Intracranial aneurysm model

The intracranial aneurysm pathogenesis rupture remains unclear. Because it is difficult to study the time course of human aneurysms and most unruptured aneurysms are stable, animal models are used to investigate the characteristics of intracranial aneurysms.

An agreed clear, precise and reproducible definition of what constitutes an aneurysm in the models would assist in their use to better understand the pathology of intracranial aneurysm and applying findings to patients [1].

Yamaguchi et al. newly established a rat model of intracranial aneurysm rupture model that features site-specific ruptured and unruptured aneurysms. In this study the authors examined the time course of changes in the vascular morphology to clarify the mechanisms leading to rupture.

Ten-week-old female Sprague-Dawley rats were subjected to hemodynamic changes, hypertension, and ovariectomy. Morphological changes in rupture-prone intracranial arteries were examined under a scanning electron microscope and the association with vascular degradation molecules was investigated.

At 2-6 weeks after aneurysm induction, morphological changes and rupture were mainly observed at the posterior cerebral artery; at 7-12 weeks they were seen at the anterior Willis circle including the anterior communicating artery. No aneurysms at the anterior cerebral artery-olfactory artery bifurcation ruptured, suggesting that the inception of morphological changes is site-dependent. On week 6, the messenger RNA level of matrix metalloproteinase-9, interleukin-1β, and the ratio of matrix metalloproteinase-9 to the tissue inhibitor of metalloproteinase-2 was significantly higher at the posterior cerebral artery, but not at the anterior communicating artery, of rats with aneurysms than in sham-operated rats. These findings suggest that aneurysm rupture is attributable to significant morphological changes and an increase in degradation molecules.

Time-dependent and site-dependent morphological changes and the level of degradation molecules may be indicative of the vulnerability of aneurysms to rupture. [2].

Hosaka et al. modified two rodent aneurysm models to create a murine model that produces consistent aneurysms and rupture and can be used for studying cerebral aneurysm formation, rupture and treatment [3].

1)
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2)
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