SERUM BYPRODUCTS OF COLLAGEN SYNTHESIS ARE NOT ELEVATED IN PATIENTS WITH MYOCARDIAL FIBROSIS DUE TO HYPERTROPHIC CARDIOMYOPATHY

ACC Moderated Poster Contributions
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Purpose: Histopathologic studies of patients with hypertrophic cardiomyopathy (HCM) have demonstrated a global increase in interstitial myocardial fibrosis. Serum byproducts of collagen synthesis, such as procollagen types I and III N-terminal propeptide (P1NP and P3NP), are elevated in active pro-fibrotic states. We measured P1NP and P3NP in peripheral arterial and coronary sinus serum of HCM patients with documented myocardial fibrosis on cardiac magnetic resonance imaging (CMR) and investigated their relationship with diastolic dysfunction.

Methods: We performed simultaneous peripheral arterial and coronary sinus sampling on 19 subjects - 10 with asymmetric septal hypertrophy due to HCM (90% male, mean age 54±12 years) and 9 healthy controls. Levels of P1NP and P3NP were determined from both sample sites. CMR with gadolinium contrast was performed on all HCM patients to confirm the presence of myocardial fibrosis. Diastolic function was assessed by transthoracic echocardiography (including septal, lateral and mean E/e').

Results: No significant differences were observed between the HCM and control groups in the levels of P1NP ( peripheral arterial 41.4±14.1 vs. 42.0±17.0 μg/L, p=0.94; coronary sinus 39.6±12.6 vs. 41.6±16.8 μg/L, p =0.78) or P3NP (4.57±0.68 vs. 4.25±0.66 μg/L, p=0.33; coronary sinus 4.76±0.68 vs. 4.43±0.86 μg/L, p=0.42). Additionally, levels did not significantly differ according to the site of sampling. While HCM patients had a significantly higher mean E/e’ than controls (11.6±2.8 vs. 8.1±2.2, p=0.01), P1NP and P3NP levels did not correlate with this measure of diastolic function.

Conclusions: Serum markers of collagen synthesis are not elevated in patients with HCM despite the presence of myocardial fibrosis and diastolic dysfunction. This suggests that active pro-fibrotic states in HCM may either be relapsing-remitting, or occurring at an earlier time in the disease process.