

MO050

GLYCEMIC CONTROL AND THE RISK OF AKI IN PATIENTS WITH DIABETES AND CKD: PARALLEL POPULATION-BASED COHORT STUDIES IN U.S. AND SWEDEN ROUTINE CARE

Yang Xu¹, Aditya Surapaneni², Jim Alkas¹, Alexander Chang³, Morgan Grams², Juan Jesus Carrero¹

¹Karolinska Institutet, Medical Epidemiology and Biostatistics, Stockholm, Sweden,

²Johns Hopkins Bloomberg School of Public Health, Department of Epidemiology,

Baltimore, United States of America and ³Geisinger Health System, Division of

Nephrology, United States of America

Background and Aims: Patients with diabetes and chronic kidney disease (CKD) have increased susceptibility to acute kidney injury (AKI), but the underlying mechanisms are not well known. We here explore the association between glyceemic control and risk of AKI.

Method: We created two parallel observational cohort studies of Swedish (SCREAM project, Stockholm, 2006-2011) and U.S. (Geisinger Health system, Pennsylvania, 1996-2018) adult patients with diabetes mellitus and confirmed CKD stages G3-G5. Glyceemic control was evaluated through repeated HbA1c measurements, which were categorized into 5 levels of glyceemic control intensity, with HbA1c 6-6.9% as referent category, and continuously using cubic splines. We evaluated the association between baseline and time-varying HbA1c levels with AKI (defined as increase in creatinine ≥ 0.3 mg/d over 48 hours or 1.5x creatinine over 7 days) using Cox proportional hazards regression and, in sensitivity analyses, Fine and Gray competing risk models accounting for death.

Results: In the Swedish cohort, there were 13932 patients with median age 76 years, 51% women, median eGFR 50.8 (Interquartile Range (IQR) 41.4-57.1) ml/min/1.73. In the U.S. cohort, there were 26520 patients with median age 71 years, 55% women and 52.1 (IQR 43.4-57.5) ml/min/1.73 m². During a median of 2.3 and 3.1 years of follow up, 3172 and 8671 AKI events were recorded in the Swedish and US cohorts, respectively. The adjusted association between baseline HbA1c and AKI was similar in both cohorts, with the lowest risk between 6-6.9% and higher risk at higher levels of HbA1c. Compared to baseline HbA1c 6-6.9%, baseline HbA1c >9% associated with a 1.28 fold (95% CI 1.11-1.47) higher risk of AKI in the Swedish cohort, and a 1.14 (95% CI 1.04-1.25) higher risk in the U.S. cohort. Conversely, baseline HbA1c <6% did not associate with AKI. When using time-varying HbA1c, AKI risk was higher for HbA1c >9% (HR 1.18, 95% CI 1.03-1.37 in Swedish cohort and 1.27, 1.17-1.37 in U.S. cohort); AKI risk was also higher for HbA1c <6% in the U.S. cohort (1.12, 1.04-1.19), but not in the Swedish cohort (1.06, 0.97-1.16).

Conclusion: Higher A1c was associated with AKI in adults with diabetes and CKD, suggesting that better glyceemic control may also reduce risk of AKI.

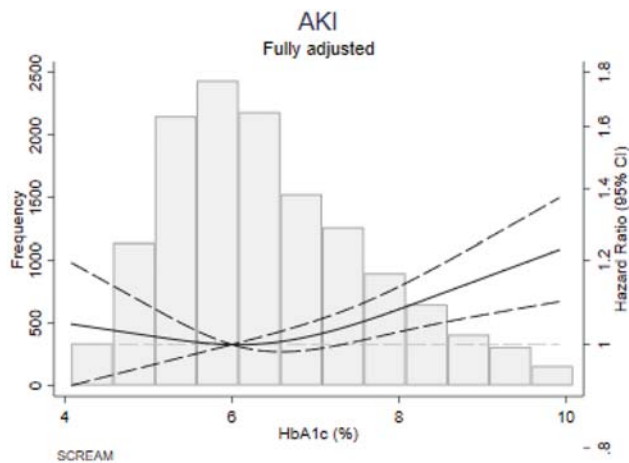


Figure. Multivariable-adjusted associations between baseline HbA1c (continuous variable) and the risk of AKI in two parallel cohorts of adults with diabetes CKD; Abbreviation: ESRD, end-stage renal disease; HbA1c, glycated hemoglobin.