Kinetics of morphofunctional changes in the center of inflammation of pulmonary tissue caused by staphylococcus aureus

Abstract

In three experimental series of experimental animals caused by inflammation model with a standard strain of Staphylococcus aureus. (Strain WOOD -46). Held control of the morphological and functional changes in the lung tissue at different times from the beginning of the introduction of Staphylococcus aureus. The histological method was used. The observations indicate a dose-dependent morphological changes in the lung tissue with inflammation caused by various doses of the standard Staphylococcus aureus.

Keywords: chronic purulent pneumonia, experiment, staphylococcus aureus, morphology

Introduction

Medical and socio-economic importance of chronic inflammation in the bronchi and lungs, is extremely high due to their prevalence in human disease patterns, increased frequency of chronic obstructive pulmonary disease and the emergence of SARS, significant mortality and disability of patients. The severity and nature of the inflammatory reaction in the lungs, the development of regenerative processes depend on the degree of functional activity of the effector cells of inflammation and their functional reserves. The central link of pathogenesis, responsible for the formation of clinical manifestations of these diseases, is chronic fetal reaction of bronchial mucosa and lung parenchyma on the background of impaired local and general immunity. Pneumonia is the most common disease occurs at any age, has certain flow characteristics at different ages and is a complex pathological processes developing in the distal lung tissue. The main manifestation of these processes is an infectious, exudative, rarely interstitial inflammation caused by microorganisms of different nature, and dominant in the whole picture of the disease.

The purpose of the study

To study the effect of various standard dosages staphylococcal infection on morphological changes in the lung tissue damaged experiment.

Material and methods

25 white mongrel rats of males with a mass of 180-200 g, contained in the vivarium condition, were observed. The model of inflammation was caused by the method of K.V. Samson. The light injected standard Staphylococcus aureus (strain WOOD -46) by puncture of all tissues of the chest wall to a depth of 1.0 cm. In different doses.

For morphological studies, the taken pieces of lung tissue taken after the face were fixed in a 10% solution of neutral formalin. The histological method was used. The observations indicate a dose-dependent morphological changes in the lung tissue with inflammation caused by various doses of the standard Staphylococcus aureus.

Results and discussion

In the first series (10 rats), 2 million units of standard Staphylococcus aureus dissolved in 1 ml were injected into the lung physiological solution in an amount of 0.2 ml.

In the second series (10 rats) was injected into the lungs 3 million units of standard staphylococcus dissolved in 1ml physiological solution in an amount of 0.2 ml.

A control group consisted of 5 healthy rats. The examination was performed 2,7,13 days from the beginning of the experiment.

Observations made in two days of experiments showed that macroscopically the internal organs, bronchial mucosa and lung lesions were found. Nevertheless, histological study of a series of sections of light laboratory rats with a model of staphylococcal pneumonia gave the following data.

It was found that in the group receiving a dose of staphylococci in the amount of 3 million units, there was a more pronounced development of the pathological process compared to the group of animals administered a dose of 2 million units of staphylococci (Figure 1) (Figure 2). These changes were more significant on the 7th day of the experiment.

In the pulmonary tissue, to this day, all the signs of purulent inflammation developed, namely, inflammatory hyperemia characterized by capillary and arterial fullness, and the character of the blood filling of the pulmonary parenchyma was uneven and more pronounced in areas of maximum destruction of tissue.

Observed mosaic destruction of lung parenchyma: in some areas of the lung is less affected by the pathological process, determined zones acute emphysema, and in other-micro atelectasis and drain foci expressed inflammatory infiltration between the alveolar walls and the
Kinetics of morphofunctional changes in the center of inflammation of pulmonary tissue caused by staphylococcus aureus

walls of bronchioles with portions of tissue destruction, depending on the dose of infection with microorganisms. In the gaps of small bronchi and bronchioles in the areas of injury were determined purulent plugs occlusive their gaps. On separate areas determined accurately expressed diapedetic small focal hemorrhage in gaps between the alveoli and the alveolar septum. Also characteristic signs of the inflammatory process were edema between the alveolar septa, sloughing of the epithelium of the alveoli into the lumens, where along with these cells, cells of an inflammatory infiltrate were determined in large numbers. Attention was also drawn to reactive hyperplasia of the lymphoid lung tissue, more pronounced in the group of animals receiving microorganisms in a dose of 3 million units.

Figure 1 Lung tissue rats on the 7th day of staphylococcus infection in a dose of 2 million. e Diniz. Inflammatory hyperemia and peribronchial reactive hyperplasia of lymphoid tissue. Uvel. 10x20., Coloration: hematoxylin-eosin.

Figure 2 Cloth of a light rat on the 7th day of infection with staphylococci in a dose of 3 million units. Purulent plugs in the lumens of bronchioles, foci of destruction of lung tissue, focal acute emphysema. Uvel. 10x10, 10x20, Color: hematoxylin-eosin.

By the 13th day of the experiment, the lesion zone was not confined to individual inflammation foci, but acquired a wider area, while the inflammation spread across the alveolar septa, acquiring the character of either interstitial or draining pneumonia. These changes were more pronounced in the group receiving staphylococcus in a dose of 3 million units. Preserved focal emphysema, purulent fuses in the lumens of individual small bronchioles, although the destructive manifestations decreased. Also, a sharp thickening between the alveolar septa was determined due to pronounced edema and inflammatory infiltration.

Among acute inflammatory cells to the 13-th day increased number of mononuclear cells. Decreased tissue destructive processes in the lung parenchyma, but also circulatory disorders accompanied by hyperemia and focal inflammatory diapedetic hemorrhages, uneven alveolar edema. Along with these changes, there was also a decrease in the manifestations of reactive hyperplasia of peribronchial lymphoid tissue, which may be a consequence of depletion of lymphoid tissue in response to the inflammatory process (Figure 3) (Figure 4). Thus, morphofunctional changes in the dynamics of observation indicate the development of inflammatory phenomena in the lung tissue, depending on the dose of the infection.

Figure 3 The tissue of a light rat on the 13th day of infection with staphylococci in a dose of 2 million units. Interstitial nature of the lesion. There is inflammatory hyperemia and focal emphysema. Focal hemorrhages. Uvel. 10x20, Coloration: hematoxylin-eosin.

Figure 4 The tissue of a light rat on the 13th day of infection with staphylococci in a dose of 3 million units. There is an inflammatory process, purulent fuses in the lumens of small bronchioles, focal emphysema. Uvel. 10x10, 10x20, Color: hematoxylin-eosin.

Conclusion

i. Morphofunctional changes in lung tissue in the experiment dose are dependent.

ii. Morphofunctional changes in lung tissue depend on the amount injected into the lungs of standard Staphylococcus aureus.

Acknowledgements

None.

Conflict of interest

Authors declare that there are no conflicts of interest.

References


Citation: Sadikova GA, Rakhmatullaev HU, Zalyalova ZS. Kinetics of morphofunctional changes in the center of inflammation of pulmonary tissue caused by staphylococcus aureus. Int Phys Med Rehab J. 2018;3(3):200–202. DOI: 10.15406/ipmrj.2018.03.00103