COPEPTIN AS A SURROGATE FOR VOLUME STATUS AND SERIAL MONITORING IN HEART FAILURE

Poster Contributions
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Background: Volume status assessment is critical in heart failure (HF). Vasopressin (AVP), is known to be upregulated when volume status is elevated. Copeptin, the C-terminal portion of the precursor of vasopressin, is a measurement of AVP. We evaluated copeptin in dyspneic patients and serial measurements in decompensated HF.

Methods: The BACH trial was a prospective study of 1641 patients presenting with dyspnea; 562 were diagnosed with decompensated HF.

Results: In all-comers, log copeptin was elevated in subjects with physical exam findings consistent with hypervolemia including elevated jugular venous pressure (1.43 +/- 0.5 vs 1.09 +/- 0.5, p<0.001), rales (1.37 +/- 0.5 vs 1.05 +/- 0.5, p<0.001), and edema (1.33 +/- 0.5 vs 1.06 +/- 0.5, p<0.001). In those with HF, copeptin demonstrated a 35.6% decrease from admission to 48 hours. Those diagnosed with HF (65.7%) whom had a decreasing copeptin also had their weight on admission to discharge decreased: 79.58 + 23.88 kg to 74.91 + 19.40 kg (p<0.001). Those with a rising copeptin did not have significant weight loss and an increased incidence of 90-day mortality: 15.4% vs 9.4% (p<0.0001). Kaplan-Meier analysis demonstrated an improved survival (p=0.021) in those with a decreasing copeptin (Figure 1).

Conclusion: Copeptin is elevated in dyspneic patients who are hypervolemic and decreases in copeptin correlates with weight loss and a decreased risk of mortality. Studies utilizing copeptin to assess euvolemia prior to discharge may assist in HF management.