

Aneurysm risk factors

- Global, regional, and national trends in the epidemiology of aortic aneurysms among women of childbearing age, 1990-2021, with predictions through 2036
- Recognition and Recurrence of Aneurysmal Bone Cyst Secondary to Giant Cell Tumor: A Case Series and Review Of The Literature
- The role of histone modifications in the development of abdominal aortic aneurysm
- Thrombelastogram are associated with coronary artery aneurysm in Kawasaki disease patients
- Prevalence and risk factors of thoracic aortic dilatation detected incidentally in adjuvant radiotherapy planning CT scans in patients with breast cancer
- Development and validation of a predictive model for postoperative hepatic dysfunction in Stanford type A aortic dissection
- Phenotype-driven risk stratification of cerebral aneurysms using Shapley Additive Explanations-based supervised clustering: a novel approach to rupture prediction
- Evaluating artificial intelligence models for rupture risk prediction in unruptured intracranial aneurysms: a focus on vessel geometry and hemodynamic insights

Genetic Factors

There is evidence suggesting a genetic component in the development of intracranial aneurysms. Individuals with a family history of aneurysms may have a higher risk. Specific genetic conditions, such as autosomal dominant polycystic kidney disease (ADPKD) and certain connective tissue disorders (e.g., Marfan syndrome), are associated with an increased prevalence of intracranial aneurysms.

Hemodynamic Stress

Hemodynamic factors, such as turbulence and increased blood flow, can contribute to the formation and progression of intracranial aneurysms. Areas of the cerebral arteries exposed to high hemodynamic stress, especially at bifurcations and curved segments, are more prone to aneurysm development.

Vascular Wall Weakness

Weakness in the arterial wall is a key factor. The integrity of the arterial wall can be compromised by structural abnormalities, such as defects in the elastic fibers or smooth muscle cells. This weakening may be congenital or acquired.

Inflammation and Degeneration

Chronic inflammation within the arterial wall can contribute to the degeneration of the vessel structure. Conditions that promote inflammation, such as atherosclerosis, may increase the risk of

aneurysm development.

Hypertension

Chronic high blood pressure is a significant risk factor for the development and rupture of intracranial aneurysms. Hypertension places additional stress on the arterial walls, making them more susceptible to weakening and aneurysm formation.

Smoking

Smoking is a modifiable risk factor associated with an increased risk of developing intracranial aneurysms. It is thought to contribute to the weakening of arterial walls and the progression of atherosclerosis.

Age and Gender

The risk of developing intracranial aneurysms tends to increase with age. Women, particularly postmenopausal women, have a higher prevalence of aneurysms. Female hormones, particularly estrogen, may play a protective role before menopause.

Certain Medical Conditions

Conditions that affect blood vessels, such as fibromuscular dysplasia, arteriovenous malformations (AVMs), and certain infections, can contribute to the development of intracranial aneurysms.

Trauma

Traumatic injury to the head or blood vessels can potentially lead to the development of intracranial aneurysms. However, this is relatively uncommon compared to other risk factors. Understanding the interplay of these factors is essential for unraveling the mechanisms behind intracranial aneurysm development. Ongoing research aims to identify new biomarkers, genetic associations, and potential therapeutic targets to better prevent, diagnose, and manage intracranial aneurysms. Early detection and intervention are crucial in mitigating the risk of rupture and improving patient outcomes.

Observational evidence identified multiple clinical and anatomic [risk factors](#) for the formation of de novo IAs, including [female sex](#), [age](#) <40 yr, [family history](#), [smoking](#) history, [multiple intracranial aneurysms](#) at first diagnosis, and IC as the initial site. More aggressive long-term angiographic follow-up with [digital subtraction angiography](#), [computed tomography angiography](#), or [magnetic resonance angiography](#) is recommended for these patients ¹⁾.

Genetically determined [HDL-C](#) and [LDL-C](#) reduce the risk of [intracranial aneurysm](#) and [ruptured intracranial aneurysm](#). The effects of different [lipid-modifying drugs](#) on IA need to be further investigated ²⁾.

Although some previous reports have demonstrated an association between lipid accumulation and degenerative changes in [aneurysm walls](#) in humans, epidemiological studies have failed to identify dyslipidemia as a risk factor for [intracranial aneurysm pathogenesis](#). Thus, Shimizu et al. examined whether an increase in serum [cholesterol](#) levels facilitates the progression of intracranial aneurysms in a [rat](#) model. Rats were given a high-fat diet (HFD) and subjected to an intracranial aneurysm model. The HFD elevated their serum cholesterol levels. The [intracranial aneurysms](#) induced at the anterior cerebral artery-olfactory artery bifurcation were significantly larger in the high-fat group than in the normal-chow group. Histological analysis demonstrated that the loss of medial smooth muscle layers was exacerbated in the high-fat group and indicated the presence of macrophage-derived foam cells in the lesions. In in vitro experiments, the expression levels of the pro-inflammatory genes induced by LPS in RAW264.7-derived foam cells were significantly higher than those in RAW264.7 cells. The combination of these results suggests that increased serum [cholesterol](#) levels facilitate degenerative changes in the media and the progression of intracranial aneurysms presumably through foam cell transformation ³⁾.

Smoking

[Tobacco cigarette smoking as an intracranial aneurysm risk factor](#)

Radiotherapy

Intracranial aneurysms after radiotherapy (RT) have previously been reported. However, the majority of studies were case reports. Therefore, we performed a nationwide study to explore the risk of radiation-induced intracranial aneurysms.

METHODS: This study included patients diagnosed with head and neck cancer (ICD9: 140-149, 161). Intracranial aneurysms formation was identified using the following ICD9 codes: nonruptured cerebral aneurysm (ICD9:4373), aneurysm clipping (ICD9:3951). Patients who did not receive curative treatment and those with intracranial aneurysms before the diagnosis of head and neck cancer were excluded.

RESULTS: In total, 70,691 patients were included in the final analysis; they were categorized into the following three groups: nasopharyngeal carcinoma (NPC) with RT, non-NPC with RT, and non-NPC without RT. Patients in the NPC with RT group had the highest risk of developing intracranial aneurysms (hazard ratio (HR) 2.57; $P < 0.001$). In addition, hypertension was also a risk factor of developing intracranial aneurysms (HR 2.14; $P < 0.01$). The mean time interval from cancer diagnosis to intracranial aneurysm formation in the NPC with RT group was 4.3 ± 3.1 years.

CONCLUSIONS: Compared with the non-NPC with RT and the non-NPC without RT groups, patients

with NPC who received RT had a higher risk of developing intracranial aneurysms ⁴⁾.

1)

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Zhang B, Dong S, Miao Y, Song G, Yuan F, Liu L, Xia S, Qin Y, Huo X, Wu Z, Miao Z, Mo D, Liu A; International Stroke Genetics Consortium (ISGC) Intracranial Aneurysm Working Group. Effects of blood lipids and lipid-modifying drugs on intracranial aneurysms. *Eur J Neurol*. 2022 Jun 21. doi: 10.1111/ene.15471. Epub ahead of print. PMID: 35726534.

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