Abstracts

P0714 RELATIONSHIP BETWEEN BIOLOGICAL AGE ESTIMATED BY SKIN AUTOFLUORESCENCE, CHRONOLOGICAL AGE, AND MORTALITY IN CHRONIC KIDNEY DISEASE

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Background and Aims: While chronological age associates with increased risk of death, there is a quest for markers of biological age in chronic kidney disease (CKD) that better reflect accumulation of tissue and cellular damage, which could contribute to shorter life span. Skin autofluorescence (SAF) is a biomarker for accumulation of advanced glycation end products in skin that associate with chronological age and with factors that may increase mortality risk. However, the predictive capacity of SAF for mortality has not been fully elucidated in CKD. We have investigated the relationship between biological age calculated by SAF, chronological age and all-cause mortality in patients with CKD stage 5.

Method: In a cohort of 199 CKD5 patients (non-dialysis CKD5, n=100, hemodialysis, n=27 and peritoneal dialysis, n=72; median age 66 years, 34% females, 21% diabetes (DM), 20% cardiovascular disease (CVD), and 34% malnourished), we calculated biological age by a formula based on SAF measurements using the AGE Reader. Framingham risk score, coronary artery calcium score, the heart rate-corrected augmentation index, body composition, nutritional status, handgrip strength, and various biochemical markers (hemoglobin, albumin, creatinine, intact-parathyroid hormone, triglyceride, cholesterol, HDL-cholesterol, high-sensitivity C-reactive protein (hsCRP), and interleukin (IL)-6) were recorded at baseline. During median follow-up of 38 months, 34 patients died, and 51 patients underwent renal transplantation. We analyzed spline curves showing sub-distribution hazard risk (sHR) for all-cause mortality with biological age calculated by SAF and chronological age by the Fine and Gray competing risk analysis.

Results: There was a significant association between biological age calculated by SAF and chronological age (rho=0.48; p<0.001). IL-6 and hsCRP were positively associated both with biological age according to SAF measurement (IL-6: rho=0.34, p<0.001; n=155 and hsCRP: rho=0.31, p<0.001; n=199) and chronological age (IL-6: rho=0.47, p<0.001; n=155 and hsCRP: rho=0.40, p<0.001; n=199). The multivariate spline curve showing sHR for all-cause mortality associated positively with chronological age (sHR: 1.04, p=0.035) and biological age calculated by SAF (sHR: 1.01, p=0.048) when adjusted for sex, DM, CVD, nutritional status, 1-standard deviation increase of hsCRP, and CKD5 groups.

Conclusion: All-cause mortality risk increased linearly with higher chronological age and SAF-estimated biological age - and with similar magnitude of sHR for the two suggesting that prediction of mortality risk based on SAF is not superior compared to chronological age in CKD. We conclude that biological age calculated by SAF and chronological age are equally robust predictors of clinical outcomes in CKD; however, both indices are influenced by the inflammatory status.