

**Cardiac Function and Biomarkers in Patients with Recoverd Peripartum Cardiomyopathy**  
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Background: Peripartum Cardiomyopathy (PPCM) is a rare cardiomyopathy and many questions regarding pathophysiology remain unanswered. Altered endothelial function and heightened oxidative stress in pregnancy may contribute to the development of PPCM. In addition, LV recovery does not guarantee normal function at subsequent pregnancies. We attempted to evaluate whether residual myocardial injury may be detected by comprehensive echo techniques like Tissue Doppler (TDI) and 2D Strain (2DS) imaging in post PPCM subjects.

Methods: We evaluated 13 women (age  $36\pm 6$  years) 11 of whom with complete LVEF recovery. Both LV and RV function assessment using standard echo, TDI, 2DS and stress echo were evaluated. The number of EPCs (CD34 and CD34/KDR), VEGF, hsCRP, IL 6, and Ox LDL antibody serum levels were quantified. Stress echo was performed in 9 patients. All biomarkers where compared to 11 age-matched controls.

Results: The mean LVEF at presentation was  $32.5\pm 8.8\%$ , at follow-up (3  $2\pm 20$  months) was  $58.6\pm 3.5\%$ . Compared to controls, patients with PPCM had a trend for lower systolic velocities on TDI (lateral  $7.9\pm 2.0$  vs.  $9.5\pm 1.9$ ,  $\rho=0.07$ , septal  $10.1\pm 1.9$  vs.  $11.2\pm 2.2$  ms,  $\rho=0.08$ ) and decreased global longitudinal strain ( $-20.5\pm 2.3\%$  vs.  $22.8\pm 2.2\%$ ,  $\rho=0.07$ ). No significant changes in the rest TDI indexes or circumferential strain were obtained. Circulating VEGF levels were significantly lower in PPCM group ( $1.41\pm 0.06$  vs.  $1.47\pm 0.03$  pg/ml,  $\rho=0.008$ ) compared to controls with no significant differences in EPCs, IL 6 and Ox LDL antibodies.

Conclusions: In this pilot study TDI and 2DS were able to identify some residual myocardial injury in patients post PPCM. This finding may lead to consider the use of these techniques to assess LV recovery when a subsequent pregnancy is desired. Attenuated production of VEGF even after the initial insult provides further insights into the pathophysiological mechanisms in PPCM, suggesting the contribution of endothelial dysfunction in the acute setting.