Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Image of the Month

Onset of ulcerative colitis during SARS-CoV-2 infection

Emma Calabrese*, Francesca Zorzi, Giovanni Monteleone, Giovanna Del Vecchio Blanco

Gastroenterology Unit, Department of Systems Medicine, University of Rome Tor Vergata, Italy

A 19-year-old, non-smoker woman with a recent history of fever, nausea, vomiting, bloody diarrhea and loss of taste and smell was admitted to the Tor Vergata Hospital. A nasofaringeal swab resulted positive for SARS-CoV-2. At entry, she had a body temperature of 38 °C, pulse of 110 beats/min, and 99% oxygen saturation. She had severe anemia but no shortness of breath or chest pain. C-reactive protein, platelets, fibrinogen and D-dimer were elevated. A chest and abdominal CT scan showed no pneumonia but increased contrast enhancement in the ileum and colon. No further pathogen was evidenced. After 1 week treatment with hydroxychloroquine, all the symptoms/signs disappeared except the severe anemia, which required a blood transfusion, and the enhanced inflammatory markers. The subsequent nasofaringeal swabs were negative for SARS-CoV-2. At day 16, a small bowel ultrasonography revealed an increased bowel wall thickening of the whole colon associated with an increased blood flow vascularization (Limberg score 4) (Fig. 1, panels A-B) and ileocolonscopy showed an extensive colitis with mucosal friability, spontaneous bleeding and tiny and large ulcerations (Fig. 1, panels C-D). Hematoxylin and eosin staining of the colonic biopsy samples showed ulcerations, crypt architectural distortion, a diffuse and active inflammatory infiltrate with crypt abscesses (Fig. 1, panels E-F). SARS-CoV2 RNA in colon/ileal and fecal samples was negative [1,2]. A diagnosis of ulcerative colitis was made and treatment with oral beclomethasone dipropionate and MMX-mesalamine was started.

The clinical spectrum of SARS-CoV-2 ranges from asymptomatic or mild respiratory disease to pneumonia with respiratory distress syndrome and/or sepsis (Covid-19), which can result in a fatal outcome. Common symptoms are fever, cough, and shortness of breath, but gastrointestinal symptoms can occur in infected patients in line with the demonstration that SARS-CoV-2 RNA can be detected in feces and some of the infected patients remain positive in stools after becoming negative in respiratory samples [3]. Notably, the human intestine expresses constitutively high levels of angiotensin-converting enzyme 2 (ACE2) and the transmembrane serine protease, which are needed for SARS-CoV-2 to gain entry into the cells. Consistently, elevated levels of fecal calprotectin have been documented in Covid19-infected patients with ongoing diarrhea even in the absence of fecal SARS-CoV-2 RNA [4]. Overall these findings suggest that SARS-CoV-2 infection can instigate an acute intestinal inflammation, which under specific circumstances (e.g. genetic susceptibility, exposure to environmental factors), can eventually evolve towards a chronic inflammatory disorder or potentially deteriorates the course of IBD [5]. The persistence of severe anemia and increased levels of inflammatory markers together with the marked mucosal inflammation, after clearance of the SARS-CoV-2, strongly support such a hypothesis.

* Corresponding author.
E-mail addresses: emma.calabrese@uniroma2.it, emmac@libero.it, emma.calabrese@uniroma2.it (E. Calabrese).

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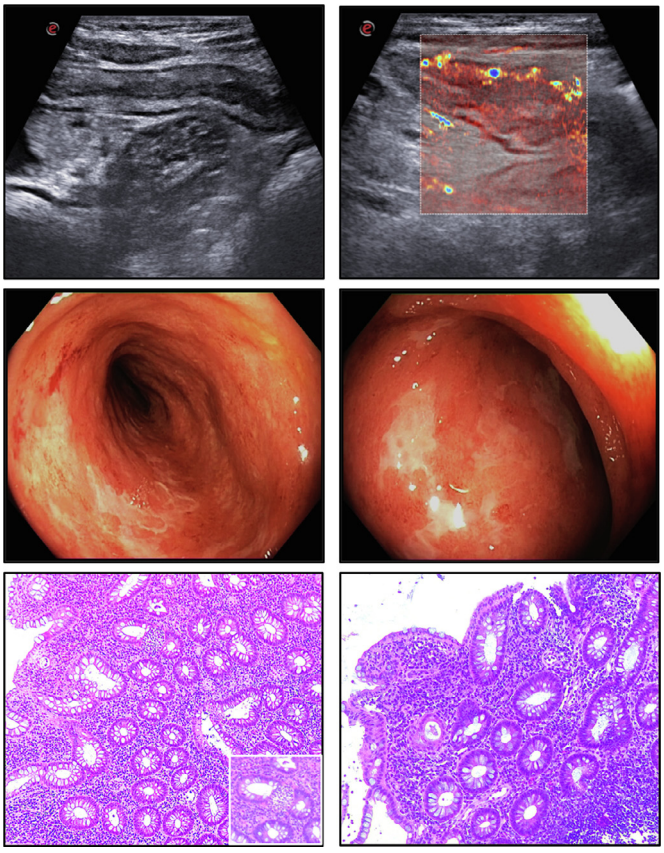

Fig. 1. Bowel ultrasonography (panels A–B), ileocolonoscopy (panels C–D) and histology (panels E–F) showing final diagnosis of inflammatory bowel disease.

Author contributions: EC, FZ, and GDVB evaluated the case, EC and GM drafted the manuscript.

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Informed consent was obtained from the patient to publish these images.

Declaration of Competing Interest

None

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