ENDOTHELIAL DYSFUNCTION AND BLOOD VISCOSITY INPATIENTS WITH UNSTABLE ANGINA IN DIFFERENT PERIODS OF A SOLAR ACTIVITY

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ABSTRACT. The origin of hemorheologic and endothelial defects in patients with unstable angina (comparing with healthy persons) is determined by a solar activity period: the blood viscosity increases in a period of high solar activity in the vessels of small, medium and macro diameters, a local decompensate dysfunction of small vessels endothelium had been fixed (microcirculation area). In the period of a low solar activity there is an increase of a blood viscosity in vessels of all diameters, generalized subcompensated endothelial dysfunction is developed (on the background of the III phase blood clotting activating). In the period of a high solar activity a higher blood viscosity had been fixed, comparing with the period of a low solar activity.

Key words: solar activity, unstable angina, blood viscosity, endothelial dysfunction.

Change of a solar activity is one of natural risk factors for a human health. Active solar phenomenon are accompanied by oscillations of the Earth electromagnetic field. Geomagnetic field of the Earth is one of the few environmental factors which influences an organism and its regulatory mechanisms at all levels, including molecular, endocellulal, intercellular, etc. Heliogeophysical activity damages a chronological sequence of informational signals which are used in our organism for coordination of biological processes rhythmic with an environment rhythmic.

It is known that a cardiovascular system is a primary target for a heliogeophysical activity (Breus, 2003). In the most of the patients with an ischemic heart disease geomagnetic irradiation provokes hypertension, arrhythmia episodes, blood rheological properties; it causes feebleness of capillary circulation, blood viscosity increase and erythrocytes aggregation change (Breus, 2003).

As rheological defects are independent risk factors of an ischemic heart disease (Backer et al., 2002), it is important to study the blood viscosity (BV) and functional properties of vascular endothelium in vessels of different diameters in patients with unstable angina (UA) in the periods of high and low solar activity (SA) during the 11-years solar cycle.

During the last 23rd solar cycle (September, 1996 – September, 2007) 310 patients with II class unstable angina by Braunwald had been examined: 129 of them – in the period of a high SA, 81 – in the period of a low SA. The groups were matched in age and sex characteristics, severity of a disease, amount of myocardial infarction episodes, arterial hypertension, etc.

There were 80 apparently healthy persons in the control group (C), they had been examined at the same period (September, 1996 – September, 2007): 40 of them – in the period of a high SA, 40 – in the period of a low SA.

To characterize the SA in the Wolf numbers there had been used the data of the Institute of space exploration of RSA. An average value of the Wolf numbers in the period of a high SA was 112,0±2,9, in the period of a low SA – 62,4±3,9 ($p<0,05$).

The BV had been estimated under the three rate-of-shear (200 sec^{-1}, 100 sec^{-1}, 20 sec^{-1}) with the help of the rheometer AKP-2, which let us measure a blood flow property in the vessels of small, medium and macro diameters correspondingly.

The functional ability of endothelial vessel wall had been estimated by the occlusion test (OT) based on a modeling of a short 3-minute local ischemia, stimulating by the fixing of a sphygmomanometer on a patient's shoulder, and the pressure 10 mm of mercury higher than a systolic pressure (Baluda V.P. et al., 1992). The substances that decrease the blood clotting and thrombocytes aggregation, influence the blood viscosity and erythrocytes functional properties, are released into the blood flow from a vascular endothelium at the occlusion test. The occlusion test models an angiospasm and let estimate a reserve of vessel wall of the subject at the short ischemia condition.

An endothelial dysfunction had been estimated by BV measuring results before and after OT under the three rate-of-shear (200 sec^{-1}, 100 sec^{-1}, 20 sec^{-1}) which reflexes the endothelium condition in the vessels of a small, medium and macro diameters.

There had been also fibrinogen measured (FG), as its increase in a blood plasma causes an increase of a BV.

It had been denoted that the character of hemorheologic and endothelial defects in patients with unstable angina (comparing with healthy persons) is determined by a solar activity period.

At a high SA in the patients with unstable angina there had been fixed an increase of a BV (comparing with healthy persons) in vessels of medium and small diametres (under the rate-of-shear 100 sec^{-1}, 20 sec^{-1} , $p<0,05$) (Table 1). But in vessels of macro diameters (200 sec^{-1}) there had not been revealed a significant change in a BV ($p>0,05$). So, rheological defects are fixed only in vessels of medium and small diametres.

During the OT in vessels of medium and macro diameters (200 sec^{-1}, 100 sec^{-1}) an endothelium reaction corresponds with reactions in healthy persons in the period of a high SA: a BV does not change before and after OT ($p>0,05$) (Table. 1). In vessels of small diameters (20 sec^{-1}) there had been fixed a statistically valid increase of a BV after OT ($p<0,05$).

Thus, at a high SA in the patients with UA (comparing with healthy persons) there had been fixed neither a BV, nor functional properties of endothelium changes in macrovessels. In the vessels of a medium diameter a blood viscosity is high, but endothelium reaction on an induced ischemia is kept. In small vessels the BV is high and an endothelial dysfunction was revealed – paradoxical increase of a BV after OT which indicates a failure of compensatory-adaptive of an endothelial properties at a spasm condition and developing of decompensated endothelial dysfunction. The given failures are not connected with an increase of procoagulative blood potency, as fibrinogen concentration in the patients with UA in the period of a high SA does not differ from the indices of healthy persons ($p>0,05$) (Table 1).

At a low SA in the patients with UA there is an increase of a BV ($p<0,05$) in vessels of all diameters (200 sec^{-1}, 100 sec^{-1}, 20 sec^{-1}) comparing with indices of healthy persons at the same period (Table 2).

During the OT a homotypic reaction had been revealed in vessels of all diameters (200 sec^{-1}, 100 sec^{-1}, 20 sec^{-1}): BV does not change before and after OT ($p>0,05$) (Table 2). At the same time in healthy persons in the period of a low SA during OT there is a decrease of a BV ($p<0,05$) (Table 2). Thus, an endothelial dysfunction progresses in vessels of all diameters in the patients with UA, the function failure is of a generalized origin. At the same time the origin of an endothelial dysfunction differs from the same one in the period of a high SA: BV does not change at a spasm condition, but in a high SA after OT there is an increase of BV (decompensated endothelial dysfunction). We had revealed endothelium function failure in the period of a low SA and it can be denoted as a subcompensated type of endothelium dysfunction.

The revealed failures come out on the background of FG level increase in the patients with UA (comparing with healthy persons) in the period of a low SA ($p<0,05$) (Table 2).

The results of the study show that unidirectional endothelium reactions on an induced ischemia in the vessels of different diameters is fixed only in the period of a low SA (generalized subcompensated endothelium dysfunction). In the period of a high SA there is a misfit in endothelium reactions on an induced ischemia in the vessels of different diameters and developing of a local decompensated endothelial dysfunction. Thus, in the period of a low SA there is more intensive retention of basic mechanisms of regulation, than in the period of a high SA.

Table 1: Blood viscosity indices in an induced ischemia and fibrinogen in the patients with unstable angina in the period of a high solar activity (M±m)

Indices	High SA	
	UA (n=129)	C (n=40)
BV 200 sec^{-1} mPa·sec before OT after OT	6,58± 0,13 6,99± 0,13	6,47±0,33 6,56±0,52
BV 100 sec^{-1} mPa•sec before OT after OT	10,50± 0,44 # 11,67± 0,56 #	7,06±0,40 7,12±0,64
BV 20 sec^{-1} mPa•sec before OT after OT	11,73±0,45# 13,52± 0,47 *#	9,62±0,46 9,69±1,00
FG, g/l	3,69± 0,08	3,59±0,10

Note: # – the difference between indices of the patients with UA and the control group is statistically valid ($p<0,05$);

* – the difference between index values before and after OT is statistically valid ($p<0,05$).

Table 2: Blood viscosity indices in an induced ischemia and fibrinogen in the patients with unstable angina in the period of a low solar activity (M±m)

Indices	Low SA	
	UA (n=81)	C (n=40)
BV 200 sec^{-1} mPa·sec before OT after OT	6,38± 0,13# 6,48± 0,19#	4,49±0,22 3,81±0,15*
BV 100 sec^{-1} mPa•sec before OT after OT	7,19± 0,18# 6,9± 0,22#	4,54±0,21 3,89±0,21*
BV 20 sec^{-1} mPa•sec before OT after OT	10,59±0,39# 10,18± 0,47#	5,29± 0,33 3,98±0,24*
FG, g/l	3,97± 0,11 #	3,09± 0,17

Note: # – the difference between indices of the patients with UA and the control group is statistically valid ($p<0,05$);

* – the difference between index values before and after OT is statistically valid ($p<0,05$).

We can see that in the patients with UA defects in microvasculature (20 sec-¹) develop independent from a period of a SA, but endothelium dysfunction in small vessels in a high SA is local but more dangerous (decompensated). In the low SA there is generalized (in vessels of all diameters), but less dangerous – subcompensated – endothelium dysfunction. The same is for BV defects (comparing with healthy persons): in the period of a high SA – in vessels of medium and small diameters, in the period of a low SA – in all vessels.

In comparison of BV in the patients with UA in the periods of low and high SA there had been revealed the following: the higher BV in the period of a high SA in macro vessels is only at the spasm conditions (after OT), in vessels of medium diameter – both at rest and at the spasm conditions (before and after OT), in small vessels – only at the spasm conditions (after OT) ($p<0,05$) (Table 3). Fibrinogen concentration in the patients with UA is higher in the period of a low SA ($p<0,05$) (Table 3).

Table 3: Blood viscosity indices in an induced ischemia and fibrinogen in the patients with unstable angina in different periods of a solar activity (M±m)

Indices	High SA (n=129)	Low SA (n=81)
BV 200 sec^{-1} mPa·sec before OT after OT	6,58± 0,13 6,99± 0,13**	6,38± 0,13 6,48± 0,19
BV 100 sec^{-1} mPa•sec before OT after OT	10,50± 0,44 ** 11,67± 0,56 **	7,19± 0,18 6,9± 0,22
BV 20 sec^{-1} mPa•sec before OT after OT	11,73±0,45 13,52± 0,47 * **	10,59±0,39 10,18± 0,47
FG, g/l	3,69± 0,08 **	3,97± 0,11

Note: * – the difference between index values before and after OT is statistically valid ($p<0,05$);

** – the difference between high and low SA is statistically valid ($p<0,05$).

Thus, the origin of hemorheologic and endothelial defects in patients with unstable angina (comparing with healthy persons) is determined by a solar activity period: the blood viscosity increases in a period of high solar activity in the vessels of small, medium diameters, a local dysfunction of small vessels (microcirculation area) endothelium is decompensate (pathological increase of BV at the induced ischemia condition). In vessels of macro diameters BV and endothelial functions are saved. In the period of a low solar activity there is an increase of a blood viscosity in vessels of all diameters, generalized subcompensated endothelial dysfunction is developed (on the background of the III phase blood clotting activating). In the period of a low solar activity in vessels of all diameters (200 sec-¹, 100 sec-¹, 20 sec-¹) blood viscosity increases and generalized subcompensated endothelial dysfunction (the absence of physiological decrease of BV in OT).

In the period of a low solar activity on the background of the III phase blood clotting activating (FG increasing), in the period of a high solar activity – without this mechanism activation.

In the period of a high solar activity in the patients with UA there is a higher BV and a misfit in endothelium vessels of different diameters (comparing with the period of a low SA).

References

Breus T.K. *Thesis of a Doctor of Physics and Mathematics*, Moscow, 2003, 45 p.

Backer T.L., Buyezere M., Segers P. et al.: 2002, *Aterioscleroses*, **165 (2)**, 367.