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Response

To the Editor:

We appreciate the insightful comments about our article[1] made by Dr Keng. The information on consolidative (C)-pattern in examination point 5 was missing in e-Table 1. The revised e-Table 1 is attached to this response letter. We defined C-pattern as areas of tissue hepatization with or without air bronchogram.[1] C-pattern in zone R5 was seen in 36 patients (61%) in the group with cardiogenic pulmonary edema (CPE), 23 (55%) in the group with ARDS, and 12 (38%) in the miscellaneous group ($P = .093$). C-pattern in zone L5 was seen in 44 patients (75%) in the group with CPE group, 22 (52%) in the group with ARDS, and 13 (41%) in the miscellaneous group ($P = .004$). These variables were not selected as statistically significant in a prediction model for CPE in the multivariate analysis.

C-pattern reflects a variety of causes such as atelectasis (eg, resorptive, compressive, and passive), consolidation, contusion, infection, mass, and pulmonary embolism.[2] It is often difficult to identify a cause of C-pattern with critical care ultrasonography alone.[3] We anticipated that C-pattern in the form of compressive atelectasis was associated with pleural effusion in the posterolateral zone. In an effort to differentiate effusion-induced compressive atelectasis from consolidation or other causes of C-pattern, we created a variable, C-pattern without significant pleural effusion (< 20 mm), in zone 5. C-pattern without significant pleural effusion in zone R5 was seen in 14 patients (24%) in the group with CPE, 16 (38%) in the group with ARDS, and 10 (31%) in the miscellaneous group ($P = .304$). C-pattern without significant pleural effusion in zone L5 was seen in 16 patients (27%) in the group with CPE, 12 (29%) in the group with ARDS, and 10 (31%) in the miscellaneous group ($P = .94$). These two variables were included in the multivariate analysis; however, they were not statistically significant for differentiating CPE from ARDS.

C-pattern was not an important variable for differentiating CPE from ARDS in patients with acute hypoxemic respiratory failure. Although variables on C-pattern were accidentally missing in the original e-Table 1, they were included in the initial multivariate analysis. Therefore, the study conclusions are the same as demonstrated in our original article: left-sided pleural effusion (> 20 mm), moderately or severely decreased left ventricular function, and a large inferior vena cava minimal diameter (> 23 mm) were predictive of CPE. Our results are consistent with previous reports on CT, which demonstrated that atelectasis and consolidation are seen in both patients with CPE and those with ARDS.[4,5]

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Additional information: The e-Table can be found in the Supplemental Materials section of the online article.

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Radiologic Criteria for the Diagnosis of High-Attenuation Mucus in Allergic Bronchopulmonary Aspergillosis

To the Editor:

We read with interest the article by Alikhan et al[1] in a recent issue of *CHEST* (December 2015). The

authors describe a case of allergic bronchopulmonary mycosis secondary to colonization of a patient's clarinet by *Alternaria*. In this article, the authors incorrectly describe high-attenuation mucus as mucus with attenuation higher than that of water.

On computed tomography (CT) scan, high-attenuation mucus is defined subjectively as mucus visually denser than the chest wall or paraspinal skeletal muscle[2-4]; it is defined objectively on a CT scan as having an attenuation value > 70 Hounsfield units. Hyperattenuating mucoid impaction is a pathognomonic feature of allergic bronchopulmonary aspergillosis (100% specificity).[5] If present at diagnosis, it is an independent predictor of poor outcomes in allergic bronchopulmonary aspergillosis.[3,6,7] In addition, several aspects of discussion in the article by Alikhan et al[1] point toward research performed by our group, such as the estimate of the prevalence of high-attenuation mucus[8] and the mechanistic explanations regarding the occurrence of masses in allergic aspergillosis.[9] However, none of our work has been cited.

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Response

To the Editor:

In response to our recent article in *CHEST*,[1] Agarwal et al aptly point out the radiologic criteria for the diagnosis of high-attenuation mucus in allergic bronchopulmonary aspergillosis. High-attenuation mucus is a very specific finding for allergic pulmonary aspergillosis (ABPA). The mucus in ABPA is often of higher attenuation. However, mucus with an attenuation > 70 Hounsfield units is much more specific for ABPA, as noted in the correspondence from Agarwal et al, and is associated with poor outcomes.[2,3] We thank the authors for this clarification, as well as the helpful citations for the interested reader.

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