Chest pain in patients with heart failure: why history may matter

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This editorial refers to ‘Relationship between angina pectoris and outcomes in patients with heart failure and reduced ejection fraction: an analysis of the Controlled Rosuvastatin Multinational Trial in Heart Failure (CORONA), by A.A. Badar et al. on page doi:10.1093/eurheartj/ehu342

Recording a patient’s medical history and being able to describe fully the so-called ‘patient story’ is becoming a lost art. Many patients are in need of care; in conjunction with this reality comes the pressure to make diagnoses and consequent treatment decisions rapidly. As a result of these pressures, essential diagnostic elements, such as recording a patient’s medical history, get left by the wayside. Companies such as Google, International Business Machines (IBM), and others are investing heavily in organizing information technology that will address the diagnostic challenges in medicine. Furthermore, these companies are striving to find novel ways of integrating medical information to allow for better decision-making. Yet, despite these technical advancements, medical professionals must remain mindful that it is very often the simplest question that informs the most crucial decision. For example, asking ‘Do you have chest pain (CP)?’ could assist in managing heart failure (HF). Interestingly, clinicians treating HF patients have typically focused on symptoms other than CP, such as congestion, dyspnoea, and oedema, counselling patients to monitor their fluid levels, watch their sodium intake, titrate their diuretics, and weigh themselves daily. But what about CP? Investigators from the Controlled Rosuvastatin Multinational Trial in HF (CORONA) now bring our focus back to this important (but often subordinate) symptom.

The origin of CP in patients with HF is probably multifactorial. There are obvious mechanisms such as left ventricular ischaemia from segmental atherosclerotic narrowing of an epicardial coronary artery, but there are less obvious factors that may also play a role, such as subendocardial ischaemia in a hypertrophied ventricle and right ventricular ischaemia in secondary pulmonary hypertension. Regardless of the mechanism, CP is common, reported in 21–25% of HF patients (who had HF of both ischaemic and non-ischaemic aetiologies) enrolled in clinical trials. At our institution, patients with HF undergoing a cardiac catheterization frequently report CP. In fact, even patients with HF of a coronary angiogram-determined non-ischaemic aetiology report a past history of CP 40% of the time. In the CORONA study, at the time of enrolment, investigators asked patients aged ≥ 60 years with symptomatic ischaemic HF with reduced ejection fraction (HFrEF) whether they had CP on a scale of 0–4 ‘during the past few days’: 46% of these patients answered ‘yes’. The authors found that patients who experienced recent CP had worse outcomes during the trial follow-up. Specifically, patients were at increased risk of acute coronary syndromes, coronary revascularization procedures, and HF hospitalizations, though somewhat surprisingly there was no association with increased cardiovascular or all-cause mortality. These findings of increased morbidity, but no associated increase in all-cause mortality, are consistent with other observational reports of patients with ischaemic HF. Yet, given the association between CP and consecutive downstream morbidity, there seem to be logical opportunities for improving patient outcomes through interventions that target CP. The history of studying treatments for stable coronary disease in other patient populations (i.e. those without HF) is a reminder to focus on understanding the benefits of medical therapy in addition to revascularization. With this in mind, we outline key unanswered questions for the medical management of these patients (Figure 1). To improve symptoms, current HF guidelines recommend medical therapy (beta-blockers and nitrates) and revascularization procedures for coronary artery disease patients with CP. Yet there is little observational or clinical trial evidence available comparing CP therapies in this patient population, thus many questions remain. What is the optimal medical regimen for patients with HF with preserved ejection fraction where the benefits of beta-blockers and nitrates are not established? Are ranolazine or other medical therapies for angina effective among patients with HF? Is there a role for device therapies such as chronic vagal nerve stimulators? Furthermore, even though not addressed in the current CORONA analysis, the prevalence of CP in patients without ischaemic cardiomyopathy raises important questions. What are the mechanisms causing these symptoms? Are patients with CP at increased risk? Can treatments for CP in patients with non-ischaemic cardiomyopathy improve outcomes?

In summary, these repeated observations of poor outcomes in patients with HF and CP expose the need for prospective studies to define the best strategy of care for this common problem. Clinical trials using a pragmatic study design would be well suited to address these questions as many of the potential therapies are...
available for other indications. As the cardiology field and the care of patients with HF become increasingly technologically oriented, and we are encouraged to use high-end technology for both diagnostic and treatment decisions, we must not forget the patient story and that the simplest questions very often provide the clearest answers.

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References


Figure 1 Chest pain (CP) in patients with heart failure (HF). This figure displays potential mechanisms for CP in patients with HF and key unanswered questions regarding treatment strategies for patients with HF and CP. LVEF, left ventricular ejection fraction.