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Reply. We would like to thank the colleagues who have submitted correspondences in response to our clinical practice update, "Update on Management of Inflammatory Bowel Disease During the COVID-19 Pandemic"¹ and take this opportunity to reply to their inquiries.

Professors Barberio, Buda, and Savarino raise the important question as to whether patients with inflammatory bowel disease (IBD) should be tested for coronavirus disease 2019 (COVID-19) before starting a biological therapy, especially because the induction dosing used in most instances is a higher dose and /or regimen than that used for maintenance of disease control. Although we agree that there may be patients with asymptomatic severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infections among patients with moderately to severely active ulcerative colitis or Crohn's disease (the target group for biological therapy), the robust data that have emerged linking systemic corticosteroids to poor patient outcomes in COVID-19,^{2,3} along with the realization that, in the absence of biologics, systemic corticosteroids have traditionally been the "go-to drugs" support a general approach of not delaying these nonsteroid therapies. Our position was reiterated by the International Organization for the Study of Inflammatory Bowel Diseases.⁴ The American Gastroenterological Association's excellent guidelines on the topic of community based asymptomatic screening for SARS-CoV-2 infection,⁵ as well as other critical analyses of testing in the absence of a known exposure raise additional concerns about rates of false-positive and -negative results, and their implications.⁶

In a second correspondence, our Italian colleagues Bezzio and Saibeni relate a case of a patient with severely active IBD who, despite initially testing negative for SARS-CoV-2 upon admission to the hospital, subsequently received intensive corticosteroid therapy and developed symptoms that were consistent with active COVID-19 (supported by a second SARS-CoV-2 test that was positive). They adeptly point out that these patients often need a multidisciplinary approach to their IBD care, with expert input from the gastroenterologists, surgeons, and infectious disease specialists to ensure an optimal outcome. We agree, and emphasize that this case report further supports for our contention to avoid delays in initiating nonsteroid therapies in patients with moderately to severely active IBD.

The final correspondence from Drs Mago, Vaziri, and Tadros highlights the dilemma of detecting active IBD in the setting of a patient with Crohn's disease or ulcerative colitis who may be infected with the SARS-CoV-2; both conditions may result in an elevation in fecal calprotectin and, owing to the identification of SARS-CoV-2 in the stool of infected individuals,⁷ routine sigmoidoscopy or colonoscopy may unnecessarily expose the medical staff to the coronavirus.⁵ We agree with the suggestion that obtaining a fecal calprotectin

may be helpful in that a normal or mildly elevated level (the authors suggest that a threshold level of $<100 \ \mu g/g$ would be a reasonable limit)⁸ would suggest that the patient does not have active IBD and, therefore, obviate the need for urgent endoscopic evaluation.

Obviously, our experience with COVID-19 in the IBD population is evolving, and so should our clinical management strategies.⁹ We appreciate these contributions from our global colleagues.

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Conflicts of interest

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Drug-induced Acute Pancreatitis: Anecdotal Evidence vs Prospective Evaluation



Dear Editors:

We have read with great interest the analysis by Meczker et al¹ reporting on 1060 patients with drug-induced acute pancreatitis (DIAP). The authors meticulously collected data from worldwide published case reports and case series of DIAP