under investigation. It has been hypothesized that an already liable intestine is prone to any infection, including COVID-19. Mucosal breakdowns from previous infections, combined with baseline elevated levels of ACE-2 and transmembrane serine proteases in the gut, provide the ideal environment for the SARS-CoV-2 virus to enter cells and trigger an inflammatory cascade severe enough to reactivate dormant UC or "generate" a de novo autoimmune response.

Immunological theories were raised to illuminate the interacting pathogenesis between COVID-19 and autoimmune diseases. COVID-19 causes a significant decrease in T-lymphocytes including CD41, CD81 and regulatory T cells which is related to the induction of apoptosis, upregulation of inflammatory cytokines and bone marrow suppression. During the recovery phase, the lymphocyte levels increase which may trigger an immune reconstitution state where the immune system is hyperactive and unable to self-regulate. There is immunosuppression that occurs during COVID-19 infection causing a transient decrease in the ability of the innate immune system cells to identify differences in tissues (6).

Additionally, the fact that our patient had a prior vaccination a few months before developing autoimmune colitis brings the attention to the question if vaccine mRNA could be related to disease progression.

Conclusion

The current case report discusses the affection of gastrointestinal tract presented as autoimmune colitis secondary to COVID-19 infection. However, the interplay between COVID-19 infection and IBD needs further investigation. As we discussed earlier COVID-19 manipulate the immune system raising the risk of developing IBD, in addition IBD facilities COVID-19 penetration through mucosal barrier.

Reference

1. Argenziano, M. G., Bruce, S. L., Slater, C. L., Tiao, J. R., Baldwin, M. R., et al. (2020). Characterization and clinical course of 1000 patients with coronavirus disease 2019 in New York: retrospective case series. bmj, 369.

2. El-Hady, H. A. N. Y., Darwish, E., Eldeeb, M. K., Abdelfattah, M., Elwazzan, D., et al. (2022). COVID-19 GIT Affection: An Updated Review. Iranian Red Crescent Medical Journal, 24(5).

3. Allocca, M., Fiorino, G., Zallot, C., Furfaro, F., Gilardi, D., et al. (2020). Incidence and patterns of COVID-19 among inflammatory bowel disease patients from the Nancy and Milan cohorts. Clinical Gastroenterology and Hepatology, 18(9), 2134-2135.

4. Calabrese, E., Zorzi, F., Monteleone, G., & Blanco, G. D. V. (2020). Onset of ulcerative colitis during SARS-CoV-2 infection. Digestive and Liver Disease, 52(11), 1228-1229.

5. Mora, M. C. F., Abushahin, A., Gupta, R., Winters, H., & Khan, G. M. (2022). Severe Ulcerative Colitis as a Complication of Mild COVID-19 Infection in a Vaccinated Patient. Cureus, 14(6).

6. Lakkasani, S., Shah, M., Mirza, N., Rayad, M. N., & Okwesili, B. S. (2021). S1890 COVID-Induced Expression of Gastrointestinal Tract Autoimmunity. Official journal of the American College of Gastroenterology| ACG, 116, S830.