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UNRUPTURED CEREBRAL ANEURYSM IN VASCULAR NEUROSURGERY

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Annotation

Vascular neurosurgery is a diverse field focused on surgical and interventional treatment of cerebrovascular disease, both the manifestations as well as the underlying causes. Specifically, the common diseases that fall in the domain of vascular neurosurgery include cerebral ischemia and ischemic stroke including occlusive vascular disease, non-lesional hemorrhagic vascular disease – which includes spontaneous, hypertensive and angiopathic intracerebral hemorrhage (ICH) – and lesional hemorrhagic vascular disease – which includes cerebral aneurysms, arteriovenous malformations, and cavernomas.

Keywords: vascular neurosurgery, cerebral ischemia, stroke, intracranial hemorrhage, syndrome.

Given this diverse disease pool, vascular neurosurgery spans multiple fields, including neurology, cardiology, intensive care, interventional radiology, and clinical genetics, and can affect involve both adult and pediatric patients. Furthermore, both cerebral ischemia and stroke, as well as intracranial hemorrhage, can be a manifestation of systemic disease, such as atherosclerosis or hypertension and amyloid angiopathy, respectively.

Equally important to the treatment of patients with SAH is the understanding of the biology of cerebral aneurysm formation. Their global prevalence is estimated at 2–6% in the general population, making them relatively common. The most frequently reported risk factors for harboring aneurysms and their risk of causing a hemorrhage have been identified as increased age, female sex, smoking, alcohol intake,

hypertension, Japanese and Finnish ancestry, and most importantly, size of aneurysm (Weir, 2002). However, debate remains as to which aneurysms pose a risk of bleeding and, therefore, which aneurysms require treatment. The biology of aneurysm formation has not been fully understood. It is thought that hemodynamic stress leading to endothelial dysfunction, macrophage infiltration, and vessel wall inflammation and subsequent loss of smooth muscle cells and remodeling lead to the formation and growth of aneurysms. Numerous clinical studies have established genetic association with genetic syndromes, e.g., autosomal polycyst kidney disease, Ehlers–Danlos syndrome, Marfan syndrome. However, most aneurysms occur sporadically, and are not hereditary. However, significantly less is known about genetic predisposition and somatic mutations that drive aneurysm formation. Large-scale genome-wide association studies on humans have identified numerous polymorphisms in genes related to endothelial function, extracellular matrix, and inflammation.

Mechanisms of aneurysm formation.

Endothelial dysfunction: IL-1β NF-κB, MCP-1, Reactive oxygen species, Nitric oxide, Nitric oxide synthase, Angiotensin II, Phosphodiesterase-4, Prostaglandin E2, Vascular cell adhesion protein 1, Intercellular adhesion molecule 1. Macrophage infiltration and vessel wall inflammation: MCP-1, NF-κB, Ets-1, MMPs, IL-1β, TNF-α, IL-8 and IL-17.

Loss of smooth muscle cells/vessel remodeling: TNF-α, IL-1β, MCP-1, MMPs, Adhesion molecules, Complement FGF, TGF, VEGF. (FGF, fibroblast growth factor; IL-1β, interleukin 1β; IL-6, interleukin 6; IL-8, interleukin 8; TGF, transforming growth factor; MCP-1, monocyte chemoattractant protein-1; MMP, matrix metalloproteinase; NF-κB, nuclear factor-κ B; TNFα, tumor necrosis factor-α; VEGF, vascular endothelial growth factor).

All in all, unruptured intracranial aneurysms (UIA) are a common finding, occurring in about 2% of the population, making them very likely to be seen by most practitioners, and present a challenge in the recommendations for optimal management and screening. The consequences of aneurysm rupture are dire, with high likelihood of significant morbidity and mortality. Most aneurysms do not rupture and patients harboring these lesions often remain asymptomatic. There are

effective surgical and endovascular interventions to prevent rupture, but these procedures carry a risk of adverse complications.

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