

Impact of type 2 diabetes on incidence and phenotype of heart failure in patients with atrial fibrillation

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Background: Type 2 diabetes (T2DM) portends adverse prognosis in patients with atrial fibrillation (AF). Whether T2DM independently increases the risk of incident heart failure (HF) in AF is uncertain. Also, HF phenotype developing in patients with vs. those without T2DM has not been characterised.

Purpose: In AF patients without a history of prior HF, we aimed to assess: 1) the impact of T2DM on the risk of new-onset HF; and 2) the association between T2DM and HF phenotype developing during the prospective follow-up.

Methods: We included diabetic and non-diabetic AF patients, without a history of HF. Baseline T2DM status was inferred from medical history, haemoglobin A1c levels and oral glucose tolerance test. Study outcome was the first hospital admission or emergency department treatment for new-onset HF during the prospective follow-up. The phenotype of new-onset HF was determined by echocardiographic exam performed following clinical stabilisation (at hospital discharge, or within a month after HF diagnosis). HF phenotype was defined as HFrEF (left ventricular ejection fraction [LVEF] <40%), HFmrEF (LVEF 40–49%) or HFpEF (LVEF ≥50%). Cox regression analyses adjusted for age, sex, baseline LVEF, comorbidities, smoking status, alcohol intake, AF type (paroxysmal vs. non-paroxysmal) and T2DM treatment was used to analyse the association between T2DM and incident HF.

Results: Among 1,288 AF patients without prior HF (mean age: 62.1±12.7 years; 61% male), T2DM was present in 16.5%. Diabetic patients had higher mean baseline LVEF compared with nondiabetic patients (50.0±6.2% vs. 57.6±9.0%; P<0.001). During the median 5.5-year follow-up, new-onset HF occurred in 12.4% of patients (incidence rate, 2.9; 95% confidence interval [CI], 2.5–3.3 per 100 patient-years). Compared with non-diabetic patients, those with T2DM had a hazard ratio of 2.1 (95% CI, 1.6–2.8; P<0.001) for new-onset HF, independent of baseline LVEF or other factors. In addition, diabetic patients had a significantly greater decline in covariate-adjusted mean LVEF (–10.4%; 95% CI, –9.8% to –10.8%) at follow-up, compared with nondiabetic patients (–4.0%; 95% CI, –3.8% to –4.2%), P<0.001. The distribution of HF phenotypes at follow-up is presented in Figure. Among patients with T2DM, HFpEF (56.9%) was the most common phenotype of HF, whereas in patients without T2DM, HF mostly took the phenotype of HFpEF (75.0%).

Conclusions: T2DM is associated with an independent risk of new-onset HF in patients with AF and confers a greater decline in LVEF compared to individuals without T2DM. HFpEF was the most prevalent presenting phenotype of HF in AF patients with T2DM.

