Dyslipidemia

Dyslipidemia (dyslipoproteinemia; formerly hyperlipoproteinemia - HLP) is a group of metabolic diseases that are characterized primarily by elevated plasma lipid or lipoprotein concentrations due to their increased synthesis or decreased degradation. However, an increase in some blood lipid fraction is often combined with a decrease in HDL cholesterol .

Dyslipidemia is one of the most important risk factors for atherosclerosis . Complications of atherosclerosis (acute myocardial infarction , stroke , ischemic disease of the lower limbs) occupy a leading position in the causes of mortality and morbidity not only in our country, but in virtually all developed countries.

Classification of dyslipidemias

Dyslipidemia (hereinafter DLP) can be classified according to various criteria:

1. Classification by cause

Primary dyslipidemia

They are genetic disorders of lipoprotein metabolism. Most HLPs are thought to be primary (eg, familial hypercholesterolemia, familial hypertriacylglycerolemia).

Secondary dyslipidemia

They are the result of another disease that disrupts lipid and lipoprotein metabolism. They may be manifested by an isolated increase in cholesterol or triacylglycerols or both. It often accompanies eg diabetes mellitus , hypothyroidism , liver disease, obesity , chronic alcoholism . Their danger lies in their long asymptomatic period, the sudden manifestation then occurs as a complication of atherosclerosis in various places or as acute hemorrhagic pancreatitis.

2. Therapeutic classification

Classification of dyslipidemias according to the European Society for Atherosclerosis (1992)

It represents a simple and practical division of DLP based on the determination of serum cholesterol and triacylglycerol levels into three groups. It is the basis for deciding on a therapeutic approach.

Hypercholesterolemia

Isolated increase in total cholesterol, especially in the LDL fraction. We usually encounter familial hypercholesterolemia and polygenic hypercholesterolemia.

- ***Familial hypercholesterolemia (FH)*** is an autosomal dominant disease caused by a genetic disorder in the production or function of LDL receptors . In homozygotes, LDL catabolism by LDL receptors is practically non-functional; in heterozygotes, the capacity of LDL receptors is halved. As a result, atherogenic LDL particles accumulate in the blood. The heterozygous form is more common and occurs in about 1 case per 500 people. Homozygotes have been severely affected since childhood, with tendon and skin xanthomas, and most of them die of myocardial infarction within the age of 20. In affected heterozygous persons, it manifests as a premature occurrence of cardiovascular diseases (coronary heart disease aged 30–50 years), and *arcus senilis corneae*, *xanthelasma palpebrarum* or tendon xanthomatosis. Total cholesterol concentrations are around 7-10 mmol / l for heterozygotes and around 15-30 mmol / l for homozygotes. The changes in the lipoprotein spectrum correspond mainly to phenotype IIa, less often to IIb (according to Fredrickson).
- Genetic as well as environmental influences apply to polygenic ***hypercholesterolemia*** . We meet it very often in industrialized countries. Total cholesterol levels do not usually exceed 8 mmol / l, but already represent an increased risk of atherosclerosis. The changes in the lipoprotein spectrum correspond mainly to phenotype IIa, less often to IIb.
- ***Secondary hypercholesterolemia*** can be found , for example, in hypothyroidism, nephrotic syndrome, in a diet rich in saturated fats.

Combined hyperlipidemia

Simultaneous increase in cholesterol and triacylglycerols.

- ***Familial combined hyperlipidemia*** is one of the most common primary HLP. It occurs in the frequency of 1:50 to 1: 100. It is based on genetically determined increased production of apolipoprotein B100. It is associated with an increased risk of vascular disease. LDL and VLDL, corresponding to phenotype IIb, are usually increased, but we also encounter phenotype IIa, IV and V.
- ***Secondary forms*** *are* found, for example, in hypothyroidism, in the treatment of corticoids.

Hypertriacylglycerolemia

Isolated increase in triacylglycerols:

- Genetic hypertriacylglycerolemia is ***familial hypertriacylglycerolemia*** , which affects about 0.2-0.3% of the population. It is manifested by an increase in VLDL, probably due to their increased production. At the same time, we find a reduced level of HDL-cholesterol. In the laboratory finding, we encounter slightly elevated triacylglycerols, usually up to 6 mmol / l at normal cholesterol concentrations. Patients are at risk of myocardial infarction.
- We can rarely encounter ***familial hyperlipoproteinemia type I*** , characterized by hyperchylomicronemia. Patients are at risk of pancreatitis caused by high levels of triacylglycerols (often over 20 mmol /), which increases its risk.
- ***Secondary forms of*** hypertriacylglycerolemia are often associated with diabetes mellitus, obesity, excessive alcohol intake or a high carbohydrate diet.

3. Fredrickson classification

The Fredrickson classification is historically the first classification of disorders of lipoprotein metabolism. It is currently being abandoned because it does not explain the root cause of the disease and is being replaced by a more modern approach, using new knowledge in the etiopathogenesis of HLP. We will discuss it briefly, because we still meet it in older literature. Based on the concentration of cholesterol and triacylglycerols in serum and electrophoretic examination of lipoproteins, HLPs were divided into 5 lipoprotein types - phenotypes (according to the WHO, type II was later divided into IIa and IIb). However, the lipoprotein type is only a current picture of the state of lipid and lipoprotein metabolism..

Laboratory

standard:

- Total cholesterol: <5 mmol / l
- LDL-cholesterol: <3 mmol / l
- TAG: <1.7 mmol / l
- HDL: in men> 1 mmol / l; in women> 1.2 mmol / l

Links

Related

- Lipoproteins (clinic)
- Lipoproteins
- Biochemical examination in hyperlipoproteinemia
- Hypolipidemics
- Hypolipidemic treatment
- Familial hypercholesterolemia

References

- BURTIS, CA and ER ASHWOOD. *Tietz Textbook of Clinical Biochemistry.* 2nd edition. Philadelphia: WBSaunders Company, 1994. ISBN 0-7216-4472-4 .
- ČEŠKA, R., et al. *Cholesterol and atherosclerosis, treatment of dyslipidemias.* 1st edition. Prague: Triton, 2005. ISBN 80-7254-738-0 .
- KLENER, Pavel, et al. *Internal Medicine.* 4th edition. Prague: Galén, Karolinum, 2011. 1174 pp. ISBN 978-80-7262-705-9 .
- MASOPUST, J. *Clinical Biochemistry. Requirement and evaluation of biochemical examinations I. and II. part.* 1st edition. Prague: Karolinum, 1998. ISBN 80-7184-650-3 .
- RACEK, J., et al. *Clinical biochemistry.* First edition. Prague: Galén - Karolinum, 1999. ISBN 80-7262-023-1 .
- SCHNEIDERKA, P., et al. *Chapters from clinical biochemistry.* 2nd edition. Prague: Karolinum, 2004. ISBN 80-246-0678-X .
- SOŠKA, V. *Disorders of lipid metabolism. Diagnosis and treatment.* 1st edition. Prague: Grada, 2001. ISBN 80-247-0234-7 .
- STERN, P., et al. *General and clinical biochemistry for bachelor's fields of study ..* 1st edition. Prague: Karolinum, 2005. ISBN 978-80-246-1025-2 .
- TÁBORSKÁ, E., et al. *Biochemistry II - Practical exercises.* 3rd edition. Brno: Masaryk University, 1999. ISBN 978-80-210-4516-3 .
- ZIMA, T., et al. *Laboratory diagnostics.* 1st edition. Prague: Galén, 2002. ISBN 80-7262-201-3 .

External Links

- Lipoproteins (czech wikipedia)
- Lipoproteins (english wikipedia)

Recomended Litrature

- FIALOVÁ, L. and M VEJRAŽKA. *Selected biochemical examinations in patients with lipid metabolism disorders* [online]. © 2005. Last revision March 20, 2008, [cited. September 7, 2009]. < https://el.lf1.cuni.cz >.
- VAVERKOVÁ, Helena, et al. Recommendations for the diagnosis and treatment of dyslipidemia in adulthood, developed by the committee of the Czech Society for Atherosclerosis. *Cor Vasa* [online] . 2007, vol. 49, vol. 3, pp. K73-K86, also available from < http://www.athero.cz/user_data/zpravodajstvi/obrazky/File/Doporuceni%20CSAT-07.pdf+>. ISSN 1803-7712.