PROTECTIVE RELATIONSHIP BETWEEN DPP4/GLP1 MEDICATION USE AND HEART FAILURE EVENTS IN PATIENTS WITH DIABETES

Poster Contributions
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Background: Recent studies suggest that anti-diabetic medications which target the Glucagon-like Peptide 1 (GLP1) pathway (i.e. Dipeptidyl Peptidase 4 inhibitors and GLP1 analogs [GLP1 agents]) may be protective in heart failure (HF). However, there are no large observational studies or randomized trials to support these preliminary findings.

Methods: To further test this hypothesis, we performed a retrospective cohort study among members of a large health system in southeast Michigan. We identified more than 19,000 adult diabetic patients with an oral diabetes medication fill between January 1, 2000 and July 1, 2012. Patients initiating treatment with GLP1 agents were matched 1:2 to controls using propensity matching which accounted for age, race, gender, coronary disease, HF, duration of diabetes, and the number of antidiabetic medications. Multivariable Cox regression was used to test the effect of GLP1 agents on time to HF hospitalization. Secondary endpoints included all-cause hospitalization and all-cause mortality.

Results: We identified 1,488 new users of GLP1 agents and 2,939 propensity matched controls. The groups were similar on all matched variables except the number of anti-diabetic medications (1.2 vs. 1.4, p<0.001) and age (60.4 vs. 61.7 years, p<0.001). Over a median observation time of 663 days there were 281 hospitalizations, of which 184 were due to HF, and 158 deaths. Use of GLP1 agents was associated with reduced risk of HF hospitalization (adjusted hazard ratio [aHR] 0.56; 95% confidence interval [CI] 0.40-0.78, p<0.001), all-cause hospitalization (aHR 0.56; 95% CI 0.42-0.73, p<0.001), and death (aHR 0.20; 95% CI 0.13-0.33, p=0.001).

Conclusions: Our findings suggest that anti-diabetic agents which enhance GLP1 activity may have additional benefit of reducing the risk of HF events among diabetic patients. Additional studies are warranted to validate this association.