

44.Alterations of regional cerebral blood flow and oxygen saturation in a rat sinus-veinthrombosis model.

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Abstract

BACKGROUND AND PURPOSE: The pathophysiology of sinus-vein thrombosis (SVT) in patients and experimental animals is still poorly understood. This study was designed to examine and further elucidate the pathophysiological sequence of events, especially the relationship between local and regional blood flow and hemoglobin oxygen saturation (HbSO₂) detected at identical locations. The use of both parameters as outcome indicators should be compared. **METHODS:** SVT was induced by ligation of the superior sagittal sinus (SSS) and slow injection of kaolin-cephalin suspension into the SSS in rats. Regional cerebral blood flow (rCBF) was assessed by laser-Doppler flowmetry together with regional HbSO₂, which was measured by a microspectrophotometric technique at 48 identical locations for 90 minutes after SVT using a scanning technique. Fluorescence angiography was performed before and 30 and 90 minutes after SVT induction. After 48 hours the animals were killed for histology. **RESULTS:** The fluorescence angiographic findings could divide animals into three groups: (1) group A, with a solitary SSS thrombus (n=8); (2) group B, with a thrombosis of SSS and cortical veins (n=10); (3) group C, animals that had undergone sham operation (n=5). Decreases of rCBF and HbSO₂ and brain damage were seen in group B but not in group A. The reduction of local HbSO₂ receded the flow decrease after sagittal sinus ligation but before thrombosis. Blood pressure in group A was found to be significantly higher after SVT than in groups B and C.

CONCLUSIONS: The brain with acute extension of thrombus from the SSS into cortical veins experiences a critically reduced supply of blood and oxygen. CBF, local HbSO₂, and repeated angiography can be helpful monitors for the early detection of critical conditions after SVT. Local HbSO₂ has a greater sensitivity to predict outcome than ICBF. Moreover, therapies directed to improve perfusion pressure or reduce vascular resistance may open further therapeutic windows during SVT progression.

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