CURRICULUM VITAE

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[학력]

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[관심분야]

Dyslipidemia-Residual Risk, Lipoprotein(a), Hypertension, Primary Prevention, Lifestyle Modification

[논문]

1. Lipoprotein(a)-related cardiovascular and all-cause mortalities in Korean adults. Eur J Prev cardiol 2023.
2. Comparison of Office Blood Pressure, Automated Unattended Office Blood Pressure, Home Blood Pressure, and 24-Hour Ambulatory Blood Pressure Measurements. J Korean Med Sci 2023.
3. Association between Low-Density Lipoprotein Cholesterol Level and Cardiovascular Outcomes in Korean Adults: A Nationwide Cohort Study. Diabetes Metab J 2023.
4. Association of environmental tobacco smoke exposure with metabolic syndrome: A longitudinal Cohort Study of 71,055 never smokers. Nutr Metab Cardiovasc Dis 2022.
5. 2022 Consensus statement on the management of familial hypercholesterolemia in Korea. Korean J Intern Med 2022.

KSoLA - Deciphering lipid enigmas: exploring TRL, triglycerides, and remnant cholesterol as residual lipid risks and treatment targets

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Lipoprotein particles in the blood can be broadly classified into six types: chylomicron, VLDL, IDL, LDL, and lipoprotein(a), except HDL, which contains one apolipoprotein B (apoB) per particle and is referred to as "ApoB-containing lipoprotein". LDLC has traditionally been the primary lipid risk factor for atherosclerotic cardiovascular disease (ASCVD). However, recent studies have highlighted the role of triglycerides and remnant cholesterol in addition to LDLC. Triglycerides (TGs)-rich lipoprotein (TRL) refers to lipoprotein particles with high TGs content. The cholesterol present in TRL particles is called TRL-cholesterol. Remnant cholesterol refers to the cholesterol in TRL-remnant particles, excluding chylomicrons and very large VLDL. However, it is typically considered to be the cholesterol in VLDL and IDL particles. In clinical practice, remnant cholesterol can be calculated by subtracting HDLC and LDLC from total cholesterol or by using the Friedewald formula to calculate TGs divided by 5 if TGs are not high.

Previous epidemiological and Mendelian randomization studies have shown that TGs and remnant cholesterol are risk factors for ASCVD morbidity and mortality, suggesting that they are as important as LDLC in terms of residual risk.

However, there is still debate regarding whether TGs can serve as a therapeutic target in patients at high risk of ASCVD. Currently, it remains unclear whether fibrates and omega-3 fatty acids are effective in reducing the risk of ASCVD events. This uncertainty may be due to differences in the study population and control groups, drug type and dose, statin use, and outcomes on other lipid markers in recent randomized clinical trials. It is generally suggested that treatment aimed at lowering TGs with fibrates or omega-3 fatty acids act on lipoprotein particle remodeling rather than clearance. Therefore, we need to see the results of recent major trials of agents targeting apoCIII or ANGPTL3.

This lecture will cover what TRL and remnant cholesterol are and their metabolism and impact on ASCVD. It will also comment on the relevance of TGs as a marker of cardiovascular risk and as a therapeutic target based on the results of recent studies.