Comprehensive Assessment of Fluid Status by Point-of-Care Ultrasonography

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Abstract
The management of complex fluid and electrolyte disorders is central to the practice of nephrologists. The sensitivity of physical examination alone to determine fluid status is limited, precluding accurate clinical decision making. Point-of-care ultrasonography (POCUS) is emerging as a valuable, noninvasive, bedside diagnostic tool for objective evaluation of physiologic and hemodynamic parameters related to fluid status, tolerance, and responsiveness. Rapid bedside sonographic evaluation can obtain qualitative data on cardiac function and quantitative data on pulmonary congestion. Advanced POCUS, including goal-directed Doppler echocardiography, provides additional quantitative information, including flow velocities and pressures across the cardiac structures. Recently, abnormal Doppler flow patterns in abdominal organs secondary to increased right atrial pressure have been linked to congestive organ damage, adding another component to the hemodynamic assessment. Integrating POCUS findings with clinical and laboratory data can further elucidate a patient’s hemodynamic status. This drives decisions regarding crystalloid administration or, conversely, diuresis or ultrafiltration and allows tailored therapy for individual patients. In this article, we provide an overview of the focused assessment of cardiovascular function and pulmonary and venous congestion using POCUS and review relevant literature.

Case
A 63-year-old man with a medical history of hypertension, obesity, and heart failure (HF) with reduced ejection fraction presents to the nephrology clinic for assessment of an elevated serum creatinine. His baseline serum creatinine was approximately 0.6–0.8 mg/dl. He complains of abdominal distension, loose stools, and dyspnea on exertion. Medications include carvedilol, lisinopril, isosorbide mononitrate, and hydralazine. Diuretics were held by the referring physician due to a rise in creatinine. Physical examination demonstrates BP of 92/59 mm Hg with a heart rate of 65 bpm. Cardio-pulmonary exam reveals pedal edema, but no obvious jugular venous distention, rales, or third heart sound. Urine sediment is bland. Review of chest roentgenogram obtained in the primary care setting shows no evidence of pulmonary edema or pleural effusions. Laboratory studies show stable serum creatinine 1.4 mg/dl after discontinuation of diuretics. What is the next step?

Introduction
Assessment of fluid and hemodynamic status is a critical skill for nephrologists, central to almost every consult from hypertension and electrolyte disorders to management of AKI and ESKD. Fluid status assessment has a storied tradition in which physical exam signs of jugular venous distention, third heart sounds, rales, and peripheral edema have been learned and reproduced by generations of physicians. These signs are helpful in extreme cases, but are insensitive for the detection of volume overload (1). Radiographic signs of volume overload, such as pleural effusions and Kerley B lines, aid in fluid status assessment, but lack sensitivity (2). Natriuretic peptides and pulmonary artery catheters also have limitations (3,4). In the past 30 years, point-of-care ultrasonography (POCUS) has expanded from a niche subspecialty skill to a cornerstone of bedside diagnosis (5,6). Ultrasound allows us to directly visualize the body in a way that was previously inaccessible. POCUS involves answering focused clinical questions using bedside ultrasonography and increases the sensitivity of the conventional physical examination (7–12). As we move forward, ultrasonographic indicators of fluid status are being developed and validated. Some are intuitive to our understanding of physiology, and others are novel markers of previously unknown significance (Figure 1). Having said that, POCUS findings should be interpreted in conjunction with other clinical parameters—such as vital signs, body weight, mucous membrane examination, capillary refill time, and axillary moisture—and not viewed as an alternative to physical examination or standard imaging studies. We describe the most well-validated indices of fluid status: focused sonographic assessment of the heart, abdominal veins, and lungs (the pump-pipes-leaks approach) (13) to gain insight into systemic hemodynamics and guide fluid management decisions.

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Lung Ultrasound

Extravascular lung water (EVLW), or the fluid content in the lung interstitium, is an important indicator of fluid status, often guiding management decisions in clinical practice. It essentially depends on left ventricular (LV) filling pressures and permeability of the pulmonary vasculature. In the recent past, lung ultrasound (LUS) has emerged as a valuable bedside tool to detect pulmonary congestion, even before it is clinically apparent (14). Moreover, LUS is technically the least challenging of all of the sonographic applications described in this review. There are data suggesting that nephrologists can be effectively trained to measure EVLW using LUS by an entirely internet-based program (15).

Interpretation

LUS primarily involves interpretation of the artifacts rather than visualization of the pulmonary parenchyma because air is strongly reflective to the ultrasound beam. In normal aerated lung, the only detectable structure is the pleura, which appears as a shimmering hyperechoic (bright) horizontal line in between the rib shadows. The shimmer or synchronous horizontal movement with respiration denotes pleural sliding. A-lines are equidistant hyperechoic horizontal lines seen on a normal LUS (Figure 2A). These are reverberation artifacts formed due to multiple reflections of the ultrasound beam between the transducer and the pleura with underlying air-filled lung. When the air content in the lung decreases due to transudate or exudate in the interstitium, vertical hyperechoic artifacts are seen, termed the B-lines (Figure 2B). These are the ultrasound equivalents of the Kerley B lines seen on a chest radiograph, and they provide a semiquantitative estimate of the amount of EVLW. B-lines arise from the pleural line, extend to the end of the image without fading, and move synchronously with lung sliding. A positive “B-line region or zone” is defined as the presence of three or more B-lines in a longitudinal plane between two ribs. Two or more positive regions bilaterally constitutes “interstitial syndrome” and indicates diffuse pulmonary edema (16). B-lines can also be seen in conditions other than cardiogenic pulmonary edema—such as focal pneumonia, acute respiratory distress syndrome, pulmonary fibrosis, and contusion—and, hence, should be interpreted in the appropriate clinical context. These conditions are typically associated with thickened/irregular pleural line and nonhomogeneous distribution of B-lines. When the air content in the lung further decreases, such as in alveolar consolidation, lung parenchyma can be visualized on ultrasound similarly to that of liver and spleen (Figure 2C). In contrast, pleural effusion appears as an anechoic (black) space above the diaphragm, typically surrounding the atelectatic or consolidated lung (Figure 2D).

Most research studies, particularly in patients with ESKD, used a 28-zone scanning technique to obtain the total B-line count, which involves scanning of the anterior and lateral chest from the second to fourth (on the right side to the fifth) intercostal spaces and from the parasternal to midaxillary lines on each side (Figure 3A). However, in routine clinical practice, an eight-zone scanning technique is frequently used in which two anterior and two lateral areas are examined on each hemithorax (Figure 3B) (17). Recently, Torino et al. (18) showed that the eight-zone technique correlates well with the classic 28-zone score and also retains its prognostic significance (discussed below).
Diagnostic Performance

The diagnostic performance of LUS to detect pulmonary congestion is far superior compared with auscultation. For example, in a study including 79 patients receiving hemodialysis who were deemed to be at higher cardiovascular risk, only about half of those with severe congestion on LUS (defined as >30 B-lines on a 28-zone scan) had crackles on lung auscultation. Likewise, in patients with moderate congestion on LUS (15 to <30 B-lines), the prevalence of crackles was only 31% (1).

In patients with acute decompensated heart failure, LUS was shown to be more sensitive for detection of pulmonary edema than chest radiography, which is the typical first-line imaging (2). In addition, LUS has demonstrated substantial correlation with cardiac catheterization–derived LV end-diastolic pressure, making it a valuable adjunct to echocardiography and clinical variables in the management of patients with HF (19).

Prognostic Significance

LUS-detected pulmonary congestion is associated with adverse outcomes, even in patients who are asymptomatic. For instance, in a multicenter observational study including 392 patients with ESKD who were on hemodialysis, those with very severe congestion (>60 B-lines on a 28-zone scan) had a 4.2-fold risk of death (hazard ratio, 4.20; 95% CI, 2.45 to 7.23) and a 3.2-fold risk of cardiac events (hazard ratio, 3.20; 95% CI, 1.75 to 5.88) after adjusting for HF class and other risk factors compared with those having mild or no congestion (<15 B-lines) (20).

Analogously, in the context of HF, residual lung congestion at hospital discharge and in the outpatient clinic has been shown to be a strong predictor of outcome (21–23).

Role in Guiding the Therapy

Because LUS provides dynamic information, the change in B-line count can be used to monitor the effectiveness of decongestive/ultrafiltration therapy and titrate it accordingly (24–27). In a recent randomized controlled trial including 71 patients with ESKD who were “clinically euvoletic,” those who underwent prehemodialysis, LUS-guided, dry-weight reduction achieved a greater reduction in weight compared with those receiving standard care, correlating with the change in number of B-lines (~5.3 in active group versus +2.2 in controls). In addition, the magnitude of 48-hour ambulatory BP reduction was significantly greater in the active group at 8 weeks (systolic ~6.61 versus +0.67 mm Hg and diastolic ~3.85 versus +0.55, P = 0.03) (28).

Similarly, in patients with acute exacerbation of HF, B-line count has been shown to consistently decrease with diuretic therapy soon after presentation, thereby guiding further management (29). In ambulatory patients with chronic HF, an eight-zone, LUS-guided diuretic therapy was associated with a significant reduction in HF-related urgent care visits compared with standard care (30). Clinical trials are in progress that may provide further insights into long-term outcomes of LUS-guided therapy (31).

Figure 2. | Common lung ultrasound findings. Sonographic images demonstrating (A) normal lung with A-lines (arrows); (B) vertical B-lines (arrows), indicating interstitial edema. Arrowhead points to the pleural line. (C) Consolidated lung (arrow); (D) pleural effusion (asterisk) above the diaphragm (arrow). ‘LOGIQ P9’ and the letter ‘P’ in the images indicate ultrasound screen orientation markers.
Focused Cardiac Assessment

Hemodynamic assessment is predicated on the basic knowledge of cardiac function and the presence of pathologies. In contrast to referral echocardiography, focused cardiac ultrasound (FOCUS) is a limited study aimed at answering specific questions, such as evaluation of the left and right ventricular function and presence or absence of pericardial effusions. Most commonly, FOCUS consists of a series of five, two-dimensional, echocardiographic clips without spectral Doppler, in which images of the heart are obtained in orthogonal planes to provide the relevant physiologic information (detailed in Figure 4) (32,33). Further clues to fluid status can be gleaned from additional studies using Doppler ultrasonography. Table 1 summarizes the image acquisition of basic echocardiographic views, common structures visualized, sonographic parameters, and pathologies assessed in each view. A brief note on common pathologies is provided below.

Pericardial Effusion

Pericardial effusion is an important cause of hypotension and hemodynamic compromise, which can be quickly identified on FOCUS. It appears as an anechoic (black) space between the two pericardial layers. Parasternal long-axis and subcostal views allow better visualization of the effusion, although it is generally identifiable on all of the standard FOCUS views (Figure 5A). In terms of severity, separation between the pericardial layers in diastole of <1 cm is considered mild, whereas 1–2 cm is considered moderate and >2 cm is considered severe effusion (34).

LV Systolic Dysfunction

Qualitative estimation or “eyeballing” of the LV ejection fraction is another key component of FOCUS, which involves observing wall thickening and motion during the cardiac cycle. In general, the LV walls should approximate by one fourth or more in parasternal views. In patients with depressed LV function, both wall thickening and inward motion are decreased. Conversely, a hyperdynamic ventricle, i.e., where ventricular walls/papillary muscles in the parasternal short axis view almost touch at end-systole, is indicative of volume depletion in the appropriate clinical context.

Relative Chamber Size

A normal right ventricle (RV) cavity diameter is less than two thirds of the LV and can quickly dilate with pressure or volume overload. Apical four-chamber and parasternal short axis views are good for assessing this. With volume overload, the RV becomes dilated, and the interventricular septum is flattened in diastole, giving the appearance of “D” to the LV in the parasternal short axis view. This is called the “D-sign” (Figure 5B). Although such patients are hypotensive, empirically administering intravenous fluids causes further compromise of the LV cavity and reduced cardiac output.

Right Atrial Pressure

Inferior vena cava (IVC) ultrasound is used to estimate right atrial pressure (RAP) and get an idea of the resistance to venous return. In patients who are spontaneously
breathing, the IVC collapses during inspiration due to negative intrathoracic pressure. An IVC diameter of ≤2.1 cm and collapsibility of >50% with a sniff indicates normal RAP of 3 mm Hg (0–5 mm Hg), an IVC diameter of >2.1 cm with <50% inspiratory collapse indicates high RAP of 15 mm Hg (10–20 mm Hg), and scenarios in between correspond to an intermediate value of 8 mm Hg (5–10 mm Hg) (35). Figure 5, C and D, demonstrates sonographic images of large and small IVC obtained from a patient with HF and volume depletion, respectively. However, these cutoffs cannot be applied in patients who are mechanically ventilated because the IVC is dilated at baseline due to positive pressure.

| Table 1. Summary of the basic echocardiographic views |
|---------------------------------------------------|
| **FOCUS View** | **Image Acquisition** | **Structures** | **Key Assessments** | **Common Pathologies** |
| PSLA | Parasternal third to fifth intercostal space, indicator to patient’s right shoulder | RV, LV, LA, LVOT, AV, MV, descending aorta | Visual assessment of LV EF | LV dysfunction, pericardial effusion, left-sided pleural effusion posterior to descending aorta |
| PSSA | Second to fourth intercostal space, indicator to left shoulder | RV, LV, interventricular septum | Visual assessment of LV EF and LV shape | LV dysfunction, septal flattening, or shift indicative of RV dysfunction, pericardial effusion |
| Apical | Point of maximum impulse, indicator to left axilla | RA, LA, RV, LV, LVOT, MV, TV | RV/LV comparison, color Doppler of tricuspid and mitral valves | RV failure, pericardial effusion, valvular regurgitation |
| SX | Below the xiphoid process, indicator to patient’s L. | RA, LA, RV, LV | Pericardium | Pericardial effusion, LV dysfunction |

Table includes the image acquisition, common structures, sonographic parameters, and pathologies assessed in each view. Note that this list of assessments/pathologies is not exhaustive, and each pathology can be seen in multiple views. FOCUS, focused cardiac ultrasound; PSLA, parasternal long axis view; PSSA, parasternal short axis view; RA, right atrium; TV, tricuspid valve; SX, subxiphoid view.
ventilation and may not collapse at all during respiration. Although novice POCUS users are enthusiastic about IVC ultrasound because it is relatively easy to learn, interpreting it in isolation is subject to numerous pitfalls. For example, small collapsible IVC is seen in normal state of health and equating it with volume depletion, without considering the clinical context, leads to unnecessary fluid administration. Moreover, the magnitude of the respiratory effort significantly affects collapsibility (for example, a frail elderly woman versus a muscular young man), altering the interpretation. Furthermore, technical factors—such as obesity, surgical dressings, increased intra-abdominal pressure, or mistaking aorta or dilated bowel for IVC—can result in errors. Depending on how the ultrasound beam is aimed, it may not depict the true diameter of the vessel. Notably, studies demonstrate moderate to poor inter-rater agreement between IVC measurements (36,37). Although IVC is a good indicator of central venous pressure (CVP), it is not reliable to assess fluid responsiveness (38). Therefore, IVC ultrasound should be interpreted in conjunction with other POCUS findings and the overall clinical picture.

Users with further training in Doppler echocardiography assess additional parameters, such as cardiac output and its response to fluid administration, LV filling pressures, and right ventricular filling pressures at the bedside (39).

Venous Doppler
Organ dysfunction in HF is closely related to venous congestion (40). CVP is the strongest hemodynamic determinant for the development of worsening renal function in patients with decompensated HF (41). Normal and abnormal patterns of flow in abdominal and central veins result from retrograde transmission of CVP (42). POCUS can enhance the clinical evaluation of venous congestion using venous Doppler in addition to IVC ultrasound (43,44). Figure 6 illustrates the technique of obtaining the sonographic images of hepatic, portal, and intrarenal veins.

Figure 5. | Common findings on focused cardiac ultrasound. (A) Pericardial effusion (asterisk) surrounding the heart, seen from the subcostal window. (B) D-sign: interventricular septal flattening seen from the parasternal short axis view. (C) Plethoric IVC (arrow). (D) Small IVC (arrow).

Hepatic Vein Doppler
Blood flow in the hepatic veins (HVs) is pulsatile, and changes in its velocity reflect changes in RAP. The normal HV flow pattern consists of two antegrade waves (a larger systolic [S] and a smaller diastolic [D] wave corresponding to CVP “X” and “Y” descent, respectively) and one or two retrograde waves (a larger “A” wave and smaller “V” wave corresponding to CVP “A” and “V” waves, respectively). Frequently, the V wave is not seen (Figure 7A) (26). Understanding the origin of hepatic flow waves aids in understanding common pathologic alterations. For example, severe pulmonary hypertension can manifest as prominent A waves and/or decreased D wave amplitude because of an increase in RV end-diastolic pressure (45). Systolic dysfunction of the RV and tricuspid regurgitation (TR) both alter the RAP during ventricular systole, leading to progressive decrease in the peak velocity of the S wave (46). In addition, severe TR can cause S wave reversal (Figure 7B) (47). Thus, HV Doppler provides relevant information about the filling pattern of the RA.
Portal Vein Doppler

As opposed to HV flow, the splanchnic circulation is an isolated vascular unit protected from the systemic circulation by the resistance of postsinusoidal sphincters (48). Thus, normal portal flow is continuous or only mildly pulsatile (Figure 7A) (49). However, pathologic increases in RAP can be transmitted through liver sinusoids into the portal vein (Figure 7B) (50). Portal vein pulsatility was originally described in patients with severe TR (51), but has now been described in multiple conditions associated with increased RAP (52,53). Increased pulsatility in portal venous flow has been associated with a higher N-terminal pro–brain natriuretic peptide (54), higher systolic pulmonary artery pressures (55), positive fluid balance (56), and RV dysfunction (56,57). Portal vein flow alterations can be quantified by the pulsatility fraction \(100\left(\frac{V_{\text{max}} - V_{\text{min}}}{V_{\text{max}}}\right)\); a pulsatility fraction \(\geq 30\%\) is considered mild, whereas \(\geq 50\%\) is considered severely elevated (54).

There are a few studies evaluating the relationship between portal vein pulsatility and patient outcomes in the setting of HF. In a landmark study by Beaubien-Souligny et al. (54), a portal vein pulsatility fraction of \(>50\%\) and severe alterations in intrarenal venous flow (IRVF) were associated with an increased risk of AKI in patients who underwent cardiac surgery. The inclusion of portal vein Doppler significantly improved AKI risk prediction. Furthermore, alterations in portal vein flow have been associated with the development of congestive hepatopathy (58), encephalopathy (59), and major complications in patients undergoing cardiac surgery (56), and may be a useful prognostic marker in patients hospitalized for acute HF (60). Together, these data suggest that sonographic evaluation of portal vein pulsatility could become a useful tool for the diagnosis and management of venous congestion. In our experience, increased portal vein pulsatility fraction associated with volume overload often improves with diuretic treatment (61,62).

Although a plethoric, noncollapsible IVC indicates venous congestion, caution must be exercised when interpreting it in patients with cardiac conditions impeding venous return (chronic RV dysfunction/TR, RV myocardial infarction, cardiac tamponade); these patients may be fluid responsive despite IVC plethora (63). The evaluation of hemodynamic AKI in these conditions can be enhanced by assessing portal vein flow; increased portal pulsatility is suggestive of congestive AKI, which can potentially improve with decongestive therapy (62). A case of portal vein flow normalization with diuresis, even in the presence of persistent severe TR, is presented in Figure 7C.

Portal venous flow cannot be relied upon in patients with cirrhosis because both absent pulsatility in the presence of severe congestion and increased pulsatility unrelated to RAP can occur (64–67). Occasionally, portal vein pulsatility can be seen in individuals who are thin and healthy (68). Given these limitations, portal vein pulsatility fraction should not be interpreted in isolation. A recent study evaluating IVC size and hepatic, portal, and intrarenal vein Doppler flow patterns found increased specificity using the combination of multiple POCUS markers to identify clinically significant venous congestion (69).

Intrarenal Venous Doppler

Similar to the portal vein, the flow pattern in intrarenal (arcuate and interlobar) veins depends on the surrounding renal parenchymal histology as much as right atrium
function (70). Iida et al. (71) used Doppler imaging to evaluate IRVF patterns in patients with HF. IRVF waveforms were divided into three flow patterns: continuous, biphasic, and monophasic (Figure 7D). The IRVF profile was altered by increases in RAP, but was not associated with changes in cardiac index. The monophasic pattern was associated with significant TR. IRVF strongly correlated with clinical outcomes, including death from cardiovascular disease or unplanned hospitalization for HF. This correlation was independent of RAP. Similar results were obtained by Puzzovivo and colleagues (72).

Supporting the role of IRVF alterations as a marker of venous congestion, experimental fluid expansion worsened the IRVF pattern and correlated with less diuretic efficiency in patients with HF with preserved ejection fraction (73). A recent study in patients with pulmonary arterial hypertension also showed adverse outcomes were associated with IRVF alterations (74). Three patients from this study who developed severe AKI with diuretic-resistant fluid overload and required RRT exhibited a monophasic IRVF pattern.

Whereas HV Doppler mainly reflects the right atrium filling pattern, portal and intrarenal venous Doppler provide additional information about right atrial filling pressure and its correlation with congestive organ injury (43). Whether interventions aimed at addressing abnormal organ flow patterns can improve relevant outcomes in patients with venous congestion remains unknown. Moreover, the cause-effect relationship between sonographic markers of venous congestion and AKI remains elusive at this time and must be evaluated by larger studies. POCUS alterations that occur in venous congestion are summarized in Figure 8.

Going Back to the Case
FOCUS revealed severely decreased LV ejection fraction and dilated RV with significantly reduced function. Numerous bilateral B-lines were present on LUS. IVC was 2.4 cm in diameter with no respiratory variation and there were no significant ascites (Figure 9A). These findings were compatible with HF with reduced ejection fraction and severe RV failure, and moderate pulmonary congestion. To evaluate for the presence of systemic venous congestion, a bedside Doppler ultrasonography was performed. HV Doppler demonstrated decreased D wave amplitude (seen in severe pulmonary hypertension and abnormal RV relaxation), portal vein pulsatility fraction of 100%, and a monophasic IRVF (Figure 9B). Although a plethoric IVC was suggestive of venous congestion, the presence of severe RV dysfunction and significant pulmonary hypertension makes it less reliable (can be chronically dilated) and the patient might still be fluid responsive. However, both portal and IRVF patterns indicated that backward transmission of RAP was significant enough to lead to abdominal organ congestion, suggesting congestive kidney injury.

Treatment and Outcome
Given these findings, diuretic therapy was restarted with dose intensification. On follow-up, the patient showed a...
steady decrease in weight (approximately 10 kg) and noticeable improvement in symptoms. Although his serum creatinine worsened from 1.4 to 1.8 mg/dl initially, it improved and stabilized at 0.6 mg/dl with continued diuresis. Follow-up POCUS evaluation of venous congestion showed markedly improved flow patterns on both portal vein (pulsatility fraction = 32%) and IRVF (biphasic pattern) (Figure 9C).

POCUS Education and Program Development

Despite the stated advantages of POCUS and growing interest among nephrologists to acquire this skill, training opportunities remain sparse at this time. Workshops and short courses organized by professional societies, such as the National Kidney Foundation and the American Society of Nephrology, provide an introduction to the technique and interpretation, but continued practice, preferably under supervision, is vital to achieve mastery. Longitudinal curricula are shown to have a favorable effect on long-term skill retention (75,76); as such, integrating POCUS training into the 2-year nephrology fellowship offers a conceivable advantage. Nevertheless, only a few nephrology programs currently train their fellows beyond kidney ultrasound (77), the key problem being lack of trained faculty. We suggest
that nephrology divisions identify faculty interested in learning/teaching POCUS and support them to pursue a structured, multicomponent certification program such as the one offered by the American College of Chest Physicians (78). Thereafter, multispecialty collaboration at the institutional level, with specialties such as emergency medicine and radiology, facilitates organizing the curriculum, training more faculty, and quality assurance. In addition, taking advantage of the online POCUS educational tools built for nephrologists, such as NephroPOCUS.com, minimizes the need to create new didactic material by individual fellowship programs. Furthermore, forming an expert panel and putting forward consensus guidelines by nephrology professional organizations would help standardize POCUS training.

Conclusion

Physical examination is limited in determination of fluid status. Augmented examination using POCUS is gaining popularity as a noninvasive bedside tool that provides a detailed insight into cardiovascular physiology and hemodynamic determinants of fluid status. Evaluation of cardiac function by POCUS, pulmonary congestion by LUS, and systemic venous congestion by abdominal venous Doppler can guide management by providing information on fluid tolerance and responsiveness. Thorough multigorgan assessment should be undertaken to offset the limitations of individual POCUS applications. Integrating POCUS findings with clinical and laboratory data allows decisions regarding crystalloid administration or diuresis to be tailored according to the individual physiology with a risk-benefit analysis of the preferred therapeutic strategy. Further studies are needed to determine whether management guided by abnormalities detected on POCUS translates to improved clinical outcomes. On a note of caution, POCUS is not a replacement to clinical judgment and is susceptible to limitations like any other technology. Improper technique, wrong interpretation, and over-reliance on an isolated finding can potentially lead to mismanagement of the patient. Therefore, the nephrology community should focus on developing POCUS training standards and a robust certification process to ensure quality.

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E. Reisinger conceptualized the study, wrote the original draft, and reviewed and edited the manuscript; and A. Koratala provided supervision.

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