Smokers paradox or not in heart failure. Just quit

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This editorial refers to ‘A smoker’s paradox in patients hospitalized for heart failure: findings from OPTIMIZE-HF†’ by G.C. Fonarow et al., on page 1983

Fonarow et al. have explored the ‘smoker’s paradox’ in a novel inception cohort of hospitalized heart failure patients included in the OPTIMIZE-HF registry, by determining the impact of current or recent smoking on outcomes during hospitalization and in the first 60–90 day period following hospital discharge.1 Adjustments for known important potential confounders were performed. Smokers were more likely to have left ventricular systolic dysfunction (59 vs. 51%), were more likely to be male (61 vs. 46%), and had higher B-type natriuretic peptide (BNP) levels (895 vs. 789 pg/mL). However, smokers were much younger (63 vs. 75 years of age), were less likely to have a previous history of heart failure admission (84 vs. 89%), and were less likely to have a diagnosis of diabetes requiring insulin therapy (14 vs. 17%). Despite this, when compared with non-smokers, the risk of in-hospital mortality was ~30% lower for current or recent smokers, while the risk for post-discharge mortality was similar between the two groups.

How could this be? Assuming the authors appropriately assembled an inception cohort, correctly classified smokers, and appropriately adjusted for important potential confounders, only one of two conclusions can be drawn: either smoking is somehow ‘protective’ in heart failure patients; or a substantial component of the adverse effects of smoking in heart failure is reversible and has a short half-life (i.e. days) so that ‘enforced’ in-hospital smoking cessation led to an imbalance of beneficial therapies in the smokers (usual therapy plus smoking cessation) compared with their non-smoking cohorts (usual therapies only). The former hypothesis (that smoking is protective) can probably be refuted immediately. There is overwhelming evidence that smoking increases the incidence of heart failure,2 as well as heart failure morbidity and mortality (including sudden death) for those who have heart failure.3,4

It can be argued that there is substantially more merit in the second argument. Constituents of inhaled tobacco damage the cardiovascular system in numerous ways: producing endothelial and platelet dysfunction; enhancing coagulation; increasing heart rate and blood pressure; increasing myocardial oxygen demand and consumption; and inducing vasoconstriction.5,6 Cigarette smoking also significantly increases carboxyhaemoglobin production, impairing oxygen carriage and release, which has a negative inotropic effect, increasing left ventricular end-diastolic pressure.6 Thus the abrupt removal of these deleterious effects of cigarette smoking following hospital admission and enforced smoking cessation, in addition to the provision of the usual heart failure therapies in smokers, might well be expected to ‘outstrip’ the impact of usual therapies alone in non-smokers.

In examining the assumptions mentioned above, it is important to note that the authors classified smokers as ‘having smoked anytime during the previous year’—according to convention set by the Joint Commission.7 This classification has the potential to categorize individuals who were actively smoking on the day of admission and those who had not smoked for 364 days as ‘smokers’. Those who had not been active smokers for many months would not have been able to experience the benefit of enforced in-hospital smoking cessation, yet they were still younger than their non-smoking cohorts, and thus would have been predicted to enjoy the prognostic benefits from being almost a decade younger. The problems associated with the mis-classification of smoking status in cohort studies have been noted before. The lack of validated smoking status at follow-up raises the potential for misclassification. The inability to quantify previous smoking exposure (cigarettes per day, pack-years, etc.) also clouds the current analysis. Although we do not know the proportion of individuals classified as smokers who quit within the year prior to admission, the fact that the 60–90 day post-discharge event rate was similar between individuals classified as smokers compared with non-smokers supports the notion that the short-term beneficial effects of enforced in-hospital smoking cessation had run their course, with those who benefitted from it having done so already (and expressed as lower incidence of death or shorter hospital stay), leaving the remaining smokers...
(some of whom might have resumed smoking post-discharge) and the non-smokers with similar benefits from ‘usual therapies’. Conversely, the decade younger age of smokers was not able to account for a continued benefit on outcome (compared with non-smokers) once the acute benefits of smoking cessation had worn off. More importantly, post-index hospitalization follow-up was only possible in the 35% of the cohort who provided appropriate consent. It is therefore extremely challenging to generalize from these results, particularly given that post-discharge smoking status was not available.

Although the authors adhered to accepted statistical methods in adjusting for potential confounders and covariates, this may not have been sufficient. For example, in the CHARM programme, increasing age [hazard ratio (HR) 1.73 per decade over 60 years of age] was a more powerful predictor of death than decreasing ejection fraction (HR 1.13 per 5% decrease below 45); yet both appear to be similarly weighted as continuous predictor variables in the current study. It is possible that using additional procedures to account for the differences in covariates such as propensity matching might have accounted for the differences in outcomes between smokers and non-smokers, but this remains unclear.

It could be argued that the main ‘take home’ message from this study is that while smoking is associated with earlier onset and more severe symptoms of heart failure, smoking cessation at the time of hospitalization for heart failure probably has a powerful beneficial effect on heart failure outcomes while in hospital, and thus systematic efforts should be made while patients are in hospital to identify smokers and begin cessation counselling immediately.

While smoking cessation is recommended by national organizations involved in the care of cardiac patients,10–12 these guidelines do not specifically address the issues related to smoking cessation for patients hospitalized for acute heart failure. In general, it has been noted that the cardiovascular community has been slow to adopt integrated, systematized approaches to smoking cessation immediately.

The systematic identification and treatment of all admitted patients according to an established protocol offers promise in enhancing the quality of care provided to cardiac patients. One such model with demonstrated efficacy is the ‘Ottawa Model’. The Ottawa model, which ensures the identification and documentation of the smoking status of all admitted patients, also mandates the provision of counselling, pharmacotherapy where appropriate, and a 6-month post-discharge follow-up using sophisticated telephone technology; it has been associated with a 15% absolute increase in self-reported cessation 6 and 12 months post-discharge. Systematic approaches to cessation in an acute coronary syndrome population have been associated with a >40% decrease in subsequent re-hospitalization and a 70% reduction in all-cause mortality.16 The benefits of smoking cessation in the treatment of heart failure are clear.4 The provision of smoking cessation treatment to all of our cardiac patients in a systematic, organized manner at the time of their admission is long overdue.

Conflict of interest: none declared.

References