ClinicSearch

Journal of Clinical Gynaecology and Breast

Chieh Chen *

Open Access

Review Article

A Narrative Review of Atherosclerosis Dyslipidemia

Chieh Chen*

Division of family medicine, Hualien Armed Forces General Hospital. Hualien city, Hualien County, Taiwan

***Corresponding Author:** Chieh Chen, Division of family medicine, Hualien Armed Forces General Hospital. Hualien city, Hualien County, Taiwan.

Received date: December 01, 2022; Accepted date: December 12, 2022; Published date: January 27, 2023

Citation Chieh Chen (2023), A Narrative Review of Atherosclerosis dyslipidemia. *Archives of Clinical and Experimental Pathology*. 2(1); **Doi:**10.31579/2834-8508/008

Abstract

Most screening tests for the elevation of plasma lipids include total cholesterol, low- density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C), and triglyceride levels. It is generally accepted that elevations in levels of total cholesterol and LDL cholesterol, and a reduction in the level of HDL cholesterol place patients at a higher risk of initial and subsequent cardiac events.

Keywords: dyslipidemia; hyperlipidemia; hypertriglyceridemia; hypercholesterolemia; HMG-CoA reductase inhibitors

Introduction

Hyperlipidemia is an increase in cholesterol and triglycerides in the blood. Dyslipidemia, such as the condition of hypercholesterolemia, hypertriglyceridemia, or a combination of the two, is a major cause of atherosclerosis. Hyperlipidemia increases the risk of coronary heart disease. Blood lipids include cholesterol, triglycerides, and phospholipids and they are fat-soluble and must combine with plasma proteins to form lipoproteins to be transported to various organs and tissues through the blood. Low-density lipoprotein cholesterol causes atherosclerotic disease. Thus, an individual's risk of atherosclerotic disease is strongly related with their lifelong accumulation of LDL-C. Accordingly, a significant increase of risk for coronary heart disease and cardiovascular mortality has been reported in young adults with long-term LDL-C ≥ 100 mg/dL (2.5mmol/L). Therefore, in order to prevent atherosclerosis and its sequelae (including myocardial infarction, ischemic stroke, peripheral arterial disease, etc.), it is necessary to act early in life. Indeed, the early manifestations of atherosclerosis are often apparent in the third decade of life, which is a striking problem with stark reality of early morbidity and mortality associated with familial hypercholesterolemia.

The bad effect for cardiovascular disease

Moreover, changes in plasma cholesterol level have been found to be directly associated with cardiovascular diseases in young adults. Cholesterol, which is a complex form of fat, is an indispensable substance in human cells for a body to maintain the integrity of cells. There are two main sources, where one-third of which is obtained from food and enters the blood after being absorbed by the gastrointestinal tract and the other two thirds are synthesized in the liver [1]. Cholesterol is the main component of cell membrane with its role in maintaining the function of cell membrane. In addition, cholesterol is also the raw materials for the synthesis of important hormones in the human body, such as for vitamin D [2]. Blood lipids in the human body include total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides, etc. [3-4]. Due to the increasing westernization of diet, the proportion of cardiovascular, cerebrovascular diseases and atherosclerosis among Chinese people is increasing on an annual basis. The results of many epidemiological investigations show that hyperlipidemia not only affects the health and physical fitness of individuals, but also affects their heart and vessels. Cardiovascular disease, diabetes, hypertension, hyperuricemia and other diseases are closely related to the incidence or mortality [5]. Among obese adults, high total cholesterol, low high-density lipoprotein cholesterol and high low-density lipoprotein cholesterol, as well as high triglycerides, indicate that decreasing lipid level in serum of the body is related to the observed arterial injuries. Moreover, as long as the blood cholesterol concentration is reduced by 1%, the risk of cardiovascular disease is reduced by 2%, and every 1 mg/dl increase in the blood HDL-C concentration can reduce the mortality rate of coronary heart disease by 3.5% [6].

Discussion

The Framingham Heart Study

The Framingham Heart Study, which has the history of more than 50 years in development since 1948, has identified risk factors for heart disease [6-7]. High cholesterol is a very important risk factor, and other risk factors include high blood pressure, smoking, obesity, diabetes, metabolic syndrome, etc. Cholesterol is a fatty substance that can accumulate in the walls of blood vessels, leading to atherosclerosis and the formation of blood clots, which can induce cardiovascular disease or stroke. Cholesterol runs in the blood in several different forms: low-density lipoprotein and high-density lipoprotein [7-8].

The similar effect with metabolic syndrome

Metabolic syndrome is a general term for a group of multifaceted metabolic abnormalities. Common clinical manifestations of such patients include: insulin resistance, glucose intolerance, hyperlipidemia, hypertension, obesity, abnormal blood coagulation (including elevated concentrations of plasminogen activator inhibitor type1 and fibrinogen in blood, etc.), and hyperuricemia [9]. People with these metabolic syndromes are prone to serious systemic chronic diseases in the future, such as: diabetes, stroke, arteriosclerosis, myocardial infarction, cancer, liver fibrosis, etc. [10]. Taiwanese diagnostic criteria for metabolic syndrome, three or more of the following five items can be diagnosed as metabolic syndrome: abdominal obesity (waist greater than 90 cm for men and 80cm for women), hypertension (systolic blood pressure greater than or equal to 130mmHg or diastolic blood pressure greater than or equal to 85 mmHg), fasting hyperglycemia (fasting blood glucose value greater than or equal to 100 mg/dL), high triglycerides (TG>=150mg/dL), low high-density lipoprotein cholesterol (HDL) (<40mg/dL in men, <50mg/dL in women). During the pathogenesis of metabolic syndrome, the accumulation of visceral fat is closely related to its cause, such as factors of obesity and insulin resistance. Genetics and the environment may also play a role in causing metabolic syndrome.

The harm of hyperlipidemia

Patients with metabolic syndrome have an increased risk of diabetes and cardiovascular disease in the future, as well as increased cardiovascular and all-cause mortality [11]. Metabolic syndrome is currently a combinatory result of several factors in play, but the underlying pathogenetic mechanism may still involve increased visceral fat and insulin resistance [12]. However, most studies on metabolic syndrome focus only on the general adult population, and given the elderly population as a special group of attention, physical aging alone may not be adequate in explaining the rising prevalence. Dyslipidemia is the general categorical term for conditions that cause cardiovascular disease, and specific diseases such as type 2 diabetes, hypothyroidism, obesity, nephropathy, alcoholism and long-term use of steroid drugs can cause hyperlipidemia [13]. Dyslipidemia is defined of high level of triglycerides, low density lipoprotein cholesterol, and total cholesterol; and yet, with lower level of high-density lipoprotein cholesterol at the same time [14].

Treatment for atherosclerosis dyslipidemia

The blood TC and LDL-C of the elderly population decreased, which may be caused by changes in body fat distribution, systemic inflammation, chronic diseases, and malnutrition. The first-line treatment strategy for hyperlipidemia still relies on adjustment of lifestyle, including changing dietary habits (by diet of increasing high-fiber intake) and increasing physical activity [15]. If such standard therapy does not resolve the condition within 3 to 6 months, the use of drugs will then be considered. HMG-CoA reduction inhibitors have a significant effect on the course of disease, and at the same time can stabilize atherosclerotic plaques formed by smooth muscle cells, fibrinogen, and intravascular cells [16]. Statins are the most effective lipid-lowering agent, which has positive effect of reducing the incidence and mortality of coronary heart disease. Recent studies have also found that in addition to a strong lipid-lowering function, statins can also strengthen the protective function of vascular endothelial cells, regulate the inflammatory response in the body, maintain plaque stability, and inhibit platelet aggregation and other cardiovascular protective effects [17]. Overall, it is beneficial for the prevention and treatment of coronary heart disease.

Conclusion

Dyslipidemia is one of the major risk factors for atherosclerosis and coronary heart disease. Treatment of dyslipidemia can reduce the incidence and mortality of cardiovascular disease, where changing dietary habits and increasing physical activity are the preferred treatment strategies. If drug treatment is required, HMG-CoA reductase inhibitors are the most effective lipid-lowering agent, which can significantly help to reduce the incidence and mortality of coronary heart disease [18]. High low-density lipoprotein cholesterol is a major risk factor for atherosclerosis, which plays an important role in coronary heart disease and other cardiovascular diseases [19]. High-density lipoprotein cholesterol concentration, plasma viscosity, fibrinogen and C-reactive protein (CRP) are all correlated with cardiovascular diseases, especially coronary heart disease [20]. Studies have shown that reducing total plasma cholesterol by 1% reduces the incidence of coronary heart disease by 2%; and 1% reduction in LDL-C reduces the incidence of coronary heart disease by 1 to 2%. Furthermore, every 1mg/dL increase in HDL-C can reduce the mortality of coronary heart disease by about 3.5% [11,21-22].

Reference

1. Handhle A, Park A. (2021). Hyperlipidaemia. Medicine. 49(9):587-591.
2. Grundy SM. (2016). Dyslipidaemia in 2015. Nat Rev Cardiol.13(2):74-75.
3. Penson PE, Pirro M, Banach M. (2020). LDL-C: lower is better for longer—even at low risk. BMC medicine.18(1): 1-6.
4. Soran H, Dent R, Durrington P. (2017). Evidence-based goals in LDL-C reduction. Clinical Research in Cardiology. 106(4): 237-248.
5. Catapano AL, Graham I, De Backer G, et al. (2016). ESC Scientific Document Group. 2016 ESC/EAS guidelines for the management of dyslipidaemias. Eur Heart J. 37(39):2999-3058.
6. Mahmood SS, Levy D, Vasan RS, Wang TJ. (2014). The Framingham Heart Study and the epidemiology of cardiovascular disease: a historical perspective. The lancet. 383(9921):999-1008.
7. Cromwell WC, Otvos JD, Keyes MJ, et al. (2007). LDL particle number and risk of future cardiovascular disease in the Framingham Offspring Study—implications for LDL management. Journal of clinical lipidology. 1(6):583-92.
8. Ference BA, Ginsberg HN, Graham I, et al. (2017). Low-density lipoproteins cause atherosclerotic cardiovascular disease. 1. Evidence from genetic, epidemiologic, and clinical studies. A consensus statement from the European Atherosclerosis Society Consensus Panel. Eur Heart J. 38(32):2459-2472.
9. Stone NJ, Robinson JG, Lichtenstein AH, et al. (2013) ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults. Circulation. 129(25):1-45.
10. Navarese EP, Robinson JG, Kowalewski M, et al. (2018). Association between baseline LDL-C level and total and cardiovascular mortality after LDL-C lowering: a systematic review and meta-analysis. Jama. 319(15): 1566-1579.
11. Executive Summary (2001) of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA. 285:2486-2497
12. Jacobson TA, Ito MK, Maki KC, et al. (2014). National Lipid Association recommendations for patient-centered management of dyslipidemia, part 1. J Clin Lipidol. 8(5):473-488.
13. Sinha RA, Bruinstroop E, Singh BK, Yen PM. (2019). Nonalcoholic fatty liver disease and hypercholesterolemia: roles of thyroid hormones, metabolites, and agonists. Thyroid. 29(9):1173-1191.
14. Tada H, Okada H, Nohara A, Yamagishi M, Takamura M, Kawashiri MA. (2021). Effect of cumulative exposure to low-density lipoprotein-cholesterol on cardiovascular events in patients with familial hypercholesterolemia. Circulation Journal. 85(11):2073-2078.
15. Michos ED, McEvoy JW, Blumenthal RS. (2019). Lipid management for the prevention of atherosclerotic cardiovascular disease. N Engl J Med. 381:1557-1567.
16. Penson PE, Pirro M, Banach M. (2020). LDL-C: lower is better for longer-even at low risk. BMC medicine 2020; 18(1): 1-6.
17. Iskandar MF, Nurmainah N, Susanti R. (2018). The risk of atherosclerotic cardiovascular disease among hyperlipidaemia patients and appropriateness of statin therapy. Pharmacology and Clinical Pharmacy Research. 3(1).

18. Jeong SM, Choi S, Kim K, Kim SM, Lee G, Park SY, Kim YY, Son JS, Yun JM, Park SM. (2018). Effect of change in total cholesterol levels on cardiovascular disease among young adults. J Am Heart Assoc. 7(12):008819.
19. Silverman MG, Ference BA, Im K, et al. (2016). Association between lowering LDL-C and cardiovascular risk reduction among different therapeutic interventions: a systematic review and meta-analysis. Jama. 316(12): 1289-1297.
20. Mach F, Baigent C, Catapano AL, et al. (2020). ESC/EAS guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. Eur Heart J. 41(1):111-188.
21. Abdullah SM, Defina LF, Leonard D, et al. (2018). Long-term association of low-density lipoprotein cholesterol with cardiovascular mortality in individuals at low 10-year risk of atherosclerotic cardiovascular disease. Circulation.138(21):2315-2325.
22. Booth JN 3rd, Colantonio LD, Howard G, Safford MM, Banach M, Reynolds K, Cushman M, Muntner P. (2016). Healthy lifestyle factors and incident heart disease and mortality in candidates for primary prevention with statin therapy. Int J Cardiol. 207:196-202.