218

Assessment of sympathetic reinnervation after transient myocardial ischemia by C11 hydroxyephedrine PET imaging

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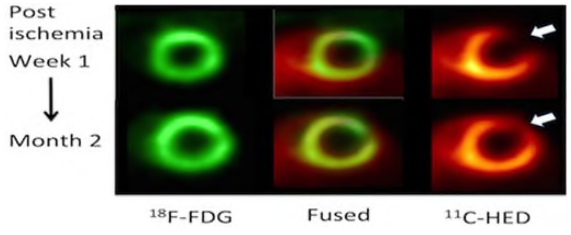
Purpose: To investigate sympathetic nerve damage and reinnervation utilizing a rat model of myocardial transient ischemia and a catecholamine analogue PET tracer, C-11 hydroxyephedrine (HED).

Methods: Transient myocardial ischemia was induced by 20 min coronary occlusion and reperfusion in Male Wistar rats. Dual-tracer autoradiography was performed sub-acutely (day 7, n=4) and chronically (>2 months, n=8) after ischemia, and in non-operated control rats (n=8) using C-11 HED as a marker of sympathetic innervation and Tl-201 for perfusion. Additional serial in-vivo cardiac C11-HED- and F-18 FDG-PET scans were performed at week 1 and after two months.

Results: After transient ischemia, a perfusion defect at the mid ventricular wall was clearly exceeded by a defect of C-11 HED at both the sub-acute and chronic phases. The sub-acute defect showed transmural pattern whereas the chronic phase defect was seen only on the endocardial portion. C-11 HED uptake ratios (vs. remote) at the endo- and epi-cardial portion of the scar were 0.26 $\pm$ 0.18 and 0.40 $\pm$ 0.28 (n.s.) at the subacute phase, and 0.35 $\pm$ 0.13 and 0.97 $\pm$ 0.10, ($p<0.001$) at the chronic phase, respectively. Tyrosine-Hydroxylase antibody nerve staining confirmed a denervation corresponding to the area of HED uptake defect. Serial in-vivo PET imaging visualized reduction of HED defect area at chronic phase consisted with autoradiography and histology.

Conclusions: Discrepant larger uptake defect with C-11 HED in comparison to the perfusion tracer after transient ischemia corresponded to the histologically identified area of denervation. Furthermore, we observed recovery of regional C-11 HED uptake from epi-cardial portion at chronic phase reflecting sympathetic reinnervation.



Abstract 218 Figure.