The Great Value of Critical Care U/S in Management of Acute Kidney Injury in ICU

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Abstract

In the last years, Critical Care U/S has become an essential branch of critical care Medicine and has gained general acceptance. Its use, both as a diagnostic tool and for hemodynamic monitoring, has increased markedly, greatly influencing contemporary cardiorespiratory management.

For example, there is RUSH protocol for shock, BLUE protocol for acute respiratory failure and SESAME protocol for cardiac arrest, all rely on critical care U/S.

A recent multinational study [the multinational AKI-EPI study] in a mixed ICU population found that 57% of patients experienced AKI according to the KDIGO criteria and that 13.5% were treated with RRT.

As judged by the American College of Radiology Appropriateness Criteria, renal Doppler ultrasonography is the most appropriate imaging test in the evaluation of AKI and has the highest level of recommendation.

Point of care U/S is safe, radiation free, noninvasive, cheap, repeatable with steep learning curve, moreover, it is now available in most critically care areas.

In this protocol we will propose a step by step approach to manage AKI by critical care U/S in ICU.

Keywords: RUSH Protocol; BLUE Protocol, AKI, US

Abbreviations

U/S: Ultrasound; AKI: Acute Kidney Injury; UB: Urinary Bladder; RI: Resistivity Index; IVC: Inferior Vena Cava; LVOT VTI: Left Ventricular Outflow Tract Velocity Time Integral; BPH: Benign Prostate Hypertrophy; SQP: Semiquantitative Renal Perfusion

Introduction

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A recent multinational study [the multinational AKI-EPI study] in a mixed ICU population found that 57% of patients experienced AKI according to the KDIGO criteria and that 13.5% were treated with RRT [1].

As judged by the American College of Radiology Appropriateness Criteria, renal Doppler ultrasonography is the most appropriate imaging test in the evaluation of AKI and has the highest level of recommendation.

Renal ultrasonography is typically the most appropriate and useful radiologic test in the evaluation of patients with AKI [2].

With B mode U/S kidney appear isoechoic or hypoechoic to nearby normal liver or spleen [3,4], increased echogenicity occurs in case of presence of abnormal materials in the kidney with high reflective power of sound waves, fibrous tissue (e.g. glomerulosclerosis, interstitial fibrosis) increases echogenicity. CKD is typically associated with increased echogenicity.

Inflammatory infiltrates may explain the increased echogenicity that occurs with acute interstitial nephritis and GN [5].

Calcium deposits and stones are very echogenic; thus, medullary nephrocalcinosis is characterized by increased medullary echogenicity and a relatively normal-appearing cortex.

Cortical necrosis is a rare cause of AKI that is classically characterized by a dark, hypoechoic renal cortex on ultrasonography. Cortical necrosis is a consequence of severe ischemia due to vascular abnormalities such as hemolytic-uremic syndrome, complications of pregnancy (e.g., eclampsia), or sepsis, which results in necrosis of tubular cells of the cortex; the necrotic cells and resultant increase in interstitial fluid cause the decrease in cortical echogenicity [6].

Kidney length is useful in the assessment of patient with AKI and may help distinguish AKI from CKD, small kidneys suggest the diagnosis of CKD [2].

In the kidney, RI is determined by assessing systolic and diastolic blood velocity in the segmental arteries and applying the following formula: Peak systolic velocity - end diastolic velocity/peak systolic velocity with the upper limits of normal value 0.7.

RI may increase in several causes of AKI, including obstruction, acute rejection, hepatorenal syndrome, and sepsis; the RI remains normal in prerenal azotemia and glomerular diseases. Studies suggest that RI may be a useful tool for predicting AKI, distinguishing ATN (or other parenchymal diseases) from prerenal azotemia and predicting severity in AKI [7].

Ultrasonography is the modality of choice to evaluate for the presence of obstruction, which accounts for 1% - 3% of cases of AKI in the ICU [8-10].

In our unit, we have a step by step protocol for approaching any patient presented with AKI.

**Discussion**

First, in any patient in our ICU who fulfill the RIFLE criteria of AKI, we start our protocol of critical care U/S in AKI management.

**First step**

If the patient is hemodynamically unstable we assess IVC if it is narrow totally collapsing, we think about prerenal cause for AKI, we start balanced crystalloid IVF guided by increase of IVC size and LVOT VTI increase, we keep IVF loading until no more increase of LVOT VTI by 10%.

We aimed for IVC either [diameter more than 2.1 and collapsibility more than 50% or diameter less than 2.1 and collapsibility less than 50%] in spontaneously breathing patient which equal to CVP of 8mmgh according to American society of Echocardiography guidelines.

In mechanically ventilated patient with controlled breath we stop giving IVF when dispensability exceed 18% [maximum diameter-minimum diameter/minimum diameter * 100 [11].
If IVC is markedly dilated and patient is hemodynamically unstable, we do Echocardiography to diagnose cardio renal syndrome and we manage accordingly.

**Second step**

We look for urinary bladder distension and dilated pelvicalyceal system to diagnose post renal failure.

**Figure 1:** Increase of IVC size after resuscitation of hemodynamically unstable patient with prerenal failure.

**Figure 2:** The same patient in figure 1 with increasing LVOT VTI after IVF.
If we find UB distension and/or dilated pelvicalyceal system, we use U/S to search for the cause.

**Figure 3:** Distended UB can be easily diagnosed by U/S.

**Figure 4:** Clear dilatation of pelvicalyceal.

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Figure 5: Sagittal view of distended UB due to BPH.

Figure 6: Transverse view revealing marked BPH.

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**Figure 7:** Big UB hematoma causing obstruction in a patient on warfarin.

**Figure 8:** Pelvic mass compressing UB and causing RT side hydronephrosis see figure 9.

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If we find UB distension in patient with urinary catheter, we search for obstructed catheter and false passage.

Figure 9: RT side hydronephrosis.

Figure 10: Distended UB with apparent catheter balloon indicating obstructed catheter.

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Ultrasonography is the modality of choice to evaluate for the presence of obstruction, which accounts for 1% - 3% of cases of AKI in the ICU.

**Step 3**

If IVC is normal and no obstruction in U/S, we check for size and echogenicity of the kidneys, if both kidneys has normal size and echogenicity, we suggest AKI and measure the renal interlobar artery RI and F/U measurement after resuscitation to differentiate between prerenal and renal failure and predict prognosis with RI value above 0.75 is going with acute persistent renal failure and value less than 0.75 is going with prerenal temporary failure.

\[ \text{RI} = \frac{\text{peak systolic velocity}}{\text{end diastolic velocity}} \]

with upper limits of normal is 0.7.

**Figure 11:** False passage urinary catheter in vagina in female patient with urine retention.

**Figure 12:** Normal interlobar RI 0.53.
Figure 13: Very high RI 0.84 in patient with septic shock and persistent acute renal failure.

Figure 14: Very high RI 0.8 in patient with hypertensive emergency and AKI.

Figure 15: Very high RI 0.85 in patient with severe obstructive shock due to PE and persistent acute renal failure.

**Figure 16:** Improving RI matching the improvement in serum creatinine in patient with transplant kidney recovered from sepsis.

**Figure 17:** Increasing diameter of IVC in patient with septic shock due to mesenteric ischemia after IVF resuscitation.

**Figure 18:** In the same patient in figure 17 increasing LVOT VTI with fluid resuscitation.

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After excluding obstructive renal failure, the causes of AKI can be divided into transient AKI resulting from decreased renal perfusion and persistent AKI, due to persistent damage of various renal structures [12].

It is usually assumed that decreased renal perfusion initially responsible for transient AKI may ultimately lead to acute tubular necrosis when it persists.

RI measured at admission was significantly higher in patients with sepsis who subsequently developed AKI [13].

This finding was recently confirmed in the postoperative setting of cardiopulmonary bypass [7]. Moreover, studies conducted in the 1990s suggested that renal RI may help to discriminate between patients with transient AKI and those with persistent AKI [14-16].

Results from three clinical studies suggest that RI may help to differentiate prerenal from intrinsic AKI [14-16].

Recent met analysis and systemic review suggest that an elevated RI may be a predictor of persistent AKI in critically ill patients [17].

Recent prospective multicenter study concluded that although statistically associated with AKI occurrence, RI and SQP perform poorly in predicting persistent AKI at day 3 [18].

**Conclusion**

Critical care U/S has a great role in the whole management of AKI in critically ill patients in ICU.

**A link for a YouTube lecture of our protocol**
https://www.youtube.com/watch?v=BcuaNix_wmc

**A link for a live study demonstrating how to apply the protocol**
https://www.youtube.com/watch?v=7W4sptnWpeY&list=PLXfXauTq7hVoyMuUSDQV6mjVQjWKcXl.60&oindex=18

**A link for playlist for a real cases of AKI management with critical care U/S**
https://www.youtube.com/watch?v=7pFvkJyFMs&list=PLXfXauTq7hVpedAI39yP_XBVUXdZCBfp
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