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MICROCIRCULATION AND BLOOD RHEOLOGY IN ARTERIAL AND VENOUS FORMS OF MESENTERIC VASCULAR OCCLUSION

N. P. Aleksandrova, V. V. Karpova, and L. N. Skuratovskaya

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KEY WORDS: microcirculation; blood rheology; occlusion

The aim of this investigation was to determine the character of the vascular response and the time course of development of disturbances of the blood rheology in acute mesenteric vascular insufficiency, due specifically to occlusion of the cranial mesenteric artery (CMA) and vein (CMV).

EXPERIMENTAL METHOD

Two series of experiments were carried out on 56 mongrel dogs weighing 18-20 kg. The control group consisted of seven dogs, in which the effect of immobilization of the animals in the supine position, laparotomy, and barbiturate anesthesia on parameters of the microhemodynamics and blood rheology was studied. A model of the disease was created by ligation of CMA (series I) and CMV (series II). Blood sampling and biomicroscopy were carried out 1 and 3 h after creation of the model. The blood viscosity was studied on a "Rotovisco RV-100" rotary viscometer (Haake, West Germany), with a shear velocity of 1 sec^{-1}. The hematocrit index was determined with a "KIT" hematocrit centrifuge (USA). The microcirculation was studied by contact biomicroscopy on the LYUMAN K-1 microscope (Leningrad Optico-Mechanical Combine), followed by photographic recording and morphometry of the specimen material on a "Mikrofot" apparatus (magnification 100). The results were subjected to statistical analysis on the PDP 11/34 computer (DES, USA).

EXPERIMENTAL RESULTS

The results are evidence that 1 h after occlusion of CMA generalized spasm of the afferent vessels of the small intestine was observed; the diameter of the arterioles was reduced from 42.6 ± 3.11 to 22.97 ± 1.1 μ ($p < 0.1$) and of the capillaries from 14 ± 0.1 to 8.1 ± 0.2 μ ($p < 0.001$). The diameter of the venules was reduced from 59.22 ± 2.8 to 40.83 ± 3.52 μ ($p < 0.01$). The capillary bed showed no significant change.

A completely different picture was observed 1 h after occlusion of CMV. The diameter of the afferent and efferent vessels was sharply increased: of the arterioles from 44.9 ± 3.1 to 57.9 ± 1.4 μ ($p < 0.1$), of the precapillaries from 11.5 ± 0.2 to 17.6 ± 1.3 μ ($p < 0.01$), and of the venules from 58.0 ± 1.7 to 98.7 ± 3.2 μ ($p < 0.01$). As a result of this reaction, dilatation of the capillaries was observed.

Persistent but nonprogressive vasoconstriction continued 3 h after acute occlusion of CMA in all components of the circulatory bed. During acute occlusion of CMV, on the other

No. 3 Department of Surgical Diseases and Department of Pathological Anatomy, Faculty of Internal Medicine, N. I. Pirogov Second Moscow Medical Institute. (Presented by Academician of the Academy of Medical Sciences of the USSR V. S. Savel'ev.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 107, No. 5, pp. 532-534, May, 1989. Original article submitted May 15, 1988.

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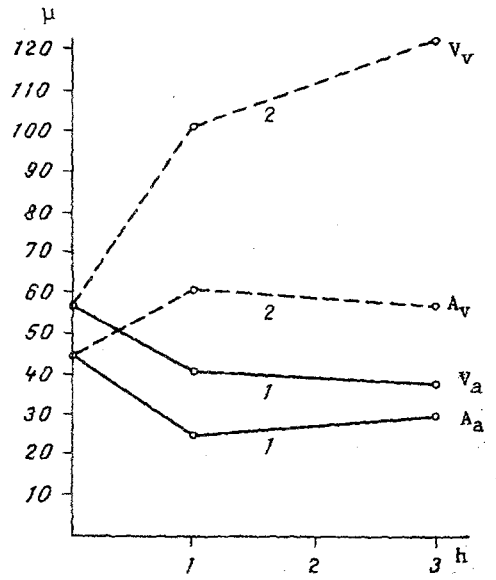

Fig. 1. Changes in diameter of vessels during disturbance of mesenteric circulation. 1) Occlusion of CMA (a); 2) occlusion of CMV (v); A) arterioles; V) venules.

hand, progressive and marked dilatation was observed: the diameter of the precapillaries compared with the previous stage of the experiment was increased up to 25.4 ± 2.6 compared with 17.6 ± 1.3 μ ($p < 0.05$); the diameter of the venules was increased to 117.8 ± 3.3 μ from 98.7 ± 3.2 μ ($p < 0.01$).

The results, reflecting the functional state of the microcirculatory bed of the small intestine during acute occlusion of CMA and CMV are evidence of a difference in the character of the vascular response to these different pathological processes.

The results of a study of the central hemodynamics are evidence that acute occlusion of CMV follows a more malignant course than acute occlusion of CMA, since at the same stages of the experiment (after 1 h and 3 h of occlusion) the cardiac output and stroke volume of the heart during acute occlusion of CMV were lower by 24 and 65% respectively than under normal conditions [3]. During occlusion of CMA, no statistically significant changes were observed in these parameters [1].

The results of investigation of the rheologic properties of the blood during acute occlusion of CMA and CMV demonstrated marked changes in both forms of occlusion.

In the case of acute occlusion of CMV, the structural viscosity of the blood and hematocrit index after 3 h of the experiment were statistically significantly greater than the corresponding parameters during acute occlusion of CMA (during occlusion of CMA the blood viscosity was 40.9 ± 4.56 cP; the hematocrit index 0.51 liter/liter; during occlusion of CMV the blood viscosity was 52.3 ± 2.47 cP and the hematocrit 0.6 liter/liter).

Acute occlusion of CMV is thus accompanied by the more rapid exhaustion of the adaptive and compensatory powers of the body compared with occlusion of CMA; this is reflected in the character and degree of change in the rheologic properties of the blood.

Our earlier parallel investigations of the microcirculatory bed in the wall of the small intestine and the bulbar conjunctive after acute thrombosis of CMA and CMV showed that, in response to occlusion accompanied in one case by local vasoconstriction of the microvessels of the intestinal wall, and in the other case by their marked vasodilatation (Fig. 1), a spastic reaction of both afferent and efferent vessels was observed at the periphery. The degree of vasoconstriction of vessels of the bulbar conjunctiva in acute thrombosis of CMA exceeded that accompanying occlusion of CMV [2, 3].

This state of affairs explains the presence of statistically significant differences in the degree of the disturbances of blood rheology and hemodynamics in the arterial and venous

forms of mesenteric vessels. This is further confirmation that a disturbance of the venous outflow from the intestine represents a greater risk to the animal than disturbance of the inflow within the same time interval.

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A DIFFERENTIAL INDICATOR METHOD OF IDENTIFYING ZONES OF ISCHEMIA AND NECROSIS IN RATS WITH EXPERIMENTAL MYOCARDIAL INFARCTION

L. N. Sernov and V. V. Gatsura

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KEY WORDS: size of a myocardial infarct; method of determination

Success in the search for therapeutic substances capable of reducing a zone of necrosis or infarction of the myocardium is largely dependent on the development of objective methods of assessment of damaged zones. Morphometric methods, most widely used at the present time, yield information on the size of zones of ischemia and necrosis [1, 6]. However, their use is limited by the complexity of the planimetric method of calculating the area of a lesion on the basis of individual sections, as well as its somewhat conventional nature.

A considerable step forward in improvement of the accuracy and simplification of the technique of mass investigations was the development of a method whereby the zone of ischemia and zone of necrosis can be estimated simultaneously as percentages of the total weight of the myocardium [4]. The basis for calculation of the zones of damage in this case is the difference in the quantity of dye contained in the hearts of rats with a myocardial infarct and intact animals. This last stage of affairs, however, is responsible for the main disadvantage of the method, namely the absence of an "internal" control, which makes the results obtained by this method much less comparable.

This paper describes a modification of the technique of determining zones of damage in rats with myocardial infarction, and which can yield original data for their calculation simultaneously in the same heart.

The principle of the method consists of using an "internal" control to identify zones of damage in each experiment on the basis of the difference between the quantity of dye in the damaged part of the myocardium and in its intact part. In this way the accuracy of the results can be greatly increased and the negative effect of possible deviations in the course of each experiment can be abolished.

As the indicator of the size of the zone of ischemia we used Evans' blue, perfusion of the isolated heart with which enables the size of the unperfused region to be estimated [3]. The indicator of the zone of necrosis was triphenyltetrazolium bromide which, like other soluble tetrazolium salts, on interaction with areas of myocardium preserving their dehydrogenase activity, is reduced into formazan [2, 5, 7].

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