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Commentary: Gastroesophageal reflux and lung allograft dysfunction: Need to improve detection and clinical reporting

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CENTRAL MESSAGE

GERD can be multifactorial and needs to be studied in the context of esophageal and gastric motility disorders that are common after lung transplantation. In addition, identification of biochemical markers of gastric content aspiration in BAL fluid will help in the study of reflux and measuring efficacy of its treatment.

Etiologies and mechanisms that result in chronic lung allograft dysfunction have been relatively elusive. Among the many potential inciting events that directly or indirectly lead to chronic lung allograft dysfunction, gastroesophageal reflux disease (GERD) is a well-known risk factor. Although the relationship between GERD and lung dysfunction may be somewhat intuitive, the exact mechanism by which gastric contents incite pulmonary inflammation is not well established and assumes aspiration of refluxed contents.

There are little data that show directly measured or recorded refluxed contents in the lungs and their downstream correlation with native or allograft dysfunction. Measurement of gastric contents in bronchoalveolar lavage (BAL) is an imperfect science, and the study of GERD in lung dysfunction has been limited by the lack of a reliable and reproducible indicator of GERD in BAL.¹⁻³ In fact, most of the evidence that implicates GERD in native or allograft lung dysfunction is indirect and comes from clinical series that study GERD and preventive effect of antireflux interventions.⁴⁻⁶

Antireflux procedures have been shown to stabilize decline in native lung function in patients with pulmonary fibrosis.⁷ These findings have also given credibility to the theory of GERD as an underlying etiology for pulmonary fibrosis. Likewise, antireflux procedures in lung transplant recipients with GERD have been shown to prevent allograft dysfunction, without any data clearly showing decrease in aspirated levels of gastric contents. Our understanding of

how severity of GERD as opposed to presence of GERD affects lung function is also limited. Although we assume that decrease in distal esophageal acid exposure time after antireflux surgery decreases aspiration of gastric contents, there is little direct proof of this.

What complicates matters even more is the fact that GERD after lung transplantation is a result of complex interactions between esophageal motility or lack thereof, gastroparesis and changes in gastroesophageal junction anatomy, and pressure gradients due to changes in thoracoabdominal pressure. Treating all patients with similar antireflux surgery without taking into account some of these other competing factors is rather simplistic and leads to more problems. For example, the mechanism for post-transplant GERD might be different in a patient with normal gastric emptying compared with one with gastroparesis (noted in up to 90% of lung transplant recipients).⁸ Therefore, the antireflux intervention should be modified accordingly. Likewise, antireflux interventions in the setting of esophageal dysmotility should accommodate for the lack of normal peristalsis.

By the same token, the results of antireflux interventions should be reported in the context of these concomitant esophageal or gastric motility problems. However, that

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has not been the case in the majority of published literature on the subject and thus limits its applicability and reproducibility. In the present article by Davidson and colleagues,⁹ the authors have amassed available data correlating antireflux surgery with preservation of allograft function. As expected, although they were able to confirm the beneficial effect of antireflux surgery, the available data were extremely variable and did not provide any of the above-mentioned context.

To make progress in the study of GERD and its effect on native or allograft lung dysfunction, we have to address the 2 major limitations discussed: (1) We need a reproducible marker of gastric contents that can be measured in BAL; and (2) we need to report GERD and antireflux procedure outcomes in the context of the organ proximal to the gastroesophageal junction and the one distal to it and not be oblivious to their dysfunction.

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Commentary: The burning questions of reflux management in lung transplantation

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Our knowledge of the complex mechanisms leading to chronic allograft rejection after lung transplantation is evolving. Early studies indicated non-alloimmune injury such as gastroesophageal reflux disease (GERD) poses risk by potentiating inflammation in the small airways

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CENTRAL MESSAGE

Surgical management of gastroesophageal reflux disease may prolong allograft function in lung transplant patients.