THE CONDITION OF INTRACARDIAC HEMODYNAMICS IN PATIENTS WITH RHEUMATIC FEVER AND CHRONIC RHEUMATIC HEART DISEASE

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Findings of inspection parameters of intracardiac dynamics at 51 patients with rheumatic heart defects in interrelation with activity of inflammatory process and character of a heart defect (25 sick mitral a heart disease, 26 sick aortal a heart defect) are given in this work.

The echocardiography was spent on the device "Toshiba SSH-40" (Japan), with use of recommendations American echocardiography society.

At comparison of parameters echocardiography at I and II degrees of activity at patients with aortal and mitral defects of the heart the most expressed changes are found out at II degree of activity of inflammatory process that confirms the influence of activity degree on parameters of central hemodynamics , and condition improvement - about adequacy of therapy.

Many researchers consider a rheumatic fever (RF) as unique cardiovascular disease, which has not been enough studied yet. [4; 9; 12; 14].

According to WHO report, acute rheumatic fever (ARF) and chronic rheumatic heart disease (ChRIH) still remain the most widespread cardiovascular diseases in children, teenagers and young age adults, affecting annually at least 12 million people and being the reason of 332 thousand of lethal outcomes [5; 18].

In the last 15-20 years, the clinical picture of RF has undergone major changes. Many authors notice the rarity of severe course of rheumatic carditis, the decrease of fatalness and reduction of disease recurrence rate, the tendency of disease to transit into monosyndromic forms, the increase of few symptomic and latent variants of current, etc. [7; 17].

According to data of many authors, the frequency of primary rheumocarditis is very great and is from 88,5 up to 100 % [1; 6]. Simultaneously, from other data the frequency of heart affection at the first attack of rheumatism enough moderated - 50-69 % [13; 11] and even low - 3-38 % [8]. These results difference can be explained hardly only from positions of polymorphism of clinical symptoms and variants of primary RF course in various regions and countries of the world.

In this connection, it is necessary to emphasize the importance of study instru-

mental methods (phonocardiography, echocardiography, dopplechocardiography) in early diagnostics of reumocarditis [2].

Carditis is the main sign of rheumatic process activity the expressiveness of which reflects a degree of inflammatory process activity. However, at the minimal activity of an inflammation or its absence by laboratory data, manifestation of carditis persists more often having a permanent current. Against a background of valvular affections rheumatic carditis can aggravate the intracardiac hemodynamics condition which has been connected with a degree of inflammation activity.

The aim of research

To study the parameters of central hemodynamics in patients with rheumatic fever and chronic rheumatic heart disease against inflammatory process activity.

Material and methods

51 patients with RF and ChRHD have been investigated aged from 23 till 37 years, of 25 have been mitral heart failure, 26 aorta heart failure.

Randomization made by the character of failure, parity of their components, and remoteness of disease and stage insufficiency blood circulation (IBC), when grouped by degree of activity.

Central hemodynamics was estimated according to echocardiography findings performed on « Toshiba SSH-40 » (Japan), equipped by electronic gauges with fre-

quency of ultrasonic waves of 2,5-3,5 MHz, by a standard technique with use of American echocardiography association recommendations (ASA) [15].

The study made before treatment and after three-month treatment course. The patients were examined with a prone position on left side. Study of structures of heart made in B-and M - modes with use of standard positions: parasternal positions on long and short axes, top five-chamber and high apical as well. They registered and then were measured more than 3 consecutive intimate cycles for breath effect leveling.

The obtained results were averaged. There measured the following parameters of heart structure: diameter of an aorta, disclosing of aortic valve, the cross-section size of left atrium at the end of diastole atrium, finite diastolic size of left ventricle (FDSLV), finite systolic size of the left ventricle (FSSLV), thickness of lateral wall of left ventricle in diastole (ThLWLV), thickness of ventricular septum in diastole (ThVSD).

The following parameters were counted against a background of the obtained data: finite- diastolic volume (FDV) and finite- systolic volume (FSV)of LV were counted by L.E.Teichgolz et all.:

 $FDV = 7 / (2,4+ FDSLV) *FDS^3$, ml, where FDS - finite- diastolic size of the left ventricle in a phase of diastole, measured on peak waves of R an electrocardiogram.

 $FSV = 7 / (2,4+FSS) *FSS^3$, ml, FSV - finite-systolic size of left ventricle in a phase of diastole:

- Fraction of emission:

FE = ((FDV - FSV)/FDV) * 100, %;

- Fraction of reduction:

FR = ((FDS- FSSShFDS) * 100, %

- Shock volume:

ShV = FDVLV - FSVLV, ml;

- Minute volume of blood:

MVB = ShV* RHC, ½/min. (rate of heart contractions)

- myocardium mass of LV:

 $MMLV = 1.04 * ((FDSLV + ThVSD + ThLWLV)^3 - FDSLV^3) - 13.6, (g), where$

ThVSD – thickness of ventricular septum, ThLWLV – diastolic thickness of lateral wall of LV, FDS - finite-diastolic size of LV.

- Myocardium mass rate of LV: $MMRLV = MMLV/P g/m^2$

Results and discussion

Table 1 noted the accurate difference between the parameters of the control and patients with rheumatic heart disease (RDH) and mitral (stenosis and failure) and aortal (stenosis and failure) heart defect. When grouped the patients with II stage of BF included. However, the change of parameters of central hemodynamics in RHD was established much earlier [3].

We analyzed the influence of inflammation activity on cardio dynamics. So, the parameters of the left atrium (LA) in mitral defect with II degree of activity were above 21,4 %; FDR - 6,6 %; FSS - on 17,3 %; FDV - 16,0 %; FSV - 48,3 %; FE – was less 6 %; FR - 8,2 %; ThLWLV and ThVS practically did not differ; MMLV was above 9,1 %. The parameters systolic arterial pressure (SAP) were above 3,9 % at II stage of activity as well; diastolic arterial pressure(DAP) was less than 1,5 %; RSC was above 14,7 % in II stage of activity.

At comparison of parameters of Echocardiography at I and II stages of activity in patients with aortal heart defect (stenosis and failure) the differences in parameters of central hemodynamics were revealed as well. So in difference from I degree of inflammatory process activity, II degree of activity at aortal defect was characterized by more marked changes from FDS which was above 14,6 %; FDV - 1,7 %; MMLV - 0,9 % whereas FR was below 5,9 %; the other parameters of the big difference were not revealed.

This circumstance still indicates the influence of a degree of inflammatory process activity on the parameters of central hemodynamics, the improvement of parameters can also testify the adequacy of therapy, which is important for the forecast of disease as a whole.

Table 1. Echocardiography parameters at patients mitral and aortic heart diseases with I and II degree of activity of an inflammation (M±M)

Z	Parameters		Mitral valv	Mitral valvular disease	Aortal valv	Aortal valvular disease
		Control (n=20)	act I (n=12)	act II (n=13)	act I (n=14)	act II (n=12)
	LA (cm)	3,08 ± 0,06	$3,45 \pm 0,04*$	$4,19 \pm 0,09*$	$3,69 \pm 0,08*$	3,86 ± 0,09*
2	FDS (cm)	$5,02 \pm 0,07$	5,75 ± 0,05*	$6,13 \pm 0,10*$	$6,13 \pm 0,11*$	7,18 ± 0,14*^
m	FSS (cm)	$3,31 \pm 0,05$	$3,69 \pm 0,08*$	4,33 ± 0,12*^	$4,10 \pm 0,12*$	$4,26 \pm 0,01*$
4	FDV (milliliter)	120,68 ± 4,31	$160,16 \pm 3,56*$	185,82 ± 7,09*^	$190,64 \pm 6,05*$	$194,01 \pm 8,02*$
S	FSV (milliliter)	45,17 ± 1,28	57,60 ± 2,54*	$85,42 \pm 6,12*$	78,18 ± 4,36*	$79,12 \pm 5,02*$
9	FE (%)	$62,24 \pm 0,91$	58,06 ± 1,48*	54,53 ± 1,44*	$60,36 \pm 1,19$	59,12 ± 2,01
7	FR (%)	$33,71 \pm 0,68$	$34,25 \pm 0,91$	$31,45 \pm 0,79^{\wedge}$	$33,10 \pm 0,86$	31,12 ± 0,75*
∞	ThLWLV (cm)	$0,90 \pm 0,02$	$1,14 \pm 0,01*$	$1,13 \pm 0,01*$	$1,21 \pm 0,02*$	$1,25 \pm 0,01*$
6	ThVSD (cm)	$0,94 \pm 0,01$	$1,04 \pm 0,02*$	$1,05 \pm 0,01*$	$1,11 \pm 0,01*$	$1,16 \pm 0,01*$
10	MMLV (g)	158,72 ± 7,23	253,14 ± 7,02	$276,12 \pm 10,12*$	$309,05 \pm 8,76*$	$312,02 \pm 6,16*$
	SAP (millimeter of mercury)	118,26 ± 2,48	120,95 ± 2,66	125,76 ± 3,17	126,66 ± 4,25	128,02 ± 3,12*
12	DAP (millimeter of mercury)	75,92 ± 1,61	67,18 ± 3,35*	$66,20 \pm 3,35*$	50,05 ± 3,69*	50,01 ± 2,14*
13	RHC (min)	$72,31 \pm 1,12$	$83,10 \pm 2,17*$	95,31 ± 3,31*^	$86,68 \pm 2,81*$	85,18 ± 1,13*

The note: * P < 0.05 - authentic distinction between parameters of the control and compared groups; $^{\Lambda}P < 0.05$ - distinctions between parameters I and II degree of activity are authentic.

Thus, the shift of cardio dynamics parameters is more marked in RHD patients with II degree of inflammatory process activity than in the ones with I degrees of inflammatory process activity .Therefore, the changes of parameters of central hemodynamics correlate with the activity of inflammation which can aggravate_BF. The hypertrophy and dilatation of LV develops at insufficiency of mitral and aortal valves when maintained adequate minute volume of blood. In conditions of a significant volumetric overload dilatation of LV starts to advance the rate of mass myocardium increase. At a microscopic level in this phase it has been observed the increase in distance among cardiomiocytes, development of myocardium sclerosis characterized for pathological simulation of LV [3].

As a rule, the rheumatic affection of mitral valve is marked in its adjusting affection. Prolonged rheumatic endocarditis leads to morphological changes of mitral valve: cusps get thicken, become rigid, grow together on comissures, tendinous fibers change, shorten. Echocardiography displays the dilatation of the left departments of heart, various directed diastolic movements of thickened mitral cusps and the absence of their systolic connection, which leads to mitral regurgitation.

Doppler study plays a very important role in diagnostics of mitral insufficiency at any degree of manifestation. The best method of the presentation of mitral regurgitation is color Doppler scanning [16] as it possesses high sensitivity, and its{his} use does not require much time. Color Doppler scanning gives the information about mitral regurgitation in real time. Though presentation about the direction and penetration depth of regurgitating jets can be obtained in pulse doppler mode, color scanning is more reliably and technically easier, especially at extrinsic regurgitation.

From the apical access mitral regurgitation looks as the flame of light blue color directed to the left atrium appearing in a systole. To register mitral failure and identify the degree of its manifestation the method of color scanning for sensitivity approximates to X-ray contrast ventriculography [10].

In rheumatic aortal insufficiency, ECG observes destructive changes and incomplete connection of aorta valves accompanied by aortal regurgitation; increase of diastolic size of LV and LA and increase of ascending part and roots of aorta as well.

In M-modal study diastolic thrill of anterior cusp of aortic valve serves as the main attribute of aortal regurgitation.

Nowadays it is established, that presence or absence of diastolic thrill of anterior cusp of mitral valve at aortic regurgitation depends on what cusp is affected, and this defines the direction of regurgitating jets.

Colour Doppler scanning reveals the big variety of the direction of regurgitating jets in different patients; when it is directed to anterior mitral cusp, trembling of cusp is observed

In severe aortal failure M-modal study registers also early closing of mitral valve [16]; this finding testifies hemodynamics disorders, requiring intensive therapeutical and surgical treatment. The examination of contractility of left ventricle, its volume, form and mass can give the valuable information for mass identification of aortal failure.

Doppler examination plays leading role in diagnostics of aortal insufficiency and mass identification. Pulse Doppler examination and particularly color scanning were more sensitive than other methods of diagnostics of aortal failure, including invasive methods.

In regurgitation revealing, occupying all diastole on doppler spectrum, control volume is moved on ventricle to find out the penetration depth of regurgitation jets into it. If the jet comes more than one third of depth of ventricle, the degree of aortal failure is rarely mild.

Thus, in softly marked RF_and carditis the diagnostic value of ECG increases and is

to carry out in dynamics in ARF and RHD, and in BF connection.

The given circumstance specifies once more to the influence of degree of inflammatory process activity on the parameters of central hemodynamics, the improvement of parameters can also testify about adequacy of therapy, that is very important for the forecast of disease as a whole.

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