Halo Formation as a Characteristic of Leukocyte Functional Activity

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Halo formation and phagocytic activity of leukocytes at different extreme states are regulated at the cellular level. Direct relationship between functional activity and halo formation is observed only during sublethal states in inflammatory diseases.

Key Words: *leukocyte activity; halo formation; terminal states*

Halo formation discovered 20 years ago became the object of studies for a group of scientists [1-4]. Observations on cancer patients suggest that halo-forming activity (HA) decreases during neutrophil activation [6], probably due to changes in glycocalyx during malignization. However modification of glycocalyx can occur in normal cells during various physiological states [10].

We evaluated phagocytic activity (PA) and the state of leukocyte glycocalyx in highly concentrated electrolyte in various terminal states.

MATERIALS AND METHODS

Experiments were performed on 140 random-bred male albino rats (300-400 g). Activity and functional capacity of leukocytes were studied in 3 experimental series. In series I, total dehydration was modeled via water deprivation [5]. In the series II the effects of exogenous hyperthermia on the studied functions were evaluated [9], and in series III experimental peritonitis was modeled [8]. The initial functional changes (stage I), peak activity of systemic reactions reflecting the resistance to extreme factor (stage II), and sublethal states (stage III) were evaluated. In series I, days 3, 6, and 10 of the experiment corresponded to these stages, in series II these were minutes 20, 75, and 120 of hyperthermia, and in series III — 3, 6, and 18 h postinjection.

Halo formation was studied as described elsewhere [6]. Halos were counted after 10 min (halo formation rate – HFR) and after 2-24 h (HA). Phagocytosis was evaluated as described previously [7]. PA (percentage of phagocytosing cells) and phagocytic index (mean number of phagocytosed particles) were evaluated.

RESULTS

In series II and III, neutrophil PA increased from stage to stage and the number of phagocytosed particles increased in parallel. In series I, PA first decreased and then returned to the control value, while phagocytic capacity of individual phagocytes increased by stage III (Table 1).

Changes in halo formation were similar during the initial stages of heating and dehydration: HFR decreased and the number of halos increased. At subsequent stages HFR varied and HA returned to the control level. During the initial stage of peritonitis HFR increased and HA decreased. In acute inflammation HA underwent phasic changes and HFR notably decreased during terminal stage (Table 2).

Differences between the studied functional states of the organism were determined not only by pathogenic factors (water deprivation, exogenous hyperthermia, pathogenic microorganisms), but also by the rate of pathological changes. Changes in halo formation

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TABLE 1. PA (%) and Phagocytic Index (PI) in Various Terminal States (*M±m*)

Experimental series		Control	Stages		
			I	II	III
Series I (dehydration)	PA	83.0±1.9	78.0±2.3*	81.0±2.3	86.0±2.1+
	PI	6.3±0.1	7.0±0.5	6.9±0.4	8.5±0.6*+°
Series II (hyperthermia)	PA	75.0±1.7	81.0±0.8*	87.0±1.1*+	93.0±0.7*+°
	PI	7.5±0.4	8.0±0.2*	10.3±0.3*+	11.4±0.4*+°
Series III (peritonitis)	PA	75.0±1.7	85.0±1.2*	86.0±1.1*	90.0±0.6*°
	PI	7.5±0.4	10.2±0.5*	11.8±0.4*+	14.9±0.5*+°

Note. Here and in Table 2: $p<0.05$ *vs.* *control, +stage I, °stage II.

TABLE 2. Halo Formation Parameters (in %) at Various Stages of Terminal States (*M±m*)

Experimental series		Control	Stages		
			I	II	III
Series I	HFR	0.61±0.05	0.39±0.05*	0.28±0.06	0.35±0.03*
	HA	6.25±1.90	12.17±1.40*	4.3±1.2+	5.13±1.06*
Series II	HFR	0.29±0.05	0.21±0.08*	0.26±0.05*	0.33±0.09
	HA	1.02±0.06	8.8±5.1*	0.50±0.09*+	1.1±0.2°
Series III	HFR	0.29±0.05	0.31±0.03*	0.24±0.02	0.13±0.03*+
	HA	1.02±0.06	0.59±0.08*	1.1±0.2+	0.50±0.05*°

were similar in hyperthermia and dehydration, while changes in PA were similar in peritonitis. This similarity can be explained by shifts in physicochemical parameters of the internal media, determining the behavior of leukocyte glycocalyx in the circulation. The increase in neutrophil phagocytic activity in acute inflammation and hyperthermia can be explained by similar temperature and/or humoral shifts.

The detected functional changes in leukocytes during exposure to extreme factors suggest that halo formation capacity of leukocytes is regulated autonomously, which is confirmed by the time course of halo formation at different terminal states. Blood plasma osmolarity is a factor modifying this characteristic. Phagocytic activity reflects general functional changes in the immune system, mediated by humoral shifts induced by high temperature or antigenic stimulation.

Halo formation and functional activity of leukocytes are not directly related. The decrease in halo formation during neutrophil activation [6] is apparently characteristic of only leukocytes. Morphofunctional characteristics of leukocytes are regulated by intracellular mechanisms. The correlation between phagocytosis reactions and halo formation is characteristic of only terminal stage of peritonitis, when the essential increase in PA and the number of phagocytosed particles is associated with a decrease in HFR and total number of halos. The increase in leukocyte nonspecific resistance to concentrated electrolyte solutions at this stage is apparently due to high "price of adaptation".

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