

Peripheral Perfusion Index As an Early Biomarker of Acute Kidney Injury: Methodological Considerations

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Dear Editor,

I read Altınsoy's article in Global Emergency and Critical Care, which investigated the peripheral perfusion index (PI) and arterial blood gas parameters as potential early indicators of acute kidney injury (AKI), with great interest [1]. The study provides preliminary evidence suggesting that PI could serve as a noninvasive, real-time biomarker for diagnosing AKI and assessing its severity in the emergency department. However, given its retrospective observational case-control design, these findings should be regarded as hypothesis-generating rather than confirmatory. I commend the author for addressing an important clinical gap, particularly given the well-documented limitations of serum creatinine as a delayed marker of renal dysfunction.

The reported area under the curve (AUC) of 0.97 for PI in predicting AKI is notably high, and this unusually strong apparent discrimination warrants cautious interpretation. Although promising, this result requires careful evaluation. The exclusion of patients with sepsis, shock, and respiratory failure—conditions commonly encountered in emergency departments and known to independently influence PI measurements—may have led to an overestimation of diagnostic performance [2]. Equally important, these exclusions raise concerns regarding spectrum and selection bias, as the resulting study population may not adequately represent the heterogeneous patient population typically encountered in real-world emergency department settings, thereby limiting the external validity of the reported findings [2]. Additionally, several physiological

factors can affect PI values, including ambient temperature, peripheral vascular tone, and the use of vasoactive medications. These factors were not systematically controlled for in this study [3]. These considerations underscore that PI is fundamentally a physiological marker of peripheral perfusion rather than a kidney-specific biomarker, a distinction that warrants caution when interpreting its apparent diagnostic association with AKI. Future studies should standardize measurement conditions and incorporate these potential confounding factors into multivariable analyses.

I would also like to highlight that the study employed a cross-sectional approach, with measurements obtained at a single time point. This limits the ability to assess changes in PI over time. Because AKI is a dynamic process, serial monitoring of PI levels may provide additional information regarding disease progression and treatment response.

I also note an important inconsistency in the original article regarding the study design: the abstract describes the study as prospective, whereas the Methods section explicitly defines it as a retrospective observational study. This discrepancy is not merely semantic, as it has important implications for participant selection, the timing of PI measurements relative to AKI diagnosis, the risk of selection and information bias, and ultimately the interpretation of the reported diagnostic performance. I would welcome clarification from the author regarding the actual study design employed. Furthermore, PI was assessed at only a single clinical time point in the study. The absence of serial measurements limits the ability to



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evaluate temporal changes in PI, within-patient variability, and responsiveness to treatment or disease progression. Because AKI is a dynamic process, serial monitoring of PI levels may provide additional information regarding disease progression and treatment response.

Another important consideration concerns the characterization of PI as an "early" marker of AKI. In the study, PI appears to have been measured in patients already diagnosed with AKI according to creatinine-based criteria. The study did not demonstrate that PI became abnormal before serum creatinine levels increased, shortened the time to diagnosis, or predicted the subsequent development of AKI. Therefore, the reported findings reflect concurrent discrimination between AKI and non-AKI groups rather than genuine early diagnostic performance, and this distinction warrants clarification.

Furthermore, although the study appropriately used the Kidney Disease: Improving Global Outcomes (KDIGO) criteria to diagnose AKI, the partial availability of urine output data for Acute Kidney Injury Network (AKIN) and Risk, Injury, Failure, Loss, and End-stage Kidney Disease (RIFLE) staging represents a methodological limitation that could affect the accuracy of severity classification [4]. In conclusion, this study contributes to the growing literature on noninvasive biomarkers for AKI, but its findings should be regarded as preliminary rather than confirmatory. Before PI can be integrated into clinical algorithms, its diagnostic utility must be confirmed through prospective studies involving consecutive and clinically representative

emergency department patients, standardized and serial PI measurements, appropriately adjusted multivariable analyses, and internal or external validation. Further multicenter prospective studies involving larger and more heterogeneous populations are warranted to confirm these findings and establish standardized PI cutoff values for clinical use.

Ethics

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