急性腎損傷生物指標之運用--黎明前的曙光

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Postoperative acute kidney injury (AKI) is a serious complication resulting in prolonged hospital stay and high mortality rate. AKI develops in 5% to 30% of post surgical patients and is associated with a mortality rate of 60%–90%. Pre-renal azotemia and ischemic acute tubular necrosis are the predominant causes of renal failure. In particular KIM-1 and NGAL are considered excellent biomarkers in urine and plasma for the early prediction of AKI; however cycle arrest biomarkers have emerged as novel markers for risk stratification of AKI. Urine TIMP-2 and IGFBP7 performed better than any other biomarkers reported to date for predicting the development of moderate or severe AKI. Biomarker combinations are required to increase diagnostic accuracy in an acute setting. NGAL, cystatin C, and FGF-23 are promising and accurate biomarkers for AKI detection. Equations combining cystatin C and SCr perform better than the equations using either cystatin C or SCr alone, especially in situations where AKI needs to be confirmed. Combining creatinine, cystatin C and urine albumin to creatinine ratio improves risk stratification for kidney disease progression and mortality.

A patient could transition from one state to another and back, depending on the nature, severity and duration of the injury. This approach permits the identification of a novel state of 'subclinical' AKI in which kidney tissue damage might occur independently of any functional change as measured by serum creatinine level and urine output. Alternatively, patients might be oliguric and have elevated serum creatinine levels in the absence of any alterations in biomarkers of tissue damage. For the practicing clinician, this approach permits a more detailed assessment of the underlying condition of the kidney and sequential measurements could guide therapeutic interventions. Various biomarkers of functional change and tissue damage could be used together for differential diagnosis and prognosis depending on the disease context, for example a combination of serum cystatin C and urine NGAL might be more informative in liver disease, whereas KIM-1 and serum creatinine level might be appropriate in patients with sepsis. combination of functional kidney markers and tissue damage markers should enhance our ability to define appropriate time windows

for interventions. Sequential testing for biomarkers and clinical evaluation would permit a better delineation of the response to interventions and, therefore, prognosis. Emerging evidence supports the concept of using biomarkers in this way; however, further validation is requiredA biomarker-integrated model of AKI is proposed, which summarizes the current state of knowledge regarding the roles of these biomarkers and the molecular and cellular biology of AKI