injection of 2×10^6 MBSCs (MBSCs group) or equal volume of DMEM (MI group), or sham operation procedure (Sham group) for 7 days. Myocardial fibrosis was evaluated by picrosirius red staining followed by a quantitative analysis of collagen volume fraction (CVF), and changes of endothelial markers (CD31 and VE-cadherin), and markers of fibroblasts (FSP-1) and myofibroblasts (α-SMA) in heart tissue were detected by immunofluroence, immnobloting and real-time RT-PCR, respectively. Cells isolated from cardiac scar were quantitatively analysed with FACS in order to reveal the effect of MBSCs on cardiac fibroblasts produced through EndMT characterised by emerging of CD31+/α-SMA+ cells.

Results In comparison with MI group, MBSCs decreased CVF by about 5% ((14.6±1.8) % vs (19.8±2.1)%, $p<0.05$)), MBSCs preserved loss of endothelial markers (CD31 and VE-cadherin) and attenuated gain of markers of fibroblasts/myofibroblasts (FSP-1 and α-SMA) after MI, and reduced CD31+/α-SMA+ cells (EndMT) by about 10% ((30.9±2.6)% vs (19.9±1.8)%, $p<0.01$).

Conclusions MBSCs can attenuate cardiac fibrosis emerged after myocardial infarction; inhibition of EndMT is a protective mechanism of MBSCs treatment that contributes to improvement of cardiac remodelling after myocardial infarction.

GW23-e0312

MENSTRUAL BLOOD STEM CELLS ATTENUATE POST-INFARCTION MYOCARDIAL FIBROSIS VIA INHIBITING ENDOTHELIAL TO MESENCHYMAL TRANSITION (ENDMT)

doi:10.1136/heartjnl-2012-302920a.83

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Objectives Menstrual blood stem cells (MBSCs) have been reported to offer superior protective effects over bone marrow mesenchymal stem cells (MSCs) on cardiac performance lost after myocardial infarction. The exact protective mechanisms of MBSCs are still elusive. The present study aimed to investigate effects and relevant mechanisms of MBSCs on myocardial fibrosis that impairs heart function by inducing cardiac remodelling, decreasing myocardial compliance, and compromising normal electrical conduction.

Methods Eighteen 10-week old male SD rats randomised to 3 groups (n=6 each), they were subjected to left anterior descending coronary artery (LAD) ligation and treated with intramyocardial