Cholesterol complications

Cholesterol as a spontaneous intracerebral hemorrhage risk factor.

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 [1)].

Maekawa et al., investigated possible associations among the presence of cholesterol crystals in embolic debris, the proportions of debris components, and postoperative cerebral embolism in patients undergoing carotid artery stenting (CAS).

Sixty-seven consecutive procedures were performed for internal carotid artery stenosis with CAS at the hospital between November 2015 and February 2018. Procedures for emergency CAS for stroke in evolution or crescendo transient ischemic attack were excluded (n = 12). The embolic debris from remaining procedures (n = 55) was stained with hematoxylin-eosin and the red blood cells, white blood cells, and fibrin were quantified by color-based segmentation. Cholesterol crystals and calcification were examined histopathologically. Diffusion-weighted imaging (DWI) was performed 1-3 days after CAS, and the images were used to classify procedures according to the presence of new lesions.

Of the 55 CAS procedures, new DWI lesions were identified after 32. One patient had symptomatic cerebral embolism. Higher proportions of patients with cholesterol crystals in embolic debris (17 vs. 78%, $p < 0.001$) and higher proportion of white blood cells (mean 2.3 [0-9.9] vs. 4.2% [0-29.9%], $p < 0.01$) were observed in the embolic debris of procedures with and without new DWI lesions.

Cholesterol crystals were common in the embolic debris from patients with postoperative ischemic lesions after CAS. These results suggest that inflammatory destabilization of the intraplaque lipid component is related to postprocedural DWI lesions [2)].

Studies report that both high and low total cholesterol (TC) elevates SAH risk. There are few prospective studies on high-density lipoproteins (HDL-C) and low-density lipoproteins (LDL-C), and apparently none concerns apolipoproteins A and B [3)].

References

1)
Shimizu K, Miyata H, Abekura Y, Oka M, Kushamae M, Kawamata T, Mizutani T, Kataoka H, Nozaki K, Miyamoto S, Aoki T. High-Fat Diet Intake Promotes the Enlargement and Degenerative Changes in the Media of Intracranial Aneurysms in Rats. J Neuropathol Exp Neurol. 2019 Jul 24. pii: nlz057. doi: 10.1093/jnen/nlz057. [Epub ahead of print] PubMed PMID: 31340038.
2)
Maekawa K, Shibata M, Nakajima H, Kitano Y, Seguchi M, Kobayashi K, Sano T, Yabana T, Miya F. Cholesterol Crystals in Embolic Debris are Associated with Postoperative Cerebral Embolism after Carotid Artery Stenting. Cerebrovasc Dis. 2019 Jan 2;46(5-6):242-248. doi: 10.1159/000495795. [Epub ahead of print] PubMed PMID: 30602147.
3)
Lindbohm J, Korja M, Jousilahti P, Salomaa V, Kaprio J. Adverse lipid profile elevates risk for subarachnoid hemorrhage: A prospective population-based cohort study. Atherosclerosis. 2018 May 5;274:112-119. doi: 10.1016/j.atherosclerosis.2018.05.011. [Epub ahead of print] PubMed PMID: 29772479.