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ISSN: 2168-975X
Brain Disorders & Therapy

Opinion Article

Cerebral Aneurysm and its Pathophysiology

Keun Hung*

Department of Neurology, Seoul National University Hospital, Seoul, Korea

DESCRIPTION

Cerebral Aneurysms (CAs) are defined by localized structural degradation of the artery wall, with loss of the internal elastic lamina and disruption of the media. They affect 3% to 5% of the general population. The most dreaded CA consequence is rupture, which is linked to a number of modifiable and nonmodifiable risk factors. Despite advancements in surgical procedures and perioperative management, aneurysm rupture still has a high mortality and morbidity rate. 2 Surgical therapies, such as microsurgical clipping and endovascular therapy, are now the only alternatives for treatment, both of which pose a significant risk of procedure morbidity. An aneurysm is a blood vessel wall that has burst open and is filled with blood. Aneurysms form when a weak spot in the vessel wall is exposed. This could be due to an inherited condition or an acquired disease. The repetitive trauma of blood flow against the vessel wall leads the aneurysm to expand by pressing against the area of weakness. According to Young-law, Laplace's increasing the area raises the tension against the aneurysmal walls, resulting in enlargement. Furthermore, a hybrid of computational fluid dynamics and morphological indices has been presented as a reliable predictor of cerebral aneurysm rupture.

Low shear stress is thought to generate massive aneurysm growth and rupture by an inflammatory reaction, whereas high shear stress causes modest aneurysm growth and rupture *via* a mural response (response from the blood vessel wall). Cigarette smoking, hypertension, feminine gender, family history of cerebral aneurysm, infection, and trauma are all risk factors for aneurysm formation. Shear stress damages the artery wall's structural integrity, triggering an inflammatory response that includes the recruitment of T cells, macrophages, and mast cells. Inflammatory mediators include interleukin 1 beta, interleukin 6, Tumour Necrosis Factor Alpha (TNF alpha), MMP1, MMP2, MMP9, prostaglandin E2, complement system, Reactive Oxygen Species (ROS), and angiotensin II. Smooth muscle cells from the artery's tunica media layer moved into the tunica intima, where their function changed from contractile to pro-inflammatory. As a result of arterial fibrosis, a decrease in the number of smooth muscle cells, and abnormal collagen synthesis, the artery wall weakens, allowing aneurysms and ruptures to occur. Cerebral aneurysms have not been related to any known gene loci.

Aneurysms with a diameter greater than 7 mm should be treated since they are more likely to rupture. Meanwhile, aneurysms less than 7 mm that form in the anterior and posterior connecting arteries are more easily ruptured than aneurysms that form elsewhere. The extracellular matrix is a dynamic structure that interacts with vascular cells to undergo constant remodeling. The mechanical strength of the big arteries is mostly dependent on the cross-linking of elastin and collagen due to the secretory function of smooth muscle cells in the cerebral artery rather than contractile action. The lifespan of elastin produced during early embryogenesis is comparable to that of humans, and it rarely undergoes wear and tear. The illness progression may not be reversible once extracellular matrix abnormalities or degradation has occurred. Smooth muscle cells and inflammatory cells produce Matrix Metalloproteinase (MMPs) and tissue inhibitors of metalloproteinase, which mediate extracellular matrix destruction and remodeling. MMPs and their inhibitors are out of balance, which leads to the genesis and progression of cerebral aneurysms.

In the study associated between *LOX* gene polymorphisms and Intracranial Aneurysm (IA) formation in a homogeneous in some population. The catalytic step in cross-linking elastin and collagen is catalyzed by *Lysol Oxidases (LOX)*. For *LOX* to function, it needs one firmly bonded copper ion. Because the amount of copper in the diet has a direct effect on *LOX* activity, copper deficit during development may reduce *LOX* activity and compromise vessel wall integrity, leading to the formation of aneurysms later in life. Copper deficiency in mice throughout development was recently shown to generate complicated vascular wall abnormalities, including thoracic aortic aneurysms and cerebral aneurysms. The thoracic aortae became enlarged with disordered elastic fibers, and the surviving mice had fusiform and saccular aneurysms. Because copper deficiency is common in infancy in cases of cow's milk feeding or infant formula with low copper content, its prevalence and outcome may be influenced by the infancy environment and food habits.

Patients with connective tissue illnesses such osteogenesis imperfecta, vascular Ehlers-Danlos syndrome, and Marfan syndrome, which are frequently associated with brain aneurysms, have been found to have a range of extracellular matrix defects.

Correspondence to: Keun Hung, Department of Neurology, Seoul National University Hospital, Seoul, Korea, E-mail: Keun@hung.kr

Received: 02-May-2022, Manuscript No. BDT-22-16959; **Editor assigned:** 04-May-2022, PreQC No. BDT-22-16959 (PQ); **Reviewed:** 18-May-2022, QC No. BDT-22-16959; **Revised:** 25-May-2022, Manuscript No. BDT-22-16959 (R); **Published:** 03-Jun-2022, DOI: 10.35248/2168-975X. 22.11.161.

Citation: Hung K (2022) Cerebral Aneurysm and its Pathophysiology. Brain Disord Ther. 11:161.