



Figure 2. (a) Numerous vascular spaces filled with blood and lymph (hematoxylin and eosin \times 40). Inset shows flattened endothelial cells (hematoxylin and eosin \times 400). (b) The pancreas is identified on right side of the field. The thick-walled vessels probably represent the feeder vessels (hematoxylin and eosin \times 40).

Moreover, disorganized proliferation of indigenous vascular tissues in the tongue and lung has been termed as a vascular hamartoma (2,4). In our case, the tumor was primarily composed of abnormally proliferated vascular tissues containing cells of 2 lineage; e.g., blood vessels and lymphatic vessels, which are indigenous to the anatomic area of occurrence; e.g., pancreas, and hence, they fulfill the criteria for being termed as vascular hamartoma of the pancreas.

Computed tomography scan and MRI are the commonest imaging modalities being used to evaluate the pancreatic lesions. Vascular tumors usually present as cystic or solid-cystic lesions showing varied degree of enhancement on contrast-enhanced computed tomography. Some of the commonly described vascular tumors of the pancreas are lymphangiomas and hemangiomas. Pancreatic hemangioma constitutes 0.1% of all pancreatic neoplasm, and they are more common in children and found to be associated with Kasabach-Merritt syndrome (5). Till date, not more than 10 cases of adult pancreatic hemangiomas have been reported. Pancreatic lymphangiomas are also rare, and around 60 cases have been reported so far. Preoperative characterization of the suspected vascular lesions of the pancreas is not always possible. Hence, in doubtful cases, surgical resection of an operable tumor seems to be beneficial for the patients.

This case also highlights the fact that these tumors can attain huge size and may become clinically apparent in the latter half of life. To the best of our knowledge, vascular hamartoma of the pancreas has not been reported before in the English language literature.

CONFLICTS OF INTEREST

Guarantor of the article: Ashesh Kumar Jha, MS.

Specific author contributions: The author is involved in every aspect of this case.

Financial support: None to report.

Potential competing interests: None to report.

Informed consent: Informed consent was obtained for this case report.

RFFFRFNCFS

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COVID-19 Patients With Hepatitis B Virus Infection

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Am J Gastroenterol 2021;116:1357–1358. https://doi.org/10.14309/ajg.000000000001034

We read with great interest of the study written by He et al. (1). The authors reported the clinical data of coronavirus 2019 (COVID-19) patients with hepatitis B virus (HBV) infection from hospitals in different regions of China. The results of this study are important, and we have some comments about it in conjunction with some of the current studies.

In this study, the authors found that COVID-19 patients with HBV infection were observed to have a lower risk of severe events including intensive care unit admission or death. However, the results of the HBV-DNA test have not been given in current study, and therefore, the clinical stage of included patients cannot be determined. The clinical outcomes of patients with chronic HBV infection or acute HBV infection can be different, and the same applies to chronic patients with or without active HBV replication. Other factors, including whether patients had other concomitant diseases, use of antiviral drugs, and combination of multiple medications, can also influence the patients' final outcomes.

We found that recent studies have described inconsistent clinical outcomes in COVID-19 patients with HBV infection. One study supports the conclusion of the current study and concludes that severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and HBV coinfection had no effect on the course and prognosis of COVID-19 (2). However, 2 studies suggest that patients coinfected with SARS-CoV-2 and HBV are more likely to have severe/ critical illness (3,4). In all these studies, patients with or without coinfection had similar mortality rates possibly because of their small samples. Therefore, the impact of HBV coinfection on COVID-19 remains unclear, and further longitudinal cohort with large sample is still needed to determine the true situation.

Interestingly, the current study also reported that the incidence of HBV infection among patients with COVID-19 seems to be lower than the incidence of HBV infection in the overall Chinese population. A hypothesis was proposed recently for this phenomenon that the exhaustion of T lymphocytes may affect HBV-infected patients' ability to respond to other viruses and then reduce the degree of "cytokine storm," thus culminating in a less severe disease of COVID-19 (5). Although different from the results of clinical observation, it prompts that the immune dysfunction may be one of the possible mechanisms to explain the clinical outcomes of these coinfected patients. We hope that future studies will provide data on patients'

immune-related indicators to help shed light on the truth.

CONFLICTS OF INTEREST

Guarantor of the article: Jin-Lin Yang, MD. **Specific author contributions:** X.-H.L. and J.-L.Y. developed the concept and designed the study; X.-H.L., K.D., and J.-L.Y. wrote the manuscript. All authors approved the final version of the manuscript.

Financial support: None to report. **Potential competing interests:** None to report.

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Sarcopenia and TIPS: How Best to Measure Muscle Mass

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Am J Gastroenterol 2021;116:1358. https://doi.org/ 10.14309/ajg.0000000000001091 We read with great interest the article by Benmassaoud et al., a retrospective study of impact of preexisting sarcopenia on the incidence of *de novo* hepatic encephalopathy (HE) and overall mortality in patients who underwent transjugular intrahepatic portosystemic shunt (TIPSS) insertion for refractory ascites (1). The authors found that baseline sarcopenia is not associated with HE or increased mortality in such patients. However, several issues need to be addressed.

First, *de novo* HE after TIPSS insertion in the present study was noted in 29.9% of patients. Although skeletal muscle index at the third lumbar vertebrae (L3-SMI) significantly predicted HE both on univariate and multivariate analyses, sarcopenia, which is assessed by the similar index, failed to predict the same, which needs an explanation. However, Nardelli et al. in their prospective study found sarcopenia to be associated with the development of HE post-TIPSS (2). The authors should also disclose the association between total psoas muscle index at the third lumbar vertebrae (L3-PMI) and the incidence of HE in the study population.

Second, the authors assessed sarcopenia in their patients using L3-SMI and L3-PMI; however, only L3-SMI was used to define sarcopenia. Golse et al. have used different muscle indices to define sarcopenia in patients with cirrhosis awaiting liver transplant (3). They have found that among psoas muscle area (PMA), PMA normalized by height or body surface area (BSA) and L3-SMI, PMA offered better accuracy than L3SMI and PMA/BSA, and the same accuracy as PMA/squared height. Thus, comparison of all these indices for the assessment of sarcopenia in patients undergoing TIPSS are needed.

Finally, the authors concluded that baseline sarcopenia as assessed by L3-SMI was not associated with an increased mortality. However, another similar study has found that sarcopenia assessed by transversal right psoas muscle thickness at the umbilical level/height (TPMPT/height) was independently associated with post-TIPSS mortality in patients of recurrent ascites and gastrointestinal bleeding (4). Moreover, patients with ascites had more severe sarcopenia and patients with gastrointestinal bleeding had a higher MELD score, with similar mortality in both the groups, thus highlighting the importance of considering MELD-sarcopenia score in these groups before considering TIPSS (5).

Overall, we congratulate the authors for conducting this study, highlighting the consequences of TIPSS placement on the body composition of cirrhotic patients. Future research should focus on the prospective evaluation of various aspects of sarcopenia and frailty assessment such as muscle strength, quality, quantity, and physical performance for better understanding of outcomes associated with post-TIPSS.

CONFLICTS OF INTEREST

Guarantor of the article: Prasanta Debnath, MD, DM.

Specific author contributions: P.D: Writing the draft; P.R: Critical revision.

Financial support: None to report. **Potential competing interests:** None to report.

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Response to Debnath and Rathi

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Am J Gastroenterol 2021;116:1358–1359. https://doi.org/10.14309/ajg.000000000001150