LIPID PEROXIDATION IN THE BLOOD IN PNEUMONIA

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It is stated in the literature that lipid peroxidation (LPO) in the plasma and blood cells in inflammatory diseases of the lungs depends on the clinical variant of the disease [1, 3]. Correlation between LPO in the blood and morphological changes of inflammation in the lungs has not been adequately studied. This paper is devoted to a study of this problem.

EXPERIMENTAL METHOD

Inflammation of the lungs was induced in rabbits by introducing a foreign body (a Kapron thread 0.5 mm in diameter and 6-8 cm long) into the trachea [4]. The foreign body was removed from the trachea of some rabbits after remaining 5 days or 1 and 3 months *in situ*. The investigation was conducted 5 days, 1 week, and 1 and 3 months after introduction of the foreign body into the trachea and 2 weeks after its removal. Healthy rabbits served as the control. Blood was taken from the marginal vein of the ear. The conjugation of diene conjugates (DC) in extract of blood plasma lipids was measured spectrophotometrically at a wavelength of 232 nm [7] and the lipid concentration in the extract was determined by the sulfophosphovanillin method [5]. Total emission of chemiluminescence (CHL) of blood plasma and erythrocytes, stimulated by hydrogen peroxide (final concentration 0.03 M) was measured under quantometric conditions during the 30 sec after addition of peroxide [6]. Blood catalase activity (CA) was determined by the method of Bakh and Zubkova [2]. Lung sections were stained with hematoxylin and eosin and with picrofuchsin by Van Gieson's method. The quantitative results were subjected to statistical analysis by Student's t test.

EXPERIMENTAL RESULTS

Focal catarrhal bronchitis with moderate destructive and exudative changes in the bronchial mucous membrane developed 5 days after introduction of the foreign body into the trachea. Changes in the blood consisted of some increase in the DC concentration and a tendency for CA to fall. One week after the beginning of the disease, diffuse catarrhal-suppurative bronchitis was observed in the lungs, with marked destructive and exudative changes in the bronchial mucous membrane. In the blood, just as at the previous time of inflammation, the DC concentration was increased and CA was reduced. CHL of the erythrocytes also was intensified and CL of the plasma showed a tendency to weaken (Table 1). After 1 month of inflammation, bronchopneumonia with abscess formation developed. A combination of even more marked (even to the extent of small foci of suppuration and necrosis) and more widespread destructive-exudative changes was observed with activation of proliferation of connective-tissue cells (macrophages, lymphocytes, fibroblasts) and initial sclerosis (formation of collagen fibers around foci of histolysis). An increased DC concentration, intensified CHL of the erythrocytes, and weakened CHL of the plasma persisted in the blood. Meanwhile, CA returned to normal. After 3 months suppurative-necrotic changes (lung abscess), proliferation of connective-tissue cells, and marked focal (organization of foci of histolysis) and diffuse (peribronchial, perivascular, and in the alveolar tissue) sclerotic changes were combined in the lungs. Besides CA, CHL of the plasma and erythrocytes returned to normal in the blood. After removal of the foreign body from the trachea, inhibition of degenerative and exudative changes took place in all cases in the lungs, and after 1 and 3 months of inflammation, activation of cell proliferation (connective-tissue cells, bronchial and alveolar

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TABLE 1. Blood LPO Parameters in Rabbits with Inflammation of the Lungs (M ± m)

Group of animals	Diene conjugates, nmoles/g lipids	Catalase activity, μmoles $H_2O_2/10^6$ erythrocytes	Chemiluminescence (relative units)	
			of plasma	of erythrocytes
Healthy	580±38	108±4	1000±58	1000±57
Sick:	(n=23)	(n=39)	(n=37)	(n=34)
after 5 days	860±194	89±9	796±50	—
	(n=4)	(n=5)	(n=4)	
P	>0,05	>0,05	>0,1	
after 1 week	930±178	88±6	784±17	1367±235
	(n=8)	(n=7)	(n=8)	(n=8)
P	<0,01	<0,05	<0,1	<0,05
after 1 month	828±157	101±12	472±164	1622±247
	(n=4)	(n=4)	(n=4)	(n=4)
P	<0,05	>0,1	<0,01	<0,01
after 3 months	431±48	112±3	980±152	975±95
	(n=6)	(n=8)	(n=9)	(n=7)
P	<0,1	>0,1	>0,1	>0,1
Recovering (2 weeks after inflammation of lungs):				
duration 5 days	699±175	94±8	1352±82	—
	(n=4)	(n=5)	(n=4)	
P	>0,1	>0,1	<0,1	
duration 1 month	512±82	112±7	905±112	—
	(n=6)	(n=5)	(n=5)	
P	>0,1	>0,1	>0,1	
duration 3 months	294±27	109±21	—	693±167
	(n=3)	(n=3)		(n=3)
P	<0,05	>0,1		>0,1

epithelium) with sclerosis of the lung tissue was observed. A distinguishing feature of these changes after 3 months of inflammation compared with 1 month was the weaker inhibition of destructive and exudative processes and the much greater degree of sclerosis of lung tissue. After 5 days of inflammation the DC concentration and CA activity of the blood were back to normal and the plasma CHL showed a tendency to rise. After 1 month of inflammation CA remained normal, the DC concentration and plasma CHL had returned to normal, and after 3 months CA and CHL of the erythrocytes still remained normal and the DC concentration showed an even greater fall.

The results given below show definite correlation between inflammatory changes in the lungs and LPO in the blood. In particular, the development of degenerative and exudative changes is accompanied by an increase in the DC concentration in the blood plasma lipids (after 5 days, 1 week, and 1 month of inflammation) and a fall in the blood CA activity (after 5 days and 1 week of inflammation). Potentiation of degenerative and exudative changes to the limit of their manifestation, in the form of suppurative and necrotic changes, corresponds to weakening of plasma CHL and strengthening of erythrocyte CHL (1 week and 1 month of inflammation). During activation of connective-tissue cell proliferation the normal blood CA level is restored (1 and 3 months of inflammation); a decrease in the DC concentration and normalization of plasma and erythrocyte CHL correspond to the development of sclerosis (3 months of inflammation). Correlation between destructive and exudative processes, on the one hand, and an increase in the DC concentration and decrease in CA activity on the other hand, is confirmed by the normalization of these parameters after removal of the foreign body from the trachea on the 5th day of inflammation. Normalization of the plasma CHL 2 weeks after 1 month of inflammation is further evidence of the connection between this parameter and the suppurative and necrotic changes. Correlation between sclerosis of lung tissue and a fall in the DC concentration is reflected in the fact that 2 weeks after 3 months of inflammation there was a further fall in the DC concentration. Changes in blood LPO are thus connected both with inflammatory changes and with repair processes in the lungs. In particular, normalization of blood LPO values can be observed both during inhibition of destructive and exudative processes and during sclerosis of lung tissue. This must be taken into consideration when inflammatory lung diseases are diagnosed on the basis of the state of LPO in the blood and in the course of LPO-corrective treatment of pneumonia.

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COLLAGEN RESORPTION BY HEPATOCYTES DURING REGRESSION OF CIRRHOSIS OF THE LIVER

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The possibility of regression of cirrhosis of the liver has been conclusively proved by many investigations [1, 2]. The study of this problem is now concentrated mainly on elucidation of the mechanism of resorption of sclerotic tissue. Since collagen destruction in the liver is considered to take place entirely extracellularly, most research has been devoted to a study of the role and degree of participation of collagenase in this process [4-8]. It is not known whether hepatocytes participate in collagen resorption and, if they do, how this participation is manifested.

In the investigation described below an electron-microscopic method was used to study the liver in experimental cirrhosis and its regression. Attention was concentrated on hepatocytes which, according to the writer's observations, play an important role in the resorption of sclerotic tissue.

EXPERIMENTAL METHOD

Noninbred male albino mice were used. Cirrhosis of the liver was induced by injection of 0.2 ml of a 40% solution of CCl_4 in olive oil into the animals subcutaneously once a week for 5 months. To stimulate regeneration, 10 days after the last injection of CCl_4 all the animals underwent resection of the left lobe of the liver. Partial hepatoectomy has been shown [1, 3, 9] to accelerate regeneration of the cirrhotically changed liver considerably. Material for study was taken during resection and 5, 10, and 15 days thereafter. Material for histological investigation was fixed in 10% formalin, for electron microscopy — in OsO_4 or glutaraldehyde, followed by postfixation in OsO_4, dehydrated, and embedded in Epon. Serial ultrathin sections were cut (50-60 from each block) and examined in the ÉMV-100L electron microscope.

EXPERIMENTAL RESULTS

A picture of cirrhosis with annular proliferation of connective tissue and the formation of pseudolobules was observed in histological sections from pieces of liver removed during resection. Electron microscopy revealed bundles of collagen fibers of different thickness, with clearly distinguishable characteristic cross striation. Lipid inclusions were present in the cytoplasm of most hepatocytes. Far fewer lipid inclusions, but many lysosomes and peroxisomes were present 5 days after resection in the hepatocytes and the lamellar complex was well developed. Some bands of fibers 10 days after partial hepatectomy consisted of collagen fibers which had lost their cross striation. The ultrastructure of the

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