Fakultät für Medizin

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**Titel des Beitrags:**
Leukocyte transmigration in inflamed liver: A role for endothelial cell-selective adhesion molecule.

**Abstract:**
BACKGROUND/AIMS: This study was designed to investigate the role of endothelial cell-selective adhesion molecule (ESAM), a recently discovered receptor expressed in endothelial tight junctions and platelets, for leukocyte migration in inflamed liver. METHODS: The role of ESAM for leukocyte migration in the liver was analyzed using ESAM-deficient mice in a model of warm hepatic ischemia-reperfusion (90min/30-360min). RESULTS: As shown by immunostaining, ESAM is expressed in sinusoids as well as in venules and is not upregulated upon I/R. Emigrated leukocytes were quantified in tissue sections. Postischemic neutrophil transmigration was significantly attenuated in ESAM-/- mice after 2h of reperfusion, whereas it was completely restored after 6h. In contrast, T-cell migration did not differ between ESAM+/+ and ESAM-/- mice. Using intravital microscopy, we demonstrate that ESAM deficiency attenuates I/R-induced vascular leakage after 30min of reperfusion. The I/R-induced elevation in AST/ALT activity, the sinusoidal perfusion failure, and the number of TUNEL-positive hepatocytes were comparable between ESAM+/+ and ESAM-/- mice. CONCLUSIONS: ESAM is expressed in the postischemic liver and mediates neutrophil but not T-cell transmigration during early reperfusion. ESAM deficiency attenuates I/R-induced...
vascular leakage and does not affect leukocyte adherence. Despite the effect on neutrophil migration, ESAM-deficiency does not protect from I/R-induced injury.

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