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Hyperlipidemia: Primary Prevention Measures to Lower the Risk of Cardiovascular Disease

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Abstract

Hyperlipidemia is a heterogenous group of disorders characterized by an excess of lipids in the blood stream. Hyperlipidemia is a risk factor for coronary disease and platelet reactivity is increased with high cholesterol levels in blood suggesting a prethrombotic risk. It is recognized as a risk factor for ischemic heart disease and coronary mortality. The World Health Organization estimates that almost 20% of all strokes and over 50% of all heart attacks can be linked to high cholesterol. Lowering blood cholesterol levels reduces coronary event among individuals with or without coronary artery disease. This article focuses on primary prevention strategies that when observed can maintain blood cholesterol levels thereby decreasing the risk of cardiovascular diseases. The positive aspect in managing this condition is that it can be entirely controlled with strict adherence to lifestyle modification. It is highly important for every individual to adhere to these preventive aspects to cut the risk of cardiovascular disease.

Keywords: Hyperlipidemia; Cardiovascular disease; Cholesterol.

Introduction

Cardiovascular diseases (CVD) are the major cause of morbidity and mortality in our society with hyperlipidemia contributing significantly to atherosclerosis. CVD in developed countries continue to grow at an epidemic proportion. There are a significant number of young adults with no clinical evidence of CVD but who have two or more risk factors that predispose them to CV events and death. Hyperlipidemia has been closely linked to the pathophysiology of heart disease and is a key independent modifiable risk factor of CVD. The rate of hyperlipidemia is not only high in developed countries but also in developing countries as a result of occidentalization of diet and lifestyle change.[1] In 2002, World Health Organization reported hyperlipidemia is associated with more than half of the global cause of ischemic heart diseases. Estimation of the prevalence of hyperlipidemia ensures proper health control planning actions for all health sectors for the prevention of CVDs.[2]

It is imperative that primary prevention efforts should be initiated at a young age to avert decades of unattended risk factors. Hyperlipidemia had been linked to CVD a century ago. The increased levels of Low Density Lipoprotein (LDL) and low levels of High Density Lipoprotein (HDL) are well known risk factors of CVD in all groups. The new National Cholesterol Education Program (NCEP)/Adult Treatment Panel III (ATP) guidelines have come as to wake up call to clinicians about primary prevention of CVD through strict lipid management and multifaceted risk management approach in the prevention of CVD.[3]

Basic Description of Lipid and Lipoprotein

Lipid is the scientific term for fats in blood. Lipid performs important functions in the body when they are present in normal levels and it causes health problems when their levels are abnormally high. Cholesterol is a fat like substance that is present in cell membranes and is a precursor of bile acids and steroid hormones. Cholesterol travels in blood in distinct particles containing lipids and proteins. Three major classes of lipoproteins are found in serum of a

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fasting individual such as: LDL, HDL and Very Low Density Lipoprotein(VLDL).

LDL makes up 60 -70% of total serum cholesterol. It is major atherogenic lipoprotein and has long been identified by NCEP as the primary target of cholesterol lowering therapy. HDL cholesterol normally makes up 20 -30% of total serum cholesterol. VLDL is a trigyceride-rich lipoproteins but contains 10 -15% of the total serum cholesterol. A fourth class lipoproteins are chylomicrons, triglyceride- rich lipoprotein formed in the intestine from dietary fat and appear in blood after a fat-containing meal.

The term hyperlipidemia means high lipid levels in blood i.e., LDL is above 130mg/dl; HDL is below 60 mg/dl; and triglyceride levels are greater than 150mg/dl. Coronary angiographic trials have demonstrated that cholesterol lowering slows the progression of coronary atherosclerosis and may even induce regression. Studies suggest that reduction in coronary events seen in angiographic trials is greater than would be anticipated for the degree of angiographic improvement induced by cholesterol lowering. Plaque stabilization due to decrease in the lipid content of the lesions most likely to rupture and improved endothelial function are two mechanisms that could partly account for reduction in coronary events with cholesterol lowering. Another mechanism is a decrease in the tendency toward platelet thrombus formation with cholesterol lowering.[4]

Progression of Hyperlipidemia to CAD

Cholesterol comes from two sources: the body and food; either one can contribute to high cholesterol. Some people inherit genes that trigger too much cholesterol production and the remaining population it is mainly caused through diet. Saturated fat and cholesterol are found in animal- based foods including meat, eggs and dairy product made up of milk. Genetic make up is reflected in family history of high cholesterol or heart disease may result in high cholesterol, although diet is carefully taken coupled with exercise.

All individuals possess cholesterol in their blood, when LDL levels become high in the blood, the excess can accumulate on the walls of the arteries. This build-up of cholesterol and other substances called plaque can narrow the artery. When plaque is formed in the coronary arteries that supply blood to heart muscle, it is termed as coronary artery disease. Next consequence of plaque is that when it breaks, it can form a clot. If a clot lodges in an artery and completely impedes that blood supply, the cells become deprived of nutrients and oxygen and ultimately lead to cell death.[5]

Clinical research studies confirm the disastrous consequences of high cholesterol levels. Coronary heart disease developed with great consistency in patients with a ratio of total cholesterol to HDL cholesterol of more than 4.5.[6] Nitric oxide is involved in dilating blood vessels to provide more blood to heart. Nitric oxide tablets are extremely effective for angina patients. DMA inhibitor of nitric oxide synthesis. Chronic hypercholesterolemia stimulates DMA production through elevation of lipid peroxides and contributes to the development of atherosclerosis.[7] In the long term, systolic blood pressure, high cholesterol levels and smoking were associated with an increased risk of carotid stenosis in the elderly population. Cholesterol levels may be associated with endothelial function, thus potentially contributing to the increased risk of macrovascular disease due to elevated cholesterol levels.[8]

Hypercholesterolemia is associated with an enhanced platelet thrombus formation on an injured artery, increasing the propensity for acute thrombosis. Platelet thrombus formation at both high and low shear rates decreased as total and LDL cholesterol levels were reduced with pravastatin. Cholesterol lowering may therefore reduce the risk of acute coronary events in part by reducing the thrombogenic risk.[9]

Elevated levels of blood lipids are well-documented risk factors for CVD. Current classification schemes and treatment levels for hyperlipidemia are based on the NCEP/ATP III guidelines. Extensive research over the past decade has raised the question whether or not ATP III guidelines are sufficiently aggressive. New guidelines from ATP IV are expected to be released in the near future, but in the mean time physicians are faced with uncertainty about how low to target LDL cholesterol whether to pharmacologically treat HDL cholesterol and triglyceride levels and how best to achieve target goals.[10]

Primary Prevention Measures to Lower the Risk of CVD

The American Heart Association recommends all adults age 20 or older have their cholesterol and other traditional risk factors checked 4-6 years. Cholesterol score is calculated using following equation: HDL + LDL + 20% of triglyceride level. A total cholesterol score of less than 180 mg/dl is considered optimal.[11]

Statin Therapy[12]

- Before starting lipid modification therapy for the primary prevention of CVD, take at least one blood sample to measure lipid profile. This includes measurement of total cholesterol, HDL, LDL, and triglyceride concentrations.
- Offer atorvastatin 20 mg for the primary prevention of CVD to people who have a 10% or greater 10- year risk of developing CVD. Estimate the level of risk using QRISK2 assessment tool.
- Start statin treatment in people with CVD with atorvastatin 80 mg and it is used in lower doses when potential drug interactions; high risk of adverse effects; and patient preference.
- Measure cholesterol levels once in three months. If 40% reduction in cholesterol levels are not achieved then discuss adherence and timing of dose; optimize adherence to diet and lifestyle measures; and consider increasing dose if started on less than atorvastatin 80 mg and the person is judged to be at higher risk because of co-morbidities, risk score or using clinical judgment.
- Before starting statin treatment baseline blood tests and clinical assessment and treat comorbidities. Include all of the following in the assessment:

- Smoking status
- Alcohol consumption
- Blood pressure
- Body mass index or measure of obesity
- Total cholesterol
- HbA_{1c}
- Renal function and Glomerular function rate
- Transaminase level
- Thyroid stimulating hormone

A meta-analysis of the use of statins in primary prevention has been performed: seven trials with 42,848 patients were included. The results showed that in patients without CVD, statin therapy decreases the incidence of major coronary and cerebrovascular events and revascularizations but not coronary heart disease mortality and overall mortality.[13]

Dietary Precautions[12]

- Read food labels and choose foods with low cholesterol and saturated trans fat. The American Heart Association recommends aiming for a dietary pattern that limits saturated 5- 6% of daily calories and reduces the percent of calories from trans fat.
- Reduce saturated fat intake to 7% of daily calories
- Reduce fat intake to 25- 35% of daily calories
- Limit dietary cholesterol to less than 200 mg per day
- Limit intake of red meat and dairy products made with whole milk to reduce saturated and trans fat. Choose skim milk, low fat dairy products. Limit fried food, and use of healthy oil such as vegetable oil.
- Increase amount of fiber intake. It can lower cholesterol levels by 10 percent.
- Other foods that can help control cholesterol include cold- water fish such as mackerel, sardines and salmon. These fish contain omega-3 fatty acids that may lower triglycerides. Soy beans, soy nuts and many meat substitutes contain a powerful antioxidant that can lower LDL. A supplement known as psyllium increases soluble fiber intake. Psyllium helps absorb water and cholesterol from intestines and may improve the ratio of HDL to LDL.
- Advise to choose whole grain varieties of starchy food; reduce intake of sugar and food products containing refined sugars including fructose; eat at least 5 portions of fruits and vegetables per day; and eat at least 4 – 5

portions of unsalted nuts, seeds and legumes per week.

Exercise[12]

- Exercise makes up another component in primary prevention of hyperlipidemia. Walking briskly for 20-30 minutes most days of the week. Exercise can lose weight, relieve stress, raise HDL, and lower LDL and triglycerides. Regular checking of weight is essential.
- Advice to do muscle strengthening activities on two or more days a week that work all major muscle groups in line with national guidance for the general population.
- Encourage to perform moderate- intensity physical activity in case of co-morbidities. Also advice about physical activity should take into account the person's needs, preferences and circumstances.

Patient Education[12]

- Explain the risks associated with hyperlipidemia such as heart attack, stroke/ disability.
- Suggest strategies to minimize damage caused by alcohol and nicotine. Men should not regularly take more than 3- 4 units a day and women should not regularly drink more than 2-3 units a day.
- Offer the intensive support service to the population who want to quit smoking
- Support to work with them to achieve and maintain healthy weight
- Emphasize importance of adhering to treatment regimen. Utilize motivational interviewing to promote readiness for treatment and list referral services.

Conclusion

The burden of hyperlipidemia is alarming when considered by the perspective of morbidity and mortality. And it is one of the four established conventional risk factors for CVDs besides cigarette smoking, diabetes mellitus, and high blood pressure. A wealth of knowledge has evolved over the past half century linking atherosclerotic changes to CAD and stroke. Blood lipid abnormalities have been identified as a primary risk factor. Although lifestyle changes can prevent or slow down the progression of CAD, pharmacological management is frequently required. With physical exercise, balanced diet, stop smoking along with medication can control blood cholesterol at a large extent and all individuals who are susceptible should take the aforementioned measures strictly and thus prevent them from coronary events and death.

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