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The State of Carbohydrate and Lipid Metabolism in as in Women

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Abstract: Free radical processes with the development of oxidative stress stimulate the expression of cytokines by T-lymphocytes and macrophages, are able to inhibit endothelium-dependent vasodilation (ESVD) of blood vessels and cause vasoconstrictor reactions [Landmesser U., Spickermann S., Dikalov S. et al., 2002; Davignon I., Ganz P., 2004; Lubos E., Handy D.E., Loscalzo D., 2005].

Pro- and anti-inflammatory cytokines mediate intercellular interactions and support local inflammation in the atherosclerotic plaque by activating endothelial cells, inducing the expression of adhesion molecules and prothrombotic activity of the endothelium [Chereshnev VA, Gusev E, 2012; Lutay MI, Golikova IP, Slobodskoi VA., 2007; Libby P., Okamoto Y., Roscha V .Z., Folco E., 2010].

Meanwhile, the most complete information about the role of oxidative stress, immune inflammation and endothelial dysfunction in the development of pathological conditions is presented on the basis of experimental work.

At the same time, clinical studies on the comprehensive assessment of immuno-inflammatory reactions taking into account the state of the POL system and antioxidant enzymes (AOF), the activity of pro- and anti-inflammatory cytokines, a complete analysis of endothelial function with the study of its vasodilating, vasoconstrictor and adhesive properties in relation to hemodynamic parameters and the severity of the course of coronary heart disease are few [Lankin VZ, Tihaze AK, Belenkov YN.,2000; Chereshnev VA, Gusev E, 2012; Yogiatzi G., Tousoulis D., Stefandis C., 2009; Lutay MI, Golikova IP, Slobodskoi VA., 2007; Libby P., Okamoto Y., Roscha V.Z., Folco E., 2010; Ivanov SG, Sitnikova MY , Shlyakhto EV., 2006; Belenkov YN, Tatenkulova SN, Mareev VY et al., 2009; Gonzalez Y.A., Selwyn A.P., 2003; Taub P.R., Gabbat-Saldate P., Maisel A., 2010; Biasillo G., Leo M., Della Bona R., Biasacci M., 2010].

There is evidence in the literature that the intensification of free radical processes in myocardial ischemia not only induces hyperproduction of cytokines, but also promotes activation and damage to the endothelium [Belardinelli R., Tiano J., Iittarri G P., 2008; Misra M.K., Sarwat M., Bhakun P. et al., 2009].

In the experiment, it was found that oxidative stress activates immune responses in the vascular wall with the expression of a number of pro-inflammatory cytokines [Yogiatzi G., Tousoulis D., Stefandis C., 2009; Lubos E., Handy D.E., Loscalzo D., 2005; Libby P., Okamoto Y., Roscha V.Z., Folco E., 2010]

Lipid peroxides and AOF have a regulating effect and contribute to the excessive production of proinflammatory cytokines in patients with coronary heart disease. The fact that there is a close

relationship between the indicators of proinflammatory cytokines and hemodynamic parameters seems to indicate that overexpression of cytokines, in particular TNF-α, in the blood and myocardium can have a damaging effect on the myocardium and can induce myocardial remodeling processes with the development of diastolic and systolic LV dysfunction.

According to the results of the Framingham study, in patients with stable angina, the risk of developing nonfatal myocardial infarction and death from coronary heart disease within 2 years is, respectively, 14.3 and 5.5% in men and 6.1 and 3.8% in women.

Angina pectoris occurs if the work of the heart and the myocardial oxygen demand exceed the ability of the coronary arteries to supply the corresponding areas of the myocardium with a sufficient amount of oxygenated blood. It is believed that pain with angina is a direct manifestation of myocardial ischemia, leading to the accumulation of under—oxidized metabolites in the heart muscle. As myocardial ischemia develops, the blood pH in the coronary sinus decreases, intracellular potassium is lost, and instead of lactate utilization, its increased production begins. Pathological ECG changes appear, the mechanical performance of the ventricles is disrupted. During an attack of angina, diastolic pressure in the cavity of the left ventricle (LV) often increases, sometimes so much that pulmonary congestion occurs or shortness of breath develops.

During the Framingham study, it was found that the frequency of coronary heart disease in middle-aged men whose blood pressure exceeds 160/95 mm Hg is 5 times higher than in men with normal blood pressure (BP) [13,31].

According to other data, an increase in blood pressure for every 10 mm Hg increases the risk of developing heart disease by 30% [85, 168].

At the same time, the negative effect of lipid disorders and atherosclerosis on the blood pressure level has been proven. Thus, when conducting a study among American residents, it turned out that the level of TG and LDL cholesterol in blood plasma is associated with the presence of arterial hypertension (AH) in them [85].

One of the most frequent and dangerous "companions" of hypertension is a violation of the lipid composition of the blood, found in 40-85% of patients with hypertension, which is very often clinically manifested by the development of various forms of coronary heart disease (CHD). The level of blood pressure and plasma cholesterol, along with age, gender and smoking, form the basis of the 10-year risk of death from cardiovascular diseases assessment scale.

According to R.G. Oganov, up to 50% of mortality in patients with hypertension is associated with the development of myocardial infarction and cerebral stroke [51, 85].

It is known that an increase in cholesterol levels in patients with hypertension by 1% increases the risk of coronary heart disease by 2% [123].

In AS, chest pain is most often similar to that in typical angina pectoris: the same localization and reaction to nitrates, but without a clear connection with provoking factors. Often the pain is described as at rest, starting from a low level of intensity, which gradually increases, persists for 15 minutes, and then its intensity slowly decreases. This characteristic should alert the doctor about the possibility that there is a coronary vascular spasm. Another atypical picture is pain, localization and characteristics of angina pectoris, which occurs during exercise, but stops some time after exercise or can be stopped by taking nitrates. Most often it occurs in patients with microvascular angina.

Classification of chest pain is used to differentiate angina pectoris.

Traditional clinical classification of chest pain

- Typical angina pectoris (proven) — there are all three complete characteristics:
- ✓ discomfort behind the sternum of a typical nature and duration
- ✓ provoked by physical or psychoemotional stress
- ✓ relieved at rest and / or taking nitrates for minutes.

- ✓ Atypical angina pectoris (probable) — the presence of two of these characteristics.
- ✓ Non—anginal chest pain - the presence of only one of the above characteristics or their absence

The pain syndrome is stopped or reduced when the load stops. Taking nitrates in the vast majority of cases completely stops the attack within 1-2 minutes.

During the objective examination and examination of patients, to differentiate angina from its atypical form, we used this classification of pain.

To study the state of carbohydrate-lipid metabolism in patients with AS, a biochemical blood test was performed.

To study the state of lipid metabolism, taking into account gender, the level of total cholesterol, high–density lipoproteins (HDL), triglycerides (TG) were determined using standard kits, the level of low-density lipoprotein cholesterol (LDL) was determined by the turbometric method.

The atherogenicity index was calculated using the following formula:

Atherogenicity index (conl. units) = (OHC – HDL) / HDL, where-

- ✓ OHC- total cholesterol;
- ✓ HDL- high-density lipoproteins. The value of no more than 3.0 was considered to be the norm of the atherogenicity index

The obtained results of a biochemical study of the patients' blood showed distinctive sides in the comparison group. Thus, the fasting glucose level in women averages 8.4± 3.6 g/l, in patients of the comparative group 6.5± 2.9 g/l. This indicates a tendency to hyperglycemia in AS in women.

Low–density lipoproteins (LDL) are a fraction of lipoproteins responsible for the transfer of cholesterol to the cells of tissues and organs. LDL is the main transprot form of cholesterol, transferring it mainly in the form of cholesterol esters. They transport lipids, including cholesterol, from one cell population to another [22].

The result of the analysis of LDL levels in patients with AS was significantly increased by 2.0 times in relation to the comparative group: 3.8±0.39 mmol/l and 1.87±0.6 mmol/l, respectively, ($P<0.05$).

Consequently, the data obtained show that starting from the age of 47, women have an increased risk of developing cardiovascular diseases as a result of the activation of the transport of cholesterol and lipids at the cellular and tissue levels.

High-density lipoproteins consist mainly of the protein part and contain a little cholesterol. Their main function is to transfer excess cholesterol back to the liver, from where they are excreted as bile acids. Therefore, HDL cholesterol (HDL cholesterol) is also called "good cholesterol".

The study of HDL levels showed their tendency to decrease in women with AS to 1.79±0.4 mmol/l against the indicators of the comparison group-2.17± 0.4 mmol/l, which confirms the state of dyslipidemia. At the same time, low HDL levels contribute to the development of atherosclerosis, which causes the development of AS.

According to the new provisions of the VII revision of the ESC/EAS Recommendations for the correction of dyslipidemia (DLP) and reduction of cardiovascular risk (SSR) in clinical practice [13, 22], a lower target level of low-density lipoprotein cholesterol (LDL) for the category of very high SSR – 1.4 mmol/l was proposed. The category of extreme risk has been introduced. [22]. This leads to the conclusion that women in both study groups have a high risk of cardiac complications. At the same time, against the background of atherosclerosis, lipids are actively transported with the formation of visceral fat.

It is known that triglycerides (TG) are the main source of energy for the body at its cellular level. Usually, TG enters the body with food. Depending on the age, they are synthesized in adipose tissue, in the liver and in the intestine.

In studies in women of the main group, TG increases to 1.8± 0.82 mmol/l versus the comparison group-1.47± 0.39 mmol/l. Although the data obtained were not statistically significant, in a comprehensive assessment they confirm an increase in the synthesis of TG in adipose tissue in women aged 50 years and older.

As a result, the atherogenicity index tended to increase by 1.54 times, confirming the fact that hypercholesterolemia in women with AS is accompanied by activation of lipid transport to internal organs and the formation of visceral fat.

The study of the nature of inflammation in AS in women allows predicting the outcome of the underlying disease and choosing tactics for further management of patients in this category. Laboratory blood parameters at the same time showed a tendency to decrease Hb to 108.6±8.6 g/l in the main group of patients, and creatinine was slightly increased to 102.7±28.6 mmol/l against the indicators of the comparison group: 117.8±9.0 g/l and 88.13±7.5 mmol/l, respectively.

Thus, the study of the biochemical spectrum of blood in AS showed that women have an increased risk of developing cardiovascular diseases from the age of 47 as a result of activation of cholesterol and lipid transport at the cellular and tissue levels. At the same time, hypercholesterolemia on the background of anemia and uremia is accompanied by activation of lipid transport to internal organs and the formation of visceral fat.

LIST OF LITERATURE

1. Активность воспалительно-деструктивных изменений в процессе формирования нестабильной атеросклеротической бляшки / Рагино Ю.И. и соавт.// Кардиология, 2007; 9: 62–67.
2. Аляви А.Л., Низомов А.А., Касимова Г.М. Клинико-диагностическое значение активации цитокинов и возможности коррекции выявленных нарушений у больных острым коронарным синдромом с подъемом сегмента ST // Тезисы Российского национального конгресса кардиологов 2012. № 0024. С. 43-44.
3. Белоконева К.П., Бичан Н.А. Провоспалительные цитокины при различных осложнениях инфаркта миокарда // Вестник Уральской медицинской академической науки. 2012. № 4. С. 87.
4. Белоцкий С.М., Авталион Р.Р. Воспаление. Мобилизация клеток и клинические эффекты. – М.: Издательство БИНОМ, 2008. – 240 с.
5. Взаимосвязи уровня фракталкина и показателей эхокардиоскопии при артериальной гипертензии, стенокардии напряжения и их сочетании. / Тарасочкина Д.С., Полунина Е.А., Севостьянова И.В., Воронина Л.П., Кантемирова Б.И. // Кубанский научный медицинский вестник. 2015. №4. URL: https://cyberleninka.ru/article/n/vzaimosvyazi-urovnya-fraktalkina-i-pokazateley-ehokardioskopii-pri-arterialnoy-gipertenzii-stenokardii-napryazheniya-i-ih-sochetanii
6. Гальцева Н.В. Реабилитация в кардиологии и кардиохирургии. // Клиницист. 2015: 2: 13–22.
7. Гареев И.Ф. Роль микроРНК в регуляции патофизиологических механизмов при артериальной гипертензии //Наука молодых – Eruditio Juvenium. 2018. №4. URL: https://cyberleninka.ru/article/n/rol-mikrornk-v-regulyatsii-patofiziologicheskih-mehanizmov-pri-arterialnoy-gipertenzii
8. Диагностика и коррекция нарушений липидного обмена с целью профилактики и лечения атеросклероза. // Российские рекомендации, VII пересмотр. 2020; 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk.
9. Закирова А.Н., Закирова Н.Э. Роль перекисного окисления липидов, антиоксидантной защиты и реологических нарушений в развитии ишемической болезни сердца. // Российский Кардиологический Журнал 2006; 2:24-7.

10. Закирова Аляра Нурмухаметовна, Закирова Нэлли Эриковна Экспрессия провоспалительных цитокинов при стабильной стенокардии // CardioСоматика. 2013. №1. URL: https://cyberleninka.ru/article/n/ekspressiya-provospalitelnyh-tsitokinov-pri-stabilnoy-stenokardii.
11. Иванов С.Г ., Ситникова М.Ю., Шляхто Е.В. Роль оксидативного стресса в развитии и прогрессировании хронической сердечной недостаточности: актуальность и возможность коррекции. // Кардиология СНГ 2006; 4:267-70).
12. Калинченко С. Ю., Апетов С. С. /Применение андрогенов у женщин в климактерическом периоде //Лечащий врач. 2009. № 3. С. 28–30
13. Калинченко С. Ю., Апетов С. С. Индивидуализация выбора заместительной гормональной терапии с учетом разных типов климактерических расстройств (с преимущественным дефицитом эстрогенов и андрогенов, по смешанному типу) //Consilium Medicum. 2012. Т. 14. № 6. С. 80–84
14. Линчак Р. М., Догадова Т. В., Жирова Л. Г. Атипичная стенокардия: особенности диагностики и лечения // Вестник Национального медико-хирургического Центра им. Н. И. Пирогова. 2011. №4. URL: https://cyberleninka.ru/article/n/atipichnaya-stenokardiya-osobennosti-diagnostiki-i-lecheniya
15. Мелатонин в комплексном лечении больных сердечно-сосудистыми заболеваниями. / Заславская Р.М., Шакирова А.Н., Лилица Г.В., Щербань Э.А. // М.: ИД «МЕДПРАКТИКА-М», 2005. 192 с. [Zaslavskaya R.M., Shakirova A.N., Lilitsa G.V., Shcherban` E.A. Melatonin v kompleksnom lechenii bol`nykh serdechno-sosudistymi zabolevaniyami [Melatonin in complex therapy of patients with cardiovascular discases]. Moscow: PH «MEDPRACTICA-M», 2005. 192 p.
16. Нарушения в системе гемостаза у больных нестабильной стенокардией. /Глухова Н.А., Паршина С.С., Афанасьева Т.Н., Потапова М.В., Федорова О.Ф. // БМИК. 2016. №8. URL: https://cyberleninka.ru/article/n/narusheniya-v-sisteme-gemostaza-u-bolnyh-nestabilnoy-stenokardiey
17. Оганов Р.Г., Масленникова Г.Я. Особенности лекарственной терапии стенокардии у мужчин // КВТиП. 2014. №2. URL: https://cyberleninka.ru/article/n/osobennosti-lekarstvennoy-terapii-stenokardii-u-muzhchin
18. Пономаренко И.В., Сукманова И.А. Клинико-гемодинамические характеристики пациентов с острым коронарным синдромом молодого возраста // Комплексные проблемы сердечно-сосудистых заболеваний. 2018. №1. URL: https://cyberleninka.ru/article/n/kliniko-gemodinamicheskie-harakteristiki-patsientov-s-ostrym-koronarnym-sindromom-molodogo-vozrasta
19. Ребров А.Л., Воскобой И.В. Роль воспалительных и инфекционных факторов в развитии атеросклероза. // Терапевт. арх. 2004; 1: 78–82.
20. Цитокиновый профиль при сочетанном течении острого инфаркта миокарда с внебольничной пневмонией. /Данько А.А., Белков С.А.,. Репетий Н.Г, Лысов А.Ю., Минкова Т.А., Матвеев А.А., Палченкова М.В. // ВМЖ. 2012. Т. XXXIII, № 11 (ноябрь). С. 59-60.
21. Цыпленкова В.Г. Критические заметки по поводу парадигмы «гибернирующий и оглушенный миокард». // Кардиология 2005; 9: 43-6).