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Blocking the Transient Receptor Potential Vanilloid-2 Channel (TRPV2) may attenuate myocardial damage after myocardial infarction

During the healing process after MI there an immense invasion of inflammatory cells occurs into the infarct area. We have studied the potential role of TRPV2 cation channel in recovery after MI. We show by gene expression array study five days post MI that there was a significant increase in TRPV2 expression compared to sham TRPV2 channel is highly expressed in phagocytes and splenocytes and moderately in the heart. We have performed LAD ligation studies in WT mice, TRPVK/O mice and in rats. On the 3rd day by RT-PCR an increase in the level of TRPV2 was shown and validated by western blot and flow cytometry. Immunohistochemical staining with anti CD68 and anti TRPV2 antibodies showed abundance of inflammatory cells on the 3rd day and also by confocal microscopy. Migration assay showed that only WT macrophages had the capacity to migrate and TRPV2 K/O mice macrophages lacked this ability. Echocardiography and pathology showed a lower reduction in LVEF in the TRPV2 K/O mice compared to control. Scar formation was smaller and the expression of BNP from LV tissue by RTPCR was higher in the K/O mice. Adoptive transfer of WT macrophages to TRPVK/O mice resulted is worse outcome than study mice injected with KO macrophages. We are currently studying the potential role of specific antibodies to TRPV2 channel and look for a small blocking molecule. Our data suggest TRPV2 channel blockers and reduction of activity of the channel in the setting of myocardial infarction may have a beneficial role.

Biography

Gad Keren is the Head of the Department of Cardiology at Tel Aviv Medical Center and is a Professor of Cardiology at the Sackler School of Medicine.

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