# KIDNEY INJURY TUBULAR BIOMARKERS FOLLOWING HEMATOPOIETIC STEM CELL TRANSPLANTATION (PILOT STUDY)

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## OBJECTIVES

Hematopoietic stem cell transplantation (HSCT) is associated with high risk of acute kidney injury (AKI). Routine diagnostics, based on the measurement of serum creatinine (Cr), may not reflect incipient tubular damage. The latter can be assessed by urinary biomarkers (BM's). The aim of this study is to evaluate changes of tubular damage BM's and their relationship to the clinical AKI following HSCT.

## METHODS

- We recruited 30 patients after allogeneic HSCT.
- Urinary concentrations of calbindin, clusterin, IL-18 (interleukin-18), KIM-1 (kidney injury molecule-1), GST-pi (glutathione S-transferase-pi), MCP-1 (monocyte chemoattractant protein-1) were measured along with serum creatinine in all patients on 5 consequent timepoints (before HSCT- week 0, on the week 1, 2, 3 and 4 after the transplantation). BM's increase was defined as doubling of BM's concentration from baseline (week 0).
- Clinical AKI was determined according to KDIGO (Kidney Disease Improving Global Outcomes) guidelines.

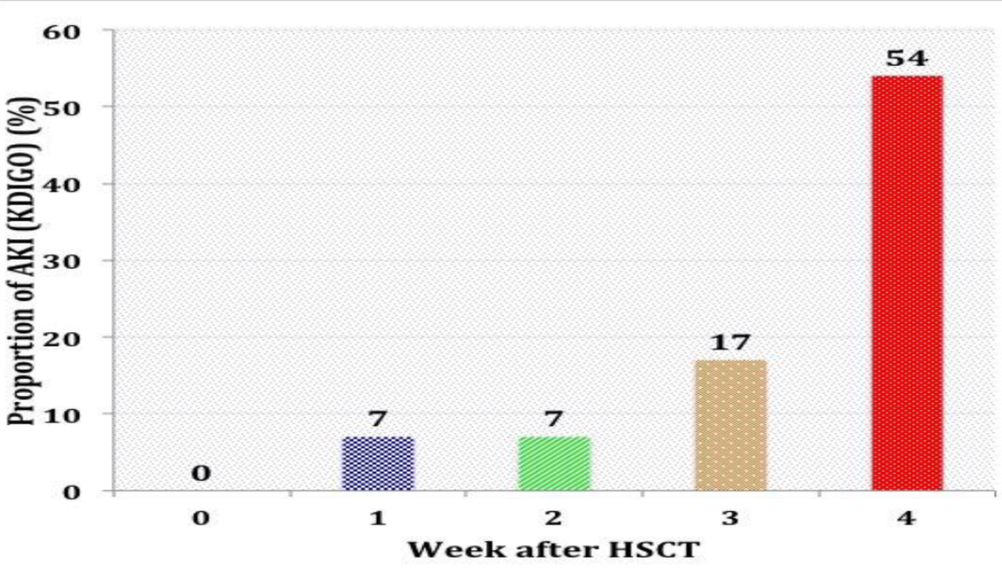


**Fig.1 Proportion of AKI (KDIGO) cases.** ($p_{ANOVA}$<0,05)
AKI-acute kidney injury; KDIGO-kidney disease improving global outcomes

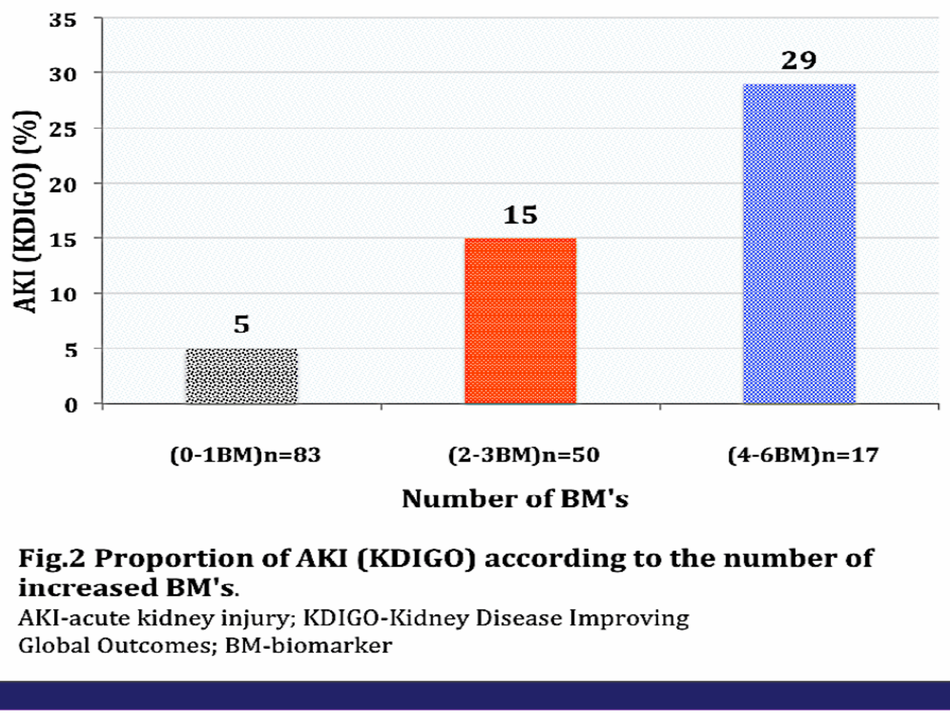


**Fig.2 Proportion of AKI (KDIGO) according to the number of increased BM's.**
AKI-acute kidney injury; KDIGO-Kidney Disease Improving Global Outcomes; BM-biomarker

## RESULTS

The cumulative proportion of AKI (KDIGO) cases on the weeks 1 and 2 after HSCT was 7%, on the week 3- 17%, on the week 4- 54% ($p < 0.05$) (Fig.1). The proportion of cases with increase of at least one BM on the week 1 following HSCT was 78%, on the week 2 - 85% on the week 3 - 90%, on the week 4 - 74%. The proportion of cases with simultaneous increase of 1-3 BM's was 49% on the week 0, on the week 1 - 55%, on the week 2 - 58%, on the week 3 - 59%, on the week 4 - 36%. The proportion of cases with simultaneous elevation of 4-6 BM's after HSCT had been progressively increasing from 6% (week 0) to 38% (week 4). The frequency of clinical AKI on the week following BM's analysis increased in relation to the number of simultaneously elevated BMs on the previous week ($p_{ANOVA}$=0,007) (Fig.2). The number of elevated BM's was associated with subsequent occurrence of clinical AKI (ROC-analysis: $S_{AUC}$=0,69; p=0.006).

## CONCLUSION

Tubular injury, as evaluated by BM's, occurs frequently and precedes clinical AKI. Further evaluation of BM's is needed for the assessment of incipient tubular injury in respect of prognosis and prevention of clinical AKI in the setting of HSCT.

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