Aneurysm wall

Rupture of a saccular intracranial artery aneurysm (IA) causes subarachnoid hemorrhage, a significant cause of stroke and death. The current treatment options, endovascular coiling, and clipping are invasive and somewhat risky. Since only some IAs rupture, those IAs at risk for rupture should be identified. However, to improve the imaging of rupture-prone IAs and improve IA treatment, IA wall pathobiology requires more thorough knowledge. Chronic inflammation has become understood as an important phenomenon in IA wall pathobiology, featuring inflammatory cell infiltration as well as proliferative and fibrotic remodulatory responses [1)].

Aspirin intake prevented inflammation of both the periadventitial tissue and aneurysm wall, irrespective of initial wall condition. Although ASA prevented significant growth in aneurysms with vital walls, this preventive effect did not have an important role in elastase-degraded pouches. In possible translation to the clinical situation, ASA might exert a potential preventive effect during early phases of aneurysm formation in patients with healthy vessels but not in those with highly degenerative aneurysm walls [2)].

Geometric or hemodynamic considerations favor the identification of rupture status; however, retrospective identification of the rupture site remains a challenge for both engineers and clinicians. A more precise understanding of the hemodynamic factors involved in aneurysm wall pathology is likely required for computational fluid dynamics to add value to current clinical decision-making regarding rupture risk [3)].

1)
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