

Development of Cerebral Vasospasm Following Traumatic Intracranial Hemorrhage: Incidence, Risk Factors, and Clinical Outcomes

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Background: Post-traumatic vasospasm (PTV) remains a poorly understood entity. Using a systematic review approach, we examined the incidence, mechanisms, risk factors, impact on outcome and potential therapies of PTV.

Methods: A search on Medline database up to 2015 performed with "traumatic brain injury" and "vasospasm" key-words retrieved 429 references. This systematic review was reported and analyzed following the PRISMA criteria and according to the relevance in human clinical practice.

Results: The research retrieved 429 references of which 226 were excluded from analysis because of their irrelevance and 87 finally included in the review.

Conclusion: Mechanical stretching, inflammation, calcium dysregulation, endothelin, contractile proteins, products of cerebral metabolism and cortical spreading depolarization have been involved in PTV pathophysiology. PTV occurs in up to 30-40% of the patients after severe traumatic brain injury. Usually, PTV starts within the first 3 days following head trauma and may last 5 to 10 days. Young age, low Glasgow Coma Score at admission and subarachnoid hemorrhage have been identified as risk factors of PTV. Suspected on transcranial Doppler, PTV diagnosis is best confirmed by angiography, CT angiography or MR angiography, and perfusion and ischemic consequences by perfusion CT or MRI. Early PTV is associated with poor outcome. No PTV prevention strategy has proved efficient up to now. Regarding PTV treatment, only nimodipine and intra-arterial papaverine have been studied up to now. Treatment with milrinone has been described in a few cases reports and may represent a new therapeutic option.