REAL-WORLD EVIDENCE OF THE RISK OF STROKE OR SYSTEMIC EMBOLISM IN CHRONIC KIDNEY DISEASE PATIENTS WITH ATRIAL FIBRILLATION

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An analysis of a large EMR dataset shows a small increased rate of stroke/systemic embolism (S/SE) in CKD patients treated for AF compared to untreated AF patients. These findings do not support treating CKD patients with AF differently than any other AF patients. Further analyses should examine whether CKD progression impact results or whether varying OAC drugs change the conclusions.

OBJECTIVES
The risk of stroke and bleeding increases monotonically by stage of chronic kidney disease (CKD). CKD and atrial fibrillation (AF) patients share common risk factors. Recent studies have found inconsistent findings regarding the effectiveness of oral anticoagulant (OAC) treatment for AF in patients with CKD (1). The following analysis used a large, federated healthcare network to examine the relative risk of stroke/systemic embolism (S/SE) in CKD patients treated for AF.

METHODS
Data from a research network representing over 17M patient-lives was used to examine the association between OAC use and S/SE among stage 1-4 CKD patients. Patients were defined by ICD9/10 and LOINC codes. Patients were 65+, had a CKD or eGFR code, and an AF code between 2012-2017. The first OAC treatment between 2013-2017 defined the index date for treated patients. Untreated patients were matched to treated patients and an induction period was imputed based on the matched patient’s time from AF diagnosis to treatment (Figure 1). Incidence rate ratios (IRRs) and 95% CIs were calculated for the occurrence of S/SE among a propensity score matched study population.

A propensity score model included the variables in Figures 3-5, as well as other bleeding events (not pictured). The standardized mean difference (SMD) comparing variables in the treated and untreated cohorts was less than 10%. A Poisson regression model was used to calculate the IRR overall and for each stage of CKD (Figure 5). Variables included in the propensity score model with a SMD of between 10-20% were also included as covariates in the final Poisson regression model to adjust for residual confounding (heart failure, beta blockers, loop diuretics, calcium channel blockers, lipid lowering agents, and aspirin cardiac glycosides). Patients were censored.

RESULTS
• Propensity score matching balanced patients on CKD stage and other baseline characteristics.
• Treated patients with any stage of CKD had an increased rate of S/SE compared to untreated patients, although this increase was small.
• CIs around the IRRs for Stages I & II CKD and Stage IV indicate a large amount of variability for these estimates.
• Effect measure modification by CKD stage is small, although patients in Stages I & II may differ from patients in Stages III & IV.

CONCLUSIONS
An analysis of a large EMR dataset shows a small increased rate of S/SE in CKD patients treated for AF compared to untreated AF patients. These findings do not support treating CKD patients with AF differently than any other AF patients. Further analyses should examine whether CKD progression impact results or whether varying OAC drugs change the conclusions.