

Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.jhep.2021.04.021>

References

Author names in bold designate shared co-first authorship

- [1] Younossi Z, Anstee QM, Marietti M, Hardy T, Henry L, Eslam M, et al. Global burden of NAFLD and NASH: trends, predictions, risk factors and prevention. *Nat Rev Gastroenterol Hepatol* 2018;15:11–20.
- [2] Sarin SK, Kumar M, Eslam M, George J, Al Mahtab M, Akbar SMF, et al. Liver diseases in the Asia-Pacific region: a lancet Gastroenterology & hepatology commission. *Lancet Gastroenterol Hepatol* 2020;5:167–228.
- [3] Mendez-Sanchez N, Arrese M, Gadano A, Oliveira CP, Fassio E, Arab JP, et al. The Latin American Association for the Study of the Liver (ALEH) position statement on the redefinition of fatty liver disease. *Lancet Gastroenterol Hepatol* 2021;6(1):65–72.
- [4] Valencia-Rodríguez A, Vera-Barajas A, Chávez-Tapia NC, Uribe M, Méndez-Sánchez N. Looking into a new era for the approach of metabolic (dysfunction) associated fatty liver disease. *Ann Hepatol* 2020 May-Jun;19(3):227–229.
- [5] **Eslam M, Newsome PN**, Sarin SK, Anstee QM, Targher G, Romero-Gomez M, et al. A new definition for metabolic dysfunction-associated fatty liver disease: an international expert consensus statement. *J Hepatol* 2020;73(1):202–209.
- [6] **Eslam M, Sanyal AJ, George J**. MAFLD: a consensus-driven proposed nomenclature for metabolic associated fatty liver disease. *Gastroenterology* 2020 Feb 8. <https://doi.org/10.1053/j.gastro.2019.11.312>.
- [7] Shiha G, Korenjak M, Eskridge W, Casanovas T, Velez-Moller P, Höglström S, et al. Redefining fatty liver disease: an international patient perspective. *Lancet Gastroenterol Hepatol* 2021;6(1):73–79.
- [8] Clayton M, Fabrellas N, Luo J, Alghamdi MG, Hafez A, Qadiri TA, et al. From NAFLD to MAFLD: nurse and allied health perspective. *Liver Int* 2021 Apr;41(4):683–691.
- [9] Fouad Y, Gomaa A, Semida N, Ghany WA, Attia D. Change from NAFLD to MAFLD increases the awareness of fatty liver disease in primary care physicians and specialists. *J Hepatol* 2021. S0168-8278(21)00101-X.
- [10] Alem SA, Gaber Y, Abdalla M, Said E, Fouad Y. Capturing patient experience: a qualitative study of change from NAFLD to MAFLD real-time feedback. *J Hepatol* 2021. S0168-8278(21)00034-9.

Nahum Méndez-Sánchez^{1,2,*}

Luis Díaz-Orozco^{1,2}

Jacqueline Córdova-Gallardo^{2,3}

¹Liver Research Unit, Medica Sur Clinic & Foundation, Mexico City, Mexico

²National Autonomous University of Mexico, Mexico City, Mexico

³Department of Hepatology, Service of Surgery and Obesity Clinic, General Hospital “Dr. Manuel Gea González”, Mexico City, Mexico

*Corresponding author. Address: Liver Research Unit, Medica Sur Clinic & Foundation and Faculty of Medicine, National Autonomous University of Mexico, Puente de Piedra 150. Col. Toriello Guerra, Tlalpan, Mexico City, Mexico. Tel.: (+525) 55424-4629, fax: (+525)55 666-4031.

E-mail addresses: nmendez@medicasur.org.mx or nah@unam.mx (N. Méndez-Sánchez)



Autoimmune hepatitis developing after coronavirus disease 2019 (COVID-19) vaccine: Causality or casualty?

To the Editor:

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection has been associated with the development of autoimmune processes.^{1–4} Molecular mimicry has been suggested as a potential mechanism for these associations.¹ In an *in vitro* study, Vojdani *et al.*⁵ showed that antibodies against the spike protein S1 of SARS-CoV-2 had high affinity against the following human tissue proteins: transglutaminase 3, transglutaminase 2, anti-extractable nuclear antigen, nuclear antigen, and myelin basic protein. As this is the same viral protein that the vaccine mRNA codes for, it is plausible that these vaccines may unmask autoimmune diseases in predisposed patients. Recently, several cases of immune thrombocytopenia (ITP) developing days after COVID-19 vaccination, have been reported to the Vaccine Adverse Event Reporting System (VAERS), reinforcing the possibility of vaccine-induced autoimmunity.⁶

We have recently treated a 35-year-old Caucasian female in her third month postpartum, who developed autoimmune hepatitis after COVID-19 vaccination. During pregnancy, she was diagnosed with gestational hypertension and started on labetalol 100 mg bid. C-section was performed without any complications,

and patient was discharged from the hospital on labetalol for blood pressure control. She resumed her job as a healthcare provider in mid-December, and received her first dose of Pfizer-BioNTech COVID-19 vaccine on January 4th. After 1 week, she started developing generalized pruritus, then choloria, and finally noticed jaundice, presenting to the emergency room on day +13 after COVID-19 vaccination.

She had a normal physical exam, except for scleral icterus, jaundice and palpable hepatomegaly. In the emergency room, laboratories were significant for: bilirubin 4.8 mg/dl, AST 754 U/L, ALT 2,001 U/L, alkaline phosphatase 170 U/L, and ammonium 61 mg/dl. Laboratory results were negative for hepatitis A, B, and C, Epstein-Barr virus (EBV), cytomegalovirus (CMV), herpes simplex virus (HSV) type 1 and 2, and HIV. At the time of submission, HEV had not been tested. Antinuclear antibody (ANA) was positive (1:1,280; homogeneous pattern). Double-stranded DNA antibodies were also positive (1:80). Other antibodies (*i.e.* anti-mitochondrial, anti-smooth muscle, liver-kidney microsomal, antineutrophil cytoplasmic antibodies) were negative. Total IgG was 1,081 mg/dl (normal range: 694–1,618 mg/dl). Ceruloplasmin, transferrin saturation, alpha-1-antitrypsin, TSH, and serum protein electrophoresis were all normal. Abdominal ultrasound with Doppler reported hepatomegaly without cirrhotic morphology, and no intra- or extra-hepatic biliary dilation.

Keywords: SARS-CoV-2; mRNA vaccines; liver injury; coronavirus.

Received 18 March 2021; received in revised form 29 March 2021; accepted 7 April 2021; available online 20 April 2021

<https://doi.org/10.1016/j.jhep.2021.04.003>