There are reports that IL-2 is effective not in all models of hypertension. For instance, no hypotensive action of IL-2 could be found on a model of renal hypertension [12]. In this case it is admissible to postulate a specific effect of IL-2 in the essential form of arterial hypertension.

Thus further proof has been obtained of the role of the immune system in the development of experimental hypertension and also, perhaps, of essential hypertension in man.

REFERENCES

1. T. E. Medvedeva, "The role of emotiogenically positive zone of the lateral hypothalamus in the regulation of the systemic hemodynamics in experimental arterial hypertension," Author's Abstract of Dissertation for the Degree of Candidate of Biological Sciences, Khar'kov (1988).
2. V. G. Pinelis, T. P. Vakulin, A. V. Kozlova, et al., Byull. Éksp. Biol. Med., No. 10, 31 (1982).
3. B. I. Shulutko and O. P. Budai, Terap. Arkh., No. 4, 125 (1987).
4. P. Bataillard, J.-C. Freiche, M. Vincent, et al., J. Hypertens., 4, Suppl. 3, 5455 (1986).
5. A. Bendich, E. H. Belisle, and H. R. Strausser, Biochem. Biophys. Res. Commun., **99**, 600 (1981).
6. D. J. Dzielak, Hypertension, **15**, 95 (1990).
7. R. A. Norman, D. J. Dzielak, K. L. Bost, et al., J. Hypertens., **3**, 261 (1985).
8. D. W. Pascual, H. Jin, K. L. Bost, et al., Hypertension, **16**, 468 (1990).
9. J. G. Svendsen, Acta Path. Microbiol. Scand., **85**, 548 (1977).
10. N. Takeichi, K. Suzuki, and H. Kobayashi, Clin. Exp. Immunol., **40**, 120 (1980).
11. N. Takeichi, D. Ba, and H. Kobayashi, Cell. Immunol., **60**, 181 (1981).
12. R. S. Tuttle and D. P. Boppana, Hypertension, **15**, 89 (1990).

IMMUNOCORRECTION IN DESTRUCTIVE PANCREATITIS

N. K. Manucharov, Yu. D. Tavdidishvili, E. N. Akhvlediani, M. D. Sychev, K. B. Tomaev, and M. M. Kiladze

UDC 616.72-002.1-085.275.2

KEY WORDS: pancreatitis; immunocorrection.

A factor in the pathogenesis of acute pancreatitis [8-10] is a marked disturbance of hemostasis, caused initially by enzymic and later by suppurative toxicosis. For that reason methods of detoxication used in the combined treatment of acute pancreatitis must be regarded as pathogenetically justified. In recent years interest of research workers in the study of the possible role of immunocorrective mechanisms in the pathogenesis of acute pancreatitis has increased. The aim of this investigation is a comparative study of the immunocorrective influence of modern methods of detoxication in acute destructive pancreatitis.

EXPERIMENTAL METHOD

Experiments were carried out on 40 male mongrel dogs weighing 13-15 kg. The model of destructive pancreatitis followed that described in [1]. Acute pancreatitis was treated by hemoperfusion (HP), plasmapheresis (PPH),

K. D. Éristavi Research Institute of Experimental and Clinical Surgery of Georgia, Tbilisi. (Presented by Academician of the Russian Academy of Medical Sciences A. D. Ado.)

Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 114, No. 8, pp. 183-185, August, 1992. Original article submitted April 15, 1991.

TABLE 1. Changes in Immunologic Reactivity of Dogs with Acute Pancreatitis and during Treatment

Group of dogs	Number of leukocytes, 10^9/liter	Number of neutrophils, %		Number of lymphocytes, %		Number of T lymphocytes, %		Number of B lymphocytes, %		Number of 0 lymphocytes, %		Number of E-RFC, %		Complementary activity, U/ml
		10^9/liter		10^9/liter		10^9/liter		10^9/liter		10^9/liter		10^9/liter		
Intact	7,31±0,70	69,55±1,94	4,76±0,44	21,52±1,77	1,52±0,10	50,45±0,8	0,91±0,02	25,0±0,8	0,39±0,03	24,55±0,12	0,22±0,04	34,8±3,02	1,63±0,12	49,91±0,37
Destructive pancreatitis	16,58±1,65	80,35±0,89	12,37±0,75	11,97±0,77	1,98±0,16	41,82±3,31	0,82±0,07	22,27±2,02	0,44±0,03	35,91±0,3	0,72±0,08	30,47±3,07	2,92±0,21	57,46±0,46
Method of ECCXS treatment	15,10±1,38	76,5±0,8	11,55±1,12	15,4±1,03	2,32±0,3	43,8±1,03	2,32±0,3	23,2±2,05	0,53±0,04	33,0±0,09	0,77±0,01	33,0±3,25	3,81±0,25	56,0±0,40
UV irradiation of autologous blood	14,01±1,16	76,9±0,97	10,70±1,04	15,4±1,5	2,15±0,11	50,03±3,6	1,08±0,10	25,6±2,01	0,55±0,05	24,4±0,23	0,52±0,05	28,0±2,2	3,01±0,29	55,1±1,48
Hemoperfusion	12,7±1,25	73,5±0,8	9,30±0,81	17,6±0,8	2,23±0,13	51,7±4,6	1,15±0,11	23,2±2,02	0,51±0,03	25,0±0,2	0,57±0,05	40,8±3,4	3,80±0,23	49,0±0,41
Plasmapheresis	11,34±0,95	72,7±0,71	8,38±0,53	18,8±0,75	2,16±0,09	47,1±3,6	1,01±0,05	23,5±1,01	0,51±0,03	29,4±0,18	0,64±0,06	28,9±2,54	2,42±0,04	50,26±0,4

UV irradiation of autologous blood, and extracorporeal connection to a xenogeneic spleen (ECCXS). The absolute (in 1 liter) and relative (in %) content of T and B lymphocytes in the dogs' peripheral blood was determined by the method of spontaneous rosette formation with sheep's and mouse red blood cells, and also by the rosette-forming ability of neutrophils, with the aim of revealing their functional activity [4, 6, 7]. A parallel study was made of the time course of the change in serum lysozyme level [3] and activity of complement by the 50% hemolysis method. Blood was taken from a vein of the dogs at all stages of the experiment: in the control (intact animals), at the height of the disease (the most marked clinical manifestations of destructive pancreatitis) and after the end of treatment.

EXPERIMENTAL RESULTS

During the period of manifest development of acute destructive pancreatitis the animals showed marked deviations of the parameters of the immunologic status (Table 1). These took the form of a significant fall in the number of T lymphocytes in the peripheral blood (from the initial 50.45 ± 0.81% to 41.82 ± 3.31%) in the peripheral blood, with predominance of lymphocytes with low avidity (rosettes with 3-5 erythrocytes). Less marked changes were recorded in the humoral component of immunity. The number of B lymphocytes fell from an initial 25.0 ± 0.8% to 22.7 ± 2.02% ($p = 0.05$). The level of 0 lymphocytes was sharply increased (from 24.55 ± 0.12% to 35.91 ± 0.3%), which is itself an unfavorable sign, for it indicates reduction of expression of E receptors of T lymphocytes, probably under the influence of endogenous factors, caused by the general toxicosis. An increase in the serum lysozyme concentration also was observed (from 41.35 ± 0.87 to 49.6 ± 0.91%). This rise was evidently connected with an increase in functional activity of the neutrophils at this period (the absolute number of rosette-forming neutrophils in the peripheral blood was almost doubled compared with the control). Activity of complement was considerably depressed compared with normal.

The methods of detoxication used in acute pancreatitis in this study proved to vary in the efficacy of their immunocorrective action. The most positive immunocorrective effect was observed after the use of the xenogeneic spleen and UV irradiation of autologous blood. In these cases the number of T and B lymphocytes in the peripheral blood returned to normal, simultaneously with an increase in avidity of the lymphocytes (a higher percentage of rosettes with 5-10 erythrocytes). The level of 0 lymphocytes was significantly depressed, evidently due to removal of endotoxins blocking the surface receptors of the lymphoid cells from the blood [5, 6].

After extracorporeal connection to a xenogeneic spleen an increase in the rosette-forming capacity of the neutrophils also was observed. This, as we know, reflects their adhesive activity. However, the plasma lysozyme level was a little lower than at the height of the disease, in all probability due to its rapid utilization. The complement level moved closer to the limits of normal fluctuations.

During plasmapheresis some increase also was noted in the activity of immunity mechanisms, but the parameters of this activity did not reach the original level. This may be due to some features of the action of the method used. During PPH several biologically active substances, including those of immune nature, are removed from the blood stream [2]. One result of this could be slowing of the recovery of the immune system in the initial period after plasmapheresis.

During the first days of hemoperfusion very slight positive shifts were observed in the blood levels of T and B lymphocytes. Marked stimulation of the rosette-forming capacity of the neutrophils and restoration of the lysozyme level were noted. Taken as a whole, these observations point to an increase in total functional activity of the neutrophils, and they are in agreement with data published previously [8-10].

Thus in acute destructive pancreatitis inhibition of the mechanisms of immunity, and mainly of its T-cell component, takes place. The most favorable and rapid immunocorrective action on these mechanisms is exhibited by UV irradiation of the blood and the use of a xenogeneic spleen.

Hemoperfusion, incidentally, has a normalizing, and even stimulating, effect on the parameters of immunity in the later period after the beginning of treatment (the 3rd and 7th days).

REFERENCES

1. V. M. Buyanov et al., Vestn. Khir., No. 9, 28 (1989).
2. E. V. Gembitskii et al., Revmatologiya, No. 3, 3 (1987).
3. I. I. Dzerzhinskaya, Immunologiya, No. 1, 64 (1982).
4. V. G. Dorofeichuk, Lab. Delo., No. 1, 28 (1968).
5. V. I. Midlenko, Vestn. Khir., No. 2, 33 (1982).
6. L. V. Poluektov et al., Vestn. Khir., No. 21, 92 (1982).
7. Zh. Sundetov, "Correlation of humoral factors and immunity," Author's Abstract of Dissertation for the Degree of Candidate of Sciences, Aktyubinsk (1973).
8. L. D. Taranenko et al., Vestn. Khir., No. 2, 35 (1990).
9. V. S. Savel'ev et al., Acute Pancreatitis [in Russian], Moscow (1983).
10. V. I. Filin, Acute Diseases and Injuries of the Pancreas. Textbook for Physicians [in Russian], Meditsina, Leningrad (1983).
11. A. B. Tsypin et al., Treatment of Septic Diseases by Connection to a Xenogeneic Spleen: Technical Recommendations [in Russian], Moscow (1988), p. 18.

EXPRESSION OF TRANSCOBALAMIN II RECEPTORS OF THE PLASMALEMMA OF HUMAN BLOOD LYMPHOCYTES STIMULATED BY MITOGENS

A. E. Oreshkin, M. V. Gudkova, and N. V. Myasishcheva

UDC 616-006.6-07:616.155.32-02

KEY WORDS: transcobalamin II receptor; human blood lymphocytes; endocytosis of Co-cyanobalamin; stimulation of division.

In mammalian cells cobalamin-dependent methionine synthetase is the key regulatory mechanism of formation of the pool of folate coenzymes that are essential for synthesis of the purine and pyrimidine bases of RNA and DNA [2, 10]. Embryonic, normal proliferating, and tumor cells take up exogenous precursors of the cobalamin coenzymes from the surrounding medium [1, 3]. Interaction of a complex of cobalamin and a blood plasma transport

Laboratory of Endogenous Modifying Factors of Carcinogenesis, Research Institute of Carcinogenesis. All-Union Oncologic Scientific Center, Russian Academy of Medical Sciences, Moscow.

(Presented by Academician of the Russian Academy of Medical Sciences N. N. Trapeznikov.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 114, No. 8, pp. 185-187, August, 1992. Original article submitted December 25 1991.