Intensity of Lipid Peroxidation: Indicator of Inflammation Severity in Chronic Bronchitis

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The content of products of lipid peroxidation and antioxidative activity were measured in exhaled air condensate and blood sera of patients with chronic bronchitis. Exacerbation of purulent bronchitis was found to be associated with an increase of heptane-soluble products of lipid peroxidation in serum and condensate, which was not observed in patients with catarrhal bronchitis. In catarrhal bronchitis, the content of heptane-soluble products is increased only in the serum; antioxidative activity increased in exhaled air condensate in catarrhal bronchitis and decreased in serum and condensate in purulent bronchitis.

Key Words: *chronic bronchitis; lipid peroxidation; antioxidative activity*

Any inflammatory process involves local accumulation of phagocytes and their activation with concomitant increase in production of oxidants. This reduces tissue antioxidant potential, which is paralleled by an increase in peroxidated lipids [2]. Therefore, measurement of lipid peroxidation (LPO) products in exhaled air condensate (EAC) has been employed for the diagnostics of bronchopulmonary inflammations [4].

We have compared the levels of LPO products in EAC and serum of patients with catarrhal and purulent chronic bronchitis.

MATERIALS AND METHODS

Twenty-four men aged 25-60 years with chronic bronchitis (8 with catarrhal and 16 with purulent) were examined. Control group consisted of 10 age-matched healthy men.

Free-radical oxidation of lipids was assessed from the level of peroxide lipids and antioxidative activity (AOA), which were measured in EAC and serum. LPO products were measured by extraction spectrophotometry in the heptane and isopropanol phases of lipid extract [1]. AOA was assessed from the capacity of the serum or EAC to suppress biogenic lipid peroxidation *in vitro*. The acute phase reaction parameters (neutrophil count and blood contents of seromucoids and C-reactive protein) were measured by routine hematological and biochemical methods. The results were statistically processed. The significance of differences was evaluated by the Student's *t* test and nonparametrical Wilcoxon—Mann—Whitney test.

RESULTS

Table 1 shows that chronic bronchitis is characterized by unidirectional shifts in serum and EAC LPO. The content of heptane-soluble LPO products increased in the serum of patients with both catarrhal and purulent bronchitis, whereas in the EAC an increase was observed only in patients with purulent bronchitis. Therefore, the level of heptane-soluble LPO products in EAC adequately reflects the severity of local inflammation in chronic bronchitis, and its changes correlate with the indicators of the inflammation severity, such as shifts in the content of C-reactive protein and leukocytosis (Table 1).

By contrast, serum content of heptane-soluble LPO products is less informative from the diagnostic

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TABLE 1. Lipid Peroxidation Parameters in Patients with Various Variants of Chronic Bronchitis ($M \pm m$)

Parameter		Healthy subjects	Catarrhal bronchitis	Purulent bronchitis
Condensate				
Heptane phase	Primary products	0.50±0.16	0.45±0.20	0.80±0.10*
	Secondary products	0.25±0.09	0.30±0.16	0.45±0.04*
Isopropanol phase	Primary products	0.60±0.02	0.26±0.15*	0.35±0.10*
	Secondary products	0.27±0.01	0.17±0.10*	0.19±0.09
	AOA, %	11.3±2.00	17.60±2.30*	9.70±1.90**
Serum				
Heptane phase	Primary products	0.62±0.03	0.75±0.02*	0.71±0.02*,**
	Secondary products	0.13±0.01	0.16±0.04	0.11±0.09
Isopropanol phase	Primary products	0.53±0.01	0.50±0.02	0.54±0.02
	Secondary products	0.37±0.01	0.32±0.02	0.78±0.02*
	AOA, %	95.2±1.00	89.40±2.60	93.10±1.70*
Acute-phase reaction				
C-reactive protein content		0.20±0.30	1.00±0.17*	1.70±0.12*,**
Seromucoids		0.15±0.01	0.29±0.03*	0.36±0.02*,**
Neutrophils, 10^9/liter		3.00±0.20	3.90±0.50	4.40±0.25*

Note. Contents of isopropanol- and heptane-extracted LPO products are expressed in the units of oxidative index which was calculated as the ratio between light absorbances (E_{232}/E_{220}) for diene conjugates (primary products) and as E_{278}/E_{220} for ketodienes and coupled trienes (secondary products). *$p<0.05$ compared with control, **compared with catarrhal bronchitis patients.

viewpoint, because in chronic purulent bronchitis it is significantly lowered compared with that in catarrhal bronchitis (Table 1). Shifts in serum and EAC AOA correspond to modified levels of heptane-soluble lipoperoxides. AOA is significantly decreased in chronic catarrhal bronchitis in comparison with the control. For EAC this value is the lowest in the patients with purulent bronchitis. Contrary to this, in catarrhal bronchitis the AOA level in the condensate is significantly higher than in the controls or in patients with chronic purulent bronchitis.

Shifts in the content of isopropanol-soluble LPO products were the least informative from the diagnostic viewpoint. In EAC they were decreased in both variants of bronchitis, and there was virtually no difference between the values in catarrhal and chronic purulent bronchitis. The only significant shift was observed in the patients with purulent bronchitis: more than twofold increase in secondary isopropanol-soluble LPO products in comparison with the control. Thus, accumulation of heptane-soluble LPO products and opposite AOA shifts in the EAC sufficiently well reflect the severity of inflammation in chronic bronchitis. A parallel decrease in the levels of isopropanol-soluble LPO products in the EAC and blood serum can be explained by hypoxia, which is common in chronic nonspecific pulmonary diseases. The data on generation of part of LPO products as byproducts of mitochondrial oxidation in health [3] support this hypothesis.

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