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ERYTHROCYTES FUNCTIONAL FEATURES IN THE 11-YEAR SOLAR CYCLE

Parshina S.S.[1], Tokayeva L.K.[1], Dolgova E.M.[1], Afanas'yeva T.N.[1], Samsonov S.N.[2], Petrova V.D.[1], Vodolagina E.S.[1], Kaplanova T.I.[1], Potapova M.V.[1]

[1] Saratov State Medical University n.a. V.I. Razumovsky of the Ministry of Health of Russia, St. B. Kazach'ya, 112, Saratov, Russia, *parshinasvetlana@rambler.ru*

[2] Institute of Cosmophysical Research and Aeronomy n.a. U.G. Shafer, Russian Academy of Sciences, Lenin Avenue, 31, Yakutsk, Russia, *s_samsonov@ikfia.ysn.ru*

ABSTRACT. There had been studied features of rheological blood failures in patients with unstable angina (UA) in periods of the high (HSA) and low solar activity (LSA) in the 23rd 11-year solar cycle. This category of patients is characterized by prethrombotic blood state, although they don't have coronary thrombosis. The research aimed to study compensatory mechanisms which block thrombosis development at the solar activity increase. There had been established that the period of the solar activity increasing in the 11-year solar cycle is characterized by an increase of a blood viscosity, comparing with the period of a low solar activity. Though, erythrocytes functional features in this case are compensatory mechanisms – erythrocyte aggregation paradoxically reduced and their deformability increases. It is probably connected with the revealed fibrinogen decrease in the period of the high solar activity. We can see that the change of a solar activity is accompanied not only by the progressing of pathologic processes, but also by an activation of adaptive changes in erythrocyte membrane so0 as to prevent thrombosis. Though, the required compensatory mechanisms were found invalid, which were shown in the decrease of an oxygen delivery to tissues, and the effectiveness decrease of the medical treatment in the period of a HSA.

Keywords: solar activity, unstable angina, blood rheological properties, erythrocytes functional features.

1. Introduction

Unstable angina (UA) is an exacerbation of an ischemic heart disease and is characterized by developing of a prethrombotic state, and rheological blood properties (blood viscosity and erythrocytes functional features), in this case, are of prime importance (Voskoboi, 1995). The external influense on haemorheological defects at UA are considered only in a few studies (Parshina, 2006). Although it is known that the cosmic weather influences significantly on a cardiovascular system condition (Samsonov et. al., 2016).

The research aimed studying erythrocytes functional features (aggregation and deformability) in the 11-year solar cycle in order to reveal compensatory mechanisms which prevent thrombosis at the solar activity (SA) increase.

2. Results and discussion

200 patients with UA had been examined. 120 of them were in the group of the high solar activity (HSA). They had been examined in the period from the 2nd up to the 5th year of the 11-year solar cycle. This period is characterized by an increment of a solar activity (SA) and high values of the Wolf's numbers (112,0±2,9). The group of the low SA (LSA) consisted of 80 patients, who had been examined in the period from the 6th up to 11th year of the solar cycle. The second half of the 11-year solar cycle was characterized by the decrease of a SA and by the low value of the Wolf's numbers (62,4±3,9; $p<0,05$ with the period of a HAS).

In the group of a HAS a higher blood viscosity (BV) had been noted in vessels of a medium diameter (on the 100 s^{-1} rates of shear), than at patients in the period of a LSA ($p<0,05$) (Table 1). At the same time the increase of a SA had not influenced on a blood fluidity in vessels of big and small diameters (on the 200 s^{-1} and 20 s^{-1} rates of shear) ($p>0,05$) (Table 1).

Table 1. Blood rheological properties, hematocrit index and fibrinogen level at patients with unstable angina in the high and low solar activity (M±m)

Parameters	Unstable angina	
	High SA (n=120)	Low SA (n=80)
BV 200 s^{-1}, mPa·s	6,58±0,13	6,38±0,13
BV 100 s^{-1}, mPa·s	10,50±0,44 *	7,19±0,18
BV 20 s^{-1}, mPa·s	11,73±0,45	10,59±0,39
EA, c.u.	1,18±0,03 *	1,46±0,03
ED, c.u.	1,55±0,06 *	1,10±0,08
Ht/ η, c.u.	6,51±0,02 *	7,09±0,16
Ht, %	40,3±0,5 *	42,9±0,3
FG, g/l	3,69±0,08 *	3,97±0,11

Notes: SA – solar activity;

* – the difference between the group of the HSA and the LSA is statistically valid, $p<0,05$.

It had been found that erythrocyte functional features are very sensitive to SA change. So, in the high SA there is lower erythrocyte aggregation activity (EAA) than in the low SA ($p<0,05$) (Table 1).

It is known that a high EAA increases a local BV in postcapillary venules, slows down the blood velocity, increases capillary tension. This leads to extravasation of fluids, tissue hypostasis, ischemia, capillars rupture, it also causes a decrease of oxygen delivery to tissues (Korkushko & Lishnevskaya, 2005). That is why revealed decrease of EAA in increase of a SA at patients with UA is an important compensatory mechanism which prevents progressing of myocardial ischemia.

Besides, there had been noted a fast growth of erythrocyte deformability (ED) in a HSA comparing with the period of a LSA ($p<0,05$) (Table 1). That is ED which let erythrocytes go through vessels the diameter of which is commensurately to their sizes. As even a 10% decrease of ED leads to serious failures in tissue oxygenation (G. Cicco, A. Pirelli, 1999), a hypothesis for a leading role of the non-ischemic hypoxia caused by ED defect, in atherosclerosis pathgenesis (Korkushko & Lishnevskaya, 2005). An increase of ED can be connected with the decrease of fibrinogen (FG) level as well, in the increase of SA (Table 1).

Hematocrit (Ht) parameter had been decreased in the group of a HSA comparing with the group of a LSA ($p<0,05$) (Table 1), which can be considered as another compensatory mechanism preventing further defect of a blood fluidity in microcirculation zone, in increase of a SA.

The analysis of the study results shows that the basic factor of a haemorheological decompensation in the SA increase at patients with UA is an increase of a blood viscosity. Erythrocytes functional features in this case serve as a compensatory mechanism – erythrocyte aggregation paradoxically reduced and their deformability increases.

At the same time the tissue oxygen delivery (Ht/η) in the HAS was lower than in the period of a LSA ($p<0,05$) (Table 1). This means that despite of the activation of compensatory haemorheological mechanisms in the increase of a SA (EA decrease and ED increase), adaptative ability at patients with UA are not enogh to eradicate an ischemic heart disease hypoxia. Taking to account the given results, we should consider haemorhelogical changes at patients with UA in the period of a HSA as a compensatory mechanism failure.

The hypothesis finds endorsement in the study of treatment effectiveness in the periods of HSA and LSA. We had established that medical treatment effectiveness depends on the period of a SA: in the LSA antianginal effect was more evident than in the HSA ($2,27\pm0,16$ points and $1,75\pm0,12$ points, $p<0,05$).

From there, an increase of a SA at patients with UA is accompanied by not only pathological processes developing, but also by an activation of compensatory changes in erythrocyte membrane which prevent thrombosis. Though, the required compensatory mechanisms were found invalid, which were shown in the decrease of an oxygen delivery to tissues, and the effectiveness decrease of the medical treatment in the period of a HSA.

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