The possible role of neuropeptide Y after spontaneous subarachnoid hemorrhage.

Neuropeptide Y (NPY), a highly potent vasoconstrictive neuropeptide, is widely expressed in the human brain, regulating vessel diameter and cerebral blood flow. Earlier studies focusing on the possible role of NPY in the context of aneurismal subarachnoid hemorrhage (SAH) and vasospasm have produced conflicting results. However, despite extensive research efforts, the pathophysiological mechanisms underlying the SAH-related vasospasm and delayed cerebral ischemia (DCI) have not been clarified. We, therefore, attempted to investigate the role of NPY in SAH-induced vasospasm in a larger, well documented patient population utilizing modern analytical tools. We focused on the release of the potent vasoconstrictor NPY in cerebrospinal fluid (CSF) and blood, and its correlation to vasospasm and stroke in the early clinical stage. Thirty-seven patients with SAH and a control group consisting of 29 patients were included. Eighteen patients developed stroke, 21 patients met the Doppler sonographical criteria for vasospasm. Twenty-nine patients had aneurysms of the anterior circulation and four patients of the posterior circulation. All patients had ventricular drainage inserted and an arterial catheter. Blood and CSF were drawn daily for NPY analysis during a 10-day interval. The levels of NPY in CSF and plasma were significantly higher after
SAH than in the control group (p = 0.001). The vasospasm group showed NPY levels in CSF which continuously ranged above the NPY levels of the non-vasospasm group (p = 0.001). Patients with stroke caused by vasospasm had significantly higher levels of NPY (p = 0.001). NPY is released excessively into blood and CSF following SAH. Patients with cerebral infarction caused by vasospasm had significantly higher levels of NPY. Our results indicate a certain role for NPY in the pathophysiology of vasospasm due to SAH and justify further studies in this area of research.