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A Quick Look at Dangerous Lung Water in Hemodialysis Patients

Marco Marano

Hemodialysis Unit, Maria Rosaria Clinic, Pompeii, Italy

Dear Editor,

Campos et al. [1] have recently provided interesting insights on respiratory functional parameters in hemodialysis patients.

As they have stated, fluid overload may adversely impact alveolar oxygen diffusion leading to hypoxemia, and from the converse point of view, reaching dry weight should improve arterial blood oxygenation. As an example they showed improvement of arterial oxygen saturation (SaO_2) with fluid removal. However, connection between fluid overload and arterial blood oxygenation – an attractive issue for nephrologists – cannot be explored looking at SaO_2 , unless it is grossly reduced, as the case they reported. Due to the sigmoid shape of oxygen–hemoglobin dissociation curve, the SaO_2 does not change significantly even if large changes in the oxygen partial pressure (pO_2) occur in the upper flat part of the curve, as almost all hemodialysis patients lie before [2] and during [1] hemodialysis treatment.

Respiratory parameters other than SaO_2 and pO_2 are currently used to explore oxygen diffusion in different clinical scenarios [3] as well as during hemofiltration [4]. In the past, the alveolar–arterial oxygen gradient (A-aO_2 , in mm Hg) has been extensively exploited in the assessment of acute lung derangement [5, 6] and was profitably used also in hemodialysis patients [7]. It allows the functional assessment of alveolar capillary barrier. As interstitial lung edema increases alveolar–arterial distance and in turn impairs oxygen diffusion, it leads to hypoxemia typically featured by large A-aO_2 . The A-aO_2 is computed as the difference between the alveolar pO_2 and arterial pO_2 , with alveolar pO_2 equal to fractional concentration of inhaled oxygen * (barometric pressure – partial pressure of water vapor in air) – (1/respiratory exchange ratio) * pCO_2 , where pCO_2 stands for partial pressure of carbon dioxide. At sea level and on room air, the gradient may be approximated as $150 - \text{pO}_2 - (1.2 * \text{pCO}_2)$; hence, no further blood sample is needed to compute A-aO_2 . Therefore, the gradient can be easily and quickly obtained at the bedside and this is also a very attractive chance. Blood sample for gas analysis should be obtained before dialysis starts from the arterial fistula needle, because vascular access recirculation affects the pCO_2 value [8]. Furthermore, during the treatment, patient recumbency and gases gain from the dialysate affect pO_2 and pCO_2 as well as the respiratory exchange ratio, hence intradialytic A-aO_2 can no longer

reflect interstitial lung edema. The A-aO_2 increases with age being the upper normal limit equal to $\text{age}/4 + 4$, but 20 mm Hg is commonly referred to as upper limit [5].

I already proposed [9] to use pre-dialysis A-aO_2 as a window onto dangerous water – that is extravascular lung water – in hemodialysis patients. It must be emphasized that the notion of dry weight and that of lung water are quite different and they may not be correlated with each other depending on cardiac output, plasma oncotic pressure, capillary permeability and others. Moreover, A-aO_2 allows the assessment of pulmonary imbibition only at a glance where most suitable techniques are straightway unavailable. Finally, increased A-aO_2 may be due to different lung diseases. Nevertheless, it allows to have a quick look at the ‘dangerous water’ at the bedside and, in particular, if pre-dialysis A-aO_2 is within the normal value, an increased lung water as cause of dyspnea may be excluded. An example of using the magnitude of A-aO_2 to guide fluid removal is shown in figure 1. After a long interdialytic period, acute respiratory failure due to overhydration, deranged respiratory functional parameters. Fluid removal obtained by a single dialysis treatment allowed to find SaO_2 near the normal value since before the next treatment, but this is not true for A-aO_2 . The gradual and progressive decrease of the gradient, assessed before the succeeding dialysis sessions better mirrored the fluid removal.

Fig. 1. Pre-dialysis assessment of SaO_2 and A-aO_2 in overhydration. Progressive fluid removal over time (1 month) was mirrored by A-aO_2 . Low SaO_2 was found only in the occurrence of breathlessness (before the first dialysis of the week).

For the sake of accuracy, I recall that in asymptomatic hemodialysis patients, early interstitial edema does not interfere with respiratory exchange but could cause a mild degree of hyperventilation so that respiratory alkalosis, rather than impaired arterial oxygenation, could be the first sign of increased lung water [10].

Disclosure Statement

None declared.

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