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P235 -Is there a rationale for biomarker stratified clinical trial for primary prevention of heart failure in chronic kidney disease?

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Introduction: Early detection of subclinical cardiac dysfunction and primary prevention of heart failure (HF) in chronic kidney disease (CKD) are KDIGO research priorities. It has been shown that NT-proBNP, a natriuretic peptide, predicts incident heart failure in asymptomatic CKD patients [J Am Soc Nephrol. 2015 Apr;26(4):946-56]. Indeed, an NT-proBNP value of >433 pg/ml was associated with >9 times higher risk of HF than a normal value. We set out to determine whether the biomarker is associated with asymptomatic cardiac dysfunction in CKD.
Methods: We studied 48 asymptomatic male CKD patients (stages 2-5) without known cardiac disease or diabetes. We evaluated peak cardiac performance to reveal sub clinical cardiac dysfunction. We measured peak cardiac power (CPOmax, expressed in Watt) using a specialised cardiopulmonary exercise test (CPX) [Nephrol Dial Transplant. 2018 Mar 1;33(3):450-458]. NT-proBNP assays were also performed. Association between variables was evaluated using Pearson's correlation. Log transformed NT-proBNP values were used for analysis. Results are presented as mean±SD. $P<0.05$ is considered significant.
Results: CKD patients had a mean age of 46.7±12.5 years. Mean NT-proBNP values across CKD stages is shown in Figure 1A. There was a gradual decline in peak cardiac function (CPOmax) with worsening kidney function (eGFR) ($r=0.54$, $P<0.10\text{-}3$). NT-proBNP demonstrated significant negative correlation with CPOmax ($r=-0.35$, $P=0.014$) (Figure 1B). NT-proBNP levels were >433pg/ml in 58.3% of the asymptomatic CKD patients but only a fifth of the study patients (20.8%) were on beta blockers.
Conclusion: The study demonstrates association between subclinical cardiac dysfunction and plasma NT-proBNP levels in CKD patients. More than a half of our study patients had an NT-proBNP value >433 pg/ml placing them at a high risk of heart failure. We studied a CKD population in the absence of any cardiac comorbidities; it is likely that the proportion could be higher in an unselected CKD population. As a significant proportion of CKD patients are at high risk of developing HF - Should we be doing a biomarker stratified clinical trial for primary prevention of HF in CKD?