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Editorial Article

Cystatin C is not Useful to Predict Approaching Acute Kidney Injury in Unstable Critical Care Patients

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Editorial

The Concept of Acute Kidney Injury (AKI) has changed very much lately. Some decades ago, it was considered a benign condition and needed only supportive treatment. But it is proven now that it may have devastating consequences. Study reported that, in the community; the patients who recovered from AKI have increased risk of death (HR:1.5) also they have increased risk to become a chronic renal failure patients (HR:1.91) in the United States of America [1]. Therefore, in the community, in the hospitals or in the Intensive Care Units patient with risk must be protected from developing of AKI. To do this we should have better biomarkers than conventional ones which are considered serum creatinine and several other urinary markers. The most important reasons of these unwanted outcomes should be delayed diagnosis of AKI. Better biomarkers should alert us beforehand should be practical and applicable in any conditions.

More than 30 different definitions were used for the definition of AKI hitherto which both caused difficulties to interpret and compare the studies. These definitions were developed based on the serum creatinine level which was considered late marker of AKI because it was not start to increase unless kidney functions decline 50% or more. It was suggested that re-evaluation of the definition of AKI was mandatory. For the consensus of the definition and improvement of the quality of studies on AKI, Acute Dialysis Quality Initiative (ADQI) group was developed. They recommended the term of AKI instead of ARF, and indicated that spectrum of AKI is broader and covers different degrees of severity of the disease. In 2002, for a uniform definition of AKI, they described three categories for severity (Risk of ARF, Injury of the kidney, and Failure of kidney function) and two classes for kidney outcome (Loss of kidney function and ESRD), which is called shortly RIFLE criteria [2].

Later, they excluded outcome categories and made some corrections and developed AKIN criteria[3] . Finally, in 2013 guideline of AKI definition was improved and took the final version; accepted by the nephrologists' in almost all around the world. But in any case, these definitions were based on the serum creatinine level so, they were good for established AKI, but not as early as to prevent and not useful to warn the upcoming AKI threat.

Many researches had being going on during the last decade to discover new biomarkers for AKI, since the conventional biomarkers were not sensitive enough to diagnose AKI beforehand. NGAL (Neutrophyl Gelatinase Associated Lipocaline) and CysC (Cystatin C) were the most studied ones among the others. Many investigators have proposed that CysC may be more sensitive to early AKI development and small changes in the GFR than conventional markers, such as creatinine.[4] On the contrary, a large multicenter study has revealed that CysC is less sensitive than creatinine for the early diagnosis of AKI [5]. We intend to investigate comparing these two biomarkers recently in Intensive Care Unit patients in point of the time of AKI developed.

The sNGAL , uNGAL and sCysC levels were determined at 48 hours of admission and surprisingly we found that sNGAL , uNGAL(AUC-ROC: 0.77, p = 0.005; 0.78, p = 0.002) but not CysC (0.54, p = 0.657)were useful for predicting of the development of AKI following 3-7 days in the ICU[6].

CysC was not found as efficient as serum and urine NGAL to show AKI risk in ICU in this Study. So, we thought that it was wise to detect urine and/or serum NGAL at the 48 hours in ICU admission to estimate AKI risk, even though this biomarker might be affected by so many factors in ICU.

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