

THE IMPORTANCE OF LIPIDS IN ISCHEMIC HEART DISEASE

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Abstract: For nearly half a century, a comprehensive system for providing care to patients with cardiovascular diseases has been functioning in our Republic. Cardiological offices and departments, as well as cardiology centers, are equipped with instruments that fully meet international standards, and global protocols for diagnosis and treatment are being implemented. Scientists of our Republic contribute significantly to the development of cardiology science. In ischemic heart disease (IHD), elevated levels of triglycerides (TG), free fatty acids, and cholesterol (CH) are observed in the blood. For the development of atherosclerosis, not only the absolute amounts of lipids are important but also their circulation characteristics in the blood. In the blood, lipids are found in complexes with proteins in the form of lipoproteins, which include cholesterol, triglycerides, phospholipids, and proteins. Their physicochemical properties depend on the concentration and type of lipids and proteins.

Keywords: IHD, cholesterol (CH), triglycerides (TG), phospholipids, atherosclerosis, fatty acids, lipoproteins, proteins.

ЗНАЧЕНИЕ ЛИПИДОВ ПРИ ИШЕМИЧЕСКОЙ БОЛЕЗНИ СЕРДЦА

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Аннотация: Уже почти полвека в нашей Республике функционирует разветвлённая система оказания помощи больным с сердечно-сосудистыми заболеваниями. Кардиологические кабинеты и отделения, а также кардиологические центры оснащены оборудованием, полностью соответствующим мировым стандартам, и внедряются международные протоколы диагностики и лечения. Учёные нашей Республики вносят значительный вклад в развитие кардиологической науки. При ишемической болезни сердца (ИБС) наблюдается повышение уровня триглицеридов (ТГ), свободных жирных кислот и холестерина (ХС) в крови. Для развития атеросклероза важны не только абсолютные количества липидов, но и особенности их циркуляции в крови. В крови липиды находятся в комплексе с белками в виде липопротеинов, в состав которых входят холестерин, триглицериды, фосфолипиды и белки. Их физико-химические свойства зависят от концентрации и типа липидов и белков.

Ключевые слова: ИБС, холестерин (ХС), триглицериды (ТГ), фосфолипиды, атеросклероз, жирные кислоты, липопротеины, белки.

YURAK ISHEMIK KASALLIGIDA LIPIDLARNING AHAMIYATI

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Annotatsiya: Respublikamizda deyarli yarim asrdan beri yurak-qon tomir kasalliklari bilan og‘rigan bemorlarga ko‘mak ko‘rsatishning rivojlangan tizimi faoliyat yuritmoqda. Kardiologik kabinetlar va bo‘limlar, shuningdek, kardiologik markazlar zamonaviy dunyo standartlariga to‘liq javob beruvchi uskuna bilan jihozlangan va diagnostika hamda davolashning xalqaro protokollari joriy etilmoqda. Respublikamiz olimlari kardiologiya fanining rivojlanishiga katta hissa qo‘shmoqdalar. Ishemik yurak kasalligi (IYK)da qon tarkibida triglitseridlar (TG), erkin yog‘ kislotalari va xolesterol (XS) miqdori oshadi. Ateroskleroz rivojlanishida lipidlarning umumiy miqdoridan tashqari ularning qon aylanishidagi xususiyatlari ham muhimdir. Qonda lipidlar oqsillar bilan birlashib, lipoproteinlar ko‘rinishida bo‘ladi, ularning tarkibiga xolesterol, triglitseridlar, fosfolipidlar va oqsillar kiradi. Ularning fizik-kimyoviy xossalari lipidlar va oqsillar konsentratsiyasi hamda turiga bog‘liq.

Kalit so‘zlar: IYK, xolesterol (XS), triglitseridlar (TG), fosfolipidlar, ateroskleroz, yog‘ kislotalari, lipoproteinlar, oqsillar.

Atherosclerosis (from Greek “athere” – mush and “skleros” – hard) is a chronic disease in which fatty infiltrates form in the walls of blood vessels, connective tissue develops, and fibrous plaques form, leading to impaired physiological function of the vessels and disruption of blood circulation in the body and individual organs. Fats in the blood (cholesterol and triglycerides) are bound to proteins forming complexes called lipoproteins (LP). The involvement of lipoproteins in atherogenesis depends on their size. The smallest lipoproteins (5–12 nm) – high-density lipoproteins (HDL) – easily penetrate and exit the arterial walls, so they do not participate in atherogenesis. Low-density lipoproteins (LDL) (18–25 nm), intermediate-density lipoproteins (IDL) (25–35 nm), and some very-low-density lipoproteins (VLDL) (about 50 nm) penetrate the arterial walls, get oxidized, and are retained there, contributing to the development of atherosclerosis. Large lipoproteins – chylomicrons (75–1200 nm) and large very-low-density lipoproteins (about 80 nm) – do not penetrate the arterial walls and do not participate in atherogenesis. There is a direct correlation between the amount of LDL and the development of ischemic heart disease (IHD). The higher the LDL, the greater the risk of IHD. There is an inverse relationship between HDL levels and IHD

development: the higher the HDL, the lower the risk of disease. Triglycerides predominantly compose chylomicrons (80–95%). They are synthesized from dietary fats in the small intestine. Very-low-density lipoproteins contain 55–80% triglycerides; however, triglycerides are not considered significant in atherogenesis since chylomicrons and very-low-density lipoproteins do not penetrate arterial walls. LDL and HDL contain small amounts of triglycerides (5–15%).

Types of Hyperlipidemias:

There are five known types of dyslipidemias:

- Type I is characterized by elevated chylomicrons and high triglyceride levels.
- Type IIa involves high levels of LDL cholesterol.
- Type IIb involves elevated levels of both LDL and VLDL, with high cholesterol and triglycerides in the blood.
- Type III shows increased chylomicrons and intermediate lipoproteins, with elevated plasma cholesterol and triglycerides.
- Type IV involves elevated triglycerides and very-low-density lipoproteins with normal cholesterol levels.
- Type V is characterized by pronounced increases in triglycerides and cholesterol.

Dyslipidemias are classified as primary (genetic) and secondary (associated with various diseases).

Plasma lipids are divided into classes by mobility and density using ultracentrifugation or electrophoresis (on polyacrylamide gel):

- Chylomicrons (CM)
- Low-density lipoproteins (LDL) or β -lipoproteins
- Very-low-density lipoproteins (VLDL) or pre β -lipoproteins
- High-density lipoproteins (HDL) or α -lipoproteins

Chylomicrons mainly contain exogenous triglycerides, LDL – cholesterol, VLDL – endogenous triglycerides, and HDL – phospholipids.

D. Frederickson classified five types of hyperlipoproteinemias, which may be primary or secondary:

- Type I – chylomicrons
- Type IIa – cholesterol and triglycerides
- Type IIb – predominantly cholesterol and β -lipoproteins
- Type III – “pathological” floating β -lipoproteins
- Type IV – endogenous triglycerides, pre β -lipoproteins
- Type V – chylomicrons and β -lipoproteins

Types IV and V are usually secondary since carbohydrate metabolism disorders can cause these conditions. Lipids in blood plasma are transported as complex complexes – lipoproteins, which are classified by density, size, and apoprotein and lipid composition into groups: chylomicrons (CM), very-low-density lipoproteins (VLDL), intermediate-density lipoproteins (IDL), low-density lipoproteins (LDL), and high-density lipoproteins (HDL). Very-low-density lipoproteins (VLDL) structurally and compositionally resemble chylomicrons and have a density of about

0.95–1.006 g/ml. Their main structural functional proteins are B-100 and apo C-I, C-II, C-III. VLDL mainly consist of endogenous triglycerides and small amounts of cholesterol esters. Increased VLDL concentration in blood leads to hypertriglyceridemia, which is often observed in insulin-independent diabetes mellitus, hypothyroidism, and obesity. If hypertriglyceridemia is accompanied by decreased HDL, it is considered a high-risk factor for atherosclerosis. Intermediate-density lipoproteins (IDL) contain a large amount of cholesterol esters and a small amount of VLDL. Their main transport proteins are B-100 and apo E. The density of IDL is 1.006–1.019 g/ml. Elevated IDL levels cause hypercholesterolemia and hypertriglyceridemia. Isolated IDL elevation is rare and associated with hereditary defects of hepatic lipoprotein lipase, which contributes to the development of atherosclerosis. Low-density lipoproteins (LDL) have a density of about 1.019–1.063 g/ml and mainly contain cholesterol esters with apoprotein B-100. Increased LDL concentration is associated with the development of coronary artery atherosclerosis. For LDL to exhibit atherogenic properties, they must undergo modification, the main one being lipid peroxidation. Oxidation of LDL alters their properties: interaction with liver receptors is disrupted, and they become active chemoattractants for monocytes. Activated monocytes penetrate the subendothelial layer of the vessel wall, transform into macrophages, which phagocytize modified LDL, forming foam cells filled with cholesterol esters. Activated macrophages and foam cells release biologically active substances – growth factors, proinflammatory cytokines, and adhesion molecules – leading to increased endothelial permeability, growth of atherosclerotic plaques, narrowing of the vessel lumen, and thrombosis upon plaque rupture. High-density lipoproteins (HDL) have a density of 1.063–1.210 g/ml and are considered anti-atherogenic because they transport cholesterol from the vessel walls and macrophages to the liver. HDL is divided into subgroups HDL-2 and HDL-3, which have a discoid shape. The level of HDL cholesterol in the blood is important: the higher the HDL level, the lower the risk of atherosclerosis. Numerous clinical and epidemiological studies confirm the important role of types II, III, and IV in the development of atherosclerosis.

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