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WHAT'S NEW IN INTENSIVE CARE

Does my patient really have ARDS?

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How often does ARDS go unrecognized in our ICUs?

A recent international observation study launched by the working group of the European Society of Intensive Care Medicine after the release of the new Berlin definition of ARDS has brought many important results [1]. One of the most surprising—and challenging—findings was the large amount of under-recognition by clinicians. Indeed, this study included all hypoxemic patients (PaO_2/FiO_2 ratio below 300 mmHg) under mechanical ventilation and the diagnosis was made automatically when criteria for the definition were fulfilled [2]. Both on admission and at discharge, the question was specifically asked whether the patient, at any time during the ICU stay, was qualified as having ARDS. Clinician recognition of ARDS ranged from only 51.3 % (95 % CI, 47.5–55.0 %) for mild ARDS to 78.5 % (95 % CI, 74.8–81.8 %) for severe ARDS. This had clear consequences since ventilatory settings were different in those with "unrecognized" ARDS. Not surprisingly, the patients with recognized ARDS were sicker in all categories. Interestingly also, the number of patients per physician or nurse in a given ICU negatively influenced this recognition. Therefore it seems important to understand why this syndrome is so often unrecognized (Fig. 1).

My patient is not hypoxemic enough

Because the cornerstone of the diagnosis is a calculated index, i.e., the PaO_2/FiO_2 ratio, one possible major source of under-recognition is the fact that this ratio is simply not calculated. When a patient receives a "safe" FiO_2, like 30 or 40 %, many clinicians will intuitively assume that these patients cannot reasonably be qualified as having ARDS. In fact, any time the PaO_2 is at or below 90 mmHg with FiO_2 30 %, the gas exchange criterion for mild ARDS is present, and any time the PaO_2 is at or below 80 mmHg with FiO_2 40 %, the gas exchange criterion for moderate ARDS is present. Electronic health record systems may help in the future to have automatic recognition of this criterion.

This is a concern since even in the mild ARDS group a reduction in tidal volume is life-saving [3]. In addition, data concerning tidal volume in non-ARDS patients tend to suggest that a "low" tidal volume could be a good default setting [4]. So, one solution may be to institute 6 ml/kg of predicted body weight tidal volume as a universal setting and readjust pressure and volume individually, especially in patients having all criteria for ARDS.

Another drawback with the PaO_2/FiO_2 ratio is that it is highly dependent on FiO_2 [5, 6]. If one institution decides to measure this index at an FiO_2 of 1 for instance, this could markedly underestimate the prevalence of the syndrome [7].

My patient has fluid overload explaining hypoxemia

A frequent (and wise) clinical thought is that patients have major fluid overload contributing to their poor respiratory status. This is certainly good clinical practice, including the fact that removing fluid quickly can help get patients off the ventilator [8], but that should not exclude the diagnosis of ARDS. The fact that "real" ARDS can have elevated high pulmonary artery occlusion pressures, has been recognized for a long time [9], and the new definition tried to be as "inclusive" as possible, simply indicating that respiratory failure should not be "fully" explained by heart failure or fluid overload [10]. Wisely applied, this definition should solve a vast majority of the cases for which the participation of fluid overload is a clinical question.

My patient needs to have severe ARDS to benefit from a dedicated approach

Although ARDS has been associated with a pathological hallmark, i.e., the presence of hyaline membranes,

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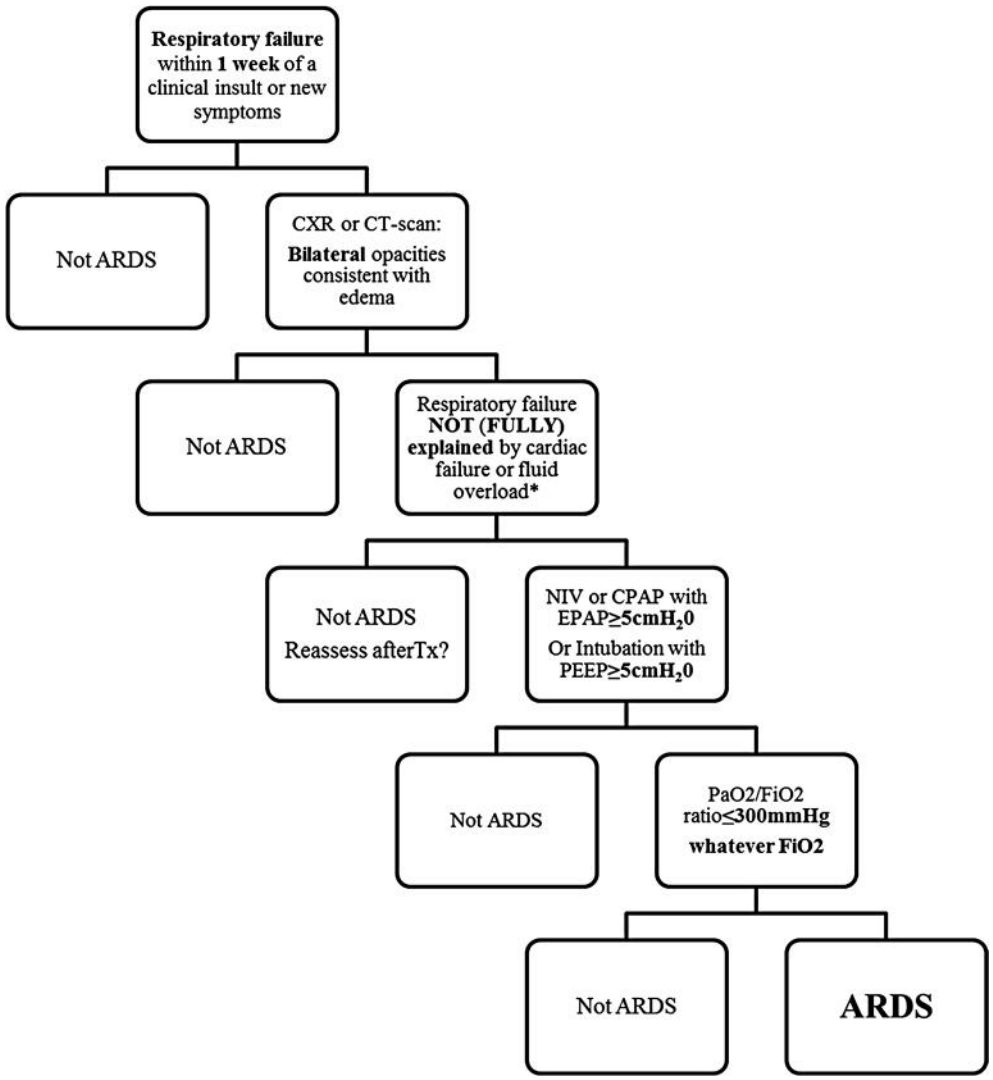

Fig. 1 Illustration of the different steps needed to make the diagnosis of ARDS in case of respiratory failure

autopsy series have shown quite divergent results, with close to 50 % of patients dying with ARDS showing no hyaline membranes on autopsy (diffuse alveolar damage) [11]. This could suggest that our definition is poorly specific. Many of our accepted animal models of ARDS, however, do not generate hyaline membranes [12]. This raises the question of what we want to achieve with a diagnosis of ARDS. If one treatment is hoping to cure endoalveolar fibroproliferation resulting from the initial insult, it makes sense to select patients with a relatively homogeneous pathophysiological process leading to this lesion. If a clinician aims at mostly protecting the lung from injurious ventilation, it may not be important to differentiate a severe bilateral consolidation from diffuse alveolar damage if, in both cases, the aerated lung is only one-third or one-fourth of a normal lung [13]. What is hyaline membrane the marker of? It has been described in human ARDS in association with high ventilatory settings (initial reports on ARDS lungs [14]), in experimental models of ventilator-induced lung injury (VILI) [15], or in severely hypoxemic ARDS patients ventilated for at least 3 days [11]. This pathologic lesion is a fairly generic marker of an alveolar insult rather than indicating any specific mechanistic target [16]. It is therefore impossible to distinguish it from VILI and hyaline membranes could be in fact mostly a marker of VILI.

Similarly, some patients have all the features of ARDS (i.e., definition criteria) but no risk factor. Once you have

formally eliminated high-pressure pulmonary edema, there are still around 10 % of patients who have no other explanation for this clinical presentation. A nice report recently described their characteristics but called these patients "mimickers of ARDS" [17]. The name may be misleading as it is simply ARDS without a risk factor, which implies the same clinical approach. This group of patients is particularly important to identify clinically as the underlying disease may have a specific therapy.

Conclusion

ARDS is the syndrome in critical care medicine for which we have the greatest evidence that our interventions can change the outcome, but it is also still a deadly one. There is evidence that injury caused by ventilation is still highly prevalent and hopefully future approaches will help to improve the outcome further. The first step, however, is to change our clinician's approach and enlarge our diagnostic scope. Not all ARDS might be treated in the same way, but individualized medicine needs at first a recognition of the problem.

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