Factors associated with the growth and rupture of cerebral aneurysms.

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Description

Cerebral aneurysm is a common cerebrovascular disease that is sometimes complicated by rupture or an inflamed mass. The initiation and development of cerebral aneurysms are degenerative progressions of the arterial wall driven by a composite interaction of biological and hemodynamic factors. Endothelial cells on the artery wall respond physiologically to blood-flow patterns. In normal conditions, these responses are accompanying with non-pathological tissue remodeling and adaptation. The assimilation of abnormal blood patterns and genetic susceptibility could lead to the pathological development of aneurysms.

Cerebral aneurysm (CA) has a high predominance and causes a fatal subarachnoid hemorrhage. Although CA is a socially important disease, there are presently no medical treatments for CA, except for surgical techniques, because the exhaustive mechanisms of CA formation remain unclear. CA is a chronic inflammatory disease of the arterial walls and several inflammation-associated factors participate in its pathogenesis. Mast cells are well recognized as major inflammatory cells correlated to allergic inflammation. Mast cells have many cytoplasmic granules that contain various cytokines. Recent studies have revealed that mast cells contribute to various vascular ailments through degranulation and release of cytokines. The number of mast cells was considerably increased in CA walls during CA formation. Inhibitors of mast cell degranulation successfully inhibited the size and medial thinning of induced CA through the inhibition of chronic inflammation, as estimated by nuclear factor-kappa B activation, macrophage infiltration, and the expression of monocyte chemoattractant protein-1, matrix metalloproteinases (MMPs), and interleukin-1β. Mast cells contribute to the pathogenesis of CA through the initiation of inflammation and that inhibitors of mast cell degranulation can be therapeutic drugs for CA.

Several factors that are blood, wall, or hemodynamics-borne have been accompanying with the initiation, progression, and rupture of intracranial aneurysms. The spreading of cerebral aneurysms around the bifurcations of the circle of Willis has provided the impetus for various studies trying to link hemodynamic factors comprise (flow impingement, pressure, and/or wall shear stress) to aneurysm pathophysiology. Hemodynamics is frequently recognized as one of the major factors in aneurysm rupture. Flow impingement, greater pressure, and abnormal wall shear stress are all signs for aneurysm rupture. Typifying wall shear stress for intracranial aneurysms at similar anatomic locations may help in understanding its role.

Hemodynamic factors seem to be associated to the distribution of aneurysms on the intracranial arterial tree and complex, slow flow patterns seem to be accompanying with aneurysm growth and rupture. However, both the prevalence of aneurysms in the general population and the frequency of ruptures in the aneurysm population are extremely low. This suggests that hemodynamic factors and purely mechanical elucidations by themselves may serve as necessary, but never as necessary and sufficient circumstances of this disease's causation. The definitive cause is not yet known, but it is likely an additive or multiplicative effect of a handful of biochemical and biomechanical factors. Arterial hypertension also plays a role in aneurysm formation, it might also be accompanying with a higher incidence of multiple aneurysms as compared to their manifestation in normotensive patients.

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