**PERIPARTUM CARDIOMYOPATHY: NEW CONCEPTS IN PATHOPHYSIOLOGY AND MANAGEMENT**

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Peripartum cardiomyopathy (PPCM) is an ididopathic cardiomyopathy presenting with heart failure (HF) due to left ventricular systolic dysfunction towards the end of the pregnancy or in the months following delivery, where no cause of HF is found. A Recent animal experimentation has shown that female mice with cardiomyocyte specific knockout of STAT3 developed PPCM. STAT3 activated by prolactin in late pregnancy is involved in protection of the heart from oxidative stress by up regulating oxidative enzymes such as Manganese superoxide dismutase (MnSOD) and in myocardial pro angiogenic effect. In the absence of STAT3 there is a decrease in LV capillary density and oxidative stress induced expression of cardiac cathepsin D that cleaves the nursing hormone Prolactin to a 16-kDa form. 16-kDa Prolactin causes Apoptosis, capillary dissociation, vasoconstriction and impaired myocyte metabolism that lead to myocardial dysfunction. Treatment with Bromocriptine, an inhibitor of prolactin, prevents the development of PPCM. Preliminary studies in women with PPCM demonstrated a significant reduction in mortality and improvement in left ventricular dysfunction as well as in symptoms of heart failure in patients treated with bromocriptine in addition to standard HF therapy compared to those who were treated with standard HF therapy alone. More information in larger number of patients will be required to establish the efficacy and safety of this novel therapy for women with PPCM.