

Short Communication

Portal Vein Pulsatility, A Better Indicator of Volume Status in COVID-19?

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Abbreviations: AKI: Acute kidney injury; RRT: Renal replacement therapy; VEXUS score: Venous EXcess UltraSound; ICU: Intensive Care Unit; US: Ultrasound; IVC: Inferior vena cava; PVP: Portal vein pulsatility; NT-proBNP: N-terminal-pro hormone B-type Natriuretic Peptide

Acute kidney injury (AKI) is common among critically ill patients with COVID-19, affecting approximately 20–40% of patients admitted to intensive, of whom 20% require renal replacement therapy (RRT) [1,2]. Little is known about the cardiovascular consequences of COVID-19 of patients requiring ICU admission. Still, hemodynamic management plays an important role, as the need for vasopressor support in 95% of mechanically ventilated patients was reported in NEJM [3]. Recently, the VEXUS score (Venous EXcess UltraSound) has gained some visibility as a

congestion score combining multiple ultrasound markers to evaluate systemic venous congestion [4]

. In our Intensive Care Unit (ICU), we evaluated the first seven patients with COVID-19, with particular focus on ultrasound (US) evaluation, including lung, heart, inferior vena cava (IVC), and portal vein. All of them were mechanically ventilated; five were men, five had AKI, of which one needed RRT, and none died. Figure 1 is a representative graph showing a median of the seven patients for each of the variables displayed (in the y-axis) throughout the time (days on

the x-axis). A quick and straightforward interpretation of this graph: as patients get hypovolemic with furosemide, creatinine and urea increases, and portal vein pulsatility (PVP) and N-terminal (NT)-pro hormone B-type Natriuretic Peptide (NT-proBNP) decreases; IVC variation and B-lines in lung US don't change significantly.

Also, we followed this seven patients for seven days and fit a linear mixed-effects model fit by maximum likelihood with normalized creatinine as the outcome variable, with fixed effects of PVP, time and NT-proBNP and random slopes for PVP, time and NT-proBNP (B-lines was not fitted in this model, as there was no correlation in the primary analysis). We found that for each 10% increase in PVP we estimate an increase of 0.5 mg/dL in creatinine (β -coefficient -0.054, SD 0.017, $p=0.0044$), as opposed to the non-significant effect of IVC variation (β -coefficient -

0.15, SD 0.137, $p=0.287$) and the marginal effect of NT-proBNP (β -coefficient 0.0016, SD 0.0006, $p=0.095$).

As lung disease progresses towards more severe forms like ARDS, the pressure to stay dry can lead to a hypovolemic status, worsening further AKI. Some indirect measures, like IVC variation and the number of B-lines, can help define and guide volume status. But maybe these measurements are not the best on COVID-19 patients, as IVC dilatation and lack of variability can result from the parenchymal and vascular lung involvement and superimposed high pressures from aggressive mechanical ventilation, and B lines can solely represent interstitial viral inflammation, more than fluid overload. Although this is a small number of patients, PVP could be a better conceptual marker of the volume status in COVID-19 patients.

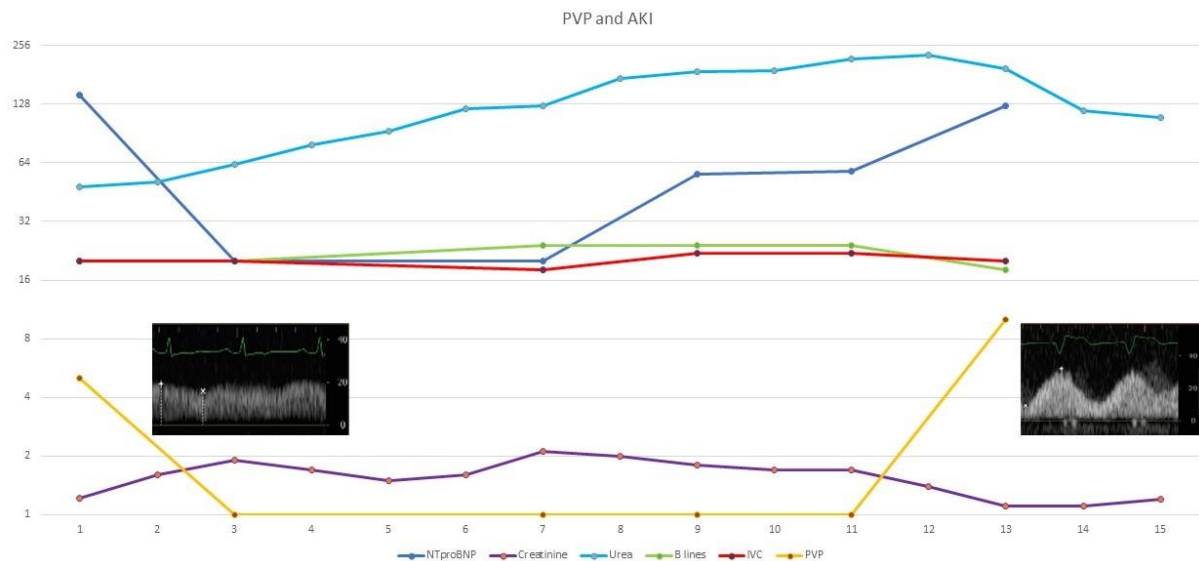


Figure 1: A representative pulsed-wave doppler of the portal vein can be seen at the beginning with a monophasic flow, indicating non-hypervolemia (possible hypo- to normovolemia), and at the end with a biphasic flow, indicating venous congestion (possible hypervolemia). PVP: Portal Vein Pulsatility; AKI: Acute Kidney Injury; NTproBNP: NT terminal of the pro-brain natriuretic peptide; IVC: Inferior Vena Cava variation.

References

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