INHIBITORY ACTION OF THE BLOOD SERUM FROM PATIENTS WITH ATHEROSCLEROSIS ON LIPOLYTIC ACTIVITY OF THE AORTIC WALL

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UDC 616.13-004.6-07:[616.132-008.939.15-02:616.15

The inhibitory action of blood serum from patients with atherosclerosis on lipolytic activity of the aortic wall of rats in experiments in vitro is not due to its heparin content. After dialysis of the serum through cellophane with a pore size of 30 Å, only its protein (undialyzed) part exhibits an inhibitory action.

A previous investigation [3] showed that the blood serum of patients with atherosclerosis can inhibit the lipolytic activity of the rat aortic wall in vitro.

In the present investigation this inhibitory action of the serum was studied in more detail, for it may be a factor promoting deposition of lipids in the arterial wall [2, 4, 11].

EXPERIMENTAL METHOD

Blood serum from patients with atherosclerosis (women aged 50-70 years) was poured in a volume of 2 ml into a cellophane bag (pore size of the cellophane about 30 Å) and dialyzed against 8 ml distilled water for 24 h. The resulting dialysate was lyophilized, and dissolved in 2 ml distilled water before the experiment. The lipolytic activity of the rat aortic wall was investigated by a modification of the method described in [3, 11]. The aortas of rats were incubated in lipid substrate (2% Tween-80 solution in 4% solution of serum albumin in Krebs–Ringer–phosphate buffer, pH 7.4), mixed in the ratio of 2:1 with the serum, with its protein part, or with its dialysate. The lipolytic activity of the aortic wall was calculated as the difference between the content of higher nonesterified fatty acids (NEFA) in the substrate before and after incubation and expressed in μmoles/ml per g aortic tissue. The NEFA content was determined by Duncombe's method [8], and the free heparin concentration in the blood by Pieptea's method [10].

EXPERIMENTAL RESULTS

The experimental results are given in Table 1.

The results given in Table 1 show that the inhibitory action of blood serum from patients with atherosclerosis on the lipolytic activity of the aortic wall cannot be explained by a decrease in its content of heparin, an activator of lipolytic processes [9], which is known to be reduced in this disease [1, 7]. The results also show that the inhibitory action of blood serum from patients with atherosclerosis on the lipolytic activity of the aortic wall is concentrated in its protein fraction. The work of Lipovetskii has shown that the lipid-mobilizing factors of the pituitary gland, which are found in the dialyzable part of the serum, activate the lipolytic activity of the rabbit aortic wall in experiments in vivo [5], and that their level in the

Laboratory of Pathochemistry of Lipid Metabolism, Department of Pathophysiology, and Department No. 1 of Internal Medicine, Central Postgraduate Medical Institute, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR, N. A. Fedorov.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 72, No. 11, pp. 27-28, November, 1971. Original article submitted March 23, 1971.

TABLE 1. Relationship between Effect of Blood Serum from Patients with Atherosclerosis on Lipolytic Activity of the Rat Aortic Wall, Heparin Concentration in the Serum, and Action of Its Protein and Nonprotein Fractions (M ± m)

Lipolytic activity of aorta during inbucation with blood sera of patients (in μmoles/ml per g)	14.7 ± 0.62 (18)[1]	7.1 ± 0.57[2] (18)
P		P < 0.001
Heparin concentration in patient's blood (in units/ml)	7.3 ± 0.44 (18)	6.9 ± 0.41 (18)
P		P > 0.1
Lipolytic activity of aorta on incubation with sera (in μmoles/ml per g)	15.2 ± 0.52 (18)	8.3 ± 0.61 (17)
P		P < 0.001
Lipolytic activity of aorta on incubation with protein fraction of the same sera (in μmoles/ml per g)	16.4 ± 0.6 (18)	11.8 ± 1.25 (17)
P		P = 0.01
Lipolytic activity of aorta on incubation with sera (in μmoles/ml per g)	14.7 ± 0.48 (23)	8.0 ± 0.65 (18)
P		P < 0.001
Lipolytic activity of aorta on incubation with nonprotein fraction of the same sera (in μmoles/ml per g)	12.2 ± 0.83 (23)	14.4 ± 1.04 (18)
		P > 0.1

Note: 1) Number of investigations shown in parentheses. 2) Depending on their action on the lipolytic activity of the aortic wall, the sera were divided into two groups; sera during incubation with which the level of lipolytic activity was higher and lower, respectively, than the mean (11 μmoles/ml per g).

serum is lowered in patients with atherosclerosis [6]. The absence of correlation between the inhibitory action of the serum and its dialysate on lipolytic activity of the aorta in the present experiments indicates that this inhibition, in the group of patients investigated, was not connected with any possible lowering of the level of lipid-mobilizing factors in their serum. Further investigations using molecular sieves must reveal the components of the protein fraction of the serum with which its inhibitory action on lipid breakdown in the arterial wall is connected in atherosclerosis.

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