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PROGNOSTIC SIGNIFICANCE OF PLASMA CONCENTRATIONS OF PROCALCITONIN IN PATIENTS WITH SUSPECTED HEART FAILURE

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Background Heart failure is associated with an increase in plasma concentrations of markers of inflammation and fibrosis. Some have hypothesised that this may reflect absorption of endotoxins from gut bacteria or bacterial translocation into the gut wall. This might provoke an increase in markers of infection, such as procalcitonin (PCT).

Methods Patients with suspected heart failure referred from the local community to a specialist clinic were invited to participate. Consenting patients underwent a systematic evaluation including prior medical history, medications, symptoms, signs, electro- and echocardiograms, standard haematology and biochemistry profiles and measurement of PCT and amino-terminal pro-brain natriuretic peptide (NT-proBNP).

Results Of 1891 patients enrolled, the median age was 72 years (IQR 64–78), 669 were women, 807 had left ventricular systolic dysfunction (LVSD), 400 had no major echocardiographic abnormalities other than LVSD, 192 had no major echo abnormality but an NT-proBNP >400 ng/l (of whom 65 had atrial fibrillation and 15 had eGFR <30 ml/min) and 492 had none of the above. Median (IQR) PCT overall was 22(17–47) pg/ml and for each of the four sub-groups was 23(18–32), 22(17–31), 24(19–35) and 20 (16–25)pg/ml respectively. Of patients with LVSD, 698 had a second blood sample taken after 12 months follow-up. Paired values in this subset were 22(18–29) pg/ml at baseline and 22(17–29) pg/ml at follow-up. The correlation coefficient between log (PCT) at baseline and follow-up is r=0.63; p<0.0001, suggesting relatively stable concentrations over time. Over a median follow-up of 5.0 (IQR 3.4–7.1) years, 783 patients died, 447 of cardiovascular causes. In univariable analysis, log(PCT) was strongly related to all-cause, cardiovascular and non-cardiovascular mortality (HR 1.91 with 95% CI (1.73 to 2.11), HR 1.94 (1.71–2.21) and HR1.89 (1.61–2.22) respectively, p<0.001 for all). In a multivariable Cox regression model, PCT provided additional prognostic information to 17 standard clinical variables (age, sex, aetiology, diabetes, COPD, symptoms, quality of life, NYHA, BMI, heart rate and rhythm, systolic blood pressure, oedema, severity of ventricular dysfunction, haemoglobin and creatinine and NT-proBNP) for all-cause mortality.

Conclusions PCT is similarly elevated in all heart failure phenotypes studied and adds prognostic information to standard variables. Whether it is just a marker of risk or sits on an important pathway driving the progression of heart failure awaits further study.