Activation of leukocyte-endothelial interactions and reduction of selective neuronal death after global cerebral ischemia.

Abstract:
The role of leukocyte-endothelial interactions (LEI) as part of the inflammatory response after global cerebral ischemia (GCI) is hardly understood and may be detrimental as well as beneficial. Objective of the current study was to investigate the cause-effect relationship of activated leukocytes for the development of ischemic brain damage. Mongolian gerbils were subjected to 15 min of global cerebral ischemia. A cranial window was implanted for quantitative analysis of the pial microcirculation focusing on leukocyte-endothelium interactions by intravital fluorescence microscopy up to 3 h of reperfusion. Subsequently the animals were daily screened for neurological deficits and the evolving brain damage was assessed histologically after 4 days. After global cerebral ischemia the number of rolling and adherent leukocytes increased 20- and>23-fold, respectively upon 3 h of reperfusion as compared to controls (P<0.05). Ischemic animals developed neurological deficits and showed a significant loss of neurons in selective vulnerable areas of the brain. The extent of leukocyte activation, i.e. the maximum number of rollers and stickers directly correlated to the number of viable neurons on day 4 in hippocampus, cortex, and striatum. We conclude that there is a relationship between activation of leukocyte-endothelium interactions and the reduction of ischemic brain damage after global cerebral ischemia.
ischemia. Activation of leukocytes may have neuroprotective potential or indicate regenerative processes.