

 **Heart Failure****MYOCARDIAL ISCHEMIA ON ELECTROANATOMICAL MAPPING IS ASSOCIATED WITH IMPAIRED BONE MARROW CD34+ CELL RESERVE IN PATIENTS WITH NON-ISCHEMIC DILATED CARDIOMYOPATHY**

Poster Contributions

Poster Sessions, Expo North

Saturday, March 09, 2013, 10:00 a.m.-10:45 a.m.

Session Title: Dilated Cardiomyopathies: From Peripartum, Cancer Therapy, Familial Cardiomyopathies to Cardiac Amyloidosis

Abstract Category: 15. Heart Failure: Clinical

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Authors: *Bojan Vrtovec, Gregor Poglajen, Matjaz Sever, Peter Cernelc, Francois Haddad, Joseph C. Wu, Advanced Heart Failure and Transplantation Ctr, Ljubljana, Slovenia, Stanford University School of Medicine, Stanford, CA, USA*

Background: The underlying mechanisms for impaired vasculogenesis in patients with non-ischemic dilated cardiomyopathy (DCM) remain unclear. We investigated a potential relation of myocardial ischemia and inadequate bone marrow CD34+ cell reserve in this patient cohort.

Methods: We performed electroanatomical mapping in 40 patients with non-ischemic DCM, LVEF<40%, and NYHA Class≥II heart failure. Myocardial areas with unipolar voltage (UV)≥9 mV and linear shortening (LLS)≥ 6% were considered normal. Those with UV≥9mV and LLS<6% were defined as ischemic, and those with UV<9 mV and LLS<6% were defined as scar. All patients underwent 5-day bone marrow stimulation (G-CSF, 5 µg/kg b.i.d.). After stimulation, peripheral blood CD34+ cell count was measured and poor CD34+ mobilization was defined as CD34+count<20 million/L.

Results: Poor CD34+ mobilization was present in 13/40 (33%) patients. Poor and good mobilizers did not differ in age (59±7 years vs. 56±9 years, P=0.28), gender (male: 84% vs. 85%, P=0.96), LVEF (26.1±8.1% vs. 29.4±7.1%, P=0.29), LVEDD (6.9±1.6 cm vs. 6.8±0.8 cm, P=0.64), serum creatinine (88±27 µmol/L vs. 86±20 µmol/L, P=0.81), bilirubine (19±12 µmol/L vs. 18±8 µmol/L, P=0.70), NT-proBNP (3442±4794 pg/mL vs. 2861±2768 pg/mL, P=0.71), TNF-alpha (4.1±2.0 pg/ml vs. 3.9±3.1 pg/ml, P=0.56) or interleukin-6 (4.9±3.1 pg/ml vs. 4.5±2.9 pg/ml, P=0.42). Global myocardial viability was comparable in poor and good mobilizers (UV: 8.72±2.12 mV vs. 9.15±2.17 mV, P=0.23). However, poor mobilizers displayed significantly higher proportion of ischemic myocardial segments (3.67±0.89 vs. 1.63±0.56 in good mobilizers, P=0.001) and segments with scar (2.01±0.70 vs. 1.10±0.32 in good mobilizers, P=0.03). We found no difference in the distribution of ischemic segments between the groups (anterior/anteroseptal region: 38% in poor mobilizers vs. 35% in good mobilizers; lateral region: 27% vs. 31%; infero-posterior region: 35% vs. 34%, P=0.89).

Conclusions: In patients with non-ischemic DCM, myocardial ischemia is associated with inadequate bone marrow mobilization of CD34+ cells. Thus, impaired bone-marrow stem cell reserve could contribute to defective vasculogenesis in DCM patients.