ICAM-1 and VCAM-1 expression following aneurysmal subarachnoid hemorrhage and their possible role in the pathophysiology of subsequent ischemic deficits.

BACKGROUND: The pathophysiology of ischemic cerebral lesions following aneurysmal subarachnoid hemorrhage (SAH) is poorly understood. There is growing evidence that inflammatory reactions could be involved in the pathogenesis of such delayed occurring ischemic lesions. The aim of this study was to evaluate adhesion molecules with regard to these lesions following SAH. METHODS: Serum and cerebrospinal fluid (CSF) samples were taken daily from 15 patients up to day 9 after SAH and evaluated for intercellular adhesion molecule-1 (ICAM-1) and vascular adhesion molecule-1 (VCAM-1). RESULTS: CSF and serum samples correlated well during nearly the whole time course (p 0.0001 and p< 0.007) but not to a delayed lesion in the CT scan. CONCLUSION: We believe that inflammatory processes are involved in the pathogenesis of cerebral vasospasm but they might only be a part of a multifactorial pathogenesis.