Dyssynchrony predicts better survival in low EF groups

**Conclusion**

Dyssynchrony (r²/C0=0.4, p<0.001) was associated with a worse prognosis (p<0.05). All dyssynchrony markers were higher in survivors (p<0.001 by sign test, upper panel). EF was not prognostic and depressed by dyssynchrony (r=-0.4, p<0.001). By examining all patients (regardless of EF); the association between dyssynchrony and better survival disappeared (p>0.05, lower panel). EF was restored to its prognostic significance (p=0.02). Taking a different approach to define poor ventricular function—using low S-wave velocity—EF had prognostic significance (p<0.05) and dyssynchrony markers were non-prognostic (p>0.05).

**Conclusion**

Dysynchrony predicts better survival in low EF groups because dyssynchrony artifically lowers EF without damaging survival. The effect is independent of CRT. Replacement of EF with dysynchrony-neutral measures of LV function, for example, peak S-wave velocity would avoid the appearance that dyssynchrony is favourable.

**010**

MULTICENTRE VALIDATION OF THE ADVERSE PROGNOSTIC IMPLICATIONS OF DECLINING SERUM ALBUMIN LEVELS IN CHRONIC HEART FAILURE

**Background**

Single-centre studies have shown that a low serum albumin at baseline forecasts enhanced mortality in chronic heart failure (CHF) possibly because it reflects aberrations (eg, inflammation, impaired nutrition, plasma volume expansion) that can exacerbate disease. We hypothesised that attenuations in serum albumin over time would be prognostically more ominous than baseline values, and would be so even in a multicentre setting.

**Methods**

We analysed the survival implications of baseline albumin and Δalbumin in a derivation cohort of 246 CHF outpatients (mean [±SD] age 63±12 years, LVEF 29±8%, 48% NYHA class >2) from University College London Hospital and then in a validation cohort of 148 CHF outpatients (age 69±12 years, LVEF 28±10%, 41% NYHA class >2) from Imperial Healthcare (St Marys Hospital and Hammersmith Hospital, London).

**Results**

In the derivation cohort, 51 (21%) patients died over 13 months. Baseline albumin independently predicted mortality (HR 0.89, 95% CI 0.84 to 0.94, χ²=18, p<0.0001). However, Δalbumin (unadjusted HR 0.89, 95% CI 0.84 to 0.92, χ²=53, p<0.0001) was even more predictive (Difference in ROC AUC for baseline vs Δalbumin 0.16, χ²=18, p<0.0001). In the validation cohort, 43 (30%) patients died. Δalbumin (unadjusted HR 0.89, 95% CI 0.86 to 0.92, χ²=44, p<0.0001) was again prognostically superior to baseline albumin with a fall >6 g/l predicting an sixfold increased risk (HR 5.64, 95% CI 3.08 to 10.31, χ²=35, p<0.0001). Addition of Δalbumin to the strongest four variable model (baseline albumin, NYHA class, Δurea, Δhaemoglobin) dramatically augmented the χ² value (45 vs 84, p<0.0001). In incremental prognostic analyses, the addition of Δalbumin to the strongest four variable model (baseline albumin, NYHA class, Δurea, Δhaemoglobin) dramatically augmented the χ² value (45 vs 84, p<0.0001). In the validation cohort, 43 (30%) patients died. Δalbumin (unadjusted HR 0.89, 95% CI 0.86 to 0.92, χ²=44, p<0.0001) was again prognostically superior to baseline albumin with a fall >6 g/l predicting an sixfold increased risk (HR 5.64, 95% CI 3.08 to 10.31, χ²=35, p<0.0001). Addition of Δalbumin to the strongest three variable model (baseline red cell distribution width, Ared cell distribution width, Δurea) also augmented the χ² value (51 vs 65, p<0.001).

**Conclusions**

A fall in serum albumin over time consistently predicts an amplified risk of death in systolic CHF and enables simple and cheap risk stratification.

**011**

HBA1C AND MORTALITY IN DIABETIC INDIVIDUALS WITH HEART FAILURE: AN OBSERVATIONAL COHORT STUDY

**Background**

Controversy exists regarding the importance of glycaemic control in patients with type 2 diabetes mellitus (T2DM) and chronic heart failure (CHF) based on conflicting reports that had used a single baseline HbA1c.