Mini Review
Volume 13:8, 2022

Journal of Molecular Biomarkers & Diagnosis

ISSN: 2155-9929 Open Access

Clinical Performance of Biomarkers in Acute Kidney Injury

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Abstract

Several biomarkers of acute kidney injury (AKI) have recently been discovered and characterised. These molecules, which can be found in urine or blood, indicate structural damage to the kidney. They are being proposed clinically as adjunct diagnostics to serum creatinine and urinary output to improve the early detection, differential diagnosis and prognostic assessment of AKI. The most obvious requirement for a biomarker is that it reflect the disease's underlying pathophysiology. As a result, a biomarker of AKI should be derived from the injured kidney and reflect a molecular process that is intimately related to tissue injury.

Keywords: Acute kidney injury • Biomarkers • Calprotectin • Kidney injury molecule 1 (KIM-1)

Introduction

Acute kidney injury (AKI) is a common and severe complication following cardiac surgery, with a 3.4% incidence and 1.9% requiring dialysis treatment. AKI occurs in 2.9% of patients receiving isolated conventional coronary artery bypass grafting (CABG) and 1.4% of patients receiving off-pump coronary artery bypass, with dialysis required in 35.8% (19/53) of AKI patients. According to a large nationwide database analysis in the United States, CABG-associated AKI requiring dialysis increased from 0.2% to 0.6%, while mortality decreased from 47.4% in 1988 to 29.7% in 200. The incidence of postoperative AKI was found in a retrospective cohort study of patients undergoing CABG in a single hospital in Brazil [1].

Description

We conducted two multicenter observational studies - discovery and validation - in critically ill patients at risk for AKI. The top two discovery markers were validated and compared to a number of previously described biomarkers in a second study (Sapphire). We enrolled 522 adults in three distinct cohorts during the discovery phase, including patients with sepsis, shock, major surgery and trauma and examined over 300 markers. We enrolled 744 adult subjects with critical illness and no evidence of AKI at enrollment in the Sapphire validation study; the final analysis cohort was a heterogeneous sample of 728 critically ill patients. Within 12 hours of sample collection, the primary endpoint was moderate to severe AKI (KDIGO stage 2 to 3) [3-4].

14% of Sapphire subjects had moderate to severe AKI. The top two biomarkers discovered were validated. Urine insulin-like growth factor-binding protein 7 (IGFBP7) and tissue inhibitor of metalloproteinases-2 (TIMP-2) demonstrated an AUC of 0.80 when combined as inducers of G1 cell cycle arrest, a key mechanism implicated in AKI (0.76 and 0.79 alone). Urine [TIMP-2][IGFBP7] outperformed all previously described AKI markers (P 0.002), with none achieving an AUC greater than 0.72. Furthermore, when added to a nine-variable clinical model, [TIMP-2][IGFBP7] significantly improved risk stratification when analysed using the Cox proportional hazards model, generalised estimating equation, integrated discrimination improvement, or net reclassification improvement. Finally, regardless of changes in the reference creatinine method, [TIMP-2][IGFBP7] remained significant and superior to all other markers in sensitivity analyses [5].

Conclusions

Two new AKI markers have been identified and validated in separate multicenter cohorts. Both markers outperform existing markers, provide more information about clinical variables and provide mechanistic insight into AKI.

Acknowledgement

None.

Conflict of Interest

There are no conflicts of interest by author.

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Date of Submission: 30 July, 2022, Manuscript No. jmbd-22-77503; **Editor Assigned:** 02 August, 2022, PreQC No. P-77503; **Reviewed:** 11 August, 2022, QC No. Q-77503; **Revised:** 16 August, 2022, Manuscript No. R-77503; **Published:** 22 August, 2022, DOI: 10.37421/2155-9929.2022.13.537

How to cite this article: Solary, Eric. "Clinical Performance of Biomarkers in Acute Kidney Injury." J Mol Biomark Diagn 13 (2022): 537.