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How does Lipid Peroxidation Affect the Development of Pneumosclerosis: Experimental Justification

I.A. Marupov[1], Sh.A. Bobokulova[2], A.O. Okhunov[3], F.M. Abdurakhmanov[2], K.Kh. Boboev[2], D.N. Korikhonov[2], I.Yu. Yakubov[2], A.Sh. Yarkulov[2], Sh.A. Khamdamov[2], Sh.R. Razzakov[4]

[1]Resident of the Master's program in plastic surgery No. 1 of the Tashkent Medical Academy, Tashkent, Uzbekistan;

[2]Assistant of the Department of General and Pediatric Surgery No. 1, Tashkent Medical Academy, Tashkent, Uzbekistan;

[3]Professor, MD, PhD, Head of the Department of General and Pediatric Surgery, Tashkent Medical Academy, Tashkent, Uzbekistan;

[4]Surgeon of the private clinic "AKFA Medline", applicant for the Department of General and Pediatric Surgery No. 1 of the Tashkent Medical Academy, Tashkent, Uzbekistan.

Abstract

Background. Pneumosclerosis is a frequent complication of chronic obstructive pulmonary disease, bronchial asthma, and chronic obstructive bronchitis. The development of these complications aggravates the course of the disease, worsens its prognosis, leads to an increase in the duration of inpatient treatment, and significantly worsens the quality of life of patients, especially in terms of age.

Methods. The experiments were carried out on 25 laboratory rabbits with a pneumosclerosis model. The intensity of lipid peroxidation in the lungs was assessed in terms of malondialdehyde and chemiluminescence intensity in blood samples obtained in various blood samples.

Results. The dynamics of the development of pneumosclerosis was characterized by an increase in the intensity of lipid peroxidation in the lungs. The intensity of these processes was expressed to a greater extent in the arterial blood sample, indicating the specificity of the identified changes for this pathological process. An increase in the intensity of chemiluminescence in arterial blood on the 10th day of pneumosclerosis development was noted by 3.6 times, and on the 20th day - by 3.3 times compared with the control values. Even in the long-term studies, this indicator was 1.7 and 2 times higher than the control values and was more pronounced than in venous blood.

Conclusion. Reproduction of the experimental model of pneumosclerosis is characterized by the intensity of lipid peroxidation processes to a greater extent in the lungs than in other organs not affected by the pathological process. These changes were identified using a separate assessment of the level of lipid peroxidation in venous and arterial blood with the calculation of the venous-arterial difference. At the same time, more pronounced changes were noted in relation to the chemiluminescence index, indicating a high role of free radical compounds in the formation of this pathological process.

Corresponding author: ISLAM ARIFZHANOVICH MARUPOV, resident of the magistracy in plastic surgery of the Tashkent Medical Academy, Tashkent, Uzbekistan, Almazar district, Farobi-2 street, 100109, e-mail: marupovislom@gmail.com

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INTRODUCTION

Pneumosclerosis is an overgrowth of connective tissue in the lungs resulting from various pathological processes. [1,2,6,7] Pneumosclerosis is a collective concept in terms of etiology, pathogenesis, and morphology. [8-10] Of paramount importance in its development are non-specific infectious processes - pneumonia, lung abscess, etc. [3,12,13,26].

All the above chronic lung diseases leading to the development of pneumosclerosis create conditions for the development of endothelial dysfunction, which is facilitated by the presence of hypoxia, increased levels of cytokines, leukotrienes, tumor necrosis factor, etc. [5]. An important element of the pathogenic effect of these humoral factors is the induction of oxidative stress, the manifestation of which is largely associated with the activation of lipid peroxidation [17,24].

It is known that excessive formation and accumulation of lipid peroxidation products is one of the factors of membrane damage and promotes the development of an inflammatory reaction in the body [19,22].

According to A. N. Klimov [4], excessive formation of free radicals makes a significant contribution to the intensive oxidation of lipids and, thus, plays a decisive role in the formation of atheromatous plaque.

Lung tissue is one of the largest total biological membranes in the body. In the works of Novozhenov V. G. [29] it was shown that lipid peroxidation affects the phospholipids of membrane cells, disrupts membrane transport, and ultimately leads to cell death, in the lungs there is a possibility of free radical reactions in the layers of a surfactant - surfactant.

Excessive accumulation of primary, intermediate and end products of lipid peroxidation have a negative impact on the function and structural integrity of cell and subcellular membranes [17,25]. In accordance with this, it is important to evaluate changes in the activity of lipid peroxidation directly in the lungs in the dynamics of the development of an experimental model of pneumosclerosis, which was the goal of this study.

MATERIALS AND METHODS

The experiments were carried out on 25 mature rabbits of both sexes weighing 1900–2600 grams, which were on the usual laboratory diet. Pneumosclerosis in rabbits was modeled according to the original technique developed by us ("Method for modeling pneumosclerosis" Patent of the Intellectual Property Agency of the Republic of Uzbekistan No. IAP 20060015 dated April 20, 2006) by endobronchial 7-day insufflation of an air powder consisting of talc dust and 300,000,000 units of staphylococcal culture. At the same time, the developing pathological process was supported by the introduced virulent culture.

Morphological studies of the lungs in the dynamics of the development of pneumosclerosis showed that on the 10–20th day of its modeling, proliferation of young undifferentiated cells of the connective tissue took place. In some places, the differentiation of young cells of the connective tissue into mature fibroblasts with the secretion of fibrous structures was noted. In the later periods of the formation of pneumosclerosis (40–80 days), there was the presence of whitish layers in the form of a grid around the vessels and bronchi.

Microscopically, the proliferation of connective tissue was seen peribronchially, periascularly, perilobularly. In the connective tissue layers, thickened vessels because of myofibrosis, sclerotic capillaries, lymphoid follicles with the expansion of lymphatic vessels, deposition of carbon pigment were detected. Emphysematous areas are found in places. The interalveolar septa were drawn together, thickened, and sclerosed. In many areas, the structure of the lungs was completely lost, the lung tissue was replaced by fields of fibrosis.

The assessment of the intensity of lipid peroxidation in the lungs in control and simulated animals on days 1, 10, 20, 40 and 80 of the pathological process was carried out by determining the accumulation of malondialdehyde and the intensity of chemiluminescence in blood samples obtained at the entrance (venous blood) and at the exit (arterial blood). blood) from the lungs. The accumulation of malondialdehyde was determined by the method of L.I. Andreev et al. [4] Chemiluminescence intensity was determined by the method of T.N. Fedorov et al. [30]

RESULTS

The results obtained showed that the intensity of lipid peroxidation in the control series of experiments in venous blood was more pronounced than in the arterial blood sample. The level of chemiluminescence in venous blood at the entrance to the lungs was 72.3±3.1 imp/10s, and in arterial blood it decreased to 49.6±2.75 imp/10s. The venous-arterial difference, which amounted to "-" 22.7±0.04 imp/10s, indicated a decrease in the level of light indicators with chemiluminescence in the endothelial system. Identical changes were noted in relation to the level of malondialdehyde, which in venous blood was 0.2±0.01 nmol/l and decreased to 0.14±0.012 nmol/l as it passed through the lungs. The venous-arterial difference, which amounted to "-" 0.06±0.001 nmol/l, indicated the retention of these compounds in the lungs.

Modeling of pneumosclerosis already on the 1st day of the pathological process led to ambiguous changes in terms of indicators characterizing lipid peroxidation. A significant increase in the intensity of lipid peroxidation in arterial blood to 79.4±0.84 imp/10s in terms of chemiluminescence intensity and to 0.31±0.011 nmol/l ($p<0.05$) in terms of malonic dialdehyde level was accompanied by a change in the value venous-arterial difference. At the same time, in the case of chemiluminescence intensity, it acquired a "+" value, that is, blood enrichment occurred at the expense of the lungs, while in the case of malondialdehyde, its level in both venous and arterial blood was the same with a zero value of the venous-arterial difference.

A similar trend of changes in the activity of lipid peroxidation was also noted on the 10th day of modeling pneumosclerosis. The intensity of chemiluminescence in this period increased both in venous

and arterial blood up to 94.6±3.1 imp/10s and 177.4±8.5 imp/10s (p<0.05), respectively (table).

The venous-arterial difference, which amounted to "+" 82.8±0.21 imp/10s, testified to the specificity of these changes, and was probably associated with the development of pneumosclerosis.

Table

Characteristics of changes in the intensity of lipid peroxidation in various blood samples in the dynamics of the development of an experimental model of pneumosclerosis

SERIES	Chemiluminescence (imp/10 s)		Malondialdehyde (nmol/l)	
	venous blood	arterial blood	venous blood	arterial blood
Control	72,3 ±3,1	49,6 ±2,75*	0,2 ±0,01	0,14 ±0,012
1 day	76,8 ±2,4	79,4 ±0,84*	0,32 ±0,011	0,32 ±0,011
10 day	94,6 ±3,1	177,4 ±8,5*	0,39 ±0,012	0,59 ±0,003*
20 day	141,9 ±4,5	162,7 ±9,5*	0,38 ±0,01	0,43 ±0,021*
40 day	101,4 ±1,2	82,4 ±5,9*	0,52 ±0,02	0,44 ±0,025*
80 day	104,8 ±2,1	102,4 ±0,5*	0,56 ±0,011	0,55 ±0,001*

*p<0.05 compared with venous blood sample at the entrance to the lungs

The level of malondialdehyde in this period also increased both in venous and arterial blood to 0.39±0.012 nmol/l and 0.59±0.003 nmol/l (p<0.05), respectively. Intensification of lipid peroxidation processes was noted, confirmed by the positive value of the venous-arterial difference, which reached “+” 0.2 ± 0.002 nmol/l at this time.

The 20th day of the development of the pathological process was characterized by intensification of lipid peroxidation in peripheral organs and an increase in the intensity of chemiluminescence in venous blood up to 141.9±4.5 imp/10s, while maintaining a sufficiently high activity in arterial blood, reaching up to 162.7±9.5 imp/10s (p<0.05). Changes in the level of malondialdehyde in this period were noted only in arterial blood, where there was a slight decrease in its level compared to the previous period to 0.43±0.021 nmol/l (p<0.05).

In the subsequent periods of observation (up to 80 days), against the background of the relative stability of the intensity of chemiluminescence in venous blood, there was a decrease in this indicator compared to the previous period. However, in a comparative aspect, these changes were significantly higher than the control values. The venous-arterial difference in this indicator acquired a "-" value and on the 40th day was "-"19.0±5.9 imp/10s (p<0.05), and on the 80th day it was "-"2.4±0.01 imp/10s (p<0.05).

The level of malondialdehyde during these observation periods continued to increase both in venous (up to 0.56±0.011 nmol/l) and in arterial (-0.01±0.0011 nmol/l) blood samples, respectively.

In general, the dynamics of the development of pneumosclerosis was characterized by an increase in the intensity of lipid peroxidation in the lungs. The intensity of these processes was expressed to a greater extent in the arterial blood sample, indicating the specificity of the identified changes for this pathological process. Thus, an increase in the intensity of chemiluminescence in arterial blood on the 10th day of the development of pneumosclerosis was noted by 3.6 times, and on the 20th day - by 3.3 times compared with the control values. Even in the long-term studies, this indicator was 1.7 and 2 times higher than the control values and was more pronounced than in venous blood.

The maximum value of the level of malondialdehyde in venous blood fell on the 40th day and on the 80th day of observation, where it increased by 2.6 and 2.8 times compared with the control values. In arterial blood, this trend was more pronounced than in venous blood samples. At the same time, if on the 10th day of the experiments the level of malonic dialdehyde was 4.2 times higher than the control values, then in the long term there was a slight decrease. However, this indicator remained higher than the control values by 3.2 and 3.9 times.

DISCUSSION

It is known that lipid peroxidation is a universal mechanism that regulates the state of cells, both in normal and pathological conditions [17,27,30]. According to modern concepts, increased lipid peroxidation is one of the leading factors of cell membrane damage in the pathogenesis of chronic respiratory diseases [21,30]. In addition, recent studies have convincingly proven the role of hypoxia in increasing lipid peroxidation.

The characteristic changes in the intensification of lipid peroxidation in the dynamics of the development of pneumosclerosis revealed by us during our studies are probably due to the influence of reactive oxygen species (superoxide anion, hydrogen peroxide, hydroxyl radical) on polyene acyls in the structure of glycerophospholipids of the biological membrane. As literature sources indicate, because of such a free radical attack, a chain process of free radical (peroxide) lipid oxidation begins, which ultimately leads to the destruction of lipids to cytotoxic water-soluble carbonyl intermediates [23,31].

Several authors believe that reactive oxygen species affect a variety of biological structures, but lipid peroxidation is of particular importance for the lungs, which is associated with the intensive lipid metabolism of this paired organ [21,29].

It is known that lung tissues contain an excess of unsaturated fatty acids, which are a substrate for lipid peroxidation [18,20,28]. According to Syromyatnikova N.V. [21], the respiratory organs are characterized by a high intensity of lipid metabolism. Lipids, lipoproteins, and fatty acids are the main source of energy in the lungs. They also provide the most important metabolic and structural processes [29].

CONCLUSION

Thus, the reproduction of the experimental model of pneumosclerosis is characterized by the intensity of lipid peroxidation processes to a greater extent in the lungs than in other organs not affected by the

pathological process. These changes were identified using a separate assessment of the level of lipid peroxidation in venous and arterial blood with the calculation of the venous-arterial difference. At the same time, more pronounced changes were noted in relation to the chemiluminescence index, indicating a high role of free radical compounds in the formation of this pathological process.

Ethical clearance - All experimental studies were reviewed, discussed, and approved by the bioethical committee of the Ministry of Health of the Republic of Uzbekistan and fully complied with the terms of the 1986 Council of Europe Convention for the Protection of Animals.

Consent for publication - The study is valid, and recognition by the organization is not required. The author agrees to open publication

Availability of data and material - Available

Competing interests - No

Financing – No

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LIPID PEROKSİDASYONI PNEVMOSKLEROZ RIVOJLANISHIGA QANDAY TA'SIR ETIShI: TAJRIBAVIY ASOSLASH.

I.A. Marupov, Sh. A. Boboqulova, A.O. Oxunov, F. M. Abdurahmonov, K. X. Boboev, D.N.Qorixonov, I.Yu.Yoqubov, A.Sh. Yarqulov, Sh.A. Hamdamov, Sh.R. Razzoqov

Toshkent Tibbiyot Akademiyasi

Abstrakt

Dolzrabligi. Pnevmoskleroz surunkali obstruktiv o'pka kasalligi, bronxial astma va surunkali obstruktiv bronxitning tez-tez uchraydigan asoratidir. Ushbu asoratlarning rivojlanishi kasallikning kechishini og'irlashtiradi, uning prognozini yomonlashtiradi, statsionar davolanish muddatining ko'payishiga olib keladi va bemorlarning hayot sifatini, ayniqsa yoshga qarab sezilarli darajada yomonlashtiradi.

Tadqiqod usullari. Tajribalar pnevmoskleroz modeli bo'lgan 25 ta laboratoriya quyonlarida o'tkazildi. O'pkada lipid peroksidatsiyasining intensivligi turli xil qon namunalarida malon dialdegid va ximilyuminesans intensivligi bo'yicha baholandi.

Natijalar. Pnevmosklerozning rivojlanish dinamikasi o'pkada lipid peroksidatsiyasining intensivligining oshishi bilan tavsiflanadi. Ushbu jarayonlarning intensivligi ko'proq darajada arterial qon namunasida ifodalangan bo'lib, bu patologik jarayon uchun aniqlangan o'zgarishlarning o'ziga xosligini ko'rsatadi. Pnevmoskleroz rivojlanishining 10-kunida arterial qonda xemiluminesans intensivligining o'sishi nazorat ko'rsatkichlari bilan solishtirganda 3,6 marta, 20-kunida esa 3,3 marta qayd etilgan. Hatto uzoq muddatli tadqiqotlarda ham bu ko'rsatkich nazorat ko'rsatkichlaridan 1,7 va 2 baravar yuqori va venoz qonga qaraganda aniqroq edi.

Xulosa. Pnevmosklerozning eksperimental modelini quzg'ashi patologik jarayondan ta'sirlanmagan boshqa organlarga qaraganda o'pkada ko'proq darajada lipid peroksidlanish jarayonlarining intensivligi bilan tavsiflanadi. Ushbu o'zgarishlar venoz-arterial farqni hisoblash bilan venoz va arterial qonda lipid peroksidatsiyasi darajasini alohida baholash yordamida aniqlandi. Shu bilan birga, xemiluminesans indeksiga nisbatan aniqroq o'zgarishlar qayd etildi, bu ushbu patologik jarayonning shakllanishida erkin radikal birikmalarning yuqori rolini ko'rsatadi.

Kalit so'zlar: pnevmoskleroz, xemiluminesans, erkin radikal birikmalar, malondialdegid, o'pkaning metabolik funktsiyasi.

КАК ВЛИЯЕТ ПЕРЕКИСНОЕ ОКИСЛЕНИЕ ЛИПИДОВ НА РАЗВИТИЕ ПНЕВМОСКЛЕРОЗА: ЭКСПЕРИМЕНТАЛЬНОЕ ОБОСНОВАНИЕ

И.А. Маърупов, Ш. А. Бобокулова, А.О. Охунов, Ф. М. Абдурахманов, К.Х. Бобоев, Д. Н. Корихонов, И. Ю. Якубов, А.Ш. Яркулов, Ш.А. Хамдамов, Ш.Р. Раззаков

Ташкентская Медицинская Академия

Абстракт

Актуальность. Пневмосклероз являются частым осложнением хронической обструктивной болезни легких, бронхиальной астмы, хронического обструктивного бронхита. Развитие этих осложнений утяжеляет течение заболевания, ухудшает его прогноз, приводит к увеличению продолжительности стационарного лечения, существенно ухудшает качество жизни пациентов, особенно в возрастном аспекте.

Методы. Эксперименты проведены на 25 лабораторных кроликах с моделью пневмосклероза. Проводили оценку интенсивности перекисного окисления липидов в легких по показателям малонового диальдегида и интенсивности хемилюминесценции в пробах крови, полученных в различных пробах крови.

Результаты. Динамика развития пневмосклероза характеризовалась повышением интенсивности перекисного окисления липидов в легких. Интенсивность этих процессов была выражена в большей степени в артериальной пробе крови, свидетельствующая о специфичности выявленных изменений для данного патологического процесса. Повышение интенсивности хемилюминесценции в артериальной крови на 10-сутки развития пневмосклероза было отмечено в 3,6 раза, а на 20-сутки – в 3,3 раза по сравнению с контрольными значениями. Даже в отдаленные сроки исследований данный показатель был выше контрольных значений в 1,7 и в 2 раза и был более выражен, чем в венозной крови.

Заключение. Воспроизведение экспериментальной модели пневмосклероза характеризуется интенсивностью процессов перекисного окисления липидов в большей степени в легких, чем в других органах, не затронутых патологическим процессом. Эти изменения были выявлены при помощи раздельной оценки уровня показателей перекисного окисления липидов в венозной и артериальной крови с подсчетом венозно-артериальной разницы. При этом более выраженные изменения отмечены по отношению к показателю хемилюминесценции, свидетельствуют о высокой роли свободнорадикальных соединений в формировании данного патологического процесса.

Ключевые слова: пневмосклероз, хемилюминесценция, свободные радикальные соединения, малоновый диальдигид, метаболическая функция легких.