EARLY DEVELOPMENT OF CORONARY ARTERY DISEASE AT A YOUNG WOMAN

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Abstract

The authors described a case of myocardial infarction at a young woman, which shows the influence of traditional risk factors and metabolic syndrome on the early development of coronary artery disease.

Keywords: Coronary heart disease, myocardial infarction, risk factors, women of young age

Introduction

Indicators of morbidity, disability and mortality due to major diseases of the circulatory system (DCS) tend to steady growth throughout the world, including Kazakhstan. Out of the total deaths worldwide DCS occupy leading positions. So according to the World Health Organization (WHO) more than 55 % of deaths and disability are caused by heart diseases. Every year the world's 17.3 million people die of cardiovascular disease, which is a leading cause of mortality among men and women. The number of deaths is projected to increase in 2030 to 23.3 million cases. Estimated at 8.6 million, including fatal cases of coronary heart disease today are women. Women under 50 who have experienced a heart attack of coronary nature are twice as likely to die than men of this age group. Women who have had a seizure at the age of 65 are more likely to die than men within a year after hospitalization: 42% versus 24%. [1]

Traditionally, the prevalence of coronary heart disease with women has been underestimated because of high rates of the disease in men at a young age. [2] The generally accepted view was that at women IHD starts later than at men when the “estrogen protection" factor disappears. Estrogens increase HDL and lower LDL and progesterone has the opposite effect. Estrogen deficiency is also accompanied by a worsening of vasospastic reactions and platelet aggregation. Estrogens increase HDL by 20-30%, as well as HDL2 fraction. This effect is mediated through increased production of apolipoprotein AI and a decrease in the rate of its clearance. Estrogens reduce LDL cholesterol by 10-20% through enhanced LDL receptor elimination by the liver cells. LDL inhibit vascular endothelium relaxation. Reduced estrogen levels leads to a decrease in the bioavailability of NO, which also leads to vasoconstriction. However, against the background of treatment with estrogen elevated triglycerides (TG) were detected. [7, 25, 26] Thus, under the influence of estrogen the change in the qualitative composition and reducing of the size of LDL is observed, but the concentration of C-reactive protein (CRP) increases. Recently it was found that CRP is not only a predictor of cardiovascular events, but the indicator of progression of atherosclerosis. CRP level stands in direct correlation with the severity of coronary artery disease at women [30, 31]. According to studies by HERS, ERA and WHI the hormone replacement therapy when used for tracking the dynamics of coronary atherosclerosis, despite favorable changes in blood lipid spectrum, resulted in no significant differences in the placebo group by

angiographic indicators such as the minimum diameter of stenotic arteries, the number of new stenoses at patients with atherosclerosis progression or regression [7, 28, 29]. Statistics of recent years show that currently the widely accepted «estrogen» protection of women "does not preclude" CHD. Recent studies have shown that women have special, specific risk factors, peculiar only to females. This dysfunction of sex hormones is associated with their central disregulation or genital diseases, multiple endocrinopathies. These disorders can lead to the development of pathological menopause and lead to the early development of CVD, particularly coronary artery disease. It should be noted that at relatively young women with surgical menopause possibility of myocardial infarction increases dramatically(9-10 times). [1, 2, 22]. In addition, conditions such as gestational diabetes and preeclampsia during pregnancy, polycystic ovaries greatly increase the chances of ischemia [1, 20].

It should be taken into consideration that the prevalence of traditional risk factors (RF) of coronary heart disease (CHD) at women in recent decades has steadily increased. In connection with the emancipation women are being more frequently exposed to multiple stresses, therefore, the female population has growing incidence of diabetes mellitus (DM), hypertension (HT), dyslipidemia (DLP) and obesity. Other urgent problems are inactivity, malnutrition and smoking. [2] According to researchers at the Ohio State University, overweight increases the risk of coronary heart disease at women by 64%, whereas at men only 46% [1]. Women are more common with dyslipidemia and glucose metabolism, and elevated levels of total cholesterol (TC) than men, the risk of cardiovascular disease at women has increased to a great extent. The relationship between the level of HDL-cholesterol and coronary heart disease is more pronounced in women than in men. [4, 7, 16] Raising HDL cholesterol by 1 mg/dL is accompanied by a reduced risk of CHD in men by 2%, and for women - 3%. Women have greater prognostic value decrease in HDL cholesterol and increase in triglycerides (TG). [2, 3, 7, 16-17] According to the Framingham study, despite lower triglyceride levels in women than men, the regression coefficient between this index and the risk of coronary heart disease was 5 times more than men. [5, 16, 17] The frequency of hypertension in women with coronary heart disease is twice higher than in men with coronary artery disease. [7] Women who smoked more than 35 cigarettes per day, the risk is 20- times higher than in non-smoking women. [7] In general, the incidence of acute myocardial at young women- smokers (under 60) 15 years ago was 2-3%, today - 7 - 8 %. [8] Sedentary lifestyle also increases the risk of developing this disease by three times. [1]

In the last 10-15 years much attention is paid to the metabolic syndrome (MS), which includes tissue insulin resistance, hyperinsulinemia, hypertension, atherogenic dyslipidemia, abdominal obesity, impaired carbohydrate tolerance or type 2 diabetes (G.M.Reaven, 1988). The clinical significance of MS is the presence of complex risk factors that create the preconditions for the development of atherosclerosis and its complications. Increase for several times the summation of individual cardiovascular risk by combining components of MS determines its basic medical and social significance. MS patients 10 times more likely to develop MI. Overall mortality is increased by 2.4 times. [23, 24]

Materials and Methods: the patient M. of 28 was examined. The patient came in December 2013 to Pavlodar city hospital # 2 with complaints of retrosternal pain of pressing nature radiating to the left arm and accompanied by a feeling of lack of air, breathlessness, sweating at walking within 100m and at rest, the pain lasted for 5 - 30 minutes, the pain ceased by itself at rest, also she had headaches, rises in blood pressure (BP) to 180 /90 mm Hg., palpitations, weakness, fatigue. From history: considers herself a patient since June 2013, when for the first time she began to mark rises in blood pressure to 180/100 mm Hg. She consulted the local therapist and according to the recommendations she had regularly tab.of indapamid 2.5mg 1 time per day, tab.of berlipril 10mg 1 time per day. In mid-November 2013 she suffered from the pressing retrosternal pain radiating to the left arm,

feeling short of breath, wheezing while walking, pain lasted for about 5 minutes, the pain ceased by itself at rest. On having appealed to the clinic, she was diagnosed with hypertension of the 2 stage and 3 risk, cervicothoracic osteochondrosis. Within a week she had had anti-inflammatory and metabolic therapy. Effect of treatment was not observed, retrosternal pain became more prolonged, she again consulted the therapist then her electrocardiogram (ECG) was taken down, which registered - sinus rhythm, right, CHSS89 min, normal position EOS, QS V1-V3, ST elevation V1-V3, (+ -) T V1-V3, (-) T V4-V6 (Fig. 1). In an emergency order she was sent to the urgent clinic, hospitalized with acute coronary syndrome. History of life: Smokes for 10 years by 12sigaret a day, does not drink alcohol. Heredity is burdened by arterial hypertension (AH) and coronary heart disease (CHD), coronary diseases and death at a young age with her relatives are not noted. During the last 2 years 20kg weight gain. Features of gynecological history: Menstruation since the age of 13, regular, moderate, with a cycle of 28 days. Married for 3 years. Pregnancies and births did not occur, did not use contraception .

Objective: The general condition is relatively satisfactory. Height – 153cm, weight 88kg, BMI (body mass index) is 37 kg/m2. Obesity of the 2 stage. Waist measurement (WM) - 115cm, measurement round the hips (HM) - 121cm, Attitude FROM / ON - 0.95. Skin of normal color, normal humidity. Shape of the chest: hypersthenic. Percussion sound over lungs: pulmonary. Auscultation: vesicular breathing, no wheezing. NPV - 17 min. Boundaries of the relative and absolute dullness of the heart within the age norm. Cardiac sounds are muffled, regular rhythm. Heart rate is 80 beats / min , PS 80 beats/min, satisfactory filling. Blood pressure of 150 /90 mm. Hg. on both hands. Abdominal palpation is soft, increased in volume due to subcutaneous fat, palpation is painless. Liver edge is of the costal arch. The spleen is not enlarged. Physiological functions are not violated. No peripheral edema.

According to laboratory studies on admission:

Total blood from 06.12.2013:

Нв, g/l	ER., x 10^{12}/L	CI	HT, %	L., x 10^{9}/L	RNC,%	N,%	E,%	M,%	L, %	Tr, x 10^{9}/L	ESR, mm/h
136	4.86	0.83	40	7.0	1	42	2	7	48	348	23

Biochemical Blood Analysis dated 06.12.2013:

TP, g/l	Blood urea, mole/l	creatininemc.mole/l	glucosemole/l	ALT, u/l	AST, eu/l	CRP
60	3,5	69,2	4,9	24,5	22,7	negative

Lipid spectrum dated 06.12.2013:

Cholesterol, mole/l	HDL, mole/l	LDL, mole/l	TG, mmole/l	Indexof atherogenicity
4,1	0,78	2,54	2,7	4,2

Troponin T at admission was - 0.2, while the second study after 6 hours troponin T - 0.15. In the ovulatory phase of the cycle study was conducted following hormones showed a reduction in the level of luteinizing hormone - 10.5 IU / L (normal 24 - 150), a decrease in prolactin levels of 68 ng / mL (normal, 4-23), a slight decrease in FSH 5.32 IU/l (normal 5.8 - 21).

According to instrumental studies:

The primary conducted echocardiography (echocardiography) revealed: ejection fraction (EF) of the left ventricle (LV) was 47%. Signs of left ventricular hypertrophy was detected. Hypokinesis of the apical, anterior- septal LV segments was observed. Mitral regurgitation of the 1st stage was fixed. Tricuspid regurgitation of the 1st was detected.

The electrocardiography (ECG) at hospitalization fixed sinus rhythm of the heart, right, CHSS89 min, normal position EOS, QS V1 - V3, lifting ST V1-V3, (+ -) T V1-

V3, (-) T V4-V6) - anterior myocardial infarction, septal area, the top of the left ventricle (Figure 1).

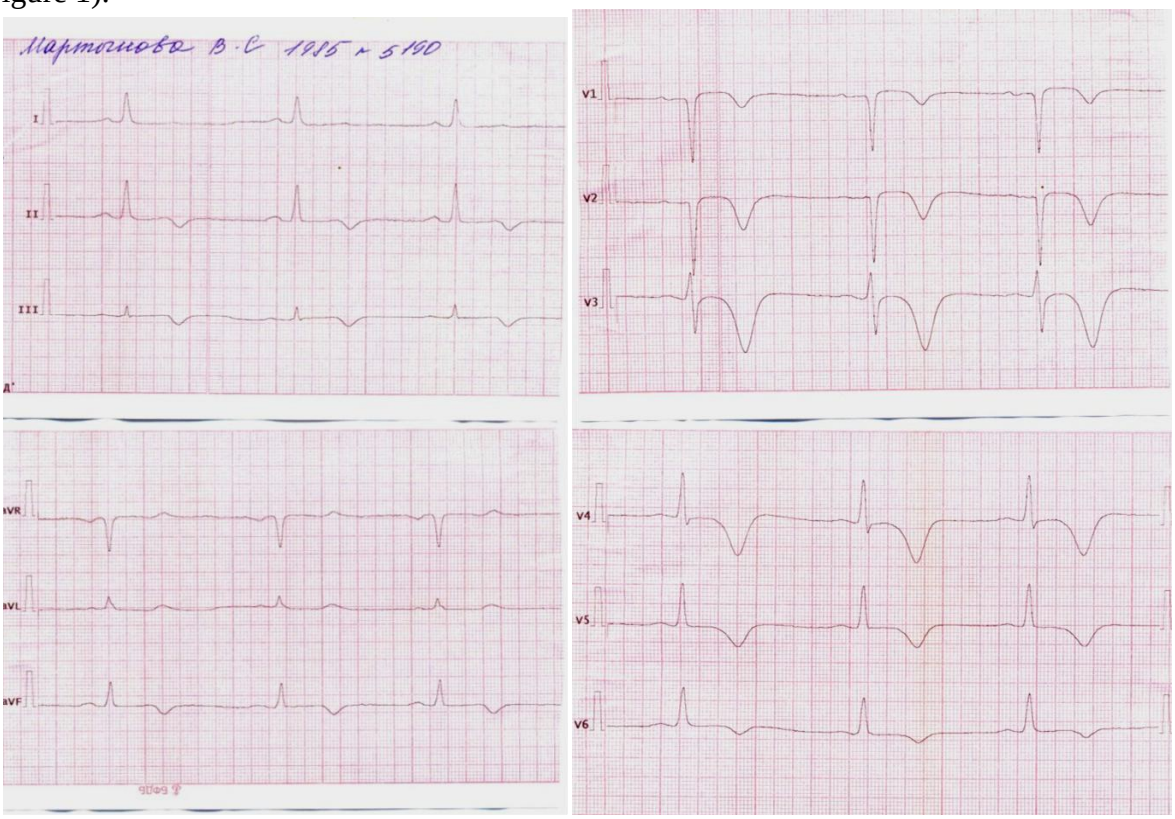
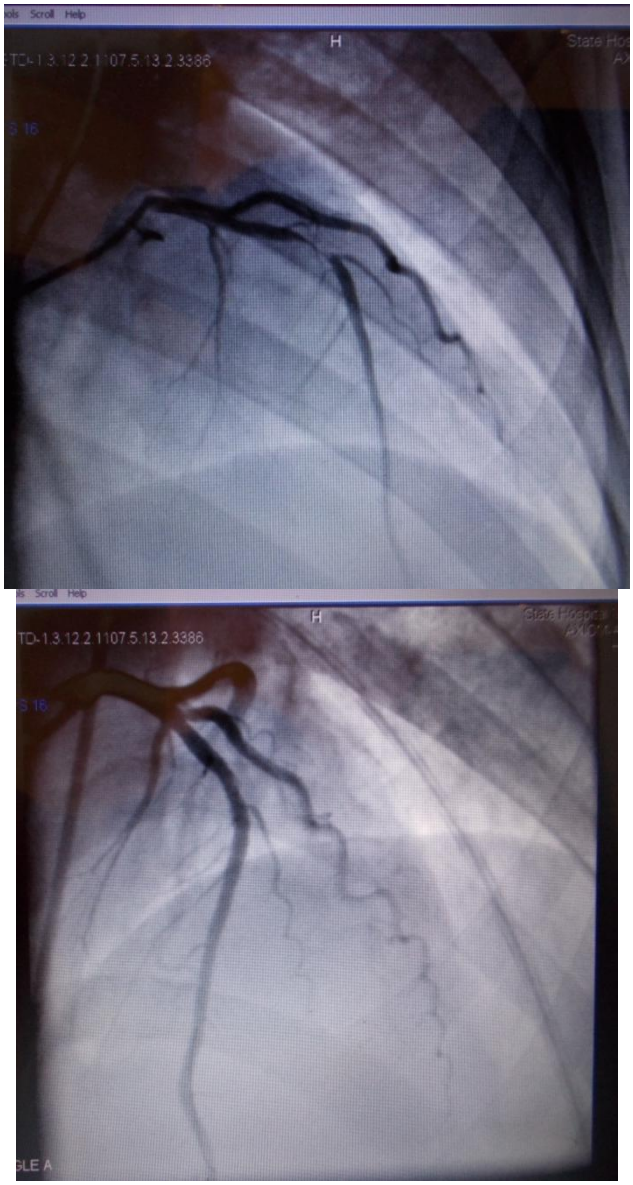


Fig.1 : The ECG of the patient M of 28, at hospitalization.

By results of daily ECG monitoring the average heart rate (HR) was 72 beats/min. Minimum heart rate was 46 beats/min at 2:29 AM. Maximum heart rate was 138 beats/min 10:57 AM. No changes were detected using the apparatus pauses > 1.5 sec. 38 episodes of tachycardia were fixed, tachycardia episode duration was 43 min. 37 seconds. Episodes of bradycardia were not detected. Three PVCs (VES), 6 atrial extrasystoles (PES) were observed. There was no evidence of arrhythmia episodes absolute.

In accordance with a coronary angiography (Figure 2a, b): the type of circulation is balanced, Left main - with equal contour passable along the entire length. LAD is with a rough outline, in the middle segment extended 99% stenosis. At the mouth of stenosis 30 %. CF is with a smooth contour and passable along the entire length. RCA is with a smooth contour and passable along the entire length.

LAD stenting was made with a stent eluted by the drug - Medtronic Resolute Integrity 2.75mm x 14mm. Satisfactory results after stenting were observed. (Figure 2a and b).



Based on the above mentioned the patient was diagnosed: CHD, myocardial infarction with ST elevation of anterior septum area, apex of the left ventricle. Single-vessel coronary lesions: stenosis of middle LAD Segment by 99%, after stenting the condition of LAD is the 1 class heart failure by Killip, Hypertension 2 risk 4.

For the time spent in hospital for ongoing background antianginal therapy the patient's health considerably improved, anginal pains were not present, though rare discomforts in the heart remained, shortness of breath was not observed, hemodynamic parameters were stable, the patient was dismissed from hospital in a satisfactory condition.

Conclusion: The presented clinical case presents interests for myocardial infarction with ST elevation occurred at a young 28 year old woman, when CHD direct myocardial infarction, has traditionally been viewed as a disease of middle aged and elderly men. The presence of risk factors such as obesity of the 2nd stage, dyslipidemia, smoking, family history, symptoms of hypoestrogenemy may have led to early atherosclerosis of coronary vessels with the development of myocardial infarction. According to the recent research the increase in the incidence of myocardial infarction and mortality from myocardial infarction at young women becomes more frequent [1, 7, 8]. Experts attribute this fact to the delay in diagnosis. Noted fact is as well connected with misinterpretation of ST segment elevation on

ECG of young women, due to lack of recognition of the increasing incidence of coronary heart disease at this group of patients [8]. Any patient, regardless of age and gender with complaints of a typical or atypical chest pain, should record electrocardiogram. Electrocardiographic signs of myocardial ischemia must be diagnosed regardless of sex and age of the patient.

References:

Coronary Artery Disease in Women: A 2013 Update - Global Heart, June 2013.
S.N.Tereshchenko, T.M. Usach, I.V. Kositsina and others //Cardiology – 2005. – # 1. – P. 98–104.
Bush T.L., Miller V.T. // Mienopause physiology and pharmacology. – New York.: Year Book Medical Publishers Inc., 1987. – P. 187–208.
Gordou D.J., Probstfield J.L., Garrison R.J. et al. // Circulation. – 1989. – Vol. 79. – P. 8–15.
Сметник В.П. // Рус. мед. журнал. – 2001. – Т. 9, № 9 (128). – С. 354–358.
S.A.Boitsov, A.V. Susekov, D.M.Aronov and others. Actual issues of the satin therapy in clinical practice. Expert Council Meeting. // Atherosclerosis and Dislipidemy — 2011. — #1. — P. 65-66.
A.A Lyaishev Peculiarities of IHD at women – Medical Encyclopedia. – Medical Articles. – Cardiology. - 2007.
Jaimison M, ST Elevation Myocardial Infarction is Underdiagnosed and Undertreated in Women, ACLS certification Institute. - May 2013
Berger JS, Elliott L, Gallup D, Roe M, Granger CB, Armstrong PW, Simes RJ, White HD, VandeWerf F, Topol EJ, Hochman JS, Newby LK, Harrington RA, Califf RM, Becker RC, Douglas PS. Sex differences in mortality following acute coronary syndromes. JAMA. 2009.- 302. - 874–882
Gulati M., Leslee J. Shaw, C. Noel Bairey Merz, Myocardial Ischemia in Women - Lessons from the NHLBI WISE Study, Clin Cardiol. 2012 March. -35(3). – P.- 141–148.
Kochaneck KD, Xu J, Murphy SL, Minino AM, Kung H-C. Deaths: Preliminary Data for 2009. National Vital Statistics Reports. 2011. – 59. – P. - 1–51.
Roger VL, Go AS, Lloyd-Jones DM, et al. Heart disease and stroke statistics--2011 update: a report from the American Heart Association. Circulation. 2011. – 123. - 18–209.
Smilowitz NR, Sampson BA, Abrecht CR, Siegfried JS, Hochman JS, Reynolds HR. Women have less severe and extensive coronary atherosclerosis in fatal cases of ischemic heart disease: an autopsy study. American heart journal. 2011. – 161. - 681–8.
Reynolds HR, Srichai MB, Iqbal SN, et al. Mechanisms of myocardial infarction in women without angiographically obstructive coronary artery disease. Circulation.2011. – 124. - 1414–25.
Mokdad AH, Ford ES, Bowman BA, et al. Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. JAMA : the journal of the American Medical Association. 2003. – 289. - 76–9.
Lerner DJ, Kannel WB. Patterns of coronary heart disease morbidity and mortality in the sexes: a 26-year follow-up of the Framingham population. Am Heart J. 1986.–111. -383–90.
Hokanson JE, Austin MA. Plasma triglyceride level is a risk factor for cardiovascular disease independent of high-density lipoprotein cholesterol level: a meta-analysis of population-based prospective studies. J Cardiovasc Risk.1996. – 3. - 213–9.
Spencer EA, Pirie KL, Stevens RJ, et al. Diabetes and modifiable risk factors for cardiovascular disease:the prospective Million Women Study.Eur J Epidemiol.2008–23. - 793–9.

Bairey Merz CN, Johnson BD, Sharaf BL, et al. Hypoestrogenemia of hypothalamic origin and coronary artery disease in premenopausal women: a report from the NHLBI-sponsored WISE study. Journal of the American College of Cardiology. 2003. -41. - 413–9.

Bellamy L, Casas JP, Hingorani AD, Williams DJ. Pre-eclampsia and risk of cardiovascular disease and cancer in later life: systematic review and meta-analysis.Bmj. 2007. – 335. - 974.

Mehta PK, Goykhman P, Thomson LE, et al. Ranolazine improves angina in women with evidence of myocardial ischemia but no obstructive coronary artery disease. JACC Cardiovasc Imaging. 2011. – 4. - 514–22.

Kennedy JW, Killip T, Fisher LD et al. The clinical spectrum of coronary artery disease and its surgical and medical managment: the Coronary Artery Surgery Study.// Circulation. 1982;66 (5 Pt 2): 11116

Prasad A., Stone G., Stuckey T. et al. Impact of diabetes mellitus on myocardial perfusion after primary angioplasty in patients with acute myocardial infarction // J. Am. Col. Cardiol. 2005. - 45. - 508-514.

Yoshioka M., Doucet E., St.-Pierre S. et al. Impact of high-intensity exercise on energy expenditure, lipid oxidation and body fatness // Int. J. Obes. 2001. - 25. – P.- 332-339.

Nabulsi A.A. et al. // New Engl. J. Med. 1993. V.- 328. P.- 1069–1075.

Soma M. et al. // Lancet. 1991. V. -337. -P. -612.

Климов А.Н., Нагорнев В.А., Денисенко А.Д. // Мед. академ. журнал. – 2005. – Т. 5. - № 2.– P.- 18–32.

Signorelli S.S., Neri S., Sciacchitano S. et al. // Maturitas. – 2001. – Vol. 39. – P. 39–42.

Rosano G.M.C., Cerquetani E., Gebara O. et al. // Eur. Heart J. – 2002. – Vol. 23. – P. 592.

Tataru M.C., Heinrich L. et al. // Eur. Heart J. – 2002. – Vol. 21. – P. 1000–1008.

Van ger Meer I.M., Moniek P.M. C-reactive protein predicts progression of atherosclerosis measured at various sites in the arterial tree. – 2002.