

Pleural fluid is surrogate for time

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I read with much interest the manuscript by Taira *et al.* (1) and like to make some comments on their results.

The authors present in their manuscript 40 patients with a primary spontaneous pneumothorax and analyse the rate of re-expansion pulmonary edema (RPE) as visible on computed tomography (CT). They describe a number of characteristics of these patients such as duration of symptoms, size of pneumothorax and radiological findings on a posteroanterior chest X-ray in erect position. They put much emphasis on presence of pleural fluid.

A common finding in patients with a pneumothorax is pleural inflammation (2,3) resulting in pleural fluid, ranging from a few millilitres to radiologically visible amounts. The authors do not discuss the reason(s) why these patients develop a pleural effusion. The by far most likely explanation is the induction of inflammation of the pleura by the presence of air in the pleural cavity. The cellular content of this fluid changes over time. After the initially influx of neutrophils and macrophages this is followed by a strong increase of eosinophils with prolonged presence of air in the pleural cavity (4). This change in differential count seems to be a reliable reflection of the duration of presence of air in the pleural cavity and therefore as a more objective proof than simply asking when symptoms started. It is well known that the moment of subjective symptoms may be many days after the first leaking of air into the pleural cavity (5).

They consider presence of pleural fluid on an erect chest X-ray as a new risk factor for the development of RPE. What the authors fail to discuss is that in about 1/3 of cases small effusions will be missed by their diagnostic approach (6). Patients who have less than 175 mL of pleural fluid, being the lower detection limit, or even much larger volumes (7), will be missed. This makes the conclusion of the authors in fact not reproducible. Although there is no

proof that more fluid reflects a longer duration of air in the pleural cavity, this seems likely and this makes their risk factor in fact a reflection of duration of existence of the pneumothorax, as reported in a much larger study (8). It would have been much better if the authors had used the optimal technique for detection of pleural fluid (ultrasound) and investigated whether differential counts of the cellular content could be a supporting method for measuring time of existence of the pneumothorax and by that a reproducible risk factor.

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