

Antioxidant status in acute stroke patients and patients at stroke risk.

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BACKGROUND AND PURPOSE: Antioxidant enzymes like copper/zinc superoxide dismutase (SOD), catalase and glutathione peroxidase (GSHPx) are part of intracellular protection mechanisms to overcome oxidative stress and are known to be activated in vascular diseases and acute stroke. We investigated the differences of antioxidant capacity in acute stroke and stroke risk patients to elucidate whether the differences are a result of chronic low availability in arteriosclerosis and stroke risk or due to changes during acute infarction. **METHODS:** Antioxidant enzymes were examined in 11 patients within the first hours and days after acute ischemic stroke and compared to risk- and age-matched patients with a history of stroke in the past 12 months ($n = 17$). Antioxidant profile was determined by measurement of glutathione (GSH), malondialdehyde (MDA), SOD, GSHPx and minerals known to be involved in antioxidant enzyme activation like selenium, iron, copper and zinc. **RESULTS:** In comparison to stroke risk patients, patients with acute ischemic stroke had significant changes of the GSH system during the first hours and days after the event: GSH was significantly elevated in the first hours ($p < 0.01$) and GSHPx was elevated 1 day after the acute stroke ($p < 0.05$). Selenium, a cofactor of GSHPx, was decreased ($p < 0.01$). GSHPx levels were negatively correlated with National Institutes of Health Stroke Scale (NIHSS) scores on admission ($r = -0.84$, $p < 0.001$) and NIHSS scores after 7 days ($r = -0.63$, $p < 0.05$). MDA levels showed a trend for elevation in the first 6 h after the acute stroke ($p = 0.07$). No significant differences of SOD, iron, copper nor zinc levels could be identified. **CONCLUSIONS:** Differences of antioxidant capacity were found for the GSH system with elevation of GSH and GSHPx after acute stroke, but not for other markers. The findings support the hypothesis that changes of antioxidant capacity are part of acute adaptive mechanisms during acute stroke. Copyright 2004 S. Karger AG, Basel

