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 **CARDIAC FUNCTION AND HEART FAILURE****DOES OBESITY AFFECT RESPONSE TO TREATMENT IN ACUTE DECOMPENSATED HEART FAILURE? A DIURETIC OPTIMIZATION STRATEGIES EVALUATION (DOSE) TRIAL SUBSTUDY**

ACC Oral Contributions

Ernest N. Morial Convention Center, Room 243

Tuesday, April 05, 2011, 8:45 a.m.-9:00 a.m.

Session Title: Novel Strategies in the Management of Heart Failure

Abstract Category: 21. Myocardial Function/Heart Failure—Clinical Pharmacological Treatment

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Authors: *Achal Gupta, Josef Stehlik, Steven McNulty, Kerry L. Lee, Edward M. Gilbert, Deborah Budge, Kevin Anstrom, David A. Bull, G. Michael Felker, Divya R. Verma, Stavros G. Drakos, Theophilus Owan, Christopher M. O'Connor, Sheldon E. Litwin, University of Utah School of Medicine, Salt Lake City, UT, Duke Clinical Research Institute, Durham, NC*

Background: Obesity is associated with an increased prevalence of heart failure. However, little is known about how obesity affects the response to treatment in acute heart failure (AHF).

Methods: DOSE trial randomized patients (pts) with AHF (n= 308) to low vs. high dose diuretic therapy. We examined the baseline characteristics of obese and non-obese pts and assessed the effect of obesity on clinical end points.

Results: Median BMI was 31.6 (25-75% = 26.8-36.7). Obese (BMI > 30; n=174) and non-obese (n=118) pts had similar gender distribution, qualifying furosemide dosage, HF hospitalization in past 12 months, ischemic etiology, history of atrial fibrillation, baseline drug treatment and serum creatinine (Cr; 1.44 vs. 1.40, p=0.97). However, obese pts were younger (66 vs. 71 years, p<0.01), more often diabetic (61 vs. 37%, p<0.0001), had higher systolic BP (119 vs. 111, p<0.01) and lower NT pro BNP levels (3404 vs. 7491, p< 0.01). While mean LVEF was higher in obese pts (37 vs. 32%), the proportion of pts with LVEF >50% was similar between the 2 groups (23% vs. 31%, p=0.15). Obese pts were not different than non-obese in terms of overall symptoms at 72 hours expressed as Global Visual Analog Scale (VAS) area under the curve (AUC) and 72 hr dyspnea VAS AUC, freedom from congestion, time to discharge and change in BNP. Obese pts had greater volume loss at 72 hours (p<0.01), but no interaction between obesity and high vs. low dose treatment strategy. Obese pts had an increase in serum Cr at 72 hours (+0.09 vs. -0.006, p<0.01), higher incidence of cardiorenal syndrome (22.2% vs. 12.3%, p=0.04) and more frequent clinical treatment failures (46.8% vs. 28.7%, p<0.01).

Conclusions: In a modern cohort of pts with AHF, over half were obese. There was no difference in the primary endpoint of Global VAS AUC and Dyspnea VAS AUC in obese vs. non-obese pts. However, the obese pts had greater incidence of acute worsening of renal function and were more likely to experience treatment failure, even though they had a greater net volume loss at 72 hours. Further work is needed to understand how the rapidly increasing prevalence of obesity affects the response to HF treatments and which treatment approaches in the obese may improve clinical outcomes.