




## CKJ REVIEW

## PoCUS in nephrology: a new tool to improve our diagnostic skills

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### ABSTRACT

Point-of-Care Ultrasonography (PoCUS) aims to include a fifth pillar (insonation) in the classical physical examination in order to obtain images to answer specific questions by the clinician at the patient's bedside, allowing rapid identification of structural or functional abnormalities, enabling more accurate volume assessment and supporting diagnosis, as well as guiding procedures. In recent years, PoCUS has started becoming a valuable tool in day-to-day clinical practice, adopted by healthcare professionals from various medical specialties, never replacing physical examination but improving patient and medical care and experience. Renal patients represent a wide range of diseases, which lends PoCUS a special role as a valuable tool in different scenarios, not only for volume-related information but also for the assessment of a wide range of acute and chronic conditions, enhancing the sensitivity of conventional physical examination in nephrology. PoCUS in the hands of a nephrologist is a precision medicine tool.

### LAY SUMMARY

Point-of-care ultrasonography (PoCUS) is defined as the correct acquisition and interpretation of ultrasound images at the patient's bedside in order to improve the sensitivity of the physical examination, personalize treatments or decide on future examinations. In nephrology this strategy was mainly framed in the study of arteriovenous fistula for haemodialysis. However, other types of applications are emerging, such as the assessment of congestion by means of pulmonary and vascular ultrasound, as well as integrating these findings with echocardiography. The use of

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PoCUS also allows the identification of one of the most common mechanisms of renal damage in the congested patient, congestive nephropathy. Using this dynamic and reproducible strategy allows improvement of the diagnostic capacity of the nephrologist at the bedside and the ability to guide treatments such as diuretics.

**Keywords:** focused cardiac ultrasound, lung ultrasound, nephrology, PoCUS, venous congestion assessment

## BACKGROUND

Point-of-care Ultrasonography (PoCUS) has emerged as a novel approach that focuses on enhancing physical examination at the patient's bedside (point of care). This tool can be used by any clinician to answer focused questions related to the diagnosis, guiding treatment or bedside procedures [1]. PoCUS is not intended to replace physical examination (PE) but aims to enhance it. The integration of PE and insonation will allow for proper guidance and interpretation of ultrasound (US) images. PoCUS helps with diagnosis, allowing quick identification of structural or functional abnormalities, providing a more accurate volume assessment, including tissue and vascular congestion [2]. Patients with kidney disease present a wide range of comorbidities, which lends PoCUS a special role. It requires training and practice, but any physician can incorporate it into their daily practice [3]. PoCUS should be limited in its scope of use for specific clinical questions and the tool should enhance the safety of a procedure, i.e. does my patient have a urinary obstruction; is shortness of breath due to pulmonary congestion; what is the best dialysis vascular access for my patient with chronic heart failure (HF) and chronic kidney disease (CKD); and can I calculate an arteriovenous fistula (AVF) flux, guide the AVF puncture

or perform a renal biopsy based in PoCUS? Furthermore, how can I assess the right ventricle function, and can I exclude a significant pulmonary hypertension or pericardial effusion [4]?

Despite the obvious advantages of using PoCUS by the clinician, multiple concerns discourage its use, especially related to overlap with other specialties (Table 1).

In this review, we will present PoCUS as an easy, fast, dynamic and reliable tool for nephrologists to incorporate into their daily renal practice.

## CONGESTION, A COMMON QUESTION IN NEPHROLOGY

Renal homeostasis requires an optimal electrolyte balance and an adequate regulation of body volume (interstitial and vascular). The relevance of volume control and specially congestion has been highlighted in different clinical scenarios of acute and chronic renal dysfunction, cardiorenal syndrome (CRS) type 1 and acute kidney injury (AKI) among others. Congestion is a common condition in nephrology and is related to organ damage (Fig. 1). An example of systemic impairment of congestion is congestive nephropathy (CN) [5]. This new

**Table 1: Myths and fact about PoCUS.**

Myth	Fact
PoCUS replaces the scheduled examination by cardiologists and radiologists	No, it does not PoCUS requires direct interaction between the clinician and the patient to answer specific questions and improves the sensitivity of the physical examination in order to address that question. The examinations performed by cardiology or radiology are complementary [1, 4]
PoCUS replaces clinical view	No, it does not PoCUS enhances clinical vision by improving the sensitivity of the physical examination. In some cases, it may reduce further diagnostic explorations [83]
PoCUS improves clinical outcomes (mortality, re-admissions)	It depends The main objective of PoCUS is to enhance physical examination. In addition, there are many factors that make it difficult to include PoCUS as an independent variable [4]
PoCUS requires US scanners with multiple probes and sophisticated equipment	No, it does not Bedside scanning requires easily portable equipment. Low-cost handheld US systems are now available that can be connected to mobile phones or tablets. In addition, images can be shared remotely for academic or clinical purposes [1, 4]
PoCUS is used for diagnostic purposes only	No, this is not the case PoCUS has diagnostic and interventional uses. Specifically in nephrology PoCUS is used to guide AVF cannulation, venous catheter placement and renal biopsies [72-75]
Can PoCUS be done without training?	No, it should not Although US scanning is relatively easy, training programmes are required to include PoCUS during undergraduate and specialist training, in addition to defining the competencies needed to develop an academic programme in PoCUS [77-79]
Should I perform PoCUS if my patient has oedema?	Yes, you should Although oedema may be associated with tissue congestion, its sensitivity for diagnosing HF is poor and does not differentiate vascular congestion. Fundamental to individualize treatment [3, 7]

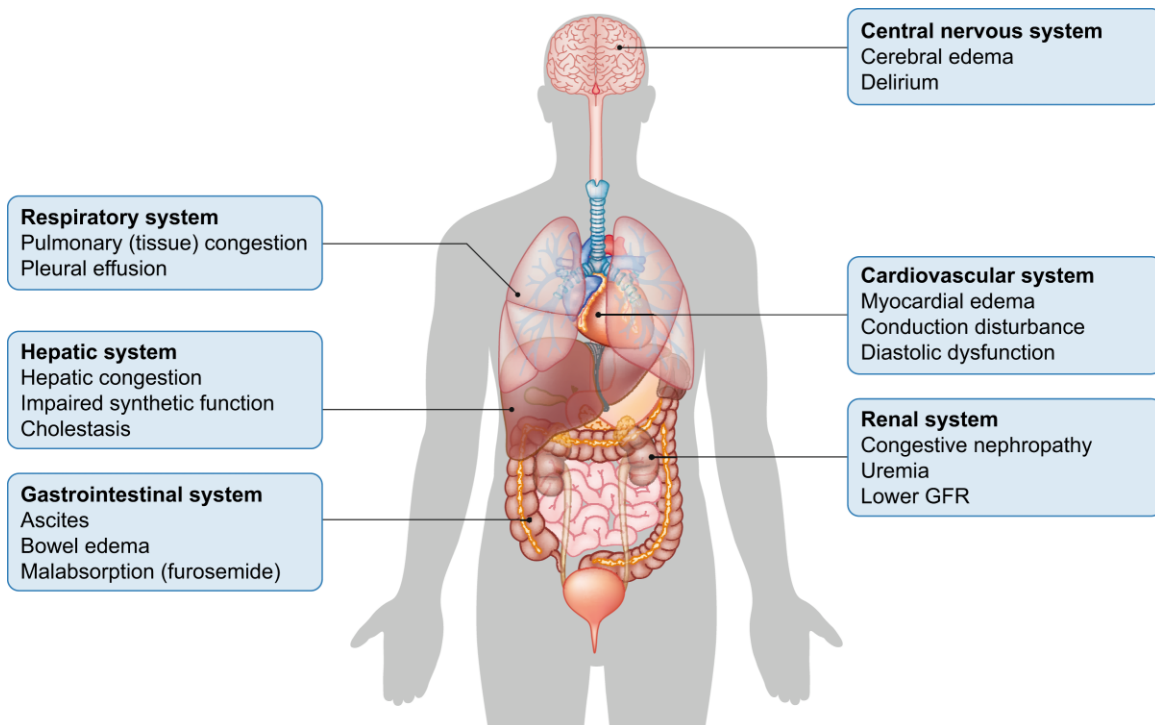


Figure 1: Systemic consequences of congestion. Abbreviations: GFR: Glomerular filtration rate. Adapted from [6].

entity has emerged as the main mechanism of kidney injury in patients with HF but probably extends to other acute and chronic settings.

Understanding the relevance of congestion in these clinical scenarios requires a conceptual evolution that has revolutionized the current understanding of acute and chronic nephrology [6].

## CONGESTION IS MORE THAN JUST OEDEMA

Beyond the acute setting, congestion in chronic HF starts in the intravascular compartment, leading to increased hydrostatic pressure that progressively leads to increased interstitial oedema and tissue congestion. However, presentation can be mixed (tissue and vascular congestion) [7].

In vascular congestion, pulmonary and cardiac filling pressures increase associated with the activation of the renin-angiotensin-aldosterone system (RAAS), the natriuretic peptide axis and the peripheral nervous system, leading to vasoconstriction and blood flow from the abdominal to the systemic circulation [8]. Increase in filling pressures in tissue congestion is progressive, leading to a rise in pulmonary, abdominal and peripheral fluid overload [9]. Global congestion can harm other organs such as the heart where it is associated with diastolic dysfunction and conduction disturbances [6], the brain where cognitive impairment and delirium have been described [10], the liver where hepatic congestion may impair synthesis, and/or bowel function where congestion has been associated with malabsorption disorders [6]. Congestion is common in the acute HF setting. The ESC-EURObservational Research Programme (EORP)-Heart Failure Association (HFA) Heart Failure Long-Term (HF-LT) registry showed that more than 80% of patients had a wet phenotype on admission and only 19.8% of congestive

patients had hypoperfusion [11]. In patients with congestion, impaired renal function was associated with increased right atrial (RAP) and central venous pressures (CVP) which correlated well with elevated serum creatinine [12–17], even better than cardiac output [17, 18]. Concerning CN, the effect of increased CVP transmitted through the low resistance renal vessels drives to renal dysfunction in encapsulated organs such as the kidneys (renal tamponade) [5, 19]. The increasing renal afterload and intrarenal pressure leads to a decrease in renal perfusion and intratubular flow, an increase in sodium and water retention mediated by activation of the RAAS, leading to tubular damage mediated by inflammatory mechanisms and decrease in the glomerular filtration rate [5]. As discussed below, CN defined as the presence of discontinuous intrarenal venous flow (IRVF) assessed by pulsed Doppler (PD) has been observed in patients with nonischaemic HF. These patients displayed a biphasic flow (26%) and a monophasic flow (23%), which was associated with elevated RAP and a worse prognosis (<40% survival at 1 year) [20]. Another interesting scenario is cardiac surgery, where a discontinuous IRVF was targeted in 34% of patients and the presence of a monophasic pattern was associated with an increased risk of developing AKI [21].

## TUBULOCENTRIC VIEW OF CONGESTION

AKI is a syndrome that comes as a consequence of multiple mechanisms which usually start with a tubular injury [22]. As mentioned, CN is associated with an intense inflammatory response and activation of neurohormonal mechanisms which are connected to alterations in tubular morphology and paired with congestion [5]. Pseudo-worse renal function and permissive AKI are two novel concepts that allow understanding that creatinine elevations during diuretic therapy are not associated

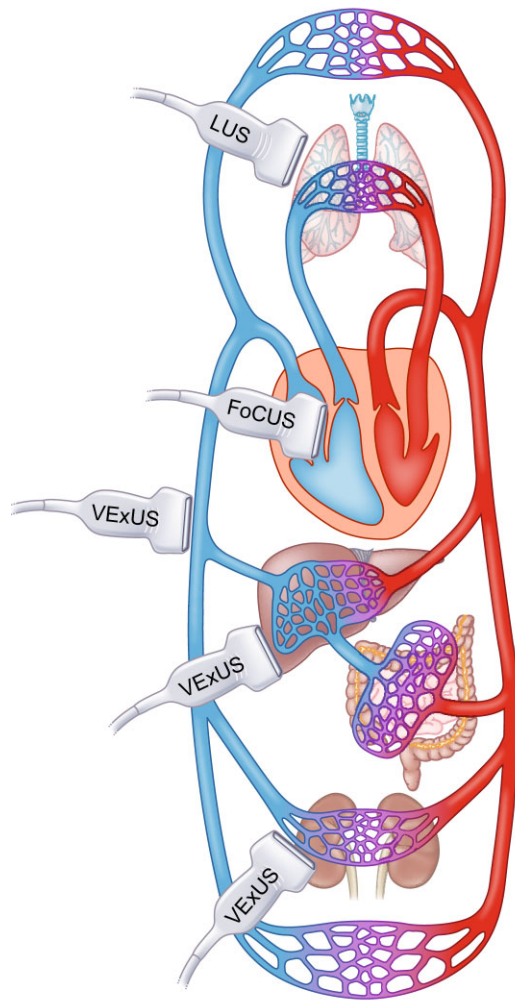


Figure 2: PoCUS strategies to assess congestion: LUS (Lung Ultrasound), VExUS (venous excess ultrasound grading system) and FoCUS (Focused Cardiac Ultrasound).

with structural damage and could be related only to functional disturbances [23–25]. Widening research into new biomarkers of tubular injury in addition to congestion assessment is essential to establish precise strategies for the prevention and progression of CN.

## PHENOTYPING CONGESTION

Physical examination is not sensitive and specific enough to differentiate between tissue and vascular congestion [7]. The multi-parametric view of congestion aims to be a holistic view that goes beyond physical examination and includes the use of US (Fig. 2) and biomarkers in order to improve the ability to correctly detect congestion, differentiate tissue and vascular congestion, establish personalized treatment strategies and predict worse cardiorenal outcomes [26]. Natriuretic peptides such as the B-type natriuretic peptide (BNP) and amino-terminal Pro-BNP (NT-ProBNP) are elevated due to increasing stretch and/or pressure of the left ventricle and act as markers of vascular congestion [27]. The higher degree of renal congestion assessed by PD of the interlobar renal vessels (elevated renal venous stasis index) has been associated with higher BNP levels [28]. Moreover, the carbohydrate antigen 125 (CA125) shows a

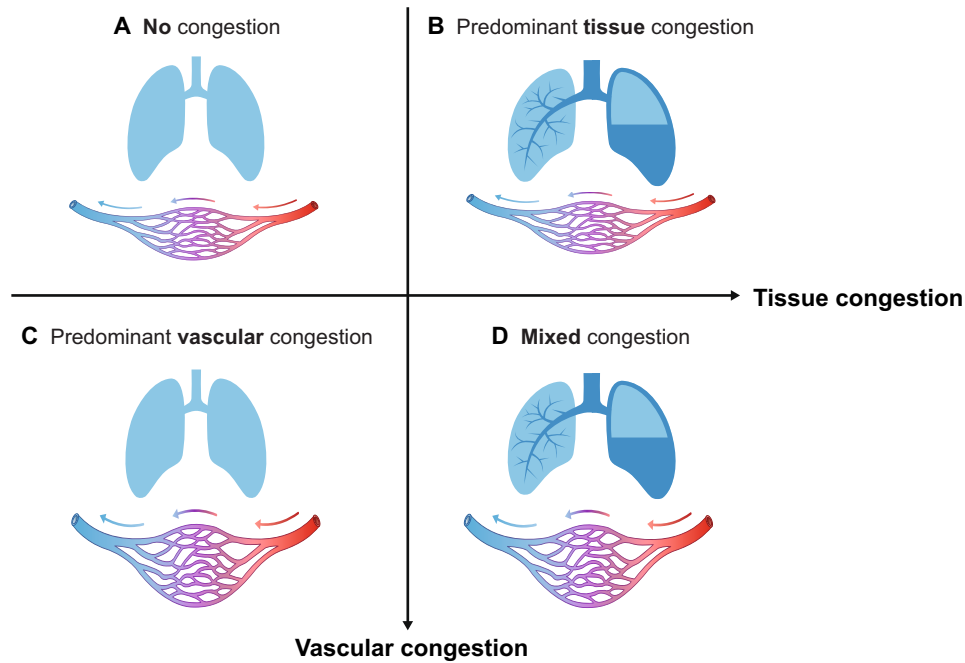
positive correlation with severity of renal vein congestion measured by PD [29] and has recently emerged as a surrogate marker of tissue congestion; it is described as an independent predictor of HF hospitalization and death [30]. Using PoCUS at least four phenotypes of congestion (Fig. 3) are generally established according to the presence or absence of tissue and vascular congestion that can be complemented with other diagnostic tools such as biomarkers of congestion or bioimpedance. This will certainly require further studies and evidence to improve profiling of phenotypes. Therefore, phenotyping congestion will allow to establishing personalized strategies with the aim to increase intravascular refilling in case of tissue congestion or to increase natriuresis in vascular congestion [7]. One of the most challenging phenotypes of congestion for the clinician is predominant tissue congestion, and fortunately new treatment options have opened up with the introduction of sodium-glucose cotransporter-2 inhibitors (SGLT2i). In addition to significantly reducing cardiac and renal events, the haemodynamic mechanism of SGLT2i mediated by its natriuretic effect is related to a significant decrease in tissue congestion and a minimal effect on vascular tone, unlike loop diuretics [31–33]. In predominantly tissue congestion, vascular refilling may be increased using hypertonic saline infusion [34] or the use of albumin infusion in those patients with hypoalbuminemia, although the latter remains controversial [35, 36]. Diuretics are the cornerstone of the treatment of tissue and vascular congestion with the aim of blocking the reabsorption of sodium and water, furosemide being the most widely used diuretic [37]. In those patients with diuretic resistance and especially those with low urine output or low urinary sodium excretion (<50–70 mmol/L) [38], strategies such as sequential nephron blockade and even ultrafiltration should be considered [39–41]. PoCUS allows not only accurate assessment of congestion but is also an additional tool to monitor treatment response.

## POCUS: AN ANSWER IN CONGESTION ASSESSMENT

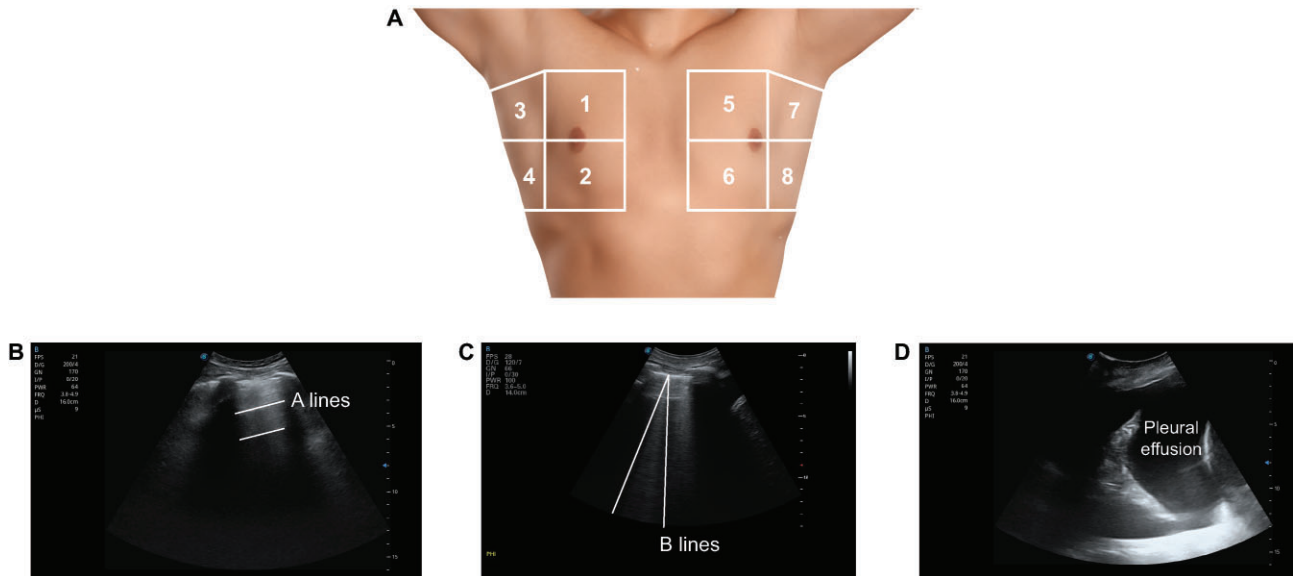
### Lung US, an answer to tissue congestion

Traditionally the lungs were believed to be organs that could not be insonated as they were mainly occupied by air. Although in fact, earlier versions of Harrison's Book of internal medicine precluded their use [42], it was not until 1994 that Daniel Lishstein described the BLUE protocol. The BLUE protocol allows bedside US diagnosis of thoracic disorders associated with acute respiratory failure [43]. Lung US (LUS) helps the interpretation of artifacts by US. The fluid content of the interstitial or extravascular lung water is an important indicator of total volume status and is strictly dependent on the filling pressure of the left ventricle [44]. LUS has been usually used to diagnose lung disorders like pneumonia, pulmonary oedema, haemo- or pneumothorax, and pleural effusion [43]. A variety of different approaches have been proposed for lung exploration that can be carried out with any type of probe including linear, convex or sectorial. A simple approach is to explore the thorax bilaterally in eight zones, anterior, lateral and posterior (Fig. 4A) [45]. In the normal status, horizontal hyperechoic equidistant lines parallel to pleura (A-lines) are seen (Fig. 4B). The basic principle of tissue congestion assessment is that in the presence of excessive lung water, the US beam is reflected by sub-pleural thickened interlobular septa, a low impedance structure surrounded by air with a high acoustic mismatch. US reflection generates hyperechoic reverberation artifacts between thickened septa and





**Figure 3:** Compartmental phenotypes of congestion by PoCUS: (A) No congestion, (B) Predominant tissue congestion, (C) Predominant vascular congestion and (D) Tissue and vascular congestion. Adapted from [7]



**Figure 4:** (A) 8-zone lung ultrasound probe positions. (B) A lines or normal lung. (C) B lines or comets tails. (D) Left pleural effusion.

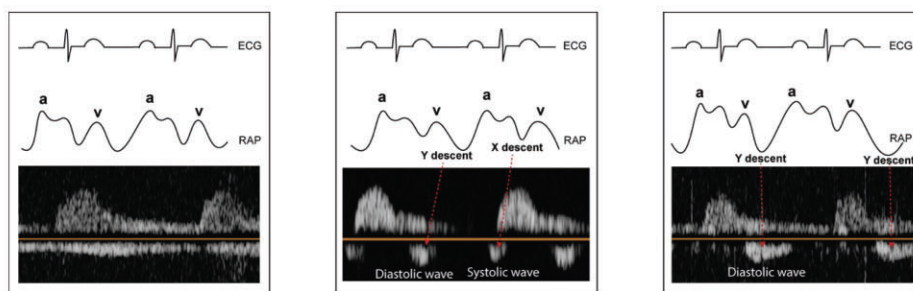
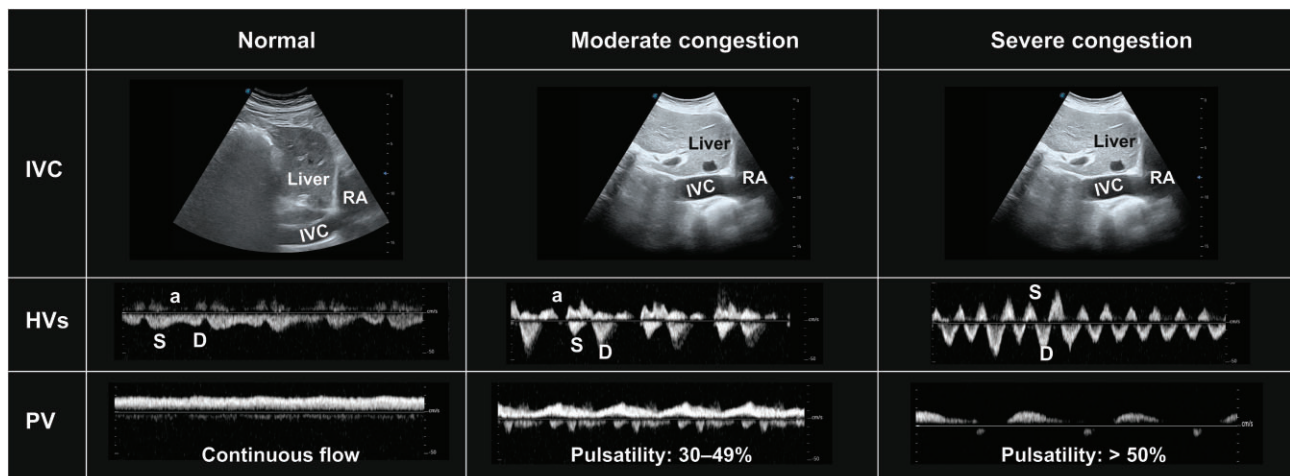
the overlying pleura, which are defined as B-lines or comet tails (Fig. 4C) [46]. It is generally considered that the appearance of three or more comets is relevant. Moreover, pleural effusion can be identified by black images and the absence of pleural sliding (Fig. 4D) [46].

In nephrology, LUS is used to assess congestion status, particularly pulmonary oedema, but it can also help to determine dry weight in dialysis patients [47]. A randomized multicenter trial on a lung ultrasound-guided treatment strategy in patients on chronic hemodialysis with high cardiovascular risk (LUST trial), established that guiding ultrafiltration using LUS is a safe strategy, and significantly decreased tissue congestion in

the intervention group without hypotension episodes. Although it failed to determine benefits in reducing the composite outcome (mortality, acute myocardial infarction or heart failure admissions), it demonstrated the ability of LUS to enhance the sensitivity of physical examination [48].

#### Venous congestion assessment: an answer in vascular congestion

Congestion assessment is not just inferior vena cava (IVC) US exploration. Identification and stratification of vascular congestion by scanning IVC and the use of PD to study flow patterns



**Figure 5:** (A) Vascular congestion patterns: Normal pattern in US congestion assessment. IVC  $< 2$ cm. HVs: normal  $S > D$  pattern. PV: Continuous flow or pulsatility  $< 30\%$  Mild-moderate congestion: IVC  $\geq 2$ cm. HVs:  $S < D$  pattern. VP: Pulsatility 30–49%. Severe congestion: IVC:  $\geq 2$ cm, HVs: Reverse S. PV: Pulsatility  $> 50\%$ . (B) Intrarenal Venous flow patterns in venous congestion: Pulsed Doppler normal and progressive congestion patterns visualised in the renal vein, in relation to the increase in RAP (with a reduced scale, 20–30 cm/sec).

of the portal vein (PV), hepatic veins (HV) and IRVF allows grading of venous congestion assessment (VExUS) [49]. The assessment of venous congestion could be performed with 3.5–5 MHz frequency probes; the transducer probe can be located in subxyphoid, subcostal and intercostal oblique planes [50]. This last plane allows the visualization of practically all venous territories and is useful when artifacts (intestine and stomach among others) prevent the correct visualization of the hepatic parenchyma in the subxyphoid and subcostal planes.

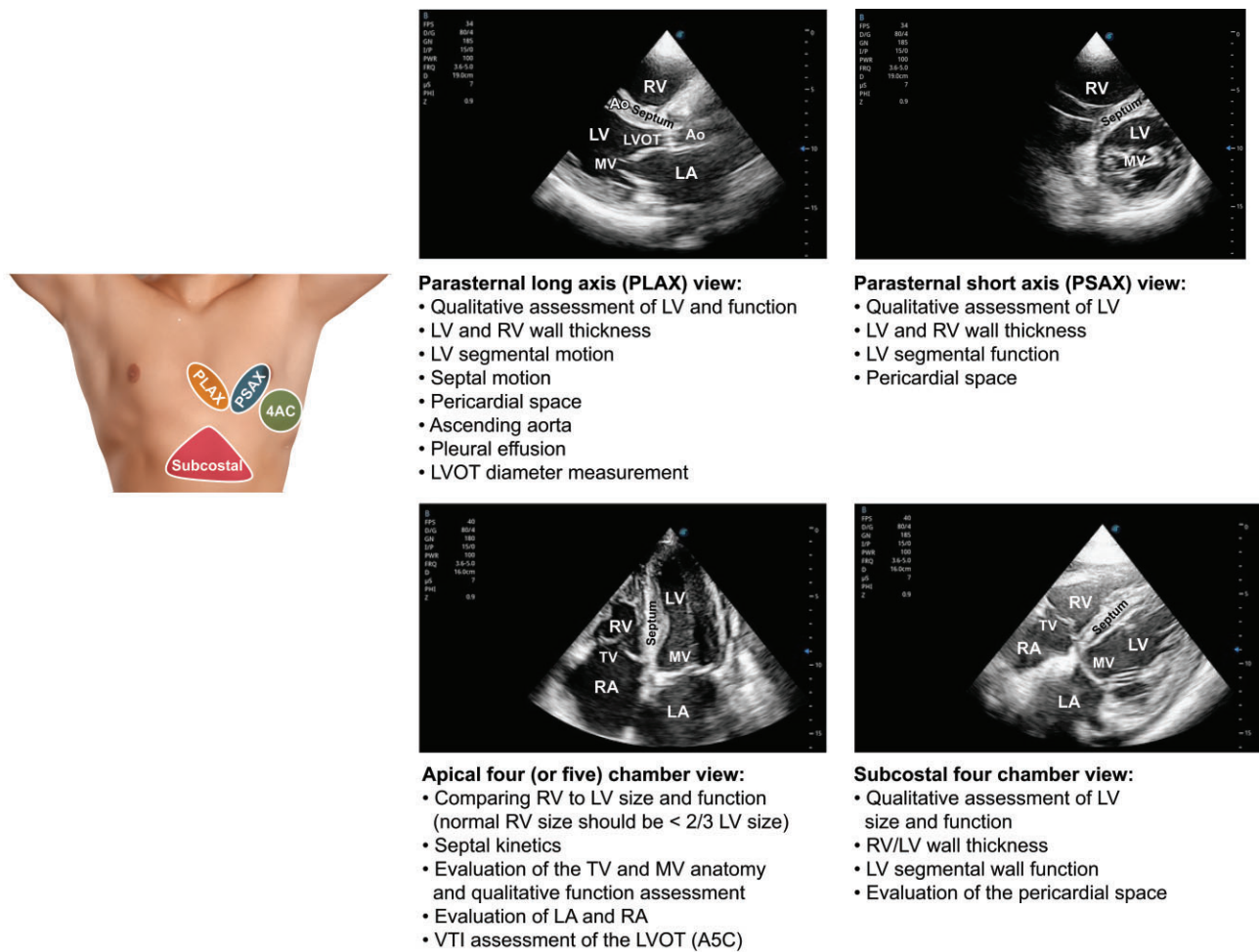
The examination begins with the visualization of the IVC in the intrahepatic portion, 2 cm from the junction with the hepatic veins. Venous congestion occurs when an IVC diameter  $\geq 2$  cm is present together with a collapsibility  $< 50\%$  while inspiring (non-invasive mechanical ventilation) [49, 50]. In the absence of these parameters, vascular congestion is ruled out. HVs drain into the IVC just before right atrium (RA) and therefore HVs have pulsatility that correlates with the cardiac cycle. Under normal conditions HVs show an aSD pattern by PD: 'a' wave corresponds to RA contraction, 'S' wave to the right ventricular (RV) contraction and 'D' wave to RV diastole. The negative S wave becomes less deep than 'D' in mild congestion ( $S < D$ ) and turns positive in severe congestion (reverse S) [49, 50]. Under normal conditions PV has no or minimal pulsatility and there is a continuous flow or minimal pulsatility ( $< 30\%$ ), increasing 30–50% in mild congestion and showing a  $\geq 50\%$  pulsatility in severe congestion (Fig. 5A) [49, 50].

Interlobar or arcuate vessels are preferably explored in the cortical region between the pyramids for the IRVF exploration. At this level, the artery and vein run together showing a pulsatile arterial wave and continuous venous flow. In the third step of

VExUS, as congestion increases, pulsatility in IRVF is observed. In mild congestion, a pulsatile flow will be detected showing a diastolic (D wave) phase deeper than the systolic (S wave). In severe congestion, the discontinuity of renal vein flow increases, showing only a D wave (monophasic flow) (Fig. 5B) [49, 50]. These findings of venous congestion are associated with elevated RAP and correspond to this new entity called CN [5, 50]. Assessing venous congestion by US has some limitations due to severe tricuspid regurgitation, pulmonary hypertension or chronic RV dysfunction, and congestion needs to be confirmed by the visualization of other venous territories (PV and RV). In addition, certain pathologies such as cirrhosis and CKD may hinder the correct interpretation of the waves [51, 52].

### Focused Cardiac Ultrasound: the heart at the middle of the congestion

Focused Cardiac Ultrasound (FoCUS) or echocardiography performed by the non-cardiologist clinician is an increasingly widespread strategy in nephrology [53]. The use of echocardiography increases the sensitivity and specificity of auscultation and allows cardiac pathology to be ruled out [54]. Echocardiographic examination is performed with a sectorial probe with frequencies ranging from 2 to 7 MHz. The standard examination is performed by placing the transducer in four basic planes: parasternal long axis (PLAX), parasternal short axis (PSAX), apical 4-chamber (A4C) and subcostal (Fig. 6). Echocardiographic assessment of fluid status includes rapid identification of morphological and functional changes in the basic image planes [55]. Rapid identification can be made systematically by starting with



**Figure 6:** Focused Cardiac Ultrasound (FoCUS): Classical planes and targets to be visualized by FoCUS. Abbreviations: LV: left ventricle, RV: Right ventricle, LVOT: Left ventricular outflow tract, MV: Mitral valve, TV: Tricuspid valve, LA: Left atrium, RA: Right atrium, VTI: Velocity time integral.

the examination of left ventricular (LV) systolic function using the visualization of the anterior leaflet of the mitral valve, allowing observation of the E-point septal separation. It is performed by measuring the distance between the anterior leaflet of the mitral valve and the septum during the early LV diastole [56]. A distance of >7 mm correlates well with a severely reduced ejection fraction [57]. Advanced users can calculate LV ejection fraction by echocardiography using M-mode and Teichholz's formula or Simpson's method [58]. The right heart has a major role in vascular congestion and although the RV is better adapted to increased volume, increased preload in the RV is associated with dilation and in some cases increased afterload which can chronically lead to RV dilation and dysfunction [59]. In the A4C plane the size of the RV can be compared with the LV size and, at the end-diastole, the basal diameter of the RV can be measured [60]. In healthy individuals, the size of the RV is approximately less than two-thirds the size of the LV and it may be present in patients with congestion RV dilatation (basal diameter >41 mm) [60]. When pressure increases secondary to congestion, a rectification of the interventricular septum gives the RV a D-shape, compared with a C-shape in the PSAX plane at the papillary muscles [59]. In addition, the RV function can be measured in M-mode by calculating the tricuspid valve systolic excursion (values below 17 mm suggest RV systolic dysfunction) [60].

Although the subcostal window is the most appropriate window to assess pericardial effusion, large effusions can also be

seen in the posterior region of the LV in PLAX and PSAX planes [53]. To rule out cardiac tamponade, one of the easiest and most sensitive signs is the distention of the IVC due to altered filling pressures (right atrial collapse in systole and RV collapse in diastole) secondary to extrinsic compression of the heart easily assessed by FoCUS [53, 55, 61].

Echocopy also allows calculation of the stroke volume (SV). The blood flow through the aortic valve or SV is calculated by measuring the velocity time integral (VTI) and LV outflow tract diameter [61]. SV is determined using the following formula  $VS = TSVI \text{ area} \times ITV$ , and if the result is multiplied by the heart rate, the cardiac output is obtained. Current US scanners devices have the ability to accurately calculate SV automatically, which requires correct measurements for the value to be reliable [61].

## IMPROVING OUR CLINICAL AND RESEARCH SKILLS WITH POCUS

The main contribution of PoCUS is to enhance physical examination by adding a fifth pillar (insonation) to the classical model (inspection, palpation, auscultation and percussion) [62], aiming to improve the sensitivity of physical examination. In a study of patients with chronic HF, the combination of classic signs such as rales, oedema and elevated jugular venous pressure showed a low sensitivity (58%) in detecting elevated pulmonary capillary



wedge pressures [63]. In addition, a recent meta-analysis showed that the sensitivity of chest radiography to correctly detect pulmonary oedema was 73%, but sensitivity increased up to 88% when LUS was included [64]. In the research agenda, the integration of a multi-parametric or holistic view of congestion [26] will allow the nephrologist to include the use of PoCUS to phenotype congestion in different acute and chronic settings (AKI, onconeurology, kidney transplantation, dialysis and glomerulonephritis, among others), and not only in CRS. Allowing the PoCUS findings to be correlated with clinical scores such as the composite congestion score [65], novel congestion biomarkers such as CA125 [29], bioelectrical impedance [66] and/or quantitative measures obtained by advanced users through FoCUS. In addition, PoCUS highlights the relevant role of subclinical congestion, i.e. those patients who have some degree of congestion assessed by PoCUS but are asymptomatic and associated with poor outcomes [67].

## MORE PoCUS RESOURCES

### Could my patient's AKI be due to lithiasis and hydronephrosis?

Renal colic is a common presentation in nephrology which involves a significant amount of resource use, including advanced imaging as well as direct and indirect costs related to patient care and lost work time. Hydronephrosis is presented as a black branching structure in the renal sinus with US but we can also find the cause of the obstruction as a stone in the renal pelvis as a high brightness structure with an acoustic shadow [68]. Even if we do not visualize the stone during the examination, the presence of hydronephrosis suggests ureteral obstruction. Recently, a prospective observational study was performed to determine the diagnostic accuracy of PoCUS in patients with renal colic and suspected hydronephrosis [69]. The authors showed a moderate sensitivity and specificity for renal colic and suggested that PoCUS might be helpful to help guide further imaging and consultation in combination with clinical course [69].

Finally, a PoCUS examination is not finished until the bladder is examined. Urine appears as an anechoic fluid and a low urinary obstruction can be discovered by PoCUS [70].

### Is it safe to create an AVF in my HF patient or does my patient with acute recurrent HF have a high-flow AVF?

AVF establishes a shunt from the arterial to the venous circulation and exposes low pressure, high capacitance venous system to the high pressure but low capacitance arterial system [71]. Immediately following its creation, there is an increase in cardiac output and a decrease in subendocardial perfusion as a consequence of reduced systemic vascular resistance, increased myocardial contractility and an increase in SV and heart rate [72, 73]. In addition, creating an AVF predisposes to increased preloading of the right chambers which in turn can lead to RV remodelling and dysfunction [74]. Usually, a permanent AVF is a haemodynamically significant left-to-right-sided shunt with a blood flow of 1–2 L/min, and further increases in blood flow can worsen cardiac function shown as a left ventricular mass, ventricular and atrial dilatation, increased pulmonary flows and increased cardiac output [75, 76].

PoCUS allows adequate assessment of RV morphology and function allowing patients with RV dysfunction who are not candidates for peritoneal dialysis to avoid the creation of proximal AVFs and to decide the best vascular access [53, 60]. Furthermore,

assessing the AVF flux by PoCUS helps to determine whether blood flow AVF exceeds the cardiac ability to maintain a functional New York Heart Association pattern and can select those patients as candidates for banding or even more, ligation of the AVF. Regular flow follows up better than a single measure could be monitored regularly in order to identify high-flux AVF [77].

### Can I start using AVF safely?

Assessing new AVF maturation can be performed using the '6 s rule'. AVF cannulation is initiated when the vein meets a diameter greater than 6 mm diameter with no depreciable narrowing of more than 20% throughout, a less than 6 mm depth and a more than 6 cm length [78]. Either way, PoCUS can be used for the assessment of alternate cannulation sites and new AVF cannulation, overwhelm difficult access cannulation and increase cannulation accuracy.

The regular use of PoCUS by haemodialysis nursing staff is essential to providing high-quality care in haemodialysis patients. It helps to ensure that vascular access is maintained in the long term while reducing discomfort and complications for patients. To avoid blind cannulation, some clinicians and nurses have turned to US to visualize vessels, particularly vessels that are new, small, mobile or tortuous [79]. The benefits of this approach for guiding needle insertion improves care, detects early complications and avoids useless punctions with the identification AVF landmarks and abnormalities, using PoCUS to view the vessel and allowing for the creation of a visual map of the AVF [78].

### Can PoCUS help performing a renal biopsy as a safe procedure?

Although renal biopsy is a training procedure in nephrology fellowship, the trend is moving to a radiology practice. Indeed, radiologists have usually performed US-guided biopsies in recent years. Nevertheless, PoCUS can help to recover the interventional procedures for nephrology practice. A well-trained nephrology team can safely carry out native and graft kidney biopsies, and with the help of US renal biopsy should no longer be an obstacle for nephrologist skills. In a recent study, Palacherla et al. showed excellent outcomes from percutaneous renal biopsies both guided and performed by nephrologists. Furthermore, they described a pre-localization of kidneys before rather than in real-time guidance during procedures [80]. They advocate for the increasing availability of PoCUS, encouraging more nephrologists to perform this essential procedure [80, 81].

## POCUS IN NEPHROLOGY TRAINING

PoCUS is being introduced into medical school curricula in the USA and Canada and it is also being offered by some nephrology training programmes [82]. Although there are not yet any competency guidelines provided by the European or American Board of Nephrology for Internal Medicine, some institutions have curricula designed to teach focused diagnostic PoCUS skills to nephrology fellows [83, 84]. From Spain, we advocate for PoCUS becoming part of the nephrology fellowship core curricula in Europe, considering that there is an unmet need in nephrology for intervention tools. The Working Group for Cardiorenal Medicine of the Spanish Society of Nephrology suggests that current general nephrology training does not seem to be sufficient to cover the rapidly evolving field of cardiorenal medicine [85]. The proposition includes an innovative programme to



enhance education of nephrologists, including tools such as PoCUS in order to well define, describe and characterize the CRS with FoCUS, VExUS and LUS [82].

Clinical nephrology practice and education needs to modernize and not lose the lead on volume status assessment, congestion and cardiorenal balance, but not only in the CRS context. As an example, NephroPOCUS.com won the 2020 American Society of Nephrology Innovation in Kidney Education award. These improvements in knowledge address two of the main barriers for PoCUS implementation: understanding the principles of the tool and knowing how to use PoCUS appropriately. The goal must be to build an international PoCUS nephrology training programme as part of regular nephrology training in collaboration with US educators. The basis of this competency-based training for nephrological PoCUS should arise from the collaboration of nephrology PoCUS training institutions, academic centres and diagnostic imaging centres becoming part of the nephrology fellowship. As PoCUS becomes more prevalent in the field of nephrology, it will likely become a crucial component of the tool kit of the modern nephrologist.

PoCUS involves multiple actions such as appropriate focused questions, identifying adequate acoustic windows and obtaining images, interpreting the images in the correct clinical setting, integrating the information appropriately to guide patient management, recognizing the limitations of our own research and seeking expert consultation or perform complementary studies when necessary. PoCUS enables the nephrologist to move from the simplistic view of physical examination learned in the 19th century to a holistic view that includes US as a precision medicine tool for the 21st century.

## DATA AVAILABILITY STATEMENT

No new data were generated or analysed in support of this research.

## CONFLICT OF INTEREST STATEMENT

Gregorio Romero-González received fees from AstraZeneca for lectures and PoCUS courses. Alberto Ortiz is the former Editor-in-Chief of CKJ.

## REFERENCES

- Díaz-Gómez JL, Mayo PH, Koenig SJ. Point-of-care ultrasonography. *N Engl J Med* 2021;385:1593–602. <https://doi.org/10.1056/NEJMRA1916062>
- Romero-González G, Manrique J, Castaño-Bilbao I et al. Congestión y ultrasonido dos retos para la nefrología de la próxima década. *Nefrología* 2022;42:501–5. <https://doi.org/10.1016/j.nefro.2021.09.013>
- Koratala A, Kazory A. Point of care ultrasonography for objective assessment of heart failure: integration of cardiac, vascular, and extravascular determinants of volume status. *Cardiorenal Med* 2021;11:5–17. <https://doi.org/10.1159/000510732>
- Koratala A, Reisinger N. PoCUS for nephrologists: basic principles and a general approach. *Kidney360* 2021;2:1660–8. <https://doi.org/10.34067/KID.0002482021>
- Husain-Syed F, Gröne HJ, Assmus B et al. Congestive nephropathy: a neglected entity? Proposal for diagnostic criteria and future perspectives. *ESC Heart Fail* 2021;8:183–203. <https://doi.org/10.1002/EHF2.13118>
- Rosenkranz S, Howard LS, Gomberg-Maitland M et al. Systemic consequences of pulmonary hypertension and right-sided heart failure. *Circulation* 2020;141:678–93. <https://doi.org/10.1161/CIRCULATIONAHA.116.022362>
- Boorsma EM, ter Maaten JM, Damman K et al. Congestion in heart failure: a contemporary look at physiology, diagnosis and treatment. *Nat Rev Cardiol* 2020;17:641–55. <https://doi.org/10.1038/s41569-020-0379-7>
- Viau DM, Sala-Mercado JA, Spranger MD et al. The pathophysiology of hypertensive acute heart failure. *Heart* 2015;101:1861–7. <https://doi.org/10.1136/HEARTJNL-2015-307461>
- Gheorghiadu M, Filippatos G, de Luca L et al. Congestion in acute heart failure syndromes: an essential target of evaluation and treatment. *Am J Med* 2006;119 (Suppl):S3–10. <https://doi.org/10.1016/j.amjmed.2006.09.011>
- Malbrain MLNG, Marik PE, Witters I et al. Fluid overload, de-resuscitation, and outcomes in critically ill or injured patients: a systematic review with suggestions for clinical practice. *Anestezjologia Intensywna Terapia* 2019;46:361–80. [www.ait.viamedica.pl](http://www.ait.viamedica.pl) (16 October 2021, date last accessed)
- Chioncel O, Mebazaa A, Maggioni AP et al. Acute heart failure congestion and perfusion status – impact of the clinical classification on in-hospital and long-term outcomes; insights from the ESC-EORP-HFA heart failure long-term registry. *Eur J Heart Fail* 2019;21:1338–52. <https://doi.org/10.1002/EJHF.1492>
- Nohria A, Hasselblad V, Stebbins A et al. Cardiorenal interactions: insights from the ESCAPE trial. *J Am Coll Cardiol* 2008;51:1268–74. <https://doi.org/10.1016/j.jacc.2007.08.072>
- Mullens W, Abrahams Z, Francis GS et al. Importance of venous congestion for worsening of renal function in advanced decompensated heart failure. *J Am Coll Cardiol* 2009;53:589–96. <https://doi.org/10.1016/j.jacc.2008.05.068>
- Damman K, van Deursen VM, Navis G et al. Increased central venous pressure is associated with impaired renal function and mortality in a broad spectrum of patients with cardiovascular disease. *J Am Coll Cardiol* 2009;53:582–8. <https://doi.org/10.1016/j.jacc.2008.08.080>
- Damman K, Voors AA, Hillege HL et al. Congestion in chronic systolic heart failure is related to renal dysfunction and increased mortality. *Eur J Heart Fail* 2010;12:974–82. <https://doi.org/10.1093/EURJHF/HFQ118>
- Testani JM, Khera AV, St John Sutton MG et al. Effect of right ventricular function and venous congestion on cardiorenal interactions during the treatment of decompensated heart failure. *Am J Cardiol* 2010;105:511–6. <https://doi.org/10.1016/j.amjcard.2009.10.020>
- Guglin M, Rivero A, Matar F et al. Renal dysfunction in heart failure is due to congestion but not low output. *Clin Cardiol* 2011;34:113–6. <https://doi.org/10.1002/CLC.20831>
- Hanberg JS, Sury K, Perry Wilson F et al. Reduced cardiac index is not the dominant driver of renal dysfunction in heart failure. *J Am Coll Cardiol* 2016;67:2199–208. <https://doi.org/10.1016/j.jacc.2016.02.058>
- Eva M, Boorsma M, Jozine M et al. Renal compression in heart failure: the renal tamponade hypothesis. *JACC Heart Fail* 2022;10:175–83. <https://doi.org/10.1016/j.jchf.2021.12.005>
- Iida N, Seo Y, Sai S et al. Clinical implications of intrarenal hemodynamic evaluation by doppler ultrasonography in heart failure. *JACC Heart Fail* 2016;4:674–82. <https://doi.org/10.1016/j.jchf.2016.03.016>
- Beaubien-Souligny W, Benkreira A, Robillard P et al. Alterations in portal vein flow and intrarenal venous flow are associated with acute kidney injury after cardiac surgery: a prospective observational cohort study. *J Am Heart*

- Assoc 2018;7:e009961. <https://doi.org/10.1161/JAHA.118.009961>
22. Ronco C, Bellomo R, Kellum JA. Acute kidney injury. *Lancet North Am Ed* 2019;394:1949–64. [https://doi.org/10.1016/S0140-6736\(19\)32563-2](https://doi.org/10.1016/S0140-6736(19)32563-2)
  23. Mullens W, Damman K, Testani JM et al. Evaluation of kidney function throughout the heart failure trajectory – a position statement from the Heart Failure Association of the European Society of Cardiology. *Eur J Heart Fail* 2020;22:584–603. <https://doi.org/10.1002/EJHF.1697>
  24. Parikh CR, Coca SG. “Permissive AKI” with treatment of heart failure. *Kidney Int* 2019;96:1066–8. <https://doi.org/10.1016/j.kint.2019.07.003>
  25. Ahmad T, Jackson K, Rao VS et al. Worsening renal function in patients with acute heart failure undergoing aggressive diuresis is not associated with tubular injury. *Circulation* 2018;137:2016–28. <https://doi.org/10.1161/CIRCULATIONAHA.117.030112>
  26. de la Espriella R, Santas E, Zegri Reiriz I et al. Cuantificación y tratamiento de la congestión en insuficiencia cardíaca: una visión clínica y fisiopatológica. *Nefrología* 42:145–62. <https://doi.org/10.1016/j.nefro.2021.04.006>
  27. Levin ER, Gardner DG, Samson WK. Natriuretic peptides. *N Engl J Med* 2009;339:321–8. <https://doi.org/10.1056/NEJM199807303390507>
  28. Husain-Syed F, Birk H, Ronco C et al. Doppler-derived renal venous stasis index in the prognosis of right heart failure. *J Am Heart Assoc* 2019;8:e013584. <https://doi.org/10.1161/JAHA.119.013584>
  29. Núñez-Marín G, de la Espriella R, Santas E et al. CA125 but not NT-proBNP predicts the presence of a congestive intrarenal venous flow in patients with acute heart failure. *Eur Heart J Acute Cardiovasc Care* 2021;10:475–83. <https://doi.org/10.1093/EHJACC/ZUAB022>
  30. Núñez J, Bayés-Genís A, Revuelta-López E et al. Clinical role of CA125 in worsening heart failure: a BIostat-CHF study subanalysis. *JACC Heart Fail* 2020;8:386–97. <https://doi.org/10.1016/j.jchf.2019.12.005>
  31. Verma S, McMurray JJV. The serendipitous story of SGLT2 inhibitors in heart failure: new insights from DECLARE-TIMI 58. *Circulation* 2019;139:2537–41. <https://doi.org/10.1161/CIRCULATIONAHA.119.040514>
  32. Verma S, McMurray JJV. SGLT2 inhibitors and mechanisms of cardiovascular benefit: a state-of-the-art review. *Diabetologia* 2018;61:2108–17. <https://doi.org/10.1007/S00125-018-4670-7/TABLES/1>
  33. de la Espriella R, Miñana G, Santas E et al. Effects of empagliflozin on CA125 trajectory in patients with chronic congestive heart failure. *Int J Cardiol* 2021;339:102–5. <https://doi.org/10.1016/j.ijcard.2021.06.045>
  34. Griffin M, Soufer A, Goljo E et al. Real world use of hypertonic saline in refractory acute decompensated heart failure: a U.S. center’s experience. *JACC Heart Fail* 2020;8:199–208. <https://doi.org/10.1016/j.jchf.2019.10.012>
  35. Phakdeekitcharoen B, Boonyawat K. The added-up albumin enhances the diuretic effect of furosemide in patients with hypoalbuminemic chronic kidney disease: a randomized controlled study. *BMC Nephrol* 2012;13:1–9. <https://doi.org/10.1186/1471-2369-13-92/FIGURES/3>
  36. Lee TH, Kuo G, Chang CH et al. Diuretic effect of co-administration of furosemide and albumin in comparison to furosemide therapy alone: an updated systematic review and meta-analysis. *PLoS One* 2021;16:e0260312. <https://doi.org/10.1371/JOURNAL.PONE.0260312>
  37. Mullens W, Damman K, Harjola VP et al. The use of diuretics in heart failure with congestion—a position statement from the Heart Failure Association of the European Society of Cardiology. *Eur J Heart Fail* 2019;21:137–55. <https://doi.org/10.1002/EJHF.1369>
  38. Tersalvi G, Dauw J, Gasperetti A et al. The value of urinary sodium assessment in acute heart failure. *Eur Heart J Acute Cardiovasc Care* 2021;10:216–23. <https://doi.org/10.1093/EHJACC/ZUAA006>
  39. Joannidis M, Klein SJ, Ostermann M. 10 myths about frusemide. *Intensive Care Med* 2019;45:545–8. <https://doi.org/10.1007/S00134-018-5502-4/FIGURES/1>
  40. Gupta R, Testani J, Collins S. Diuretic resistance in heart failure. *Curr Heart Fail Rep* 2019 16:2. 2019;16:57–66. <https://doi.org/10.1007/S11897-019-0424-1>
  41. Wilcox CS, Testani JM, Pitt B. Pathophysiology of diuretic resistance and its implications for the management of chronic heart failure. *Hypertension* 2020;76:1045–54. <https://doi.org/10.1161/HYPERTENSIONAHA.120.15205>
  42. Kasper D, Fauci A, Hauser S et al. *Harrison’s Principles of Internal Medicine*, 19e. Vol. 1. New York, NY:Mcgraw-Hill, 2015.
  43. Lichtenstein DA, Mezière GA. Relevance of lung ultrasound in the diagnosis of acute respiratory failure: the BLUE protocol. *Chest* 2008;134:117–25. <https://doi.org/10.1378/chest.07-2800>
  44. Hubert A, Girerd N, le Breton H et al. Diagnostic accuracy of lung ultrasound for identification of elevated left ventricular filling pressure. *Int J Cardiol* 2019;281:62–8. <https://doi.org/10.1016/j.ijcard.2019.01.055>
  45. Reisinger N, Lohani S, Hagemeyer J et al. Lung ultrasound to diagnose pulmonary congestion among patients on hemodialysis: comparison of full versus abbreviated scanning protocols. *Am J Kidney Dis* 2022;79:193–201.e1. <https://doi.org/10.1053/J.AJKD.2021.04.007>
  46. Reisinger N, Koratala A. Quantitative lung ultrasonography for the nephrologist: applications in dialysis and heart failure. *Kidney360* 2021;3:176–84. <https://doi.org/10.34067/KID.0003972021>
  47. Marini C, Fragasso G, Italia L et al. Lung ultrasound-guided therapy reduces acute decompensation events in chronic heart failure. *Heart* 2020;106:1934–9. <https://doi.org/10.1136/HEARTJNL-2019-316429>
  48. Zoccali C, Torino C, Mallamaci F et al. A randomized multicenter trial on a lung ultrasound-guided treatment strategy in patients on chronic hemodialysis with high cardiovascular risk. *Kidney Int* 2021;100:1325–33. <https://doi.org/10.1016/j.kint.2021.07.024>
  49. Beaubien-Souligny W, Rola P, Haycock K et al. Quantifying systemic congestion with point-of-care ultrasound: development of the venous excess ultrasound grading system. *Ultrasound J* 2020;12:16. <https://doi.org/10.1186/S13089-020-00163-W>
  50. Argaiz ER. VExUS Nexus: bedside assessment of venous congestion. *Adv Chronic Kidney Dis* 2021;28:252–61. <https://doi.org/10.1053/J.ACKD.2021.03.004>
  51. Koratala A, Reisinger N. Venous excess Doppler ultrasound for the nephrologist: pearls and pitfalls. *Kidney Med* 2022;4:100482. <https://doi.org/10.1016/j.xkme.2022.100482>
  52. Rola P, Miralles-Aguar F, Argaiz E et al. Clinical applications of the venous excess ultrasound (VExUS) score: conceptual review and case series. *Ultrasound J* 2021;13:1–10. <https://doi.org/10.1186/S13089-021-00232-8>
  53. Goyal P, Minardi J, Sakhujia A. Cardiac ultrasound for the nephrologist: know thy heart to know thy kidneys. *Adv*

- Chronic Kidney Dis 2021;28:208–17. <https://doi.org/10.1053/J.ACKD.2021.04.001>
54. Marbach JA, Almufleh A, di Santo P et al. Comparative accuracy of focused cardiac ultrasonography and clinical examination for left ventricular dysfunction and valvular heart disease. *Ann Intern Med* 2019;171:264–72. <https://doi.org/10.7326/M19-1337>
  55. Hall MK, Coffey EC, Herbst M et al. The “5Es” of emergency physician-performed focused cardiac ultrasound: a protocol for rapid identification of effusion, ejection, equality, exit, and entrance. *Acad Emerg Med* 2015;22:583–93. <https://doi.org/10.1111/ACEM.12652>
  56. Silverstein JR, Laffely NH, Rifkin RD. Quantitative estimation of left ventricular ejection fraction from mitral valve E-point to septal separation and comparison to magnetic resonance imaging. *Am J Cardiol* 2006;97:137–40. <https://doi.org/10.1016/J.AMJCARD.2005.07.118>
  57. McKaigney CJ, Krantz MJ, la Rocque CL et al. E-point septal separation: a bedside tool for emergency physician assessment of left ventricular ejection fraction. *Am J Emerg Med* 2014;32:493–7. <https://doi.org/10.1016/J.AJEM.2014.01.045>
  58. Lang RM, Badano LP, Victor MA et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr* 2015;28:1–39.e14. e14. <https://doi.org/10.1016/J.ECHO.2014.10.003>
  59. Sanz J, Sánchez-Quintana D, Bossone E et al. Anatomy, function, and dysfunction of the right ventricle: JACC State-of-the-Art review. *J Am Coll Cardiol* 2019;73:1463–82. <https://doi.org/10.1016/J.JACC.2018.12.076>
  60. Rudski LG, Lai WW, Afilalo J et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography: endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. *J Am Soc Echocardiogr* 2010;23:685–713. <https://doi.org/10.1016/J.ECHO.2010.05.010>
  61. Veitla V, Bhasin B. Focused cardiac assessment in kidney care. *POCUS J* 2022;7:45–50. <https://doi.org/10.24908/POCUS.V7IKIDNEY.14996>
  62. Narula J, Chandrashekhar Y, Braunwald E. Time to add a fifth pillar to bedside physical examination: inspection, palpation, percussion, auscultation, and insonation. *JAMA Cardiol* 2018;3:346–50. <https://doi.org/10.1001/JAMACARDIOL.2018.0001>
  63. Stevenson LW, Perloff JK. The limited reliability of physical signs for estimating hemodynamics in chronic heart failure. *JAMA* 1989;261:884–8. <https://doi.org/10.1001/JAMA.1989.03420060100040>
  64. Maw AM, Hassanin A, Ho PM et al. Diagnostic accuracy of Point-of-Care lung ultrasonography and chest radiography in adults with symptoms suggestive of acute decompensated heart failure: a systematic review and meta-analysis. *JAMA Network Open* 2019;2:e190703. <https://doi.org/10.1001/JAMANETWORKOPEN.2019.0703>
  65. Ambrosy AP, Pang PS, Khan S et al. Clinical course and predictive value of congestion during hospitalization in patients admitted for worsening signs and symptoms of heart failure with reduced ejection fraction: findings from the EVEREST trial. *Eur Heart J* 2013;34:835–43. <https://doi.org/10.1093/EURHEARTJ/EHS444>
  66. Siriopol D, Hogas S, Voroneanu L et al. Predicting mortality in haemodialysis patients: a comparison between lung ultrasonography, bioimpedance data and echocardiography parameters. *Nephrol Dial Transplant* 2013;28:2851–9. <https://doi.org/10.1093/NDT/GFT260>
  67. Rivas-Lasarte M, Maestro A, Fernández-Martínez J et al. Prevalence and prognostic impact of subclinical pulmonary congestion at discharge in patients with acute heart failure. *ESC Heart Fail* 2020;7:2621–8. <https://doi.org/10.1002/EHF.12842>
  68. Moses AA, Fernandez HE. Ultrasonography in acute kidney injury. *POCUS J* 2022;7:35–44. <https://doi.org/10.24908/POCUS.V7IKIDNEY.14989>
  69. Sibley S, Roth N, Scott C et al. Point-of-care ultrasound for the detection of hydronephrosis in emergency department patients with suspected renal colic. *Ultrasound J* 2020;12:1–9. <https://doi.org/10.1186/S13089-020-00178-3/FIGURES/4>
  70. Taus PJ, Manivannan S, Dancel R. Bedside assessment of the kidneys and bladder using point of care ultrasound. *POCUS J* 2022;7 (Kidney):94–104. <https://doi.org/10.24908/POCUS.V7IKIDNEY.15347>
  71. Stern AB, Klemmer PJ. High-output heart failure secondary to arteriovenous fistula. *Hemodial Int* 2011;15:104–7. <https://doi.org/10.1111/J.1542-4758.2010.00518.X>
  72. Duque JC, Gomez C, Tabbara M et al. The impact of arteriovenous fistulae on the myocardium: the impact of creation and ligation in the transplant era. *Semin Dial* 2015;28:305–10. <https://doi.org/10.1111/SDI.12313>
  73. Basile C, Vernaglione L, Casucci F et al. The impact of haemodialysis arteriovenous fistula on haemodynamic parameters of the cardiovascular system. *Clin Kidney J* 2016;9:729–34. <https://doi.org/10.1093/CKJ/SFW063>
  74. Gumus F, Saricaoglu MC. Assessment of right heart functions in the patients with arteriovenous fistula for hemodialysis access: right ventricular free wall strain and tricuspid regurgitation jet velocity as the predictors of right heart failure. *Vascular* 2020;28:96–103. <https://doi.org/10.1177/1708538119866616>
  75. Basile C, Vernaglione L, Casucci F et al. The impact of haemodialysis arteriovenous fistula on haemodynamic parameters of the cardiovascular system. *Clin Kidney J* 2016;9:729–34. <https://doi.org/10.1093/CKJ/SFW063>
  76. Nakhoul F, Yigla M, Gilman R et al. The pathogenesis of pulmonary hypertension in haemodialysis patients via arterio-venous access. *Nephrol Dial Transplant* 2005;20:1686–92. <https://doi.org/10.1093/NDT/GFH840>
  77. Stoumpos S, Mark PB. Should we ligate arteriovenous fistulas in asymptomatic patients after kidney transplantation? *Circulation* 2019;139:2819–21. <https://doi.org/10.1161/CIRCULATIONAHA.119.040361>
  78. Kruger Gomes L, Patel H, Agrawal N et al. Point of care ultrasound of the hemodialysis vascular access. *POCUS J* 2022;7 (Kidney):105–9. <https://doi.org/10.24908/POCUS.V7IKIDNEY.15348>
  79. Schoch M, Bennett PN, Currey J et al. Point-of-care ultrasound use for vascular access assessment and cannulation in hemodialysis: a scoping review. *Semin Dial* 2020;33:355–68. <https://doi.org/10.1111/SDI.12909>
  80. Palacherla J, O'Neill WC. Point-of-Care ultrasound for native kidney biopsies. *Kidney360* 2020;1:527–9. <https://doi.org/10.34067/KID.0001962020>
  81. Soomro QH, Amerling R. Point-of-care ultrasound in nephrology. *Curr Opin Nephrol Hypertens* 2021;30:176–83. <https://doi.org/10.1097/MNH.0000000000000681>
  82. Reisinger N, Panebianco N. Nephrology fellowship clinician-performed ultrasound curriculum. *POCUS J* 2022;7 (Kidney):7–8. <https://doi.org/10.24908/POCUS.V7IKIDNEY.14980>

- 
83. Mullangi S, Sozio SM, Segal P et al. Point-of-care ultrasound education to improve care of dialysis patients. *Semin Dial* 2018;**31**:154–62. <https://doi.org/10.1111/SDI.12664>
  84. Ma IWY, Liu G. Point-of-care ultrasound in internal medicine: establishing standards for Europe. *Eur J Intern Med* 2020;**75**:28–9. <https://doi.org/10.1016/j.ejim.2020.03.006>
  85. Díez J, Navarro-González JF, Ortiz A et al. Developing the subspecialty of cardio-nephrology: the time has come. A position paper from the coordinating committee from the Working Group for Cardiorenal Medicine of the Spanish Society of Nephrology. *Nefrología* 2021;**41**:391–402. <https://doi.org/10.1016/j.nefro.2021.02.003>