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Role of Hypertension in Aneurysms

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Hypertension may directly or indirectly contribute to aneurysmal rupture. Hypertension may weaken the aneurysmal wall by directly increasing mechanical stresses. Additionally, activation of the local renin–angiotensin system by systemic hypertension can cause vascular inflammation and remodeling and should contribute to aneurysmal rupture. Certain polymorphisms within the genes related to the renin–angiotensin system are reported to be associated with aneurysmal rupture.

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Recently, we've developed a mouse model of aneurysm that morphologically and histologically resembles human intracranial aneurysms. During this model, aneurysmal rupture causes neurological symptoms which will be easily detected by an easy neurological examination. This model provides a singular opportunity to conduct preclinical studies for identifying therapeutic targets for the prevention of aneurysmal rupture. Using this mouse model of aneurysm, we examined the roles of systemic hypertension and therefore the local renin–angiotensin system within the mechanisms for the rupture of intracranial aneurysms.

To induce systemic hypertension, we used de-oxycorticosterone acetate (DOCA)-salt.19 Mice underwent unilateral nephrectomy followed by an implantation of DOCA pellet 1 week later; 1% NaCl beverage was started on an equivalent day as DOCA pellet implantation. Mice received one injection of elastase (0.035 U) into the spinal fluid at the proper basal cistern on an equivalent day as DOCA pellet implantation. Aneurysm was defined as a localized outward bulging of the vascular wall, whose diameter was greater than the parent artery diameter.

To detect aneurysmal rupture, 2 blinded observers performed daily neurological examination as previously described. Neurological symptoms were scored as follows: 0-normal function; 1-reduced eating or drinking activity demonstrated by a weight loss >2 gm of weight (≈10% weight loss) >24 hours; 2-flexion of the torso and forelimbs on lifting of the entire animal by the tail; 3 circling to 1 side with a traditional posture at rest; 4-leaning to 1 side at rest; 5-no spontaneous activity. We've shown that this neurological testing system is sensitive and specific for detecting aneurysmal rupture during this model. Mice were euthanized once they developed neurological symptoms (score 1–5). All asymptomatic mice were euthanized 21 days after aneurysm induction. The brain samples were perfused with PBS, followed by a gelatin containing blue dye to see cerebral arteries. Two blinded observers assessed aneurysm formation and subarachnoid hemorrhage. Rupture rate was defined because the total number of mice with ruptured aneurysms divided by the amount of mice with any aneurysms. Shows a mouse with normal cerebral arteries, an un-ruptured aneurysm from a mouse that was asymptomatic throughout the experimental period, and a ruptured aneurysm with subarachnoid hemorrhage from a mouse that became symptomatic 12 days after aneurysm induction.

Our previous study found that aneurysm formation happens during the primary 6 days after aneurysm induction, and aneurysmal rupture start occurring ≈7 days after aneurysm induction during this model. We found that by treating the mice with an experimental agent ranging from 6 days after aneurysm induction, we could test whether the experimental agent can reduce the rupture rate. Therefore, during this study, treatments with antihypertensive agents were started 6 days after aneurysm induction and continued for two weeks.

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