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Objective: This study aimed to evaluate the impact of CKD age-defined definition on all-cause mortality and cardiovascular (CV) outcomes in a rural population over 136months long-term follow-up (2005-2022).

Design and method: A total of 1175 participants from the ENAH project were stratified by guideline-defined CKD (eGFR <60ml/min/1.73m^2) and age-defined CKD. The age-adjusted CKD cut-off accounted for physiological renal decline with age. Primary outcomes included mortality and CV events (myocardial infarction, atrial fibrillation, heart failure, stroke, transient ischemic attack, and haemodialysis).

Results: Participants with guideline-defined and age-defined CKD were older (73 vs. 49, 73 vs.47years, respectively; p<0.001). Systolic blood pressure, BMI and visceral obesity were higher in CKD groups regardless the used definition than in non-CKD subjects (p<0.001 for all). All-cause mortality was higher in guideline-defined CKD group than in age-defined CKD group, but not statistically significant (64.9% vs.58.6%; p=0.320). All-cause mortality was significantly lower in non-CKD group (18.0%; p<0.001 for both). Kaplan-Meier analysis showed reduced survival in both CKD groups. For guideline-defined CKD and for age-defined CKD, the log-rank test was positive HR 5.51; 95%CI: 3.36-9.04 and HR 6.93; 95%CI: 4.87-9.87, respectively (log-rank test p<0.001 for both). For non-fatal composite outcomes, HR was 1.81 (95%CI: 1.00-3.27, p=0.011) in guideline CKD and 1.57 (95%CI: 1.03-2.41, p=0.014) in age-defined CKD. Univariate analysis showed guideline-defined CKD (HR 5.62, p<0.001) and age-defined CKD (HR 7.29, p<0.001) predicted mortality and non-fatal outcomes, but in multivariate analysis elevated estimated pulse wave velocity (ePWV) (HR 2.11, p=0.014) and age (HR 1.12, p<0.001) were the only independent predictors for overall mortality.

Conclusions: CKD significantly predicted mortality and CV events in rural populations. The CKD age-defined definition identifies additional high-risk individuals. The population at greatest risk of misdiagnosis includes younger patients with eGFR near 60ml/min/1.73m^2. ePWV, which is a hallmark of CKD, was an independent predictor of mortality. The age-definition of CKD could improve CKD risk stratification and ePWV might be used as a useful tool.

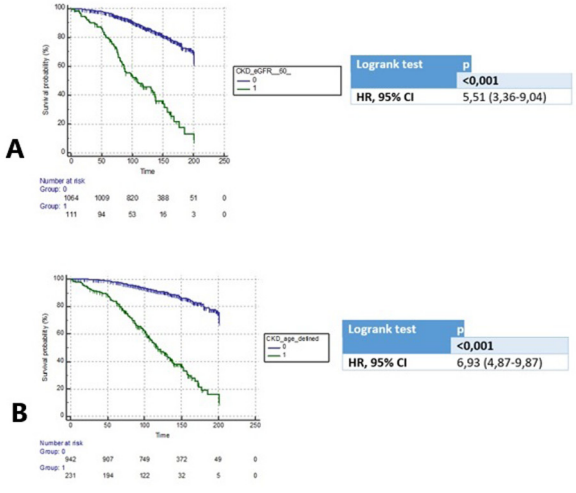

Figure 1. Kaplan Meier survival analysis for fatal outcome depending on the presence of CKD (A) defined by guidelines as eGFR < 60 ml7min/1.73m² and (B) defined as age-defined definition

PROGNOSTIC SIGNIFICANCE OF VISIT-TO-VISIT ULTRAFILTRATION VOLUME VARIABILITY IN HEMODIALYSIS PATIENTS

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Objective: Patients on chronic hemodialysis (HD) have considerably higher mortality compared with the general population. Cardiovascular (CV) disease is the primary reason for death in these patients. Suboptimal extracellular fluid management increases the CV risk of HD patients. We aimed to study the effct of visit-to visit UV variability on CV events and mortality in chronic HD patients.

Design and method: 173 chronic HD patients were included (median age: 63±13years; 53% men). Ultrafiltration volume (UV) variability has been analyzed retrospectively for 24months. The standard deviation (SD) and coefficient of variation (CV) were calculated using the indices of UV variability. CV is the SD divided by the mean. The obtained parameters were SD and CV of the UV: UVSD and UVCV. Routine transthoracal echocardiography was performed.

Results: Patients were divided in groups based on the median of UVSD: low UVSD (< 568ml) and high UVSD (>= 568ml) group, and also based on the median of UVCV: low (<0.29) and high UVCV (>= 0.29) group. All-cause mortality was higher in high compared to the low (21/84 vs. 9/89; p<0.001) UVSD group. Similarly, mortality was higher in the high UVCV group compared to the low UVCV group (18/78 vs. 12/95; p=0.005) after 24month. Major adverse CV event rates (MACE) were also higher in the high compared to the low UVSD group (20/84 vs. 8/89; p<0.001) respectively. Similarly, MACE rate was also higher in the high UVCV group compared to the low UVCV group (15/78 vs. 13/95; p=0.029) after 24month. UVSD correlated with parathormone (PTH) level (r = 0.416; p = 0.015) and UVCV with total cholesterol (r = 0.419; p = 0.015). Left ventricular end-diastolic (LVEDD) and end-systolic diameter (LVESD) were higher in the high UVCV group compared to the low UVCV group (49.95 vs. 52.08; p=0.013 and 32.19 vs. 34.13; p=0.034).

Conclusions: High UVSD and UVCD are associated with increased all-cause mortality and MACE rates in chronic HD patients. CV changes caused by increased UF volume variability during HD may contribute to higher CV morbidity and mortality in these patients.

EVALUATION OF RACE-FREE EGFR EQUATIONS IN INDIVIDUALS OF DIFFERENT ETHNICITY

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Objective: Race-free equations for estimating glomerular filtration rate (eGFR) using serum creatinine (eGFRcr), cystatin C (eGFRcys), or both (eGFRcr-cys) are digital tools used worldwide in managing CKD. However, their performance in population-based screening of low-grade CKD in Blacks and non-Blacks is unknown.

Design and method: The new race-free CKD-EPI and EKFC eGFR equations were evaluated in four diverse cohorts: African-PREDICT (341/380 healthy Black and White South Africans), FLEMENGHO (709 White Flemish), NHANES (1760/7931 Black and non-Black Americans), and 401 Black African patients hospitalised in Mbuji Mayi, Democratic Republic of Congo.

Results: Intraindividual discordance between eGFRs was substantial and larger in Black than non-Black NHANES and African-PREDICT participants. In NHANES, eGFRcr-cys (CDK-EPI) was greater than eGFRcr, but smaller than eGFRcys, while eGFRcr (EKFC) was smaller than eGFRcys. Replacing eGFRcr-cys by eGFRcr moved up to 25% Black and up to 15% non-Black NHANES participants to a higher (worse) eGFR KDIGO stage. In African-PREDICT and FLEMENGO, the mClcr (measured creatinine clearance) to eGFR ratios fell outside the expected 1.1–1.2 band in 50% of eGFR estimates. In NHANES, irrespective of the eGFR formulation applied, multivariable hazard ratios for total or cardiovascular mortality in relation to CKD grade and compared to the average population risk were all lower than unity for grade 1 CKD and greater than unity for grade >=3 with a trend p-value from stage 1 to >=3 of p<0.0001 without any racial difference (0.11<=p<=0.98). These NHANES findings were consistent, if CKD stage was replaced by eGFR and in subgroup analyses. Whereas eGFRcys and eGFRcr-cys refined models, eGFRcr did not.

Conclusions: While mortality outcomes do support use of race-free eGFR equations in population-based studies, large intraindividual variability between eGFR estimates might lead to KDIGO eGFR stage misclassification and calls for caution in opportunistic or systematic CKD screening in asymptomatic individuals with prevention as objective.