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Prognostic value of soluble suppression of tumorigenicity 2 in chronic kidney disease: a systematic review and meta-analysis

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Abstract

Cardiovascular disease represents the main complication of chronic kidney disease. Robust biomarkers of increased cardiovascular risk in the chronic kidney disease population are currently lacking. Soluble suppression of tumorigenicity 2 levels have been associated with incident heart failure, as well as with adverse outcomes in patients with cardiovascular disease. The present systematic review and meta-analysis aims to shed light on the prognostic role of soluble suppression of tumorigenicity 2 in chronic kidney disease patients, evaluating its potential association with overall survival, cardiovascular events and kidney disease progression.

Troubleshooting

1 Objective To determine the association of soluble suppression of tumorigenicity 2 (sST2) levels with survival, kidney disease progression and cardiovascular disease in patients with chronic kidney disease.

2 Eligibility criteria The population of the study will consist of adults with diagnosed with chronic kidney disease. Both pre-dialysis and dialysis (hemodialysis or peritoneal dialysis) patients will be held eligible. Kidney transplant recipients will be excluded. The exposure of interest will be serum sST2 levels. The primary outcome of interest will be all-cause mortality. Secondary outcomes will include kidney disease progression, cardiovascular mortality, major adverse cardiovascular events. Cohort (prospective and retrospective) and case-control studies and will be held potentially eligible. Descriptive, cross-sectional, animal and in vitro studies, as well as case reports/series and review articles will be excluded.

3 Literature search Literature search will be performed by systematically searching from inception PubMed, Scopus, Web of Science and CENTRAL (Cochrane Central Register of Controlled Trials). In addition, Google Scholar will be screened to provide grey literature coverage, while the full reference lists of the included studies will be examined to recognize potential missing articles. No date/language restrictions will be applied.

4 Data extraction The following data will be extracted: year of publication, country, eligibility criteria, sample size, study design, type of population, participants' age, sex, percentage of hypertension, diabetes mellitus, estimated glomerular filtration rate, history of cardiovascular disease, as well as the necessary information regarding the outcomes of interest.

5 Quality assessment The risk of bias of the included studies will be evaluated with the ROBINS-I tool, adjusted for exposure studies, taking into account the following domains: confounding, selection of participants, classification of exposures, departures from intended exposures, missing data, measurement of outcomes and selection of the reported results.

6 Data analysis All outcomes will be evaluated qualitatively. Pre-piloted forms will be used to capture all the necessary information regarding the outcomes of interest. Serum sST2 levels could be evaluated as a continuous variable or as a binary one in case cut-off values are introduced. For time-to-event endpoints, hazard ratios will be extracted. Statistical significance will be defined by the two-sided p-value threshold of 0.05. analysis will be performed in case of at least 3 studies per outcome. Confidence intervals will be set at 95%. Conventional meta-analysis will be conducted by comparing the highest to the lowest sST2 category. Random-effects statistical models will be fitted, using the maximum likelihood method. Subgroup analysis will be conducted by separately examining pre-dialysis and dialysis patients. Dose-response meta-analysis will be conducted to define the potential exposure-response relationship between serum sST2 levels and mortality risk. In particular, a non-linear model using restricted cubic

splines will be applied in a one-stage approach. Restricted cubic splines were located at the 25th, 50th and 75th percentiles of the serum sST2 level distribution.