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Extra Vascular Lung Water Assessment for Intraoperative Monitoring During Kidney Transplantation Surgeries

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Abstract: Background: Optimal fluid therapy is considered as very crucial to decrease delayed graft function after renal transplantation. The intraoperative period of renal transplantation is divided into two phases. The first phase is the one prior to the reperfusion and the second is after reperfusion of the graft. Prior to reperfusion, physicians should not consider the patients as hypervolemic as they are usually adequately dialyzed. Frequency of haemodialysis and weight gain between haemodialysis and the amount of ultrafiltration during each session will decide the patients' volume status. The patient is predisposed to be hypovolemic . Extravascular lung water (EVLW) is the amount of water that is contained in the lungs outside the pulmonary vasculature. It corresponds to the sum of interstitial, intracellular, alveolar, and lymphatic fluid, not including pleural effusions. Lung ultrasonography is accurate in detecting pulmonary edema and overcomes most limitations of traditional diagnostic modalities. Whether use of lung ultrasonography-guided management has an effect on cumulative fluid balances and other clinical outcomes remains unclear.

Keywords: *lung ultrasound, Extra Vascular Lung Water*

Introduction

Optimal fluid therapy is considered as very crucial to decrease delayed graft function after renal transplantation. The intraoperative period of renal transplantation is divided into two phases. The first phase is the one prior to the reperfusion and the second is after reperfusion of the graft. Prior to reperfusion, physicians should not consider the patients as hypervolemic as they are usually adequately dialyzed. Frequency of haemodialysis and weight gain between haemodialysis and the amount of ultrafiltration during each session will decide the patients' volume status. The patient is predisposed to be hypovolemic [1].

Resuscitation fluids are used during hypovolemia to expand the intravascular volume, for improving cardiac output and perfusion pressures. The characteristics of ideal intravenous fluid for resuscitation include the following: 1) should tend to stay in vascular compartment. 2) should be isotonic to plasma 3) solutes' concentration as same as of human plasma 4) Inexpensive [1].

Extravascular lung water (EVLW) is the amount of water that is contained in the lungs outside the pulmonary vasculature. It corresponds to the sum of interstitial, intracellular, alveolar, and lymphatic fluid, not including pleural effusions [1].

An increase in EVLW is the pathophysiological hallmark of hydrostatic pulmonary edema and acute respiratory distress syndrome (ARDS) [2].

EVLW is also high in many septic shock and critically ill patients [3].

For many years, this variable of paramount importance in the pathophysiology of critical illness could only be measured ex vivo. The emergence of transpulmonary thermodilution has opened up the area of EVLW investigation in the clinical setting [1].

During recent years, many studies have been dedicated to EVLW in the field of critical care and ARDS research. They have focused on the validation of its measurement and on its value for the characterization of lung edema, for the prognostic stratification of critically ill patients, for the evaluation of lung-targeted treatments, and for the strategy of fluid management [1].

The physiology of lung water:

Physiologically, there is a normal leakage of fluid and solutes from the pulmonary microvessels into the pulmonary interstitial tissue. Fluid and solutes do not reach the alveoli because of the tight junctions of the alveolar epithelium. This net outward fluid filtration from microvessels to the interstitium is governed by Starling's law, which mainly includes the gradient of hydrostatic and oncotic pressures between the vascular and interstitial spaces and the filtration coefficient of the alveolocapillary barrier (Fig. 1). [1].

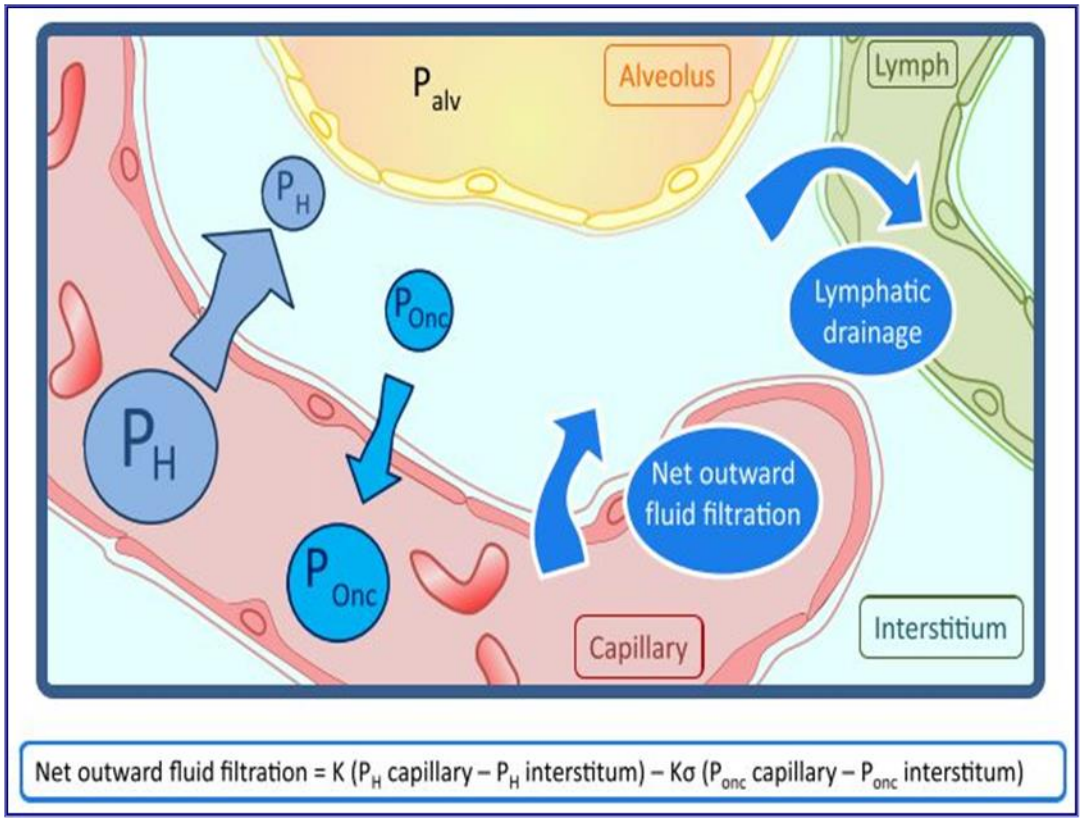

Figure (1): Physiology of lung water: There is a physiological net outward fluid filtration from microvessels to the interstitium governed by the Starling's law, which is strictly controlled by the lymphatic drainage system (Palvalveolar pressure, PH hydrostatic pressure, Ponc oncotic pressure, K filtration coefficient of the alveolocapillary barrier, Kσ reflection coefficient of the alveolocapillary barrier) [1]

To preserve their function of oxygenation, the lungs must be kept dry. The volume of EVLW is strictly controlled by the lymphatic drainage system, which constantly removes EVLW from the interstitial tissue and pours it into the superior vena cava through the thoracic duct. In the normal lung, the value of the normal EVLW indexed to the body weight (EVLWI), which results from the equilibrium between fluid leakage and lymphatic drainage, is <7 mL/kg of body weight [4].

Increased interstitial EVLW can occur as the result of increased pulmonary microvascular hydrostatic pressure or of decreased blood oncotic pressure, or as the result of increased permeability of the alveolocapillary barrier, as typically in ARDS [1].

Measurement of EVLW:

EVLW can be estimated by computed tomography or magnetic resonance imaging, but such techniques are not appropriate for convenient and repeated assessment. Isotopic methods can only be used for research, and electrical impedance tomography is currently not sufficiently validated [5].

Lung ultrasonography is increasingly used to detect lung edema. Nevertheless, its ability to quantify the volume of EVLW is not established. There is no defined method for grading the severity of typical ultrasonography signs of lung edema. Moreover, ultrasonography may be limited by the fact that it assesses lung edema in some specific regions and not in the whole organ [1].

As a first clinical alternative, transpulmonary thermo-dye dilution, which can be considered as the in vivo gold standard for EVLW measurement, has been developed and validated versus gravimetry in animals and human beings [6].

The technique requires a central venous catheter inserted in the superior vena cava and a thermistor-tipped arterial catheter placed in the femoral artery. The thermo-dye dilution is performed by injecting simultaneously through the central venous catheter a cold indicator (cold saline) and a colorimetric indicator (indocyanine green). The volume of distribution of the cold indicator includes the intravascular and the extravascular spaces of the intrathoracic compartment, whereas the colorimetric indicator is strictly an intravascular indicator. Thus, the measurement of EVLW is obtained by subtracting the volume of distribution of these two indicators (Fig. 2). Nevertheless, this method is cumbersome and costly and has been advantageously replaced by the transpulmonary thermodilution technique [1].

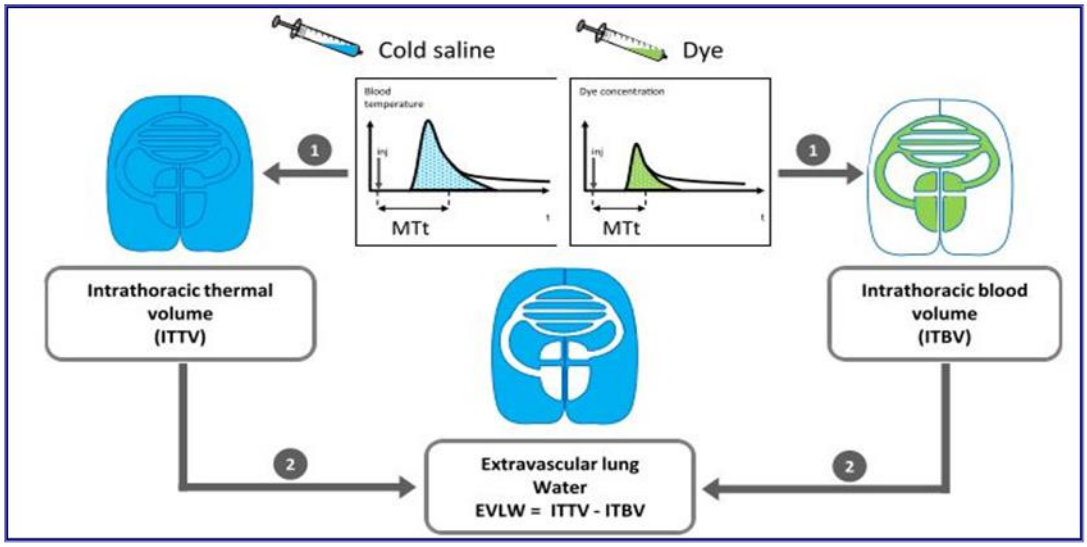

Figure (2): Measurement of extravascular lung water by thermo-dye dilution: MTt, mean transit time. [1]

Validation of EVLW estimated by transpulmonary thermodilution:

The validity of the measurement of EVLWI by transpulmonary thermodilution has been established and consolidated in recent years. EVLWI measured by transpulmonary thermodilution was reasonably correlated

with the value measured by thermo-dye dilution in humans and with the value obtained through gravimetry in animals [7].

The issue of EVLW indexation:

Historically, EVLW measurement has been indexed to the weight of patients at the time of the measurement. However, the main determinant of lung volume is not the patient's weight, but rather the patient's height and perhaps the gender [8].

Two recent studies have suggested that EVLW should be indexed to the height only. Height was the only biometric parameter independently associated with EVLW measurements [9].

Indexing EVLW to the weight underestimates EVLW in the case of overweight, a condition that is common in critically ill patients due to the positive fluid balance [8].

EVLWI was better correlated with lung injury score and oxygenation and was a better predictor of mortality in acute lung injury (ALI)/ARDS patients if indexed to predicted rather than to actual body weight [10].

Limitations of EVLW measurement by transpulmonary thermodilution:

The limitations of EVLWI measurement by transpulmonary thermodilution and the conditions in which it is not reliable have been better characterized in (Table). [1].

Table (): Limitations of extravascular lung water measurement by transpulmonary thermodilution [1]:

Overestimation of EVLW
• Positive end expiratory pressure (potential). • Lung resection (proven).
Underestimation of EVLW
• Positive end expiratory pressure (potential). • Pulmonary vascular occlusion (proven). • Heterogeneous lung injury (potential). • Pleural effusions (plausible).

The accuracy of EVLWI measurement depends on the volume of distribution of the cold indicator. This means that transpulmonary thermodilution can only detect EVLW in lung regions that are reached by the cold indicator, i.e., in well-perfused areas. In experimental studies, the occlusion of large pulmonary arteries led to underestimation of EVLWI by transpulmonary thermo-dye dilution compared with gravimetry, which was corrected after the reopening of the arteries [11].

However, underestimation of EVLWI was observed only when large vessels were occluded, not small vessels. The clinical implication is that the value of EVLWI measured by thermodilution is likely underestimated in the case of pulmonary embolism. During ARDS, the occlusions of the pulmonary vasculature that could result from vascular remodeling, micro thrombi, hypoxic vasoconstriction, or positive end-expiratory pressure (PEEP) mainly affect the small vessels. This suggests that the accuracy of EVLWI measurement should not be impaired by vascular occlusion in this clinical instance [12].

In theory, the effects of PEEP on EVLWI measurement are complex and result from opposite mechanisms. First, PEEP could alter the reliability of transpulmonary thermodilution by having opposite effects on the diffusion volume of the cold indicator. On the one hand, high levels of PEEP could reduce the cold indicator diffusion volume by squeezing pulmonary microvessels, leading to an underestimation of EVLWI. On the other hand, high levels of PEEP could increase the cold indicator diffusion volume by recruiting some atelectatic lung regions and reducing hypoxic vasoconstriction, leading to an overestimation of EVLWI.

Second, PEEP could actually change the volume of EVLWI through opposite mechanisms. By decreasing cardiac output, PEEP may reduce the pulmonary microvascular hydrostatic pressure and hence EVLWI. Also, during ARDS, by increasing the central venous pressure, PEEP could impede the lymphatic drainage of EVLWI. By contrast, during hydrostatic pulmonary edema, PEEP could contribute to the decrease in EVLWI by improving cardiac function through its own hemodynamic effects [13].

Assessment of the lung has always been considered off-limits for ultrasound, since it is standard textbook knowledge that because ultrasound energy is rapidly dissipated by air, ultrasound imaging is not useful for the evaluation of the pulmonary parenchyma [14].

The concept that ultrasound cannot be employed for evaluating the lung is linked to the presence of air, which determines a high acoustic mismatch with the surrounding tissues, causing a complete reflection of the ultrasound beam, preventing the creation of direct imaging of the pulmonary parenchyma [15].

In a normally aerated lung, the only detectable structure is the pleura, visualized as a hyperechoic horizontal line. It is debated whether this line represents an artifact due to a reflection phenomenon at the interface between alveolar air and the soft tissues of the thoracic wall, or it images the real pleura. The pleural line moves synchronously with respiration. This dynamic horizontal movement is called lung sliding [15].

In addition, there are some hyperechoic, horizontal lines arising at regular intervals from the pleural line (the A-lines). When combined with lung sliding, these reverberation artifacts represent a sign of normal or excessive content of air in the alveolar spaces (Fig.) [14].

When the air content decreases and lung density increases due to the presence of exudate, transudate, collagen, or blood in the lung, the acoustic mismatch between the lung and the surrounding tissues is lowered, and the ultrasound beam can be partly reflected at deeper zones and repeatedly. This phenomenon creates some vertical reverberation artifacts known as B-lines (Fig.) [16].

Multiple B-lines are considered the sonographic sign of lung interstitial syndrome, and their number increases along with decreasing air content and increases in lung density [17].

When the air content further decreases, such as in lung consolidations, the acoustic window on the lung becomes completely open, and the lung may be directly visualized as a solid parenchyma (Fig.) [16].

For similar reasons, no B-lines can be seen in the context of a PNX, since B-lines can be visualized only at an air-tissue acoustic interface when the visceral pleura is opposing the parietal pleura. Another sign helps rule out PNX, the lung pulse, which refers to the subtle rhythmic movement of the lung upon the parietal pleura, synchronous with cardiac beats. Like the respiratory movement, this cardiac movement of the lung cannot be detected by ultrasound if air is present between the visceral and parietal pleura [18].

How to perform lung ultrasound (LUS):

LUS can be performed on the whole chest, just laying the probe in the intercostal spaces, avoiding the ribs. The probe can be positioned both longitudinally, perpendicular to the ribs, and obliquely along the intercostal spaces. The longitudinal approach allows visualization of the so-called “bat-sign” (Fig.). In a longitudinal view, the bat sign identifies the upper and lower ribs (the wings of the bat), and, a little deeper, the pleural line (the back of the bat). The oblique approach allows visualizing a larger part of the pleural line, which is not interrupted by the rib shadows (Fig.) [14].

The diagnostic approach based on LUS can vary according to different settings and clinical situations, following the main principles of what is today known as “point of care ultrasound”. For example, in a stable patient with acute spontaneous pleuritic pain, ultrasound examination will start from the painful chest area, focusing on signs of focal pleural and parenchymal abnormality [19].

The clinical suspicion and pretest probability will guide the diagnostic process to rule in or rule out with high accuracy several conditions, such as PNX, pleuritis, pneumonia, lung peripheral infarction [20].

In this setting, a highly specific sign is the lung point, which represents the transition point between the typical sonographic pattern of PNX (absence of lung sliding and of B-lines) into the normal pattern of lung sliding, and

depicts the physical limit of PNX as mapped on the chest wall [20]. However, the lung point can be employed to detect the extension of PNX, but not its volume [14].

The sum of the B-lines found on each scanning site yields a score denoting the extent of the pulmonary interstitial syndrome. Zero is defined as a complete absence of B-lines in the investigated area. When B-lines are few in a scanning site, they can be easily counted. When they are more numerous, they tend to be confluent and it is less easy to clearly enumerate them. To obtain a semi-quantification of the sign, you can consider the percentage of the scanning site occupied by B-lines (i.e., the percentage of white screen compared to black screen below the pleural line), and then divide it by ten (i.e., 30% corresponds to about 3 B-lines, 70% corresponds to about 7 B-lines, and so on) [14].

It is advisable to always scan the costophrenic angles on both sides and to assess the "curtain sign", that is the relative movement of the lung, which covers the sub diaphragmatic organ during inspiration (the liver on the right side, or the spleen on the left side), and the diaphragm [14].

This is the region where free pleural effusion can be detected. Identification of pleural effusion is the most established application of LUS. The effusion should first be sought in dependent zones (i.e., lateral and posterior chest). By LUS, it is possible to differentiate pleural effusion from atelectasis, consolidations, masses, or an elevated hemi-diaphragm that sometimes may hardly be distinguished on a chest X-ray. LUS images pleural effusion as an anechoic or hypoechoic space between the two pleural layers, with the lung appearing either aerated or consolidated. LUS for the diagnosis of pleural effusion is especially valuable in critically ill patients, showing better sensitivity and reliability than bedside chest X-ray [21].

Ultrasound can detect the effusion, evaluate its volume, provide information on its nature, and indicate the appropriate area for thoracocentesis. Moreover, LUS outperforms CT scan in the diagnosis of complex effusion due to its ability to distinguish septa and fibrin inside the collection [22].

Position:

LUS can be performed in any position (supine, lateral decubitus, or prone), since lung abnormality distribution does not change so rapidly that significant information would be missed (apart from pleural effusion) just by changing the patient's position. The supine position is perfect for scanning the anterior chest, whereas the lateral chest may be examined in the semi-supine position (on the left decubitus to scan the right axillary lines and on the right decubitus to scan the left axillary lines). The ideal position for scanning the posterior chest is with the patient sitting on the bed, his/her back turned to the operator (Fig.) [14].

Indeed, it is also possible to scan patients while they are standing, sitting, or lying flat, without significant differences in results. The only real limitation is when LUS needs to be extended to the dorsal chest of a patient lying down who is intubated in the intensive care unit, or a patient who is unconscious and cannot be moved. In these situations, using small probes that may be better placed between the bed and the patient may allow the best result [14].

Probes:

The LUS examination can be performed using any commercially available 2-D scanner. Different transducers have been used, such as phased array (cardiac), convex (abdominal), micro convex, and linear (vascular) probes. Higher frequencies and macro probes are useful for the evaluation of the pleural line and subpleural space, so should be preferred for assessing PNX. Phased-array probes can be successfully employed to detect pleural effusion, due to low frequency and consequent ability to provide a deeper view of the chest. However, these latter probes have limitations in detecting PNX, and when a detailed examination of the subpleural space is needed. The convex and micro convex probes are the most universally used, due to their intermediate frequency values, which allow a reasonable visualization of the pleural line and subpleural space, without losing the overview of the chest [14].

B-lines can be detected by all these different probes, but again, low-frequency probes are probably the best for this application. Although the number of B-lines may be slightly different when using different probes in a specific chest site, the overall clinical picture does not change by changing the transducer [23].

Interpretation of LUS:

Interpretation of LUS images is usually not very challenging. A LUS pattern showing absent lung sliding, or multiple B-lines, or a lung consolidation may not be enough to establish a specific diagnosis, since it can be linked to different pathologic conditions [24].

The clinical condition of the patient is probably the most important feature that helps interpret LUS findings and influence patient management. For instance, in an unstable patient with signs and symptoms of hemodynamic shock or cardiac arrest, the absence of any movement of the pleural line, either respiratory (lung sliding) or cardiac (lung pulse), coupled with the absence of B-lines raises such a high suspicion of PNX that may lead to the placement of a chest tube, even without the need for a more extensive ultrasound examination or for other diagnostic techniques [19].

On the other hand, such a simplified protocol is not advisable in a stable patient, where there is time to extend the examination looking for adjunctive signs that enhance the specificity of the ultrasound diagnostic process. The distribution of B-lines and pleural line characteristics are also crucial to increasing the specificity of LUS. B-lines due to cardiogenic pulmonary edema are usually bilateral, start appearing in the dependent zones and usually diffusing or recovering symmetrically. B-lines due to pulmonary fibrosis generally start at the posterior lung basis and are often associated with irregularity of the pleural line and subpleural small consolidations (Fig.) [14].

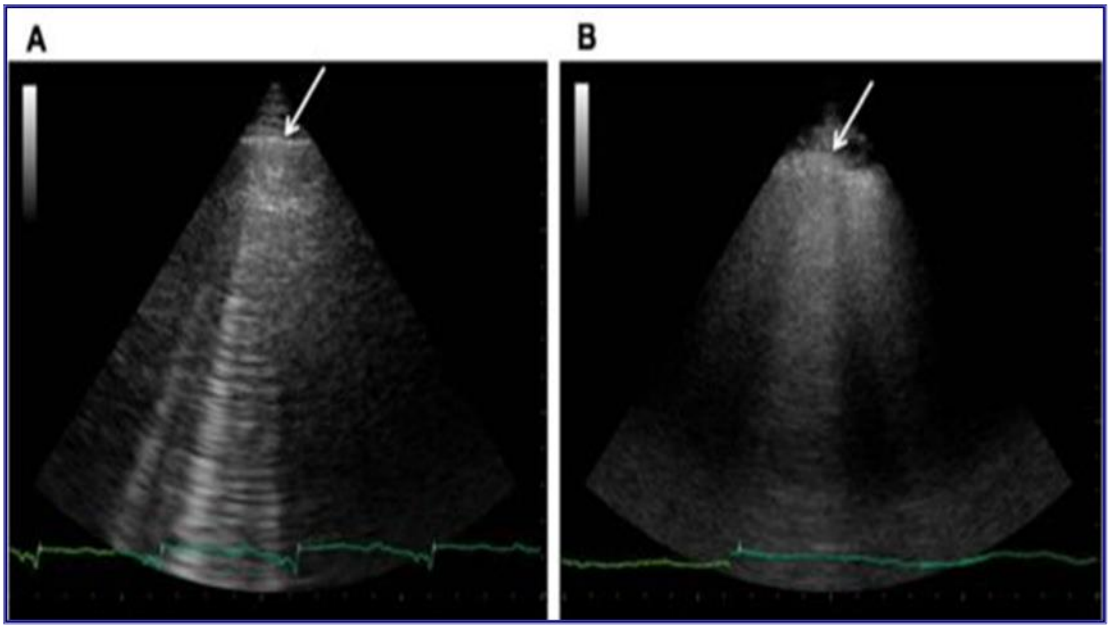

Figure (3): Multiple B-lines in cardiogenic pulmonary edema and lung fibrosis: A. Multiple B-lines in a patient with cardiogenic pulmonary edema: the arrow indicates a normal pleural line. B. Multiple B-lines in a patient with pulmonary fibrosis: the arrow indicates the abnormal pleural line which looks irregular [14].

In contrast to pulmonary edema due to congestion or overhydration, acute lung injury/ARDS shows a dishomogeneous and irregular pattern, featuring many subpleural consolidations, highly fragmented pleural line, and intense hyperlucent multiple B-lines alternating with spared areas. This irregular distribution of B-lines contrasts with that observed in cardiogenic pulmonary edema, where B-lines are usually detected in more homogenous distribution, which is gravity-related, and it is quite rare to visualize subpleural consolidations [25].

The presence of a lung consolidation with blurred margins in a patient with fever will raise a high suspicion of pneumonia, whereas a triangular-shaped consolidation with the absence of any color-Doppler signal, in a patient with chest pain and clinical risk factors for thrombo-embolic disease, will raise the suspicion of a peripheral pulmonary infarction. In the case of multiple bilateral B-lines that resolve in days during ordinary

treatment, or even in a few hours by an acute diuretic load, the cardiogenic or volume overload origin of B-lines is strongly suggested [15].

Similarly, in end-stage renal disease patients, B-lines decreasing or even disappearing after either a hemodialysis or peritoneal dialysis session, indicate resolution of pulmonary congestion due to overload [26].

Additional information derived from a bedside ultrasound evaluation of other organs may also be helpful. This approach has been recently described in patients with undifferentiated hypotension, where the integrated point of care multiorgan ultrasonography of the heart, inferior vena cava, lungs, and abdomen significantly agreed with a final clinical diagnosis obtained by retrospective chart review [19].

Limitations:

LUS limitations are essentially patient-dependent. Obese patients may be more difficult to examine due to the thickness of their ribcage and soft tissues. The presence of subcutaneous emphysema or large thoracic dressings alters or precludes the propagation of the ultrasound beams to the subpleural lung parenchyma. It should be emphasized that LUS does not rule out pulmonary abnormalities that do not reach the pleura. This physical limitation is especially important when ruling out consolidations, since some consolidations, especially in the case of tumors, can be medially located and surrounded by aerated lung, which will prevent their visualization by sonography [14].

The pulmonary interstitial syndrome from different etiologies sometimes may spare (although rarely) the subpleural space. A focal interstitial syndrome can sometimes be the “peripheral alarm” of a more medial pathological condition, for example, in the case of perilesional interstitial edema, due to either inflammation or impaired lymphatic drainage [14].

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