VASOPROTECTION IN ACUTE STROKE

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One of the therapeutic targets in acute stroke has to be vasoprotection, both to limit the effects of the causative vascular lesion but also to limit the damage in brain microcirculation and consequently in the brain tissue. During acute stroke the blood-brain barrier is severely disturbed and in this complex mechanism inflammation and endothelial dysfunction play important roles. These aspects seem to be of particular interest in chronic hypertension because even before the acute stroke, in these patients there are important modifications in brain microcirculation. These functional and structural modifications of the endothelium with tight junctions and of lamina basalis explain the local prothrombotic transformation followed by microvascular obstruction and local decreased blood flow and also the BBB increased permeability with edema and sometimes-hemorrhagic transformation. Some of the drugs usually used in secondary prevention of stroke as statins, IECA and angiotensin-receptors blockers (sartans) have pharmacological properties which could be useful also in the treatment of acute stroke associated with other well established therapeutic modalities. This statement is sustained by data from both experimental studies in different models of acute stroke, but also by results of some clinical trials.