

CARDIOVASCULAR DISORDERS IN EARLY FORMS OF CHRONIC RESPIRATORY FAILURE

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The progress of chronic cor pulmonale as a cardiovascular syndrome of manifested respiratory failure results from consecutive general-pathological microcirculatory changes. The latter can be considered early criteria for respiratory insufficiency evolution.

The aim of the present work is to study the cardiovascular disorders in latent and first stage respiratory failure (RF). Some general-pathological microcirculatory changes related with the pathophysiology of the respiratory insufficiency are also the object of our investigation.

Material and methods

Our study covered 30 patients with latent RF (LRF) and 70 ones with 1st stage RF (RF₁). Mean age was 45.9 ± 4.1 years in the first and 52.4 ± 1.8 years in the second group. Patients' selection was carried out according to indexes reflecting the changes in the acid-base balance. LRF patients had at normal acid-base profile hypoxemia (PaO_2 below 70 mm Hg) after veloergometric submaximal loading. 1st stage RF patients had hypoxemia at rest (PaO_2 below 70 mm Hg) and normal PaCO_2 (3). Most patients (80 per cent) had chronic obstructive pulmonary disease with predominant B-form (bronchitis form). The following indexes were investigated in patients and healthy controls: blood viscosity by using of rheo-viscosimeter of Höpner, capillary resistance after Kuchmeister, antithrombin III (AT III), acid-base profile, hematocrit, ECG, spirogram, working test and routine paraclinical examinations as well. Because of the different character of methods applied the number of control individuals from «healthy» groups varied but with almost equal age distribution and male:female ratio similarly to the patients under investigation.

Results and discussion

The levels of the indexes studied in control persons were presented on table 1.

1. Hemorheological changes.

Hematocrit rates were in normal ranges (44.93 per cent) in patients without disorders of acid-base profile. Hematocrit was 48.42 per cent in the patients of the second group. There existed a moderate correlation between SaO_2 and hematocrit (table 2).

Erythrocytes influenced upon viscosity not only by hematocrit but also by rigidity changes. In order to exclude hematocrit effect on viscosity we determined the relative viscosity, i. e. viscosity of a patient related to viscosity of healthy individuals divided by patient's hematocrit and multiplied by 100 per cent.

Table 1

Control values of indices studied

Indices	Control group n	males n	females n	Mean age years	Mean values
Hematocrit	200	154	46	52.0±1.8	42.73±1.92
Capillary resistance	200	154	46	52.0±1.8	17.9±2.4 cec (for 2—3 pete- chiaie)
Absolute blood viscosity	32	25	7	48.6±1.7	3.26±0.24 cP
Antithrombin III ^{fd}	30	21	9	54.2±0.9	21.80±3.9 mg %

Table 2

Hematocrit levels and correlation in respiratory failure

Patients n=100		Ht	pH	PaCO ₂	PaO ₂	r	SaO ₂	r
1 latent RF n=30	\bar{x}	44.93	7.39	40.30	88.57		92.6	
	S	6.48	0.03	3.28	3.53	-0.21	2.73	-0.05
	S \bar{x}	1.18	0.006	0.60	0.64		0.50	
2 1 st degree RF n=70	\bar{x}	48.42	7.39	41.65	63.01		90.37	
	S	5.05	0.03	2.37	4.78	-0.08	2.36	-0.36
	S \bar{x}	0.60	0.004	0.28	0.37		0.28	
p 1 to 2				>0.05	<0.001		<0.05	

By this way we could obtain information about both hematocrit and erythrocyte rigidity (2, 4).

If there was no deviation in the sense of increased rigidity of erythrocytes when biochemical indexes were concerned, the relative viscosity of any person examined should be 100 per cent. Absolute viscosity of our patients was within the limits of the confidential intervals in healthy persons. However, it was slightly increased in the group with hypoxemia (105.36 per cent) when related to hematocrit.

2. Influence of capillary resistance

The investigation of the capillary resistance was performed at hospitalizing of our patients. The changes in the course of the disease of this parameter were related to oxygen pressure in arterial blood. Capillary resistance showed rates which were the closest ones to the normal level (20.65 sec) when LRF patients were concerned. However, it was statistically significantly increased ($p < 0.01$) up to 22.16 sec in patients with hypoxemia as compared with that of the first group.

Capillary resistance was determined by the status of the vascular wall and erythrocytes. Probably, hypoxemia contributed to intensive calcium sedimentation on the endothelial wall (5).

3. Changes of the level of AT III, thrombocytes, and fibrinogen.

Mean AT III levels were in the lower limit of the normal range — 18.44 ± 3.05 mg % — in LRF patients. They were 16.59 ± 3.06 mg %, i. e. 76.1 per cent of the normal ones in the patients of the second group. There was a moderate correlation between PaO_2 and AT III ($r=0.43$).

Hypoxia in chronic respiratory insufficiency is not only hypoxic but also circulatory — of congestive type. Hepatic alterations are probably one of the reasons for reduced AT III synthesis.

Thrombocytes and fibrinogen were in normal ranges in our patients. Alveolar hypoxemia stimulated the liberation of catecholamines and prostaglandins which were powerful aggregating factors in the whole circulation (8).

4. Degree of expressiveness of ECG changes

Certain changes in heart position and pulmonary electroconductivity set in a long time before exhausting of the compensatory mechanisms of gas metabolism in a case of chronic bronchitis and pulmonary emphysema (1, 6, 7). In our LRF patients there were no ECG changes after submaximal loading. There were dynamic changes of T-wave in the patients of the 2nd group. Biphasic P or P with rising amplitude persisting up to 60 min long in the restitution period was often present. P-wave changes of the type of P-pulmonale and P-Gotie occurred at the average in 20 per cent of the patients studied. Negative or biphasic T-waves, T-depression (in the second, third and AV-leads), and $S_1.S_2.S_3$ — syndrome were present in the same frequency, too. The syndrome of $T_D < T_j$ appeared after submaximal loading in three patients of this group that could be considered an expressed myocardial hypoxia.

Cardiovascular changes in early forms of RF are results of a series of intravascular, vascular, and probably of extravascular alterations. A certain threshold is required for the progress of hypoxia and its stimulating effect on erythropoiesis. This threshold coincides with SaO_2 reduction. Our investigations show that hematocrit is the strongest factor influencing upon blood viscosity. Capillary resistance is sensitive to blood-gas abnormalities. It depends on erythrocyte changes as well as on structural alterations in the vascular wall.

AT activity reduction is a chief factor which enhances the thrombotic risk in RF patients.

We could draw the following conclusions:

1. RF alters the hemorheological status by the way of increased blood viscosity, polyglobulia and disturbed functional activity of erythrocytes.
2. AT III synthesis belongs to the most sensitive link of hemostasis regulation in case of RF.
3. ECG changes provide information not only about the myocardial lesion but also about the severity of the respiratory insufficiency.

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НАРУШЕНИЯ СЕРДЕЧНО-СОСУДИСТОЙ СИСТЕМЫ В РАННИХ СТАДИЯХ ХРОНИЧЕСКОЙ ДЫХАТЕЛЬНОЙ НЕДОСТАТОЧНОСТИ

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РЕЗЮМЕ

Было исследовано 100 больных с латентной хронической дыхательной недостаточностью, с дыхательной недостаточностью первой степени, а также с некоторыми общепатологическими и сердечно-сосудистыми изменениями микроциркуляции. Установлено, что дыхательная недостаточность вызывает изменения гемореологического статуса путем повышения вязкозитета крови, полиглобулии и нарушенной функциональной активности эритроцитов. Установленные электрокардиографические изменения дают представление не только о патологических изменениях сердечной мышцы, но и о тяжести дыхательной недостаточности. Устанавливается, что синтез АТ III является одним из наиболее чувствительных звеньев регуляции гемостаза при дыхательной недостаточности.