Can Lung Comets Be Counted as “Objects”?  

We have read with interest the study carried out by Mallamaci et al. (1) in *JACC*. The authors evaluate pulmonary vascular congestion in a group of patients in chronic dialysis through a score derived from the number of ultrasound lung comets in the anterior chest regions (2–5).

No doubt exists that a “wet lung” will present a proportionally larger number of comets, reflecting the increase in its extravascular lung water. Cardiogenic pulmonary edema typically manifests with ultrasound lung comets that are initially more evident at lung bases and then extend to the middle and superior fields in relation to the increase in capillary venous pressure.

However, the hypothesis proposed by the authors (1), that lung comets originate from the subpleural interlobular edematous septa, is less convincing. First, the 7-mm distance observed between the comets does not correspond to anatomic observations, which show a variable distance from 10 to 30 mm between the interlobular septa (6,7). Second, we have demonstrated that analogous artifacts might be observed when insonating a foam object, in the absence of identifiable structures (8). Third, from the biophysical point of view, reverberations on subpleural interlobular septa (as proposed in Figure 1 of the article [1]) are unlikely as interlobular septa to project from the pleural surface at right angles and certainly are not parallel to it, as would be necessary to create repetitive specular reflections or “transducer reverberations” (9). Finally, in our experience, the creation of ultrasound lung comets is an angle- and movement-dependent phenomenon.

This is evident from clinical practice and from the analysis of a porous and wet polyurethane phantom insonated at 90° and 45° (unpublished data). Insonation at an angle is less capable of producing artifacts analogous to the ones observed in the pathologic lung (Fig. 1), supporting the hypothesis that comets are the expression of random microfocal reverberations or “internal reverberations.”

Increasing degrees of lung congestion leads to the formation of increasing concentrations of lung comets up to their coalescence (Fig. 2). We believe counting these artifacts to be imprecise in a tissue that is more-or-less porous. Furthermore, it is unlikely that a numerical estimate of such comets, which are not correlated to identifiable structures (as septi) but are based on an angle- and movement-dependent geometry, could be used as an instrument for classification of pathology. Instead, we believe that a better physical knowledge of the acoustic phenomena produced by porous and aerated tissues is necessary to correctly interpret images of lung pathology that are nothing more than “errors” of interpretation of real signals by the echographic machine.

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