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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Baseline Disease Activity and Steroid Therapy Stratify Risk of COVID-19 in Patients With Inflammatory Bowel Disease

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New York City is the epicenter of the US coronavirus disease 2019 (COVID-19) pandemic caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) with local population infection rates estimated at 25%.[1] The impact of COVID-19 on patients with inflammatory bowel disease (IBD) within an epicenter is not well understood. Our study aims were to compare clinical outcomes between COVID-19 patients with and without IBD and to investigate the prevalence and risk factors of COVID-19 in IBD patients.

Methods

A matched cohort design was used to compare clinical outcomes in COVID-19 patients with or without IBD. The source cohort of all COVID-19-positive patients at 2 New York hospitals has been described previously[2] (Supplementary Material). The exposure of interest was defined as a pre-existing diagnosis of IBD. Cases (COVID-19 patients with IBD) were matched for decade of age and sex in a 1:2 ratio to unexposed controls (COVID-19 patients without IBD). Outcomes of interest were clinical manifestations of COVID-19, and intensive care unit admission, endotracheal intubation, and death among admitted patients. COVID-19 was defined as confirmed (positive SARS-CoV-2 polymerase chain reaction), or highly suspected (new-onset fever >37.8°C and more than 1 new symptom, including cough, sore throat, dyspnea, anosmia, or diarrhea, with a known close contact with COVID-19).[3]

A separate longitudinal cohort of active IBD patients was used to estimate the prevalence of COVID-19 in IBD patients and evaluate the effects of disease activity and treatment on risk of COVID-19 infection. Exposures of interest were IBD type, clinical, biochemical, and endoscopic indices of disease activity, and IBD treatment. The outcome of interest was diagnosis of COVID-19, as defined above. The details of methods and description of cohorts are available in the Supplementary Material.

Results

Eighty confirmed or highly suspected COVID-19 cases with IBD were matched with 160 COVID-19 controls without IBD. Disease characteristics for the IBD cases are reported in Supplementary Table 1. IBD cases and controls had similar prevalence of comorbidities (Table 1), except IBD cases had significantly lower body mass index, chronic obstructive pulmonary disease, and asthma, but higher prevalence of malignancy and immunosuppressive medication use. At presentation, vital signs, hypoxemia, and inflammatory markers were similar between cases and controls (data not shown). IBD cases more frequently presented with gastrointestinal symptoms of diarrhea (45% vs 19%; $P < .001$), and abdominal pain (20% vs 5%; $P = .001$) compared with non-IBD matched controls (Table 1). The primary outcome, a composite of death, ICU admission, or intubation, was similar but numerically lower in IBD cases compared with matched controls (24% vs 35%; $P = .352$) (Table 2). Among IBD cases, diagnosis of ulcerative colitis (UC) was associated with emergency department visit or admission (adjusted odds ratio, 12.7; $P = .009$) in multivariable analysis adjusted for age, fever, and gastrointestinal symptoms. Additionally, the proportion of patients on vedolizumab or receiving no biologic therapy was numerically higher among IBD cases needing emergency department visit or hospitalization compared with those who did not (no biologic: 29%; vedolizumab: 30%; ustekinumab 8%; tumor necrosis factor antagonist: 6%; $P = .197$; Supplementary Tables 2 and 3).

In a separate longitudinal cohort of active IBD patients (n = 119; median age was 44 years, 66 were female, 65 had Crohn's disease, and 54 had UC), 24.4% (n = 29) met criteria for COVID-19 (9 confirmed and 20 highly suspected), consistent with rates estimated in the general population of New York City (Table 3).[1,4] The distribution of age, sex, race/ethnicity, smoking, IBD type, disease location, or extra-intestinal manifestations was similar between the IBD patients with or without COVID-19. New-onset diarrhea (19.3% vs 11.1%; $P < .001$) and abdominal pain (12.6% vs 8.9%; $P =$

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Abbreviations used in this paper: **COVID-19, coronavirus disease 2019; IBD, inflammatory bowel disease; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; UC, ulcerative colitis.**

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Table 1. Characteristics of Ambulatory and Inpatient Patients With COVID-19 and Inflammatory Bowel Disease and Matched Controls

Variable	Total (N = 240)	IBD		*P* value
		Controls (n = 160)	Cases (n = 80)	
Age, *y*, mean (SD)	48.7 (17.9)	48.9 (17.7)	48.3 (18.3)	NA
Body mass index, kg/m,^{2} mean (SD)	27.5 (8.1)	29.2 (10)	25.7 (5.2)	.048
Inpatient, n (%)	51 (21.3)	34 (21.3)	17 (21.3)	NA
Male, n (%)	135 (56.3)	90 (56.3)	45 (56.3)	NA
Comorbidities, n (%)				
Hypertension	52 (21.7)	38 (23.8)	14 (17.5)	.248
Diabetes	24 (10)	20 (12.5)	4 (5)	.074
Chronic kidney disease	12 (5)	7 (4.4)	5 (6.3)	.542
Cardiovascular disease	15 (6.3)	10 (6.3)	5 (6.3)	1
COPD/asthma	21 (8.8)	19 (11.9)	2 (2.5)	.032
Obstructive sleep apnea	2 (0.8)	1 (0.6)	1 (1.3)	.624
Venous thromboembolism	6 (2.5)	3 (1.9)	3 (3.8)	.396
Cancer	13 (5.4)	4 (2.5)	9 (11.3)	.012
Chronic liver disease	7 (2.9)	2 (1.3)	5 (6.3)	.054
Solid organ transplantation	2 (0.8)	1 (0.6)	1 (1.3)	.624
Presenting symptoms, n (%)				
Fever	156 (65)	103 (64.4)	53 (66.3)	.775
Cough	150 (62.5)	96 (60)	54 (67.5)	.237
Shortness of breath	93 (38.8)	70 (43.8)	23 (28.8)	.028
Myalgia/fatigue	51 (21.3)	44 (27.5)	7 (8.8)	.002
Anorexia	33 (13.8)	26 (16.3)	7 (8.8)	.11
Altered mental status	5 (2.1)	5 (3.1)	0 (0)	.114
Nausea	36 (15)	24 (15)	12 (15)	1
Vomiting	20 (8.3)	10 (6.3)	10 (12.5)	.103
Diarrhea	67 (27.9)	31 (19.4)	36 (45)	<.001
Abdominal pain	24 (10)	8 (5)	16 (20)	.001
Anosmia	14 (5.8)	7 (4.4)	7 (8.8)	.179
Dysgeusia	11 (4.6)	7 (4.4)	4 (5)	.814
Medication history, n (%)				
Chronic steroids	11 (4.6)	1 (0.6)	10 (12.5)	.004
Immunosuppressant	22 (9.2)	0 (0)	22 (27.5)	<.001
Statin	19 (7.9)	15 (9.4)	4 (5)	.224

NOTE. *P* values are calculated using conditional logistic regressions or Mantel–Haenszel test for in matched controlled cohort, and Student *t* and χ^2 tests in SMART-IBD cohort.
COPD, chronic obstructive pulmonary disease; NA, not applicable.

.03) were significantly more frequent in IBD patients with COVID-19 than without. A higher proportion of IBD patients with COVID-19 had clinically active UC (92.9% vs 62.5%; $P =$ .035), endoscopically active Crohn's disease (92.3% vs 45.7%; $P = .004$) or UC (85.7% vs 48.4%; $P = .018$), and elevated baseline biomarker levels (C-reactive protein >0.9 mg/dL; $P = .01$; fecal calprotectin >50 μg/mg; $P = .002$) compared with those without COVID-19. Proportional baseline corticosteroid use was higher among COVID-19 patients ($P = .04$), but no overall differences were noted based on biologic, immunomodulator, or aminosalicylate use. Of 83 patients receiving biologic therapy, COVID-19 infection was similar across therapeutic classes ($P = .315$), with fewer overall cases among patients on ustekinumab (13.8%) compared with vedolizumab (30.4%), tumor necrosis factor antagonists (25.0%), or tofacitinib (42.9%).

Discussion

With the onset of the COVID-19 pandemic, there was initial concern that IBD and immunosuppressive medications would place patients at high risk for infection with and complications from SARS-CoV2. Using one of the largest reported cohorts of COVID-19–positive patients, this matched case–control analysis reveals IBD patients did not experience more severe COVID-19. Older age was a risk for emergency care or hospitalization. Although UC was associated with greater risk of severe disease in our cohort, neither baseline IBD activity nor biologic medication predicted need for higher level of care. Reflecting lower rates of obesity and pulmonary disease, IBD patients with COVID-19 experienced less dyspnea or severe outcomes than matched non-IBD controls. However, the increased prevalence of gastrointestinal manifestations of COVID-19[5] within the IBD population highlights

Table 2. Clinical Outcomes of Patients With Inflammatory Bowel Disease Admitted With COVID-19 and Matched Controls

Outcome	Total (n = 51)	IBD		P value
		Controls (n = 34)	Cases (n = 17)	
ICU admission	14 (27.5)	11 (32.4)	3 (17.6)	.226
Death	2 (3.9)	2 (5.9)	0 (0)	.221
Intubation	13 (25.5)	11 (32.4)	2 (11.8)	.117
ICU admission, intubation or death	16 (31.4)	12 (35.3)	4 (23.5)	.352
Length of stay, *d*, (median [SD])	6 (2.9)	6 (3.4)	6 (1.7)	.426

NOTE. Data are n (%). *P* values are calculated using conditional logistic regressions or Mantel–Haenszel test for in matched controlled cohort, and Student *t* and χ^2 tests in SMART-IBD cohort.
ICU, intensive care unit.

Table 3. Characteristics of the Longitudinal Inflammatory Bowel Disease Cohort

Variable	Total of IBD cases (N = 119)	COVID-19		P value
		Yes (n = 29)	No (n = 90)	
Age				.295
<40 y	51 (42.9)	10 (34.5)	41 (45.6)	
>40 y	68 (57.1)	19 (65.5)	49 (54.4)	
Male	53 (44.5)	12 (41.4)	41 (45.6)	.694
Smoking status				.238
Never smoker	84 (70.6)	18 (56)	66 (68)	
Current	11 (9.2)	2 (6.9)	9 (10)	
Former	24 (20.2)	9 (20.2)	15 (31)	
IBD type				0.291
UC	46 (38.7)	14 (48.3)	32 (35.6)	
Crohn's disease	69 (58)	15 (51.7)	54 (60)	
Unclassified	4 (3.4)	0 (0)	4 (4.4)	
Biomarkers				
C-reactive protein >0.9 g/dL	35 (29.4)	14 (48.2)	21 (23.3)	.010
Fecal calprotectin >50 μg/mg	66 (64.7)	24 (88.9)	42 (56)	.002
Symptoms				
New diarrhea	23 (19.3)	13 (44.8)	10 (11.1)	<.001
New abdominal pain	15 (12.6)	7 (24.1)	8 (8.9)	.031
Treatment[a]				
Biologic therapy				.804
Yes	84 (70.6)	21 (72.4)	63 (70)	
No	35 (29.4)	8 (27.6)	27 (30)	
Steroids	35 (29.4)	13 (44.8)	22 (24.4)	<.036
Budesonide	22 (18.5)	6 (20.7)	16 (17.8)	.73
Aminosalicylate	38 (31.9)	11 (37.9)	27 (30.9)	.43
Immunomodulators	5 (4.2)	2 (6.9)	3 (3.3)	.41
Combination therapy	4 (3.4)	2 (6.9)	2 (2.2)	.25
Patients with Crohn's disease				
Clinical disease activity				.060
HBI >4	51 (75)	14 (93.3)	37 (69.8)	
HBI ≤4	17 (25)	1 (6.7)	16 (30.2)	
Endoscopic disease activity				.004
SES-CD >6	28 (58.3)	12 (92.3)	16 (45.7)	
SES-CD ≤6	20 (41.7)	1 (7.7)	19 (54.3)	
Patients with ulcerative colitis				
Clinical disease activity				.035
PMS >1	33 (71.7)	13 (92.9)	20 (62.5)	
PMS ≤1	13 (28.3)	1 (7.1)	12 (37.5)	
Endoscopic disease activity				.018
MES >1	27 (60)	12 (85.7)	15 (48.4)	
MES ≤1	18 (40)	2 (14.3)	16 (51.6)	

NOTE. Data are n (%). *P* values are calculated using conditional logistic regressions or Mantel–Haenszel test for in matched controlled cohort, and Student *t* test and χ^2 tests in SMART-IBD cohort.
HBI, Harvey Bradshaw Index; MES: Mayo endoscopic subscore; PMS: partial Mayo score; SES-CD, Simple Endoscopic Score-Crohn's disease.
[a]Biologic therapy includes anti–tumor necrosis factor medications, Vedolizumab, ustekinumab, and tofacitinib; steroids and budesonide include both oral and rectal formulations; aminosalicylates include mesalamine and sulfasalazine; Immunomodulators include azathioprine, 6- mercaptopurine, and methotrexate.

the need for COVID-19 evaluation in IBD patients with new gastrointestinal symptoms.

These data provide early evidence tracking incident infection and clinical COVID-19 in a longitudinal IBD cohort. Despite similar overall infection rates in IBD patients and the general pandemic epicenter population, moderate-to-severe IBD activity and corticosteroid use were found to be associated with higher rates of COVID-19. Limitations in SARS-CoV-2 testing and asymptomatic carriage might underestimate the true prevalence in this cohort. These data support societies' guidelines to continue effective steroid-sparing IBD therapy in the epicenter of a pandemic to minimize active disease.

Our data support the emerging idea that IBD and/or medications used for its treatment are not associated with severe outcomes in COVID-19.[3,4] Within the IBD subset of the inpatient cohort, severe sequelae of COVID-19 were lower than in matched non-IBD controls. Despite the small number of admitted IBD patients, these findings are consistent with a possible blunting of the cytokine release syndrome, associated with severe morbidity and mortality in COVID-19,[6] by altered immune function or immunosuppressive therapy, which can limit disease progression. No significant differences were detected regarding biologic type on COVID-19 risk. These data support the need for further study of intestinal inflammation associated with SARS-CoV-2 infection and gastrointestinal symptoms.[7,8]

Supplementary Material

Note: To access the supplementary material accompanying this article, visit the online version of *Gastroenterology* at www.gastrojournal.org, and at https://doi.org/10.1053/j.gastro.2020.05.066.

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Supplementary Methods

Matched Cohort Study

The source cohort consisted of 1386 with confirmed or highly suspected COVID-19 patients who presented to our tertiary academic center in New York (including Jill Roberts Center for IBD) or an affiliated smaller nonacademic hospital in New York, between February 1 and April 30, 2020.

COVID-19 was defined as confirmed (positive SARS-CoV-2 polymerase chain reaction), or highly suspected (new-onset fever >37.8°C and more than 1 new symptom including cough, sore throat, dyspnea, anosmia, and diarrhea, with a known close contact with COVID-19). Cases consisted of COVID-19 patients with a diagnosis of IBD (exposed, n = 80), and were matched in a 1:2 ratio to the COVID-19 patients without IBD (nonexposed, n = 160) according to their decade of age and sex. Demographic and clinical data including comorbidities relevant to COVID-19 were extracted. Patients were considered to have gastrointestinal manifestations at presentation if they reported any of the symptoms of nausea, vomiting, diarrhea, or abdominal pain at time of presentation. For IBD patients, clinical disease activity at baseline was determined using the Harvey Bradshaw Index for Crohn's disease or the partial Mayo Score for UC. Endoscopic activity within 6 months was determined if a colonoscopy report was available and reported as Simple Endoscopic Score for Crohn's Disease or Mayo endoscopic subscore for UC.

The main exposure in the matched cohort study was presence of IBD. Outcomes of interest were clinical manifestations of COVID-19 on presentation (including fever, cough, dyspnea, myalgia or fatigue, anorexia, altered mental status, nausea, vomiting, diarrhea, abdominal pain, anosmia, and dysgeusia), as well as the composite of intensive care unit admission, endotracheal intubation, or death among admitted patients. Detailed definitions of individual variables have been described elsewhere.2 Among cases with IBD, the association between disease type or IBD treatment and need for emergency visit or admission was evaluated in secondary analysis. IBD treatment was categorized into biologic therapy (including tumor necrosis factor antagonists, vedolizumab, or ustekinumab), or no biologic use.

Longitudinal Cohort of Active Inflammatory Bowel Disease Patients

This consisted of 119 patients with active IBD who were followed prospectively at our center (SMART IBD longitudinal cohort). The exposures of interest were IBD type, clinical, biochemical, and endoscopic indices of disease activity, and IBD treatment. The outcome of interest was diagnosis of COVID-19, as defined above. Clinical IBD disease activity was determined using partial Mayo score for UC and Harvey Bradshaw Index for Crohn's disease. Biochemical disease activity was evaluated using C-reactive protein, albumin, and fecal calprotectin levels. Endoscopic disease activity was evaluated using last recorded Simple Endoscopic Score for Crohn's Disease or Mayo endoscopic subscore for UC, and baseline medications used to treat IBD were recorded, as for the matched cohort. IBD treatment was categorized into biologic therapy (including tumor necrosis factor antagonists, vedolizumab, or ustekinumab), oral or rectal steroids (excluding budesonide), oral or rectal budesonide, aminosalicylates (including mesalamine and sulfasalazine), immunomodulators (including azathioprine, 6- mercaptopurine, and methotrexate), and combination therapy (biologics plus immunomodulators)

Statistical Analysis

Descriptive statistics were reported as mean (SD), median (interquartile range), or counts and proportions. Conditional logistic regressions and Mantel–Haenszel test were used to compare variables between COVID-19 patients with and without IBD in the matched cohort, and multivariable logistic regression was used to evaluate the effect of IBD type and treatment on emergency visit or hospitalization. In longitudinal IBD cohort, groups were compared using χ^2 test for categorical variables and Student t test for continuous variables. All analyses were based on nonmissing data and missing data were not imputed. All tests were 2-tailed with a significance level of $\alpha = .05$. Analyses were performed with STATA, version 13.0 for Windows (StataCorp LP, College Station, TX) and SPSS, version 25.0 (IBM Corp, Armonk, NY).

Supplementary Table 1. Disease Characteristics of COVID-19 Cases With Inflammatory Bowel Disease

Characteristic	Data,[a] n (%)
IBD type	
UC	26 (40.6)
CD	38 (59.4)
CD	
CD location	
Ileal	7 (18.4)
Colonic	8 (21.1)
Ileocolonic	22 (57.9)
Perianal disease	8 (21.1)
Prior intestinal resection	18 (47.4)
Clinical disease activity CD	
Active (HBI >4)	24 (63.2)
Moderate to severe (HBI >7)	15 (41.7)
Endoscopic disease activity CD	
Active (SES-CD >4)	22 (84.6)
Moderate to severe (SES-CD >6)	19 (73.1)
UC	
UC extent	
Proctitis	2 (7.7)
Left-sided	8 (30.8)
Pancolitis	16 (61.5)
Ostomy or pouch	3 (11.5)
Clinical disease activity UC	
Active (PMS >1)	13 (52)
Moderate to severe PMS >4)	5 (20)
Endoscopic disease activity UC	
Active (MES >0)	16 (61.5)
Moderate to severe (MES >1)	14 (53.8)
C-reactive protein >0.9 mg/dL	16 (25.8)
Fecal calprotectin >50 μg/mg	26 (48.1)
Biologic therapy	
Tumor necrosis factor antagonist	16 (25)
Vedolizumab	10 (15.6)
Ustekinumab	12 (18.8)
Tofacitinib	1 (1.6)
Dual (vedolizumab + tofacitinib)	1 (1.6)
Trial drug	3 (4.7)
None	21 (32.8)
Thiopurines (6-mercaptopurine, azathioprine)	4 (6.3)
Methotrexate	3 (4.7)
Combination therapy	4 (6.3)
Oral/rectal aminosalicylate	20 (31.3)
Any steroid	13 (20.3)
Comorbidity[a]	21 (32.8)

CD, Crohn's disease; HBI, Harvey Bradshaw Index; MES, Mayo endoscopic subscore; PMS, partial Mayo score; SES-CD, Simple Endoscopic Score-CD.
[a]Data available on 64 of the 80 cases.

Supplementary Table 2. Emergency Department Visit or Hospitalization According to Inflammatory Bowel Disease Treatment Among COVID-19 Cases With Inflammatory Bowel Disease

		ED visit or hospitalization		
Biologic use[a]	Total, n	Yes, n (%) (n = 11)	No, n (%) (n = 48)	*P* value
No biologic	21	6 (28.6)	15 (71.4)	.197
Tumor necrosis factor antagonist	16	1 (6.3)	15 (93.8)	—
Vedolizumab	10	3 (30)	7 (70)	—
Ustekinumab	12	1 (8.3)	11 (91.7)	—

ED, emergency department.
[a]Details of treatment available for 64 of 80 cases. Three patients on clinical trials, 1 patient on tofacitinib, and 1 patient on dual biologic were excluded due to small sample sizes.

Supplementary Table 3. COVID-19 Diagnosis According to Inflammatory Bowel Disease Treatment in the Longitudinal Inflammatory Bowel Disease Cohort

		COVID diagnosis		
Biologic class	Total, n (N = 83)	Yes, n (%) (n = 20)	No, n (%) (n = 63)	*P* value
Tumor necrosis factor antagonist	24	6 (25)	18 (75)	.315
Vedolizumab	23	7 (30.4)	16 (69.6)	—
Ustekinumab	29	4 (13.8)	25 (86.2)	—
Tofacitinib	7	3 (42.9)	4 (57.1)	—