

Title:

Degenerative cervical myelopathy: Chronic trauma leads to chronic alterations of Angiopoetin II, an endogenic angiogenetic mediator in CSF. A notice for extended angiogenesis?

Objective: Aim of this study is to identify alterations of the proangiogenic protein Angiopoetin II (ANG II) in the cerebrospinal fluid (CSF) in patients with chronic spinal cord injury (SCI) compared to a control group.

Methods: Pre- and postoperative patients with DCM (n=49; 21 female; mean age 62.9 ± 11.28) were included. CSF samples were taken pre- and postoperatively. A control group of patients (n=45; 17 female; mean age 61.06 ± 14.54) with abdominal aortic aneurysm (AAA) was established. AAA patients received a CSF drainage preoperatively.. The neurological status of all participants was evaluated prior surgery (NDI and mJOA). Controls with any neurological deficit or history of neurological diseases were excluded. Samples were examined by ELISA testing. Protein-concentrations of ANG II in CSF (pg/ml) were analyzed.

Results: Both groups did not differ in terms of age and gender distribution. Groups differed regarding their neurological status (mJOA: DCM 10.96 ± 3.1 , AAA 17.28 ± 1.26 , $p < 0.001$; NDI: DCM 40.2 ± 21.1 , AAA 6 ± 8.3 , $p < 0.001$, and in preoperative ANG II concentration: DCM 276 ± 90 , AAA 463 ± 240 , $p < 0.001$). 3 months after surgery ANG II values were almost unaltered in DCM patients, compared to preoperative values (277 ± 65 , $p = 0.998$). Subgroup correlation analysis of clinical data showed no significant differences in concentrations of ANG II.

Conclusion: In patients with DCM concentrations of ANG II lower than in controls were found pre- and persistent postoperatively. These results correspond probably to immune mediated secondary harm in chronic spinal cord injury. Chronically reduced angiogenetic activity could be a relevant part of DCM pathogenesis and secondary harm mechanisms to the spinal cord.