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Review Article

# A Review on Dyslipidemia: Types, Risk Factors and Management

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## ABSTRACT

Dyslipidemia is characterized by abnormally elevated cholesterol or fats (lipids) in the blood. Cardiovascular disease (CVD) is becoming more prevalent worldwide and is one of the leading causes of death. Dyslipidemia is an important risk factor for cardiovascular disease other factors are hypertension, diabetes mellitus, and smoking. Presently statins and fibrates are the major anti-hyperlipidemic agents for the treatment of elevated plasma cholesterol and triglycerides respectively, with remarkable side effects on the muscles and the liver.Lifestyle interventions remain a key component for the management of dyslipidemias. The present review will highlights types, risk factors and management available for dyslipidemia.

**Keywords:** Dyslipidemia, CVD, Statins, Fibrates

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## INTRODUCTION:

Dyslipidemia defined as dysregulated plasma lipids, including abnormally elevated plasma triglycerides, total cholesterol and LDL-cholesterol reducing HDL-cholesterol. Dyslipidemia is a lipoprotein metabolism disorder that shows increasing cholesterol level, triglyceride level. Dyslipidemia is potential risk factors for further developing cardiovascular disease[1]. Some anti-dyslipidemia drugs currently available in the market include statins, fibrates, niacin, ezetimibe, and bile acid binding resins[2].

### Classification & Etiology of Dyslipidemia

Dyslipidemia is classified as primary (genetic & most common in children) or secondary dyslipidemia (due to lifestyle & common in adults)[3]. The causes behind primary dyslipidemia are single or multiple gene mutations that result in overproduction or defective clearance of TG & LDL cholesterol, & underproduction or excessive clearance of HDL. The most important causes of secondary dyslipidemia are alcohol overuse, a sedentary lifestyle with excessive dietary intake of saturated fat, cholesterol, & trans-fats[4]. Some medical conditions found to be associated with secondary dyslipidemia such as diabetes mellitus, chronic kidney disease, primary biliary cirrhosis and other cholestatic liver diseases[5].

### Symptoms

Dyslipidemia usually show symptoms like confusion & dyspnea, impairment of balance tendinous xanthomas (elbow, knee tendons), aphasia difficulty in speaking, sensation of tickling, tingling, burning, pricking but can lead to symptomatic vascular diseases, including coronary artery disease and peripheral arterial disease. High levels of TGs (> 1000 mg/dL) can cause acute pancreatitis. Severe hyper-triglyceridemia (> 2000 mg/dL) can give retinal arteries & veins a milky white appearance (lipemia retinalis). Extremely high lipid levels also can give a lactescent (milky) appearance to blood plasma[6].

### Physiological Consequences of Dyslipidemia

### Cardiovascular Disease:

Continue high lipid intake results abnormal lipid profile in blood & it can lead to lipid deposition in blood vessels thus generate much type of consequences in body. It may be coronary artery disease mean fat deposition in coronary artery thus weakened blood supply & nutrient to heart.Fat deposition on artery, can cause bulge & weakness on artery wall can lead to rupture it & form catastrophic condition called aneurysm[7].Other serious situations are stroke, gangrene, & atherosclerosis[8].

### Other Disorders:

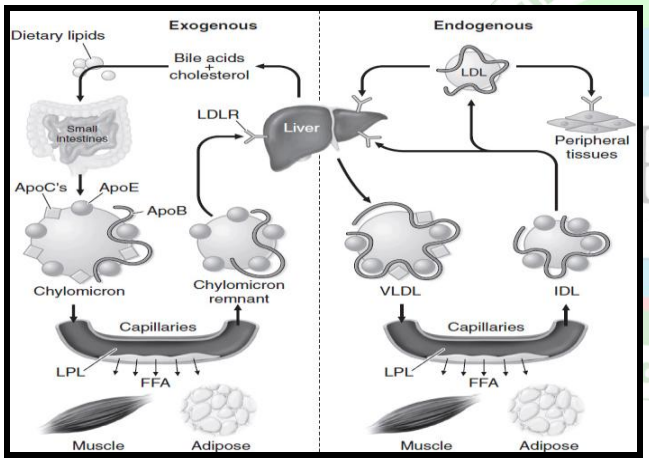
Lipid-disorders directly as well as indirectly promote many diseases such as type 2 diabetes mellitus, and a number of common cancers, PCOS in females[9] mental illness like bipolar disorder, schizophrenia[10], stress & physical inactivity[11]. Dyslipidemia also promotes prostatic growth & contractility that represent important risk factors for the development of benign prostatic hyperplasia[12].

### Dyslipidemia and Obesity:

In every country incidences of obesity is continuously growing, & often dyslipidemia occurs in parallel. Globally, around 2.8 million people die in each year as a result of being overweight or obese. [13]. If the current trend continues, 86.3% of adults will be overweight by 2030 & number of deaths will be so high[14].

### Dyslipidemia: Mechanisms

Numerous pathways, enzymes, proteins & factors are involved in the process of Lipid metabolism thus it is very complex mechanism & only one abnormality can lead to dyslipidemia, but three main pathways are responsible for the uptake, transport & storage of lipids in to the body, included the exogenous pathway, the endogenous pathway, & reverse cholesterol transport pathways. [15]




### Management:

The first line treatment of abnormal cholesterol in dyslipidemia is usually to take low saturated & trans fats containing diet, & high intake of fruits & green vegetables, nuts, seeds, stop smoking, drinking & increase exercise in daily routine. Liver makes all type of cholesterol as per body need. Cholesterol also comes in body from dietary sources such as animal-based foods like milk, eggs, & meat. Lipid or cholesterol lowering drugs included 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inhibitors commonly termed "statins", Fibric acid derivatives, niacin/nicotinic acid, bile acid sequestrates cholesterol absorption inhibitors.[16]

### HMG-CoA Reductase Inhibitor (Statins):

Statins inhibit HMG-CoA reductase thereby, suppress cholesterol biosynthesis. Currently available statins are lovastatin, simvastatin, pravastatin, atorvastatin and rosuvastatin etc.[17]HMG-CoA reductase is the key enzyme of cholesterol biosynthetic pathway & catalyzes the change of HMG-CoA to mevalonate, as a rate-limiting step in cholesterol biosynthesis.[18]

### Fibrates:

Clofibrate was the first fibrate drug; it is developed in Japan 1960s. Peroxisome proliferators activated receptor (PPAR) activation is one of the hallmarks of fibrate drugs action.[19]

### Niacin/Nicotinic acid:

Niacin or vitamin B3 is available as a prescription medication for lipid lowering & as an over-the-counter for supplement. The lipid-lowering effects of niacin were first noted in 1955; niacin reduced total cholesterol, LDL-C & increased HDL-C by different mechanisms.[20]

### Bile-acid binding resins:

The bile acid resins are the safest & oldest agents of all lipid lowering drugs. Three bile acid resins are mainly available cholestyramine, colesevelam and colestipol.[21]

### Cholesterol Absorption Inhibitors:

Cholesterol absorption inhibitors decrease the absorption of dietary & biliary cholesterol into the intestine. Therefore reduced amount of intestinal cholesterol reached to the liver results in increased activity of hepatic LDL receptor, which leads to elevated clearance of LDL cholesterol.[22]

### Lipid-Regulating Agent:

Omega-3-acid ethyl ester comes in class of medications called lipid-regulating agents and can be used it together with life style changes to treat hypertriglyceridemia. [23]

## CONCLUSION:

Dyslipidemia refers to unhealthy levels of one or more kinds of lipid in blood. Several factors can lead to dyslipidemia such as smoking, obesity, sedentary lifestyle and consumption high fatty food.. Lifestyle changes may help to get cholesterol and triglyceride levels under control. Daily exercise and weight loss may also improve cholesterol profile. Statins or fibrates and a healthy lifestyle, usually manage dyslipidemia.

## REFERENCES:

1. Shankar, Kripa, et al. "Cucumismelo ssp. Agrestis var. Agrestis Ameliorates High Fat Diet Induced Dyslipidemia in Syrian Golden Hamsters and Inhibits Adipogenesis in 3T3-L1 Adipocytes." Pharmacognosy magazine 11.Suppl 4 (2015): S501.
2. Varshney, Salil, et al. "Rohitukine inhibits in vitro adipogenesis arresting mitotic clonal expansion and improves dyslipidemia in vivo." Journal of lipid research 55.6 (2014): 1019-1032.
3. Roth, Gregory A., et al. "High total serum cholesterol, medication coverage and therapeutic control: an analysis of national health examination survey data from eight countries." Bulletin of the World Health Organization 89.2 (2011): 92-101.
4. Innerarity, T. L., et al. "Structural relationship of human apolipoprotein B48 to apolipoprotein B100." Journal of Clinical Investigation 80.6 (1987): 1794.
5. Sicree, R., J. Shaw, and P. Zimmet. "Prevalence and projections." Diabetes atlas 3 (2006): 16-104 Ogden, Cynthia L., et al. "Prevalence of childhood and adult obesity in the United States, 2011-2012." Jama 311.8 (2014): 806-814.)
6. Collison, Kate S., et al. "Dietary trans-fat combined with monosodium glutamate induces dyslipidemia and impairs spatial memory." Physiology & behavior 99.3 (2010): 334-342.
7. Bryant, Laura M., et al. "Lessons learned from the clinical development and market authorization of Glybera." Human gene therapy Clinical development 24.2 (2013): 55-64
8. Leong, B. D., et al. "Prevalence of peripheral arterial disease and abdominal aortic aneurysm among patients with acute coronary syndrome." Med J Malaysia 68.1 (2013):

9. Fulghesu, Annamaria, et al. "Obesity-related lipid profile and altered insulin incretion in adolescents with polycystic ovary syndrome." Journal of Adolescent Health 46.5 (2010): 474-481.
10. Saravane, D., et al. "Drawing up guidelines for the attendance of physical health of patients with severe mental illness." L'Encephale 35.4 (2009): 330-339.
11. Costa, Rafaela, et al. "Handling of adolescent rats improves learning and memory and decreases anxiety." Journal of the American Association for Laboratory Animal Science 51.5 (2012): 548-553.
12. Vikram, Ajit, Gopabandhu Jena, and PoduriRamarao. "Insulin-resistance reduces botulinum neurotoxin-type A induced prostatic atrophy and apoptosis in rats." European journal of pharmacology 650.1 (2011): 356-363.
13. Hanson, Michael A., et al. "Crystal structure of a lipid G protein–coupled receptor." Science 335.6070 (2012): 851-855.
14. Sladek, Robert, et al. "A genome-wide association study identifies novel risk loci for type 2 diabetes." Nature 445.7130 (2007): 881-885
15. Sultan, F., et al. "Lipoprotein lipase and hepatic lipase activities in a hypercholesterolaemic (RICO) strain of rat. Effect of dietary cholesterol." Biochemical Journal 266.2 (1990): 349-353.
16. Huang, Yadong, et al. "Overexpression of apolipoprotein E3 in transgenic rabbits causes combined hyperlipidemia by stimulating hepatic VLDL production and impairing VLDL lipolysis." Arteriosclerosis, thrombosis, and vascular biology 19.12 (1999): 2952-2959
17. Innerarity, T. L., et al. "Structural relationship of human apolipoprotein B48 to apolipoprotein B100." Journal of Clinical Investigation 80.6 (1987): 1794
18. Abumrad, Nada A., and Nicholas O. Davidson. "Role of the gut in lipid homeostasis." Physiological reviews 92.3 (2012): 1061-1085.
19. Rader, Daniel J., and Helen H. Hobbs. "Disorders of lipoprotein metabolism." Harrisons Principles of Internal Medicine 16.2 (2005): 2286.
20. Karpe, Fredrik, Julian R. Dickmann, and Keith N. Frayn. "Fatty acids, obesity, and insulin resistance: time for a reevaluation." Diabetes 60.10 (2011): 2441-2449.)
21. Klop, Boudewijn, et al. "A physician's guide for the management of hypertriglyceridemia: the etiology of hypertriglyceridemia determines treatment strategy." Panminervamedica 54.2 (2012): 91
22. Datta, Dharam V., and Sheila Sherlock. "Cholestyramine for long term relief of the pruritus complicating intrahepatic cholestasis." Gastroenterology 50.3 (1966): 323-332.)
23. Sharifi, Faranak, et al. "The efficacy of Ezetimibe added to ongoing Fibrate-Statin therapy on postprandial lipid profile in the patients with type 2 Diabetes mellitus." Journal of Diabetes & Metabolic Disorders 12.1 (2013): 24.