PPFE? To date, there are no other reported cases of PPFE associated with solid organ transplantation, with the exception of lung transplant. It has been reported that the other form of cGVHD (bronchiolitis obliterans) can occur after other organ transplants, such as kidney transplants (5). Although the pathogenesis is unclear, bone marrow–derived cells targeting the allograft might result in a similar process when directed at the respiratory epithelium.

To better understand the association between PPFE and liver transplantation, we suggest additional information and further analysis of the patient. First, did the patient have pleural thickening or PPFE-like lesions in the lung before liver transplantation? We recommend evaluating the chest X-ray or computed tomographic scan before liver transplantation and compare with any available computed tomographic scans performed during the follow-up period. This information would be helpful in ascertaining the onset of the disease. Second, it would be interesting to determine whether there are histopathologic findings of chronic rejection from autopsy specimens. Chronic rejection of the liver is characterized by the presence of diffuse biliary epithelial senescence changes, foam cell obliterative arteriopathy, or bile duct loss (vanishing vile duct syndrome) (6). In addition, if bronchiolitis obliterans is present in the lung, PPFE may be a manifestation of chronic lung allograft dysfunction. Third, is it possible that the liver transplant procedure mechanically affects the development of PPFE? The PPFE lesions in this case were asymmetric (right bigger than left) apical fibrosis and volume loss. We suspect that pulmonary complications after liver transplantation, including atelectasis, pneumonia, or diaphragmatic injury, might have led to the occurrence of PPFE.

The identification of disease onset, radiologic changes, evidence of an immune response in the lung and liver specimens, and the impact of lung complications after liver transplantation would bridge the gap between PPFE and liver transplantation. Furthermore, a large-scale longitudinal study is warranted to clarify the occurrence and significance of PPFE in various organ transplant recipients. ■

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Reply to Watanabe *et al.*

From the Authors:

We thank Watanabe and colleagues for their interest in our work and correspondence regarding our paper (1). We agree wholeheartedly with their highlighted importance of the potential relationship among liver failure, liver transplantation, and the development of pleuroparenchymal fibroelastosis (PPFE). We also wish to clarify that we reported an association between liver transplant and PPFE, not necessarily a causation. We cannot say for certain that the transplant caused the lung disease, but it remains one potential explanation, in addition to the possibility of a shared risk factor for both liver failure and PPFE. Their letter posed four questions, which we address below.

First, did the patient have evidence of abnormal lungs with an abnormal chest radiograph before liver transplantation and the development of PPFE? Indeed, the patient had a normal chest radiograph before liver transplant. Given the space limitations for this type of article, we were unable to present the baseline radiograph in the manuscript but provide it here (Figure 1). The onset of any pleural–parenchymal disease occurred after liver transplant.

Second, did the patient have evidence of chronic allograft dysfunction as a potential trigger of PPFE after liver transplant? We have reconfirmed that the patient had no evidence of chronic liver rejection at the time of death. His liver transplant team confirmed that although he had evidence of rejection around the time of PPFE recognition 3 years after transplant, there was no evidence of rejection at the time of death (another 3 yr later), with long-term normal graft function. Whether chronic allograft dysfunction presented a catalyst for the development of subsequent PPFE, we cannot say. This highlights the notability of this case and suggestion for long-term pulmonary follow-up after liver transplant. The postmortem autopsy was lung limited; thus, there is no further tissue available for evaluation.

Third, did the patient have evidence of bronchiolitis obliterans as evidence of chronic allograft dysfunction? The autopsy (performed by A.F.) specifically noted that there was no evidence of bronchiolitis obliterans in the lungs.

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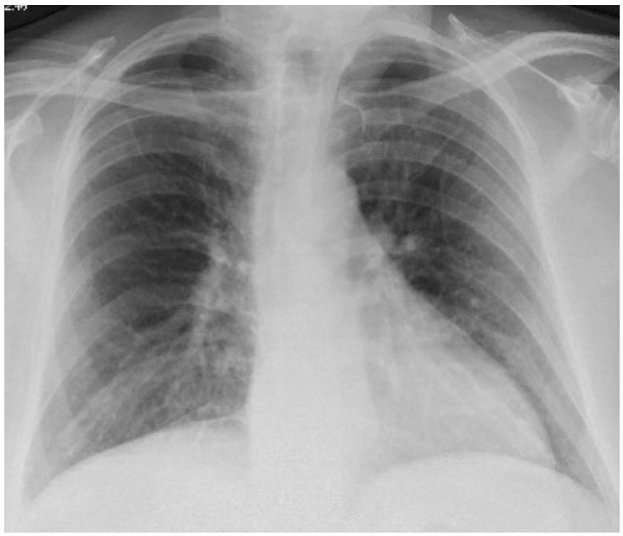

Figure 1. Chest radiograph before liver transplantation.

Fourth, may the abnormalities have arisen asymmetrically as a consequence of liver transplantation? We cannot exclude that other triggers prompted the development of PPFE in this particular individual, which underlies the suggestion for focused follow-up after liver transplant. Although atelectasis, diaphragmatic injury, or pneumonia complicate the course of many patients, PPFE remains rare. Thus, we propose the potential relationship between these complications of liver failure and liver transplant and the development of PPFE with the publication of this case.

We agree with Watanabe and colleagues that the entity of PPFE, whether idiopathic or secondary to bone marrow, lung, or potentially liver transplant, warrants further study to characterize potential predisposing factors such as graft failure, transplant-related complications, or telomere dysfunction. We hope that this patient's unique case contributes additional impetus for such important work. ■

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Erratum: Synergistic Association of House Endotoxin Exposure and Ambient Air Pollution with Asthma Outcomes

The authors have recently discovered errors in the article by Mendy and colleagues (1), published in the September 15, 2019, issue of the *Journal*. The authors informed the *Journal* that the spatiotemporal data in their article were linked incorrectly. This incorrect data linkage was the result of participant locations based on a list of census tracts from the year 2000 being inadvertently matched against air pollution readings based on a list of census tracts from the year 2010 (rather than the year 2000). This mismatch of years resulted in 475 participants not being matched to air pollution data, and thus being excluded from the analysis. The authors additionally state that it is possible that an unknown number of participants could have been matched to air pollution data at a different location than their actual residence. The correction of this incorrect linkage using 2000 census data instead of 2010 resulted in all 475 previously excluded participants being reintroduced to the analysis and the presumed correction of previously mismatched locations. The corrected dataset contained a higher proportion of participants from the Western region (31.0% vs. 25.9%) and a higher proportion of participants living in nonmetro areas (49.2% vs. 45.6%).

The authors believe that all other outcome and covariate distributions changed minimally, suggesting they had been missing at random due to the incorrect data linkage and that the revised data do not change the overall findings and conclusions. However, there have been extensive changes to the data listed in Tables 1, 2, and 3; Figures 1 and 2; and all the tables in the online supplement associated with the article. Therefore, the *Journal* is replacing the online version of the article with a corrected version. In addition, a document showing all the changes to the data will be posted as an online supplement to the original article.

The authors would like to apologize to the readership for any confusion caused by these errors. ■

Reference

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