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CHOLESTEROL AND ITS ASSOCIATION WITH HEART DISEASE

Research Submitted To The Council Of Al-Hilla University College It Is Part Of The Requirements For Obtaining A Bachelor's Degree In Medical Physics

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بِسْمِ اللَّهِ الرَّحْمَٰنِ الرَّحِيمِ

(قَالَ إِنَّمَا الْعِلْمُ عِنْدَ اللَّهِ وَأُبَلِّغُكُمْ مَا أُرْسِلْتُ بِهِ)

صدق الله العلي العظيم

سورة الاحقاف - الآية ٢٣

Dedication

I dedicate this research, which is the summary of my undergraduate studies : my wonderful mother & father who supported me to reach what I am my dear brother and my sister my friends for their support in all my work my dear teachers, I will stay grateful.

Acknowledgment

First and foremost, all thanks, loyalty, devotion, thanksgiving and praise for Allah. And His Messenger and the Gate of the City of Knowledge, Imam Ali Ibn Abi Talib (peace be upon them). I would like to thank my honorable professor who helped me complete my research .I would like to express my deepest respect and gratitude for your patience, encouragement, meaningful instructions, value, advice and kindness within and throughout the work period on this project. I also extend my thanks and gratitude to all the other doctors for helping me provide all the work to make me this level.

Abstract

Cholesterol is a substance found naturally in the body, where it is used in building cell membranes and producing some sex hormones. Cholesterol is produced in the liver and transported to the blood, carried with special lipoproteins. There are two main types of cholesterol based on its lipoprotein: LDL cholesterol and HDL cholesterol. HDL cholesterol is called good or benign cholesterol because it picks up cholesterol from the walls of the arteries and returns it to the liver. Thus, it protects against atherosclerosis, coronary heart disease, and heart attacks, As for LDL cholesterol, it is called bad cholesterol because it is associated with atherosclerosis, as the cholesterol-carrying lipoprotein LDL deposits cholesterol on the walls of the arteries, leading over time to hardening and narrowing of these arteries, especially the coronary arteries that feed the heart muscle, and may cause heart attacks and coronary heart disease.

Blood cholesterol should be checked every 5 years after the age of 20, and treatment recommendations for high cholesterol depend on the level of total cholesterol, LDL cholesterol, HDL cholesterol, and the presence of other risk factors for coronary heart disease. Food should be low in cholesterol and saturated fats. Food rich in cholesterol includes meat, especially organic meat (liver - marrow - brain - kidneys - heart...), in addition to milk and its derivatives (butter - cream), animal ghee and egg yolks. As for food rich in unsaturated fats, such as some vegetable oils (corn oil, olive oil, sunflower oil), it can reduce blood cholesterol.

Lifestyle changes include losing weight, stopping smoking, abstaining from drinking alcohol and exercising regularly.

As for drug treatment, it may not be used except under medical supervision, because only the doctor decides the type, duration, and dose of treatment, especially since there are several drug groups that can lower blood cholesterol. The use of medication is not inevitable in treating high blood cholesterol, as in many cases it is sufficient to adhere to a diet and exercise regularly, in addition to stopping smoking and drinking alcohol, and losing excess weight.

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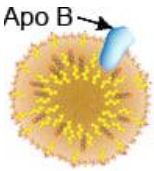
1.1 INTRODUCTION

Cholesterol is a waxy substance that the liver makes. People need cholesterol to support their health. It helps the body carry out important functions like digesting fatty foods and making hormones.

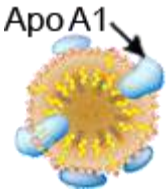
If a person has a total cholesterol level above 200 milligrams per deciliter (mg/dL)Trusted Source, they have high cholesterol. Doctors measure a person's total cholesterol level as the total of three substances in their bloodstream. [1]

There are two main types:

LDL (Low Density Lipoprotein) is the 'bad' cholesterol – increases the risk of cardiovascular disease, LDL transports cholesterol to the cells. The body's cells take up the LDL cholesterol via an LDL receptor (this is the process which is faulty in FH). This controls the amount of cholesterol in the blood. The protein component of LDL is called apolipoprotein B (Apo B).



HDL (High Density Lipoprotein) is the 'good' cholesterol – helps to protect against cardiovascular disease, HDL transports cholesterol from the cells to the liver for removal. HDL can also act as an antioxidant. The major protein component of HDL is called apolipoprotein A1 (Apo A1). [2]



1.1.1 The Relationship Between High Cholesterol and Heart Disease

When there is too much cholesterol in your blood, it builds up in the walls of your arteries. Over time, this buildup narrows the arteries and restricts blood flow to the heart. When the blood supply to a portion of the heart is completely cut off a heart attack results. [1]

Factors that Affect Your Cholesterol Levels,Many factors can affect your cholesterol level. Some of them you can control and others you can't:

- The Foods You Eat: Saturated fat and trans fats raise LDL levels. Saturated fats are mainly found in foods that come from animals, such as meat and dairy products. Trans fats are in foods made with hydrogenated or partially hydrogenated oils, such as margarine, packaged snacks, fast food, and baked goods.

- Your Weight: Being overweight is a major risk factor for heart disease and increases cholesterol. Losing excess weight can help lower your LDL and total cholesterol.
- Regular Exercise: The American Heart Association recommends getting 150 minutes or more of moderate-intensity physical activity .
- Smoking Habits: Smoking is a widely recognized risk factor for heart disease. [3]
- Age and Gender: For both women and men, cholesterol levels tend to rise with age. Before the age of menopause, women generally have lower total cholesterol levels than men of the same age. After that point, the gap narrows.
- Family History: High cholesterol and risk of heart disease may be in your genes. Familial Hypercholesterolemia (FH) is a hereditary condition that limits the liver's ability to metabolize or remove LDL cholesterol. As a genetic disorder, FH makes managing cholesterol and risk of heart disease through just eating right, controlling weight,

exercising regularly, and not smoking inefficient. Medication is always necessary to manage it. [2]

The last factor may be a surprise to many. In fact, 90 percent of individuals with FH do not know they have the disorder. Individuals with FH have a 20 time higher risk for heart disease compared to individuals without FH, which makes spreading awareness and knowledge about this disorder extremely important.[3]

1.1.2 High LDL cholesterol causes cardiovascular disease

If there is too much LDL cholesterol in the blood, it can deposit in the walls of the arteries giving rise to cardiovascular disease. These deposits are known as plaque or atheroma (pronounced ath-er-O-ma) and the process is called atherosclerosis (pronounced ath-er-O-skler-O-sis).

As shown in Figure a below, over time the plaque causes the arteries to narrow and eventually become blocked. This causes a lack of oxygen to the part of the organ supplied by the artery and will result in tissue damage, see Figure b below. Plaques can also rupture, forming a blood clot, which rapidly blocks the artery. [4]

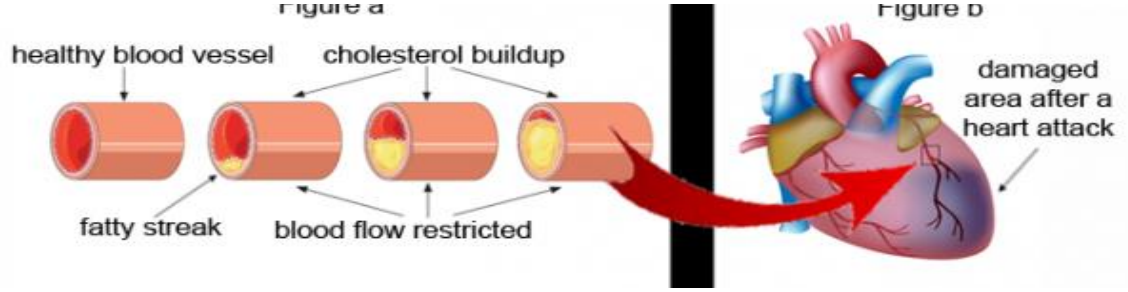


Fig 1: High LDL cholesterol causes cardiovascular disease

Depending on where the cardiovascular disease is located and how severe it is it can cause , angina or heart attacks if it occurs in the heart (known as heart disease, coronary heart disease (CHD) or coronary artery disease (CAD)), see Figure b above strokes if it occurs in the brain , other circulation problems (peripheral artery disease) if it occurs in other parts of the body e.g. leg pain when walking (intermittent claudication) and erectile dysfunction in men. The higher your LDL cholesterol, the greater your risk of getting cardiovascular disease. [5]

1.2 LITERATURE REVIEW

1.2.1 The relationship between blood cholesterol and heart disease

The relationship between blood cholesterol and heart disease is well-established, with the lowering of serum low-density lipoprotein (LDL)-cholesterol being the primary target of preventive therapy. Furthermore, epidemiological studies report lower risk for heart disease with higher concentrations of high-density lipoprotein (HDL)-cholesterol. [6]

There has also been considerable interest in studying the relationship between dietary cholesterol intake and heart disease risk. Eggs are one of the richest sources of cholesterol in the diet. However, large-scale epidemiological studies have found only tenuous associations between the intake of eggs and cardiovascular disease risk. Well-controlled, clinical studies show the impact of dietary cholesterol challenges via egg intake on serum lipids is highly variable, with the majority of individuals (~2/3 of the population) having only minimal responses, while those with a significant response increase both LDL and HDL-cholesterol, typically with a maintenance of the LDL/HDL cholesterol ratio. [7]

Recent drug trials targeting HDL-cholesterol have been unsuccessful in reducing cardiovascular events, and thus it is unclear if raising HDL-cholesterol with chronic egg intake is beneficial. Other important changes with egg intake include potentially favorable effects on lipoprotein particle profiles and enhancing HDL function. Overall, the increased HDL-cholesterol commonly observed with dietary cholesterol feeding in humans appears to also coincide with improvements in other markers of HDL function. However, more investigation into the effects of dietary cholesterol on HDL functionality in humans is warranted. [7]

1.2.2 Lipids and atherosclerosis

Coronary heart disease (CHD) results from atherosclerosis. This is a slowly progressive disease of the large arteries that begins early in life and develops over a period of many years. The early plaques begin as fatty streaks with variable amounts of smooth muscle cell proliferation, lipid accumulation, fibrous proliferation, and progression to a fibrous plaque.27 During plaque development, there may be periods of quiescence and regression interspersed with progression. Clinical disease results from progressive narrowing of the lumen of the affected vessel, hemorrhage into the plaque, or embolism-thrombus formation at the site of the plaque with vessel occlusion. Disease may go undetected until the first heart attack, and the first heart attack is often fatal. [8]

Epidemiologic, genetic, experimental, and clinical studies have provided a wealth of data supporting a causal relationship between high blood cholesterol levels and coronary heart disease. It must be kept in mind, however, that elevated blood cholesterol is only one of several important risk factors contributing to an increased predisposition to CHD . It has now been established with certainty that the reduction of elevated cholesterol levels will decrease the rate of CHD. Two clinical studies are important to note in this regard. [9]

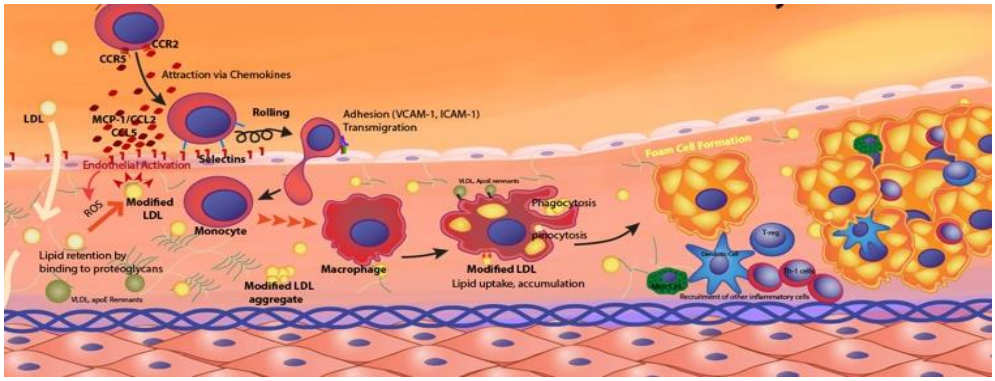


Fig 2: The Role of Lipids and Lipoproteins in Atherosclerosis.

1.2.3 Atherosclerosis and cholesterol hypothesis

The hypothesis of cholesterol suggests that lipids play a major role in the development of atherosclerosis. The 4S trial (Scandinavian Simvastanin Survival Study) showed that while there was significant reduction in total cholesterol level, LDL cholesterol level, and decrease in major coronary events, HDL cholesterol level was elevated in simvastatin receiving group. After 4S study, REVERSAL, ASTEROID, and SATURN studies revealed that parallel plaque regression was observed with aggressive lipid-lowering therapy and reduction in major cardiovascular events was achieved. These similar studies have proven the relationship between hyperlipidemia and atherosclerosis [6].

Statins reduce macrophages and extracellular lipid accumulation in atherosclerotic plaque region and increase the content of collagen in the extracellular matrix which result in intimal calcification. Statins also stabilize inflammation and coagulation cascade after plaque rupture.

1.2.4 Atherogenic dyslipidemia

In atherogenic dyslipidemia which is the result of an increase in triglyceride levels, triglyceride content is increased. The primary source of triglycerides is the VLDL. While LDL molecules are more easily oxidized, HDL molecules are more easily eliminated from the kidneys. Metabolic syndrome, type 2 diabetes, insulin resistance, abdominal obesity, and polycystic ovary syndrome are associated with atherogenic dyslipidemia. [10]

In the case of atherogenic dyslipidemia, since chylomicrons have no effect on atherosclerosis, non-HDL cholesterol level is used rather than triglyceride level. Although levels of LDL, VLDL, and chylomicron residues can be determined by detecting Apo B levels, there is limited access and standardization for the detection of Apo B level. In the ESC dyslipidemia guide, non-HDL cholesterol calculation is recommended instead of measuring ApoB levels in the presence of hypertriglyceridemia. (Class 2a) In a study conducted by Puri et al., the level

of non-HDL cholesterol rather than LDL cholesterol significantly correlated with atheromatous progression when the triglyceride level rises above 200 mg/dl. In the NICE guideline, all individuals are focused on evaluating non-HDL cholesterol exclusively from LDL cholesterol. [11]

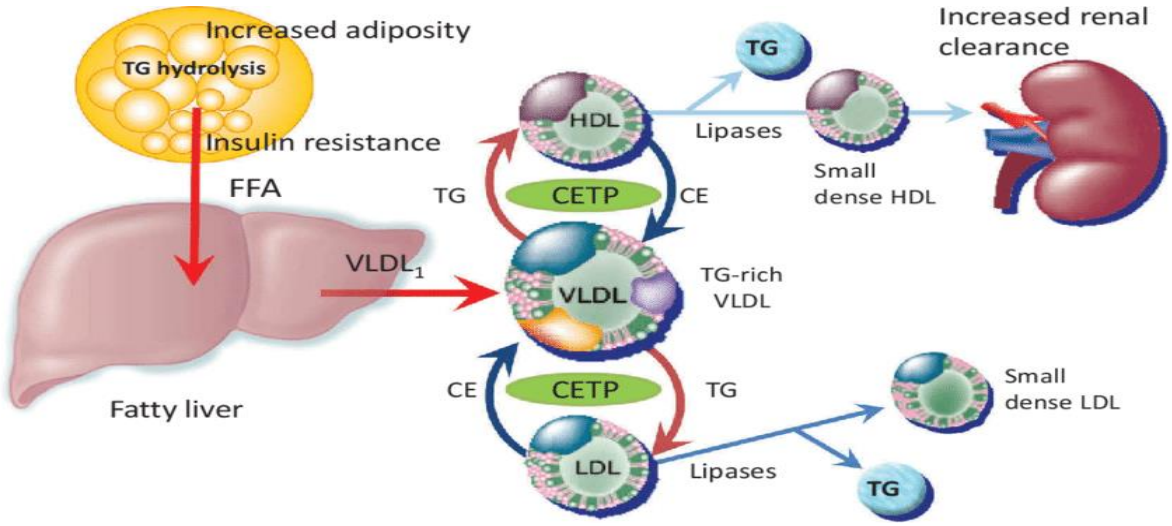


Fig 3 :Mechanism of atherogenic dyslipidemia in type 2 diabetes.

1.2.5 Treatment of hypercholesterolemia

Hyperlipidemia is defined on the basis of measurements of serum or plasma levels of lipids and comparison between age- and sex-matched population norms. An abnormally high level of a biologic substance is usually considered to be that level above the upper 5 percent of the population (95th percentile). The use of this criterion to define normal cholesterol levels in the United States is considered unreasonable because a large fraction of the population probably has levels of blood cholesterol that are too high, especially in view of the fact that CHD is the major cause of death. [11]

The average blood cholesterol level for middle-aged adults in the United States is about 215 mg/dL. The NIH Consensus Development Conference Statement on Lowering Blood

Cholesterol to Prevent Heart Disease3' recommends treatment of individuals with blood cholesterol levels above the 75th percentile (upper 25 percent of values). The consensus panel defined two levels of hypercholesterolemia "high risk" and "moderate risk." The high risk (severe hypercholesterolemia) category is defined as the upper 10 percent (90th percentile) as determined by the Lipid Research Clinics Prevalence Study. [11]

This group includes individuals with hereditary forms of high blood cholesterol, which require the most aggressive treatment. The moderate risk (moderate hypercholesterolemia) category is defined as between the 75th and 90th percentiles. This group includes a large number of individuals whose elevated blood cholesterol is due, in part, to their diet. The intensity of treatment is determined by the cholesterol level, clinical history, family history, and the presence of other coronary risk factors. Both the genetic and acquired forms of hyperlipidemia usually occur because of defects in one of the four sites of physiologic regulations10, increased hepatic triglyceride and VLDL production, decreased VLDL and chylomicron catabolism due to deficient lipoprotein lipase activity, decreased removal of remnants and catabolism, and impaired catabolism of LDL because of a deficiency in key LDL receptors. [10]

1.2.6 Diet Therapy

Diet therapy is the mainstay of treatment of patients with all forms of hyperlipidemia. The dietary prescription must be formulated on an individual basis and is dependent on the clinical situation. In the United States, mild to moderate hypercholesterolemia with an elevated level of LDL is often related to dietary habits with excesses in the ingestion of cholesterol and saturated fats. [12]

Thus, it is extremely important that the first step in evaluating and managing patients with hyperlipoproteinemia include a careful dietary history and that patients receive careful

detailed dietary instructions, preferably from a dietition. In many patients with primary hyperlipoproteinemia, blood lipid levels will return to normal on a dietary regimen alone. The average American man consumes about 500 mg of cholesterol daily. About 40 percent of calories in the average American diet come from fat, and 42 percent of this fraction is saturated fat. The American Heart Association and the Atherosclerosis Study Group of the Inter-Society Commission for Heart Disease recommend a threephase dietary approach. The initial phase (phase I) is a prudent diet for the general population at large. [12]

It consists of caloric reduction to attain ideal body weight, reduction of total cholesterol intake to 250 to 300 mg/d, a reduction in total fat intake to 30 percent of total calories, and a reduction in saturated fat intake to 10 percent or less of total calories to achieve a dietary polyunsaturated-to-saturated fat ratio of 1.0 rather than the usual 0.3. Fifty-five percent of calories should be eaten as carbohydrate and 15 percent as Patients with insufficient response to phase I and/or who have family histories of hyperlipidemia should move to phase II. The phase II diet restricts fat to 25 percent of total calories and cholesterol consumption to 200 to 250 mg/d. Carbohydrate intake is increased to 60 percent and protein intake is kept at 15 percent. [12]

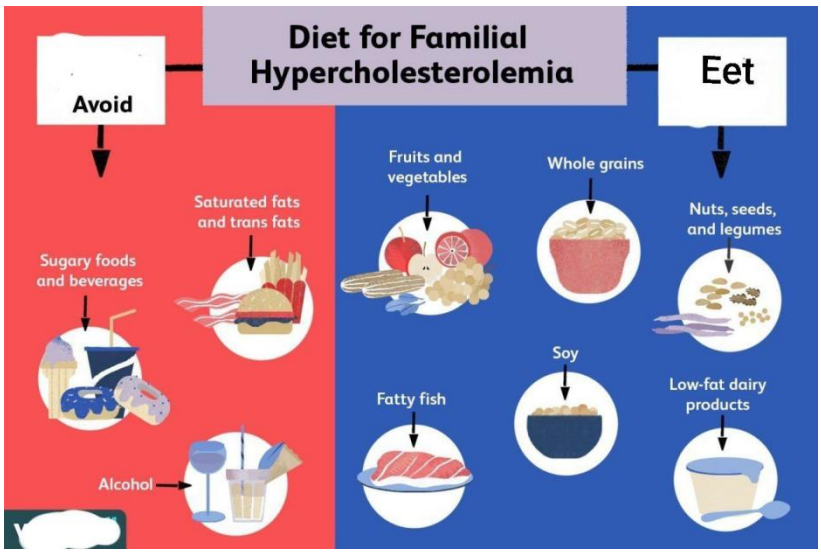


Fig4: Diet Therapy

1.2.7 Exercise

Exercise is frequently recommended as a means of modifying plasma lipid levels, although definitive answers await further investigation. On the basis of present evidence from cross-sectional and longitudinal studies, it appears that an exercise threshold needs to be surpassed before changes are realized. The level at which HDL cholesterol increases appears to be more than 8 to 15 miles ofjogging each week, and up to nine months may be required before the effects become apparent on clinically available tests. Thus, an absolute amount of additional energy expenditure (1,000 kcal/wk) appears necessary, and changes in HDL levels appear unlikely to occur in patients who do not achieve this level of activity. Elevated HDL, low LDL, and high HDL-to-total-cholesterol ratios have been found in men and women who habitually engage in endurance-type exercise. [13]

1.2.8 Drug Therapy

Patients who are unresponsive to dietary treatment may require additional aid with specific drug therapy.33-35 The hypolipidemic effects of currently available drugs are usually achieved through one of two mechanisms: increased clearance of lipoproteins, or decreased production of lipoproteins. Cholestyramine resin and colestipol hydrochloride increase lipid clearance, whereas clofibrate, nicotinic acid, and probucol decrease lipoprotein reduction. Cholestyramine is a bile acid sequestrant that is effective in reducing total cholesterol and LDL cholesterol levels. It is nonabsorbable from the gastrointestinal tract, has few systemic effects and and a low level of significant toxicity. It is the treatment of choice for patients with familial hypercholesterolemia. [13]

The usual dosage is 12 to 24 g/d. The most frequent side effects are gastrointestinal, which are usually ameliorated by a reduction in dose. Colestipol is also a bile acid sequestrant with an effect on plasma cholesterol and LDL similar to that of cholestyramine. It is also indicated

for the treatment of familial hypercholesterolemia. The average dosage is 5 g orally three times daily with a maximum dose of 30 grams. Probucol is effective in patients with mild to moderate elevations of LDL. Its mechanism of action is unclear and may be related to a lowering of LDL synthesis. When used in combination with colestipol, probucol has been reported to be effective in treating heterozygous familial hypercholesterolemia. Because probucol also lowers HDL levels, its usefulness in the prevention or control of coronary disease must be considered limited. [14]

Gemfibrozil is structurally similar to clofibrate and has a similar effect on lipoprotein metabolism. It decreases plasma levels of total cholesterol, LDL, and VLDL, but increases HDL concentration. The most common side effects are minor gastrointestinal symptoms and transient elevations in hepatic enzymes. Gemf'ibrozil potentiates the effects of warfarin and is contraindicated in patients with liver or kidney disease. It appears to be less lithogenic than clofibrate. There is considerable enthusiasm about the effects of gemfibrozil on increasing HDL, but the clinical significance of this and its effects on atherogenesis remain to be established. [14]



Fig5 : Gemfibrozil

Clofibrate is useful in the treatment of disorders characterized by increased VLDL and IDL. Its usefulness is limited in controlling elevations of LDL, and in some patients, it may increase levels of this lipid. In the Coronary Drug Project, this drug was associated with an increase in cholelithiasis, arrhythmias, angina, thromboembolism, and intermittent claudication in patients who had myocardial infarctions. [14]



Fig 6 : Clofibrate

Experim-ental Drugs: Many other drugs are currently being tested as lipid lowering agents.36 Among these are oral neomycin, compactin, and mevinolin. At a dose of 1 g twice daily, oral neomycin appears to be a safe, well-tolerated, and efficacious drug for lowering LDL cholesterol. Compactin is an agent that inhibits the activity of the enzyme 3-hydroxy-3-methylglutaryl coenzyme A reductase. [15]

1.3 CONCLUSIONS

As with smoking and hypertension, high blood cholesterol has been clearly established as one of the major modifiable risk factors associated with CHD. The recent evidence from the Lipid Research Clinics Coronary Primary Prevention Trial and the National Heart, Lung and Blood Institute Type II Coronary Intervention Study has shown that lowering cholesterol levels lowers CHD RISK and decreases the progression of atherosclerosis. Diet therapy alone may lower blood cholesterol levels by 10 to 15 percent and the combination of diet and drugs may result in a decrease of 30 percent or more. Furthermore, for each 1 percent that cholesterol is decreased, there is a 2 percent decrease in CHD risk. Thus, it is important that patients be advised about the risks of high blood cholesterol and the benefits of reducing it. A much more aggressive posture (as has occurred with hypertension) needs to be taken by physicians, a position important not only for the care of individual patients but for the general public as well.

1.4 RECOMMENDATIONS

- To achieve healthy dietary patterns, consumers are advised to eat a dietary pattern characterized by fruits, vegetables, whole grains, low-fat or fat-free dairy products, lean protein sources, nuts, seeds, and vegetable oils.
- Given the relatively high content of cholesterol in egg yolks, it remains advisable to limit intake to current levels. Healthy individuals can include up to a whole egg or equivalent daily. A 3-oz serving of shrimp is equivalent to about a whole egg.
- Patients with dyslipidemia, particularly those with diabetes mellitus or at risk for heart failure, should be cautious in consuming foods rich in cholesterol.

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جمهورية العراق وزراة التعليم العالي

والبحث العلمي

كلية الحلة الجامعة

قسم الفيزياء الطبية



الكوليسترول وارتباطة بأمراض القلب

بحث مقدم الى مجلس كلية الحلة الجامعة ضمن متطلبات الحصول على درجة البكالوريوس في الفيزياء الطبية

مقدم بواسطة

نرجس حيدر عبدالرضا

زينب علي عبيد

مريم عباس كامل

سجاد كاظم كاصد

بأشراف

د . احمد سلام عبود

الخلاصة

الكولسترول هو مادة موجودة بشكل طبيعي في الجسم، حيث يستخدم في بناء أغشية الخلايا وإنتاج بعض الهرمونات الجنسية،. يتم إنتاج الكولسترول في الكبد وينتقل إلى الدم، محملاً بالبروتينات الدهنية الخاصة. هناك نوعان رئيسيان من الكولسترول على أساس البروتين الدهني: كوليسترول LDL وكوليسترول .HDL

يسمى الكولسترول HDL بالكوليسترول الجيد أو الحميد لأنه يلتقط الكولسترول من جدران الشرايين ويعيده إلى الكبد وبالتالي فهو يحمي من تصلب الشرايين، وأمراض القلب التاجية، والنوبات القلبية،

أما الكولسترول LDL فيسمى بالكولسترول السيئ لأنه يرتبط بتصلب الشرايين، حيث يقوم البروتين الدهني الحامل اللكوليسترول LDL بترسب الكولسترول على جدران الشراين، مما يؤدي إلى وقت حدوث تصلب وتضيق هذه الشرايين، وخاصة الشرايين التاجية التي تغذي عضلة القلب، وقد تسبب النوبات القلبية وأمراض القلب التاجية.

يجب فحص نسبة الكوليسترول في الدم كل ة سنوات بعد سن العشرين، وتعتمد توصيات علاج ارتفاع نسبة الكوليسترول على مستوى الكوليسترول الكلي، والكوليسترول الضار، والكوليسترول الحميد، ووجود عوامل خطر أخرى للإصابة بأمراض القلب التاجية. يجب أن يكون الطعام منخفض الكوليسترول والدهون المشبعة. تشمل الأغذية الغنية بالكوليسترول اللحوم، وخاصة اللحوم العضوية االكبد النخاع - المخ - الكلى - القلب)...،
بالإضافة إلى الحليب ومشتقاته (الزبدة - القشدة والسمن الحيواني وصفار البيض.
أما الأغذية الغنية بالدهون غير المشبعة، مثل بعض الزيوت النباتية (زيت الذرة، زيت الزيتون، زيت دوار الشمس، فيمكن أن تقلل من نسبة الكولسترول في الدم.

تشمل تغييرات نمط الحياة فقدان الوزن والتوقف عن التدخين والامتناع عن شرب الكحول وممارسة الرياضة بانتظام

أما العلاج الدوائي فلا يجوز استخدامه إلا تحت إشراف طبي، لأن الطبيب وحده هو الذي يقرر نوع العلاج ومدته وجرعته، خاصة أن هناك عدة مجموعات دوائية يمكنها خفض نسبة الكولسترول في الدم، إن استخدام الأدوية ليس أمرا حتميا في علاج ارتفاع نسبة الكولسترول في الدم، إذ يكفي في كثير من الحالات الالتزام بنظام غذائي وممارسة الرياضة بانتظام، بالإضافة إلى التوقف عن التدخين وشرب الكحول، وفقدان الوزن الزائد