Dyslipidemia

Hyperlipidemia is abnormally elevated levels of any or all lipids and/or lipoproteins in the blood.

It is the most common form of dyslipidemia (which includes any abnormal lipid levels).

Conventional lipid-lowering agents, including statins, ezetimibe, fibrates, bile acid sequestrants, nicotinic acid, bempedoic acid and Omega-3 fatty acid, are essential to the management of dyslipidemia. However, these agents have been shown to increase the level of plasma proprotein convertase subtilisin/kexin 9 (PCSK9), a serine protease associated with increased cardiovascular risk.

A review of Luo et al. aimed to investigate the impact of commonly available conventional lipid-lowering agents on circulating PCSK9 levels and lipid profiles.

This protocol was conducted in accordance with the Preferred Reporting Items for Systematic Review and Meta-Analysis Protocols guidelines. A systematic literature search will be conducted in the following databases: MEDLINE, Cochrane Central Register of Controlled Trials (CENTRAL), EMBASE, Web of Science, SCOPUS and ScienceDirect. Additional information will be retrieved from clinical trial registries or from reference list searches. Published and peer-reviewed randomised controlled trials with adults receiving statin, ezetimibe, fibrate, bile acid sequestrant, nicotinic acid, bempedoic acid or Omega-3 monotherapy or in combination for at least 2 weeks, with availability of plasma PCSK9 at the beginning and end of treatment or the net changes in values, will be included. Study selection, data extraction and assessment of the risk of bias will be independently conducted by two investigators. Continuous data will be presented as a standardised mean difference with 95% confidence interval (CI) and dichotomous data as risk ratios with 95% CI. Subgroup analysis and sensitivity analysis will be performed when sufficient studies are included. Publication bias will be assessed with a funnel plot and Egger's test. [1].

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 aneurysms. 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 [2].

1)
Luo J, Huang T, Xu R, Wang X, Yang Y, Li L, Zhang X, Zhang Y, Yang R, Wang J, Yang H, Ma Y, Yang B, Wang T, Jiao L. Impact of conventional lipid-lowering therapy on circulating levels of PCSK9: protocol

for a systematic review and meta-analysis of randomised controlled trials. BMJ Open. 2022 Sep 8;12(9):e061884. doi: 10.1136/bmjopen-2022-061884. PMID: 36691198.
2)
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.

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