Nimodipine for vasospasm

Nimodipine, a calcium channel antagonist, is so far the only available therapy with proven benefit for reducing the impact of DID. Aggressive therapy combining hemodynamic augmentation, transluminal balloon angioplasty, and intra-arterial infusion of vasodilator drugs is, to varying degrees, usually implemented. A panoply of drugs, with different mechanisms of action, has been studied in SAH related vasospasm. Currently, the most promising are magnesium sulfate, 3-hydroxy-3-methylglutaryl-CoA reductase inhibitors, nitric oxide donors and endothelin-1 antagonists [1].

There are different drugs to treat cerebral perfusion pressure which are administrated orally or intra-arterially. While orally administrated, these drugs often do not reach their therapeutic concentration or they need a longer time to act. By intracisternal administration of these drugs, less time is needed to reach the appropriate therapeutic concentration. Papaverine is an alkaloid, which causes vasodilatory induction of cerebral and cardiac vessels through direct effect on the cells of smooth muscles. Mechanism of papaverine effect is the inhibition of cyclic adenosine monophosphate and cyclic guanosine 3 and 5 monophosphate intra-arterially [2].

Prophylaxis with nimodipine, hypertension, hypervolemia, and hemodilution (triple H) have been improved the outcome of the patients, however, they could not completely remove the effects of vasospasm [3] [4] [5].

1)
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