Background Plasma volume (PV) expansion is a hallmark feature of worsening heart failure that is notoriously underestimated by clinical examination. While radioisotope assays optimally quantify PV status, numerous haemodialysis-based equations also exist for its estimation. The prognostic utility of such formulas in chronic heart failure (CHF) is unknown.

Methods We analysed the relation between estimated PV status and mortality in 246 outpatients with CHF (mean ± SD age 67 ± 13 years, NYHA class 2 ± 1, LVEF 28 ± 8%). PV status was calculated (Hakim RM, et al) by subtracting the patients actual PV ((1-haematocrit) × (a + (b × weight)); a and b are gender-specific constants) from their ideal PV (c × weight); c=gender-specific constant.

Results Median (±IQR) PV status was —261 ± 550 ml with 78% and 21% of patients having PV contraction and expansion, respectively. Patients with PV excess had significantly higher creatinine and lower albumin levels. Over a median follow-up of 15 ± 16 months, 36 (15%) patients died. PV status predicted mortality (HR 1.001, 95% CI 1.001 to 1.002, p = 0.0007) and conferred a 75% reduced hazard for death (HR 0.25, 95% CI 0.07 to 0.87, p < 0.0001, Abstract 104 figure 1B).

Conclusions Calculating plasma volume status in CHF patients appears prognostically useful and suggests that dehydration is better tolerated than volume excess in these individuals and that targeting therapy to achieve a plasma volume status ≤178 ml might increment survival.

BCS Abstracts 2011

104 PROGNOSTIC UTILITY OF CALCULATED PLASMA VOLUME STATUS IN CHRONIC HEART FAILURE

doi:10.1136/heartjnl-2011-300198.104

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Background Brain natriuretic peptides have been shown to be reliable indicators of left ventricular failure and markers of risk in cardiac disease. However, patients with chronic obstructive pulmonary disease (COPD) are also known to have elevated concentrations of brain natriuretic peptides in the absence of overt cardiac disease, likely due to right ventricular strain. This has been shown to have prognostic value and has a potential role in the management of the condition; for example, it has been suggested that it could be used to guide the initiation of non-invasive ventilation. The aim of this study was to identify clinical and echocardiographic determinants of the polypeptide N-terminal pro-Brain Natriuretic Peptide (NT pro-BNP) in patients with stable COPD.

Method Arterial blood gases, plasma NT pro-BNP and transthoracic echocardiographic parameters were studied in 140 patients with stable COPD attending a respiratory outpatient clinic.

Results Of the 140 patients, 65 (46%) were male, 26 (19%) received home oxygen therapy, 115 (82%) were current smokers, 38 (27%) were prescribed diuretics and 15 (11%) had a left ventricular ejection fraction <45%. Patients with cor pulmonale (n = 6) were more likely to have left ventricular systolic dysfunction (p < 0.001), reduced tricuspid annular plane systolic excursion (p = 0.017) and higher pulmonary artery systolic pressures (p = 0.01). The median (IQR) NT pro-BNP concentration was 16.2 (25.4) pmol/l. Concentrations were significantly higher in those with a dilated left atrium, aortic stenosis, left ventricular systolic dysfunction, right ventricular impairment, atrial fibrillation and those prescribed diuretics and ACE inhibitors. Significant predictors of NT pro-BNP were a dilated left atrium, aortic stenosis and left ventricular systolic dysfunction. NT pro-BNP was an excellent discriminator of RV impairment (C statistic = 0.90).

Conclusions NT pro-BNP readily identifies patients with stable COPD who have right ventricular dysfunction. However, several other clinical variables also associated with increased NT pro-BNP concentrations are prevalent in this population. This is likely to confound clinical decision making.

106 CHF PATIENTS ARE VITAMIN D DEFICIENT AND HYPERPARATHYROID, WITH LEVELS OF EACH RELATED TO MARKERS OF SEVERITY

doi:10.1136/heartjnl-2011-300198.106

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Background The vitamin D-parathyroid (PTH) axis is increasingly recognised as potentially being involved with many of the features of the syndrome of CHF. We wanted to explore the relationship between vitamin D and PTH levels in a group of CHF patients and relate these to markers of severity.

Methods We analysed serum 25(OH) vitamin D3 levels in 406 consecutive attendees of the Leeds Advanced Heart Failure clinic (510 men) and correlated these to clinical markers of severity.

Results Mean age (SE) was 69 (5) years, mean left ventricular ejection fraction (LVEF) 31 (2)%), mean serum creatinine 117 ± 24 µmol/l (2.4), median vitamin D levels (IQR) 30 (20–45) nmol/l (normal for skeletal health >75 nmol/l) and mean parathyroid levels 8.8 (6.2–13.5) pmol/l (normal <6.5 pmol/l). Aetiology was ischaemic...
heart disease in 63% and 23% had diabetes mellitus. Patients were optimally treated (84% on β-blockers, 88% on ACE inhibitors, and 46% on spironolactone). The mean daily dose of furosemide was 60 (3) mg. Very few patients (5%) were sufficient in vitamin D. Patients with worse symptoms as measured by NYHA status had lower vitamin D levels than non-diabetics (p=0.001) and there was a negative correlation between vitamin D and fasting glucose levels (r=0.15; p=0.02). There was no relationship between vitamin D levels and age, calcium, creatinine or CRP, and no differences between those patients taking and those not taking β-blockers and ACE inhibitors. In 8 unselected patients we found a negative relationship between tumour necrosis factor-alpha (TNF-α) levels and vitamin D (r=0.62; p=0.05). Although there was no relationship between vitamin D levels and baseline LVEF, in a subgroup of 150 patients followed up one year after titration to optimal CHF therapy, there was a significant positive relationship between change in LV dimensions and vitamin D levels at the time of the baseline scan (p<0.05).

Conclusions The vitamin D-PTH axis is abnormal in CHF, related to the severity of the condition. Our data suggest that reverse remodelling in response to optimal drug titration is greater in those with higher vitamin D levels. Whether vitamin D deficiency is causally related to CHF remains unknown and requires a long-term, randomised, placebo-controlled study in CHF patients with efficacy and mechanistic outcomes, using a dose of vitamin D capable of normalising both vitamin D and PTH levels.

Background Red cell distribution width (RDW) is a surrogate of many aberrations (inflammation, malnutrition, iron deficiency (ID)) that may drive chronic heart failure (CHF) progression. While an elevated RDW and iron deficiency at baseline predict mortality in CHF, little is known about the prognostic implications of their temporal trends.

Methods We analysed the relation of red cell indices on first consultation and over time with mortality in 274 outpatients with CHF (mean (±SD) age 70±14 years, LVEF 28±8%, NYHA class 2±1, 54% ischaemic). The combination of a rising RDW and a falling mean cell volume (MCV) identified evolving ID.

Results On initial consultation, an RDW >15%, Hb<12.5 g/dl, and MCV <80 fl were evident in 41%, 46%, and 8% of patients. Over a median (±1QR) follow-up of 15±17 months, 60 (22%) patients died. On Cox proportional hazards analyses, a higher RDW independently predicted increased mortality (HR 1.21, p<0.0001). Over time, 51%, 55%, 40%, and 23% of patients had a rise in RDW, a fall in Hb, a fall in MCV, and evolving ID, respectively. A rising RDW predicted death (HR 1.18, p=0.002) independently of baseline RDWs and changes in Hb, with an absolute increase >1% conferring a twofold escalated risk of mortality (Abstract 107 figure 1A). Evolving ID was also associated with poorer survival (HR 2.89, p<0.0001, Abstract 107 figure 1B).