**Statistical analysis (Work on)**

* How data is reported (mean/ SD, and count/ percentage)
  + Data was reported as means ± standard devotions and as counts and percentages.
* Statistical test used, software used and version ( i.e R)
  + Relative risk
* How many people were missing clinical data (report as percentage)
* Mention imputations
* How did we create tertials of uAng2?

**Examples:**

Quantitative data were expressed as means with their corresponding standard errors or as individual data points as indicated in the figures. Means were compared for significance by two-tailed t test or two-way ANOVA as appropriate. P-values of 0.05 were considered statistically significant. Statistical analyses and graphical representation of numeric data were performed using Prism software (Graph-Pad Software, Inc.) or R version 3.6.1.

We summarized baseline participant characteristics across tertiles of baseline serum sTNFR-1 concentrations with mean and SD for continuous variables and number and percentage for categorical variables. We graphically examined the univariate distribution of sTNFR, and we evaluated the cross-sectional association of sTNFR-1 with eGFR at baseline via linear regression. For the primary analysis, we used Cox regression to evaluate the association between baseline sTNFR-1 and incident ≥40% decline in eGFR over a median (interquartile range) of 9.3 (interquartile range, 8.5–9.7) years of follow-up in a series of nested models, which controlled for potential confounding factors: age, sex, race/ethnicity, education, site of enrollment, body mass index, hypertension, systolic BP, diabetes, urine albumin-to-creatinine ratio, baseline eGFR, IL-6, and high-sensitivity C-reactive protein (hsCRP) concentrations. We tested the proportional hazards assumption of the Cox regression and found no violations; inspection of the Schoenfeld residuals likewise did not raise concerns.[22](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6218870/#B22) We also estimated the annualized proportional decline in eGFR across strata of baseline sTNFR-1 concentrations using a linear mixed model approach with random intercepts to account for the within-person correlation occurring with repeated measurements; diagnostic inspection for the distribution of random effect variances revealed no gross departures from normality. Change in eGFR was computed on the basis of the slope across all visits. Finally, we evaluated the association of serum sTNFR-1 concentrations and RAC scores >0 via logistic regression, controlling for potential confounders.

We also performed subgroup analyses to explore whether the associations between serum sTNFR-1 concentrations and ≥40% incident eGFR decline were different between participants on the basis of age, sex, race/ethnicity, diabetes, hypertension, and baseline CKD status defined as an eGFR<60 ml/min per 1.73 m2. Approximately 3% or fewer of analyzed participants were missing information on covariates, such as education and urine albumin-to-creatinine ratio; these subjects’ values were multiply imputed using chained equations,[23](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6218870/#B23) which were then combined using Rubin rules to account for the variability in the imputation procedure.[24](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6218870/#B24) For all analyses, a two-tailed P value of <0.05 was taken as evidence of statistical significance. All statistical analyses were performed in R 3.4.0 (R Core Team 2015)

**Statistical Analyses** (Zoie)

Patient characteristics were summarized across tertiles of baseline uAng2 concentrations with mean and SD for continuous variables and number and percentage for categorical variables.

For the primary analysis, we performed a relative risk (RR) analysis to evaluate the association between baseline uAng2 and clinical outcomes (AKI, dialysis and mortality) adjusting for age, sex, race/ethnicity, BMI, IMV and COVID status. A pearson correlation test was done to determine if uAng2 was correlated with plasma Ang2 (pAng2), urinary NGAL (uNGAL), and urinary KIM-1 (uKIM-1).

About 33.3% of patients in tertile 1 were missing uAng2 measurements and these values had to be imputed. Imputation was done by dividing uAng2 concentration by baseline urine creatinine. Rational for imputation…

All statistical analyses were performed in R [version] (name of software)

**Statistical Analyses** (Jordan)

We summarized baseline participant characteristics across tertiles of urinary Ang-2 concentrations normalized to urinary creatinine with mean and SD for continuous variables and number and percentage for categorical variables. The primary analysis of this paper used a log-linear regression to find relative risk exposures for several clinical outcomes of interest using uAng-2 concentrations, with controls for potential confounders: age, sex, race/ethnicity, invasive mechanical ventilation, COVID status, and BMI. To account for clustering in the dataset, robust standard errors were employed in the model. Additionally, a Pearson’s correlation was calculated to evaluate the association of several biomarkers (urinary KIM-1, urinary NGAL, and plasma Ang-2) with uAng-2.

The 192 participants were stratified into three equal tertiles based on their concentration of uAng-2. Within the whole group, approximately 19% of participants had uAng-2 concentrations that were below the lower limit of detection (LLOD) of the assay; their concentrations were imputed using (0.5) \* LLOD. For all analyses, a two-tailed P value of <0.05 was used as evidence for its statistical significance. All statistical analyses were performed in R 4.2.2 (R Core Team 2022)