and is on the speakers' bureau for Collective Acument and Gore.

David Pereyra[1]
Mattias Mandorfer[2]
Julie Heimbach[3]
Patrick Starlinger[1,4]

[1]*Department of General Surgery, Division of Visceral Surgery, Medical University of Vienna, General Hospital Vienna, Vienna, Austria*
[2]*Department of Internal Medicine III, Division of Gastroenterology and Hepatology, Vienna Hepatic Hemodynamic Lab, Medical University of Vienna, Vienna, Austria*
[3]*Department of Surgery, Division of Transplantation Surgery, Mayo Clinic, Rochester, Minnesota, USA*
[4]*Department of Surgery, Division of Hepatobiliary and Pancreas Surgery, Mayo Clinic, Rochester, Minnesota, USA*

Correspondence
Patrick Starlinger, Department of General Surgery, Division of Visceral Surgery, Medical University of Vienna, Währinger Gürtel 18-20, 1090 Vienna, Austria.
Email: patrick.starlinger@meduniwien.ac.at

ORCID
David Pereyra https://orcid.org/0000-0001-6700-9163
Mattias Mandorfer https://orcid.org/0000-0003-2330-0017
Patrick Starlinger https://orcid.org/0000-0002-4666-5337

REFERENCES

1. György GP, Pereyra D, Rumpf B, Hackl H, Köditz C, Ortmayr G, et al. The von Willebrand factor facilitates model for end-stage liver disease–independent risk stratification on the waiting list for liver transplantation. Hepatology. 2020;72:584–94.
2. Jachs M, Hartl L, Simbrunner B, Bauer D, Paternostro R, Scheiner B, et al. Decreasing von Willebrand factor levels upon nonselective beta blocker therapy indicate a decreased risk of further decompensation, acute-on-chronic liver failure, and death. Clin Gastroenterol Hepatol. 2021:S1542-3565(21)00726-6. https://doi.org/10.1016/j.cgh.2021.07.012. [Online ahead of print]
3. Mandorfer M, Schwabl P, Paternostro R, Pomej K, Bauer D, Thaler J, et al. Von Willebrand factor indicates bacterial translocation, inflammation, and procoagulant imbalance and predicts complications independently of portal hypertension severity. Aliment Pharmacol Ther. 2018;47:980–8.

Letter to the editor: Reactivation of HBV triggered by SARS-CoV-2 in a patient with cirrhosis

To the editor,
HBV reactivation can occur spontaneously, but it is more frequently triggered by immunosuppressors, immunologic diseases, or transplantation. It can also be caused by coinfection with HCV or HDV, or even by elimination of HCV in patients treated with direct-acting antivirals. In patients receiving antivirals for the treatment of chronic hepatitis, reactivation can also be caused by low levels of the drug (due to lack of therapeutic adherence or interaction with other treatments) or new mutations of the virus.[1]

Reactivation is characterized by a sudden rise of HBV DNA, which can be followed by an elevation of alanine transaminase (ALT) levels (with or without bilirubin) several weeks after.[2] It should be treated with a nucleoside/nucleotide analogue (tenofovir or entecavir) as early as possible, regardless of ALT levels. Despite antiviral treatment, up to 25%–50% of patients may still develop severe hepatitis, hepatic failure, or even death.[3]

CASE REPORT

A 37-year-old male was admitted with asthenia, hyperoxia, and jaundice for the last 4 days. The patient had a cirrhosis caused by HBV chronic infection; he had never developed previous decompensations and was taking tenofovir-disoproxil-fumarate (TDF). Initial laboratory tests were made (Figure 1), and acute-on-chronic liver failure was diagnosed. He was taking TDF properly and denied consuming alcohol, drug abuse, or unprotected sexual intercourses. A complete study for acute liver injury was made (including viral and autoimmunity tests), and all results were normal.

On admission, the patient was tested for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), resulting positive. He assumed a risk contact 10 days before, but no breathing symptoms since then. DNA HBV was tested, and 4 million copies were detected (previously undetectable); treatment with entecavir was started. The virus was also tested for mutations but none of them were identified. Unfortunately, liver function worsened progressively as shown in Figure 1, leading finally to death 3 weeks later.

DISCUSSION

In our case, other more common causes were initially excluded: Medication adherence was appropriate, serology tests were negative, no new immunosuppressive

	6 MONTHS BEFORE	ADMISSION	1 WEEK AFTER	2 WEEKS AFTER	3 WEEKS AFTER
ALP (U/L)	83	179	155	147	134
GGT (U/L)	22	64	55	55	48
GOT (U/L)	38	2211	3275	837	375
GPT (U/L)	41	1691	2512	834	301
TBil (mg/dl)	0.97	19.7	31.6	42.4	44.2
DBil (mg/dl)	-	13.41	20.55	29.2	32.9
IBil (mg/dl)	-	6.29	11.05	13.2	11.3
PA (%)	83	46	45	37	34
INR	1.12	1.66	1.73	1.98	2.1
HBV-DNA	Undetected	4 million copies	-	106.000 copies	-

FIGURE 1 Serological evolution before and after admission to the hospital. Abbreviations: ALP, alkaline phosphatase; DBil, direct bilirubin; GGT, gamma-glutamyl transferase; GOT, glutamic oxaloacetic transaminase; GPT, glutamate pyruvate transminase; IBil, indirect bilirubin; INR, international normalized ratio; PA, prothrombin activity; TBil, total bilirubin

therapies were initiated, and viral mutations were excluded. For this reason, and judging by the temporary coincidence with infection by SARS-CoV2, we propose the coinfection with this virus as the cause of HBV reactivation. The underlying pathogenesis of transient reactivation of HBV in COVID-19 remains unclear, although it could be similar to HCV or HDV, causing reactivation of HBV replication.

In summary, we would like to highlight the singularity of our case report, as it is the first patient with cirrhosis with chronic infection of HBV who presumably developed a reactivation of HBV triggered by SARS-CoV-2.

Marta Librero Jiménez
María Ángeles López Garrido
María Carmen Fernández Cano

Hospital Universitario Virgen de las Nieves, Granada, Spain

Correspondence
Marta Librero Jiménez, Hospital Universitario Virgen de las Nieves, Granada, Spain.
Email: marlibjim@gmail.com

REFERENCES

1. Smalls DJ, Kiger RE, Norris LB, Bennett CL, Love BL. Hepatitis B virus reactivation: risk factors and current management strategies. Pharmacotherapy. 2019;39:1190–203.
2. Shi Y, Zheng M. Hepatitis B virus persistence and reactivation. BMJ. 2020;370:2200.
3. Ekpanyapong S, Reddy KR. Hepatitis B virus reactivation: what is the issue, and how should it be managed? Clin Liver Dis. 2020;24:317–33.

Letter to the editor: Spontaneous recovery in patients denied early liver transplantation for severe alcoholic hepatitis—Need a closer look!

To the editor,

We read with interest the retrospective study by Musto et al. regarding the spontaneous recovery (SR) and outcome of patients denied early liver transplantation (LT) for severe alcohol-related hepatitis (SAH).[1] Early LT lead to significant improvement in outcomes. Despite