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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Clinical Features and Outcomes of Coronavirus Disease 2019 (COVID-19) Patients With Chronic Hepatitis B Virus Infection

Dear Editor:

We read with great interest the recent article published in *Clinical Gastroenterology and Hepatology* by Fan et al,[1] in which they described the characteristics of coronavirus disease 2019 (COVID-19)–related liver damage. Since the outbreak of COVID-19, liver injury has attracted widespread attention, which might be caused by pre-existing liver disease, virus infection of liver cells, and certain medications. The effect of virus infection and antiviral drugs on liver injury has been considered, however, the report and diagnosis on chronic liver disease is insufficient in this emergent situation. In a recent large cohort study, only 1.3% of COVID-19 patients recorded a history of chronic viral hepatitis,[2] which was significantly lower than the population prevalence in China.[3] Patients with pre-existing liver disease are a high-risk population for COVID-19. In this study, we aimed to report the clinical course of COVID-19 patients with chronic hepatitis B virus (HBV) infection and provide a reference for clinical treatment of patients.

From January 24, 2020, to February 29, 2020, patients with confirmed COVID-19 and chronic HBV infection were admitted to 2 designated hospitals for COVID-19. COVID-19 was confirmed by the detection of severe acute respiratory syndrome coronavirus 2 RNA in throat swabs by reverse-transcription polymerase chain reaction. Patients with abnormal liver enzyme levels at admission or a history of chronic liver diseases might undergo HBV assays. HBV infection was defined by a positive test result for hepatitis B surface antigen. HBV-infected patients are classified as hepatitis B virus carriers, chronic hepatitis B, and hepatitis B cirrhosis.[4] We defined liver injury as any parameter exceeding the upper limit of normal value of alanine aminotransferase (ALT), aspartate aminotransferase (AST), and total bilirubin (TBIL).[5] The definition of severity degree and the clinical management of COVID-19 patients were in accordance with the practice guidelines issued by China.[6] This study was approved by ethics commissions, and written informed consent was waived.

All of the patients in this study had a COVID-19 exposure history. Of the 23 patients, the mean age was 44.7 $\pm$ 11.5 years, and 15 (65.2%) were male. Only 6 (26.1%) patients reported a history of being a HBV carrier, 4 (17.4%) patients reported a history of chronic hepatitis B, but the remaining patients (56.5%) denied a history of HBV infection. The patients reported no other underlying diseases. However, laboratory tests at admission showed that 15 patients were HBV carriers, 7 (30.4%) had chronic hepatitis B, and 1 (4.4%) had hepatitis B cirrhosis. At admission, 6 (26.0%) patients had liver test result abnormalities, of which 2 patients were HBV carriers, 3 patients had chronic hepatitis B, and 1 patient had hepatitis B cirrhosis. Ten patients had increased liver enzyme levels during hospitalization, with AST, ALT, and TBIL ranges of 44 to 277 U/L, 52 to 575.1 U/L, and 17.5 to 309.18 μmol/L, respectively. All patients were mild/moderate on admission, but 3 (13.0%) patients progressed to severe, and 2 (8.7%) progressed to critically ill. The 23 patients were treated with antiviral drugs and 13 were treated with liver-protecting drugs. After treatment, all patients were discharged. The comparison of HBV carriers and patients with chronic hepatitis B/hepatitis B cirrhosis is shown in Table 1. The results showed no significant differences in all clinical features except for sex, exposure history, activated partial thromboplastin time, AST, ALT, γ-glutamyl transpeptidase (GGT), TBIL, and direct bilirubin. Notably, no differences were found in disease severity or length of hospital stay between these 2 groups.

Table 1. Comparison of Clinical Characteristics and Laboratory Findings Between COVID-19 Patients With HBV Carriers and Hepatitis B/Cirrhosis

Characteristics	Total	HBV carriers (n = 15)	Hepatitis B/cirrhosis (n = 8)	*P* value
Age, *y*, mean (SD)	44.7 (11.5)	47.1 (11.0)	40.0 (11.6)	.165
Sex, n (%)				
Male	15 (65.2)	7 (46.7)	8 (100)	.019
Female	8 (34.8)	8 (53.3)	0	
Exposure history, n (%)				
Contact with confirmed patient	13 (56.5)	11 (73.3)	2 (25.0)	.039
Contact with a resident from Hubei	10 (43.5)	4 (26.7)	6 (75.0)	
Disease severity, n (%)				
Moderate	18 (78.3)	12 (80.0)	6 (75.0)	.088
Severe	3 (13.0)	3 (20.0)	0	
Critically ill	2 (8.7)	0	2 (25.0)	
Blood routine test				
White blood cell count, $\times 10^9$/L, mean (SD)	6.3 (1.9)	6.7 (2.0)	5.5 (1.4)	.170
Neutrophil count, $\times 10^9$/L, mean (SD)	4.4 (1.7)	4.6 (1.8)	4.1 (1.2)	.474
Lymphocyte count, $\times 10^9$/L, mean (SD)	1.4 (0.5)	1.5 (0.5)	1.2 (0.5)	.140
Lymphocyte count, n (%)				
$<1.1 \times 10^9$/L	7 (30.4)	4 (26.7)	3 (37.5)	.657
$1.1–3.2 \times 10^9$/L	16 (69.6)	11 (73.3)	5 (62.5)	
Hemoglobin level, *g/L*, mean (SD)	138.1 (22.5)	132.8 (22.5)	148 (20.3)	.126
Platelet count, $\times 10^9$/L, mean (SD)	192.8 (53.1)	201.3 (53.0)	176.9 (52.9)	.303
Infection-related biomarkers				
C-reactive protein level, *mg/L*, mean (SD)				
C-reactive protein, n (%)				
<5.0 mg/L	15 (65.2)	10 (66.7)	5 (62.5)	1.000
≥5.0 mg/L	8 (34.8)	5 (33.3)	3 (37.5)	
Procalcitonin level, *ng/mL*, mean (SD)	0.2 (0.3)	0.1 (0.1)	0.4 (0.4)	.065
Procalcitonin, n (%)				
0–0.1 ng/mL	12 (52.2)	10 (66.7)	2 (25.0)	.074
>0.1 ng/mL	10 (43.5)	4 (26.7)	6 (75.0)	
Not available	1 (4.4)	1 (6.7)	0	
Coagulation function				
Prothrombin time, *s*, median (interquartile range)	13.2 (11.8–13.2)	12.3 (11.6–13.0)	14.8 (12.3–13.4)	.086
APTT, *s*, mean (SD)	33.5 (10.7)	29.0 (6.6)	42.6 (11.8)	.003
D-dimer, *μg/mL*, median (interquartile range)	0.8 (0.16–0.49)	0.4 (0.11–0.47)	1.6 (0.16–0.54)	.408
D-dimer, n (%)				
0–0.5 μg/mL	17 (73.9)	12 (80.0)	5 (62.5)	.549
>0.5 μg/mL	5 (21.7)	3 (20.0)	2 (25)	
Not available	1 (4.4)	0	1 (12.5)	
Blood biochemistry				
Lactate dehydrogenase level, *U/L*, mean (SD)	192.1 (76.7)	182 (45.8)	206.2 (112.4)	.614
Creatine kinase level, *U/L*, median (interquartile range)	88.4 (43.3–70.8)	58.5 (39.0–70.5)	143.9 (43.0–152.0)	.968
Creatine kinase MB, *U/L*, mean (SD)	10.4 (10.5)	11.3 (9.8)	8.8 (12.2)	.610
Myoglobin level, *ug/L*, median (interquartile range)	48.0 (19.7–38.1)	29.1 (19.7–37.0)	83.3 (20.1–48.5)	.317
Serum creatinine level, *μmol/L*, mean (SD)	74.6 (16.3)	70.4 (13.7)	82.4 (18.8)	.093
Serum uric acid level, *μmol/L*, mean (SD)	300.2 (87.7)	303.9 (90.5)	293.1 (87.7)	.785
Serum uric acid level, n (%)				
Male, <208.3 μmol/L; female, <149 μmol/L	2 (8.7)	1 (6.7)	1 (12.5)	1.000
Male, 208.3–428.4 μmol/L; female, 149–369 μmol/L	18 (78.3)	12 (80.0)	6 (75.0)	
Male, >428.4 μmol/L; female, >369 μmol/L	3 (13.0)	2 (13.3)	1 (12.5)	
Glucose level, *mmol/L*, mean (SD)	7.1 (3.0)	7.5 (3.6)	6.5 (1.4)	.443
Glucose, n (%)				
4.3–5.9 mmol/L	9 (39.1)	7 (46.7)	2 (25.0)	.596
>5.9 mmol/L	13 (56.5)	7 (46.7)	6 (75.0)	
Not available	1 (4.4)	1 (6.7)	0	
HBV serum markers				
HBsAg level, n (%)				
<0.05 IU/mL	1 (4.3)	0	1 (12.5)	.585
≥0.05 IU/mL	21 (91.3)	14 (93.3)	7 (87.5)	
Not available	1 (4.3)	1 (6.7)	0	

Table 1. Continued

Characteristics	Total	HBV carriers (n = 15)	Hepatitis B/cirrhosis (n = 8)	*P* value
HBsAb level, n (%)				
<10.00 mIU/mL	20 (87.0)	12 (80.0)	8 (100.0)	.684
≥10.00 mIU/mL	2 (8.7)	2 (13.3)	0	
Not available	1 (4.3)	1 (6.7)	0	
HBeAg level, signal-to-cutoff, n (%)				
<1.00	18 (78.3)	13 (86.7)	5 (62.5)	.168
≥1.00	4 (17.4)	1 (6.7)	3 (37.5)	
Not available	1 (4.3)	1 (6.7)	0	
HBeAb level, signal-to-cutoff, n (%)				
>1.00	6 (26.1)	2 (13.3)	4 (50.0)	.131
≤1.00	16 (69.6)	12 (80.0)	4 (50.0)	
Not available	1 (4.3)	1 (6.7)	0	
HBcAb level, signal-to-cutoff, n (%)				
<1.00	2 (8.7)	0	2 (25.0)	.111
≥1.00	20 (87.0)	14 (93.3)	6 (75.0)	
Not available	1 (4.3)	1 (6.7)	0	
Liver function test				
AST level, *U/L*, median (interquartile range)	31.6 (15.0–36.8)	22.0 (14.5–30.0)	51.0 (20.6–69.1)	.028
AST level, n (%)				
Male, 14–40 U/L; female, 13–35 U/L	19 (82.6)	15 (100)	4 (50.0)	.008
Male, >40 U/L; female, >35 U/L	4 (17.4)	0	4 (50.0)	
ALT level, *U/L*, median (interquartile range)	38.6 (17.0–42.0)	23.4 (13.0–25.0)	67.1 (20.3–106.9)	.030
ALT level, n (%)				
Male, 9–50 U/L; female, 7–40 U/L	19 (82.6)	14 (93.3)	5 (62.5)	.103
Male U/L, >50; female, >40 U/L	4 (17.4)	1 (6.7)	3 (37.5)	
GGT level, *U/L*, median (interquartile range)	32.3 (13.5–41.0)	23.1 (9.8–25.8)	48.4 (27.5–67.3)	.024
GGT level, n (%)				
Male, 10–60 U/L; female, 7–45 U/L	17 (73.9)	12 (80)	5 (62.5)	.549
Male, >60 U/L; female, >45 U/L	5 (21.7)	2 (13.3)	3 (37.5)	
Not available	1 (4.4)	1 (6.7)	0	
ALP level, *U/L*, mean (SD)	73.0 (24.7)	70.0 (23.2)	78.1 (27.9)	.471
ALP, n (%)				
40–129 U/L	20 (87.0)	13 (86.7)	7 (87.5)	1.000
>129 U/L	2 (8.7)	1 (6.7)	1 (12.5)	
Not available	1 (4.4)	1 (6.7)	0	
TBIL level, *μmol/L*, median (interquartile range)	24.9 (7.2–13.9)	10.2 (6.7–11.8)	50.6 (10.0–18.3)	.014
TBIL level, n (%)				
3.4–17.1 μmol/L	18 (78.3)	13 (86.7)	5 (62.5)	.168
>17.1 μmol/L	4 (17.4)	1 (6.7)	3 (37.5)	
Not available	1 (4.4)	1 (6.7)	0	
DBIL level, *μmol/L*, median (interquartile range)	12.9 (2.8–5.4)	3.2 (2.0–4.0)	29.8 (3.6–8.5)	.014
DBIL level, n (%)				
0–5 μmol/L	17 (73.9)	13 (86.7)	4 (50.0)	.033
>5 μmol/L	5 (21.7)	1 (6.7)	4 (50.0)	
Not available	1 (4.4)	1 (6.7)	0	
Albumin level, *g/L*, mean (SD)	42.3 (8.7)	42.1 (7.0)	42.5 (11.9)	.921
Globulin level, *g/L*, mean (SD)	26.4 (4.7)	25.8 (4.1)	27.4 (5.8)	.448
Treatment				
Antiviral drug	23 (100.0)	18 (100.0)	5 (100.0)	
Interferon	20 (87.0)	12 (80.0)	8 (100.0)	.526
Arbidol (Jiangsu, China)	10 (43.5)	5 (33.3)	5 (62.5)	.221
Peramivir	8 (34.8)	5 (33.3)	3 (37.5)	1.000
Lopinavir/ritonavir	7 (30.4)	3 (20)	4 (50.0)	.182
Chloroquine	6 (26.1)	4 (26.7)	2 (25.0)	1.000
Ribavirin	6 (26.1)	4 (26.7)	2 (25.0)	1.000
Oseltamivir	2 (8.7)	1 (6.7)	1 (12.5)	1.000
Liver-protecting drug	13 (56.5)	7 (46.7)	6 (75.0)	.379
Diammonium glycyrrhizinate	8 (34.8)	5 (33.3)	3 (37.5)	1.000
Reduced glutathione	6 (26.1)	2 (13.3)	4 (50.0)	.131
Compound glycyrrhizin	3 (13.0)	2 (13.3)	1 (12.5)	1.000
Magnesium isoglycyrrhizinate	3 (13.0)	1 (6.7)	2 (25.0)	.269
Glucurolactone	1 (4.3)	0	1 (12.5)	.348

Table 1. Continued

Characteristics	Total	HBV carriers (n = 15)	Hepatitis B/cirrhosis (n = 8)	*P* value
Antibiotics	20 (87.0)	13 (86.7)	7 (87.5)	1.000
Herbal medicine	20 (87.0)	13 (86.7)	7 (87.5)	1.000
Glucocorticoid	6 (26.1)	5 (33.3)	1 (12.5)	.369
Noninvasive ventilation	3 (13.0)	2 (13.3)	1 (12.5)	1.000
Admission to intensive care unit	5 (21.7)	3 (20.0)	2 (25.0)	1.000
Comorbidities				
ARDS	2 (8.7)	1 (6.7)	1 (12.5)	1.000
Deep venous thrombosis	1 (4.3)	1 (6.7)	0	1.000
Upper gastrointestinal hemorrhage	1 (4.3)	0	1 (12.5)	.348
Liver failure	1 (4.3)	0	1 (12.5)	.348
Renal insufficiency	1 (4.3)	0	1 (12.5)	.348
Prognosis				
Discharged	23 (100.0)	15 (100.0)	8 (100.0)	.911
Length of hospitalization, *d*, mean (SD)	15.1 (7.4)	15.0 (7.8)	15.4 (7.0)	

NOTE. *P* values were calculated by *t* test, Mann–Whitney *U* test, chi-squared test, or the Fisher exact test as appropriate.
ALP, alkaline phosphatase; ALT, alanine transaminase; APTT, activated partial thromboplastin time; ARDS, acute respiratory distress syndrome; AST, aspartate aminotransferase; COVID-19, coronavirus disease 2019; DBIL, direct bilirubin; GGT, γ-glutamyl transpeptidase; HBcAb, hepatitis b core antigen; HBeAb, hepatitis be antibody; HBeAg, hepatitis be antigen; HBsAb, hepatitis b surface antibody; HBsAg, hepatitis b surface antigen; HBV, hepatitis B virus; MB, isoenzyme; TBIL, total bilirubin.

The 5 severe/critically ill patients (cases A–E) all were male. These 5 patients were admitted to the intensive care unit, 3 required noninvasive mechanical ventilation and all were treated with corticosteroids. During intensive care unit treatment, case A developed deep venous thrombosis, cases B and C developed acute respiratory distress syndrome, and case D developed upper gastrointestinal hemorrhage, liver failure, and renal insufficiency. Figure 1 shows follow-up liver function tests of the 5 patients during hospitalization. The

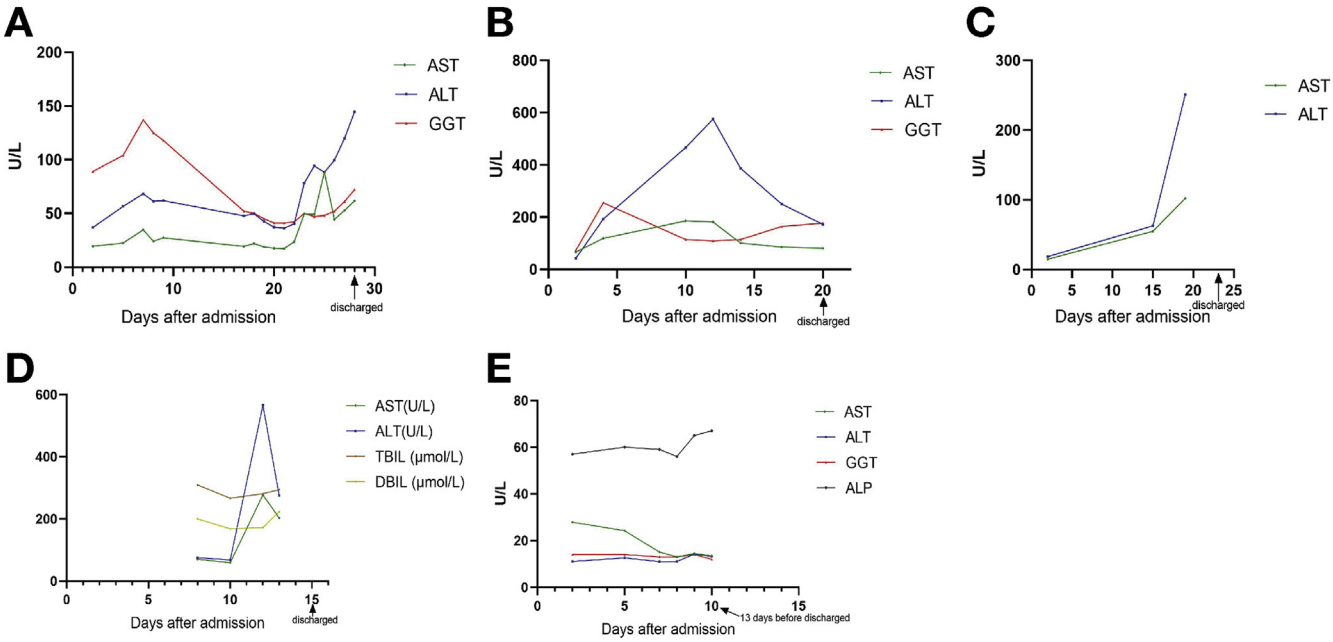

Figure 1. Temporal liver function test of cases A–E during hospitalization. (*A*) In patient A (male, 51 y; hepatitis B virus carrier, severe), AST and ALT levels increased to 3.2 times baseline in the first week and decreased to near normal on day 14 after admission with the use of liver-protecting drugs. However, 5 days later, AST and ALT levels increased again to 3.2 and 4.0 times baseline, respectively. (*B*) In case B (male, 29 y; chronic hepatitis B, critically ill), AST and ALT levels increased to 2.7 and 13.7 times baseline at day 12 after admission, and then decreased with the use of liver-protecting drugs. In the last test, AST and ALT levels were 1.2 and 4.1 times baseline, respectively. (*C*) In case C (male, 64 y; hepatitis B virus carrier, severe), AST and ALT levels increased continuously to 6.9 and 13.6 times baseline, respectively. (*D*) In case D (male, 44 y; hepatitis B cirrhosis, critically ill), AST and ALT levels increased to 4.0 and 7.5 times baseline at day 12 after admission, respectively, and then decreased to 1.7 and 1.9 times baseline owing to the use of liver-protecting drugs. TBIL and DBIL stabilized at a higher level, exceeding the upper limit of normal value. (*E*) In case E (female, 47 y; hepatitis B virus carrier, severe), liver function tests of the patient were normal. ALP, alkaline phosphatase; ALT, alanine transaminase; AST, aspartate aminotransferase; DBIL, direct bilirubin; GGT, γ-glutamyl transpeptidase; TBIL, total bilirubin.

increase in ALT level was significantly higher than AST. The ALT increase is the primary indicator of liver injury in COVID-19.[7,8] The GGT increase was not significant. Before discharge, liver function of the 5 patients could not return to baseline.

Because of the emergence of COVID-19, the report and diagnosis on chronic HBV infection is insufficient, therefore, the impact of HBV infection on liver injury might be underestimated. Most HBV carriers with COVID-19 will not progress to becoming severely or critically ill. However, 26% of patients had abnormal liver function test results at admission, 19% of whom progressed to being severely or critically ill, which was not associated with HBV infection status. Accordingly, we recommend dynamic monitoring of liver function in COVID-19 patients with liver test abnormalities at admission. ALT and AST levels, especially ALT levels, are preferred parameters that should be used to monitor liver function during hospitalization.

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Conflicts of interest
The authors disclose no conflicts.

Most current article

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COVID-19 and Inflammatory Bowel Disease: Questions on Incidence, Severity, and Impact of Treatment?

Dear Editor:

Since the emergence of coronavirus disease 2019 (COVID-19) (severe acute respiratory syndrome–associated coronavirus 2 [SARS-CoV-2]) infection in December 2019, numerous questions arise on the specific risk of COVID-19 in patients with inflammatory bowel disease (IBD), in particular those treated by immunosuppressants and/or biologics.

In a large cohort of IBD patients from Nancy University Hospital (France) and Humanitas (Milan, Italy), Allocca et al[1] reported 15 COVID-19–positive patients (9 Crohn's disease [CD] patients, 3 with active disease, 14 with immunosuppressants and/or biologics), representing a cumulative incidence of 0.0025; all had a favorable outcome (only 5 were hospitalized, not requiring intensive care support). During the same period, we identified 7 patients with proven infection. Four were male, median age was 54 years (34–60), 5 had CD, 5 had inactive disease, and 4 had comorbidities (obesity in 4, hypertension in 1, chronic pulmonary obstructive disease in 1). Diagnosis was based on polymerase chain reaction nasopharyngeal swab testing in 4 and enzyme-linked immunosorbent assay in 3. One patient was asymptomatic. Two needed short-term hospitalization without intensive care support. Two patients were on combination therapy (ustekinumab/azathioprine and vedolizumab/methotrexate), 1 had ustekinumab and 20 mg a day prednisolone for CD flare-up, 2 had anti–tumor necrosis factors (TNFs) (1 infliximab, 1 adalimumab), 1 had tofacitinib (10 mg twice a day), and 1 was on no treatment. Taken together, these results are reassuring for IBD patients and their treating physicians.

However, IBD patients with COVID-19 can have negative outcomes. Bezzio et al[2] reported endotracheal intubation in 8% and death in 8% of 79 COVID-19–infected IBD patients. Negative outcome was associated with age older than 65, comorbidities, and active disease.[2] The association with older age and comorbidities has also been reported in the international IOIBD SECURE registry (http://www.covidibd.org).[3,4] In describing the characteristics and outcomes of the first 525 cases in IOIBD SECURE registry, older age and having $\geq$2 comorbidities were positively associated with COVID-19 severity.[3] Systemic corticosteroids or mesalamine increased the risk of severe infection (adjusted odds ratio, 6.9; 95% confidence interval [CI], 2.3–20.5, and 3.1; 95% CI, 1.3–7.7, respectively), whereas anti-TNFs were not associated with worse outcome.[3] These results seem to persist (although with no statistical analysis yet) in the updated data from the IOIBD SECURE registry, showing that among 1170 reported IBD patients with COVID-19 infection, 4% had died, with a clear increase in patients older than 60 and/or having $\geq$2 comorbidities.[4] The difference in risk of severe infection and/or death related to treatment is more difficult to analyze with these raw data.[5] In fact, it would be of outstanding interest to have data from whole IBD patients' cohort(s) including both infected and noninfected patients to better outline the protective (or harmful) effect of the different treatments after adjusting for the other known risk factors.[6]