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CLINICAL AND MORPHOLOGICAL CHARACTERISTICS OF A NEW METHOD FOR MODELING ACUTE PURULENT-DESTRUCTIVE LUNG DISEASE

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Abstract: In order to maximize the adaptation of the experimental model to clinical conditions, preliminary sensitization of the macroorganism was performed by creating a focus of peripheral inflammation prior to introducing a microbial suspension into the lung tissue. Upon introduction of microbial suspensions into the pulmonary tissue, even in the early stages of the experiment, necrobiotic, dyscirculatory, and inflammatory changes develop in the lesion. These changes spread during subsequent periods of the experiment in the form of an expanding focus of necrosis and hemorrhage, and a diffusely spreading inflammatory process into the surrounding tissues with the appearance of microabscesses. The inflammatory infiltrate initially appeared immediately around the necrosis, then spread into the surrounding zones of the lung tissue in the form of focal neutrophilic-leukocyte inflammation and abscess formation.
Keywords: *acute lung abscess, purulent-destructive lung disease, lung gangrene, etiopathogenesis, prevalence, diagnostic and treatment methods.*

КЛИНИКО-МОРФОЛОГИЧЕСКАЯ ХАРАКТЕРИСТИКА НОВОГО СПОСОБА МОДЕЛИРОВАНИЯ ОСТРОГО ГНОЙНО ДЕСТРУКТИВНОГО ЗАБОЛЕВАНИЯ ЛЕГКИХ

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Аннотация: в целях максимального приближения процесса к клиническим условиям перед введением взвеси микробов в легочную ткань производилась предварительная сенсибилизация макроорганизма путем создания очага периферического воспаления. При введении в легочную ткань микробных взвесей даже в ранние сроки эксперимента в очаге поражения развиваются некробиотические, дисциркуляторные и воспалительные изменения, распространяющиеся в последующие сроки опыта в виде расширения очага некроза и кровоизлияния, диффузно

распространенного воспалительного процесса в окружающие ткани с появлением микроабсцессов. Воспалительный инфильтрат первоначально появлялся непосредственно вокруг некроза, затем распространялся в окружающие зоны легочной ткани в виде очагового нейтрофилно-лейкоцитарного воспаления и абсцедирования.
Ключевые слова: острый абсцесс легких, гнойно-деструктивное заболевание легких, гангрена легких, этиопатогенез, распространенность, метода диагностики и лечения.

ЎПКАНИНГ ЎТКИР ЙИРИНГЛИ-ДЕСТРУКТИВ КАСАЛЛИГИНИ МОДЕЛЛАШТИРИШНИНГ ЯНГИ УСУЛИ КЛИНИКО - МОРФОЛОГИК ХУСУСИЯТЛАРИ

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Аннотация: Жараённи клиник шароитларга имкон қадар яқинлаштириш мақсадида, ўпка тўқимасига микроблар суспензиясини (взвеси) юборишдан олдин, периферик яллиғланиш ўчоғини ҳосил қилиш орқали макроорганизмни олдиндан сенсибилизация қилиш (сезувчанлигини ошириш) амалга оширилди. Ўпка тўқимасига микроблар суспензияси юборилганда, ҳатто тажрибанинг дастлабки босқичларида ҳам, шикастланиш ўчоғида некробиотик, дисциркулятор ва яллиғланиш ўзгаришлари ривожланади. Кейинги муддатларда эса бу жараён некроз ва қон қуйилиши ўчоғининг кенгайиши, атрофдаги тўқималарга диффуз тарқалган яллиғланиш ва микроабсцесслар пайдо бўлиши кўринишида давом этади. Яллиғланиш инфилтрати дастлаб бевосита некроз атрофида пайдо бўлиб, кейинчалик ўпка тўқимасининг атрофдаги зоналарига ўчоқли нейтрофил-лейкоцитар яллиғланиш ва абсцесс ҳосил бўлиши кўринишида тарқалади.
Калит сўзлар: Ўпканинг ўткир абсцесси, ўпканинг йирингли-деструктив касаллиги, ўпка гангренаси, этиопатогенез, тарқалиши, диагностика ва даволаш усуллари.

INTRODUCTION

Although it is generally accepted that the primary cause of acute purulent-destructive lung disease (APDLD) is the penetration of pathogenic microflora into the lung tissue, substantial evidence has accumulated indicating that the presence of microflora in the lung parenchyma by no means solely determines the occurrence of a lung abscess. This is true despite the fact that it would be difficult to imagine a better thermostat and nutrient medium for the growth of pathogenic microflora than the lung [1, 2, 7, 8, 9].

Numerous cultures taken from lung tissue when operations are performed under aseptic conditions and the suture is absolutely airtight frequently yield the growth of various microflora, while the postoperative period proceeds without any hint of complication [2, 5, 6]. From this, it logically follows that in most cases, the body's protective forces are sufficient to suppress the action of the aggressive agent.

MATERIALS AND METHODS

The first variant of the APDLD model involved the introduction of a virulent microbial culture (30 million microbial bodies per 1 g of rat body weight) directly into the lung tissue via injection through the chest wall (Series 1). In this series, the administration of relatively smaller doses (up to 30 million microbial bodies per 1 g of weight) did not induce APDLD in 75% of cases, and the animals recovered. Autopsies performed 7–10 days after infection revealed a highly limited adhesive process at the site of infection injection in 40% of cases, and a complete absence of any traces of prior inflammation in 45% of cases.

Only in the remaining 10–15% where the disease developed was the typical picture of fibrinous-purulent pleurisy observed. When the infectious agent was administered in larger doses

(60–65 million microbial bodies per 1 g of rat body weight), the rats died in 90% of cases within the first few hours from so-called bacterial sepsis (Series 2). Upon autopsy, these rats exhibited the presence of up to 2.2 ± 0.8 mL of hemorrhagic effusion in the pleural cavity, severe engorgement of internal organs, and multiple focal hemorrhages in the visceral and parietal pleura. In short, although an inflammatory process is formally present, we are essentially dealing with sepsis, where the lung serves as the portal of entry.

Thus, summarizing these experiments, several interesting points can be noted: with intrapulmonary introduction of microflora, APDLD—as a disease in the clinical sense of the term, with its characteristic clinical picture—generally does not occur. If the forces of aggression clearly overpower the forces of defense, sepsis occurs with a lightning-fast course and animal death within the first few hours; if, however, the protective mechanisms prove stronger in the resulting conflict, the macroorganism wins, in most cases leaving no traces of the battle on the battlefield.

Perhaps the issue lies in the fact that the nature of infection differs sharply from natural conditions, given that over the past 30 years sufficient information has accumulated regarding the dominant role of various anaerobic flora in the occurrence of APDLD [1, 3, 4, 6, 9]. To clarify this issue, we conducted another series of experiments in which lung tissue infection was performed using a 5% suspension of rat autocal (fecal matter) (Series 3).

The results of this series of experiments were generally similar to those described above, namely: at relatively large doses (0.5 mL per 100 g of animal weight), the development of bacterial sepsis with a fatal outcome within the 1st day after infection occurred in 85% of cases. When smaller doses were administered (0.2 mL per 100 g of weight), the outcomes varied (Series 4): out of 21 rats, 2 (9.5%) died of bacterial sepsis, in 14 (66.7%) the process took an abortive course, and only in 5 (23.8%) did a typical picture of APDLD emerge.

Reflecting on these facts led us to three conclusions. First, massive lung infection, regardless of the nature of the microflora, does not lead to the development of an abscess in the clinical sense of the term, but rather to the hematogenous generalization of the infection. The clinical picture of the disease in this case is determined by sepsis, which has little in common with APDLD. The severity of the condition is so profound that APDLD simply does not have time to develop—the lungs play the role of a portal of entry for the infection, much like what occurs with any septic focus, such as the palatine tonsil in acute tonsillitis.

The experimental results also demonstrated that the alternative to the occurrence of bacterial sepsis is typically not lung abscess formation in the clinical sense, but rather a rapid recovery outcome, indicating the suppression and elimination of the infectious agent.

Thus, introducing an aggressive agent into the lung of a healthy animal does not cause APDLD in most cases. This second conclusion logically led us to a third: APDLD, as a specific form of body response to a conflict between the lung and an aggressive agent, arises as a consequence of altered body reactivity resulting from a preceding disease. This is fully consistent with clinical observations indicating that the most frequently encountered forms of APDLD are associated with acute respiratory viral infections, pneumonias, and other non-specific lung diseases. The very fact that an inflammatory process unfolds in an organ according to a destructive pattern indicates the presence of a hyperergic reaction of the body; consequently, the conflict between the aggressive agent and the lungs in all these cases occurs against the background of altered body reactivity.

Thus, the results of experimental studies indicate that the APDLD program is not realized in the absence of an initial hyperergic reaction of the body, the manifestation of which is an acute inflammatory process. In other words, logic forces us to conclude that the presence of an acute inflammatory process in the body can rightfully be considered a preliminary phase of APDLD.

Experimental confirmation of this was obtained in a new series of experiments, which differed from the previous ones in that, to increase the reproducibility of the model by bringing the process closer to the clinical course, 0.3–0.5 mL of a 10% ammonia solution was preliminarily injected into the subcutaneous tissue of the animal's lumbar region. After 48 hours, the development of a necrotic focus was noted at the injection site. On the same day, under superficial ether anesthesia, 0.5–0.7 mL of a 5% suspension of rat autocal was injected into the IV intercostal space on the right along the midaxillary line (Series 5).

The development of APDLD was monitored dynamically, starting from the first day after the injection of the microbial suspension. Over the subsequent 4 days, the rats developed the clinical presentation of APDLD: adynamia, tachypnea, depression, and hyperthermia. Autopsy revealed the comprehensive pathological picture of APDLD.

Histological examinations of lung and pleural biopsy samples obtained during autopsy at various stages of APDLD established the following: on day 2 of the experiment, massive necrobiotic, dyscirculatory-dystrophic, and acute inflammatory changes were detected in the zone of puncture and autocal inoculation.

Directly in the center of the lesion, massive hemorrhages into the alveolar and interalveolar tissues were observed, accompanied by necrobiotic and necrotic changes in the alveolar epithelium and interalveolar connective tissue, along with the destruction of vascular walls.

Within the necrotic mass and hemorrhages, hematogenous and macrophage cells were identified, predominantly around the necrosis, alongside infiltrated macrophages, neutrophilic leukocytes, and occasional eosinophils. In the periphery of the necrosis and hemorrhages, pronounced edema and loosening of the interalveolar tissue, as well as paralytic dilation of blood and lymphatic vessels, were noted.

As a result of the spread of the inflammatory process around the affected focus, a perifocal inflammatory field appeared, morphologically characterized as an acute serous-purulent inflammatory disease. Hyperplasia of lymphoid cells, locally forming lymphoid follicles, was observed around large vessels and bronchi.

In distant zones of the alveolar tissue, vascular hyperemia with minor diapedetic hemorrhages was recorded, alongside edema and loosening of the interalveolar tissue, with small foci of inflammatory infiltrate appearing in places.

On day 4 of APDLD reproduction following decapitation, a purulent liquid exudate with a foul, fecal-putrid odor was found in the pleural cavity. The parietal pleura was dark gray, showing putrid decay in some areas. In these segments, fusion of the pleura with the lung parenchyma was observed.

Microscopic examination at this stage of the experiment revealed a significant expansion of the lung tissue lesion due to an increase in the zone of necrosis and hemorrhage at the center of the focus.

The necrosis appeared as a dark blue disintegrated mass, within which violet-colored microbial colonies were identifiable in places. Along the periphery of the necrotic focus, the disintegration of inflammatory cells and alveolar tissue led to the expansion of the necrotic zone. A wide zone of infiltration by neutrophilic leukocytes was detected around this focus, resulting in abscess formation.

Neutrophilic infiltrates extended significantly further into the surrounding areas of the lung tissue than in the previous experimental period, forming new inflammatory foci, microabscesses, and necrosis. Directly surrounding the lesion, the alveolar tissue was diffusely infiltrated by inflammatory cells, erythrocytes, and plasma proteins, encompassing several alveoli, acini, and even segments of lung tissue.

The alveolar epithelium exhibited dystrophic swelling, destruction, and desquamation. Around large vessels, the development of inflammatory granulation tissue consisting of juvenile connective tissue cells, macrophages, neutrophils, and thin-walled vessels was observed.

In distant areas of the lung tissue, unlike the previous period, the volume of the inflammatory process was significantly increased and manifested as focal inflammatory lesions presenting as abscesses and segmental pneumonia. Concurrently, the vessels within the inflammatory foci were significantly dilated and engorged with blood; the alveolar lumens and interalveolar septa were diffusely infiltrated with neutrophilic leukocytes and macrophages, accompanied by the development of dysatelectasis and microabscesses.

RESULTS AND DISCUSSION

Thus, when autocal is introduced into the lung tissue, necrobiotic, dyscirculatory, and inflammatory changes develop in the lesion even in the early stages of the experiment. These changes expand during subsequent periods in the form of an expanding zone of necrosis and hemorrhage, and a diffuse spread of the inflammatory process into surrounding tissues with the emergence of microabscesses.

The inflammatory infiltrate initially appears immediately around the necrosis and autocal, then spreads into surrounding zones of the lung tissue in the form of focal neutrophilic-leukocyte inflammation and abscess formation.

Pronounced changes in the lung tissue occurred in 22 (73.2%) out of 30 rats in this series; 2 (6.7%) animals developed serous pleurisy, 2 (6.7%) rats died from bacterial shock, and in 4 (13.4%) animals APDLD did not develop.

Consequently, the preliminary reproduction of destructive inflammation resulted in the development of a typical APDLD picture in two-thirds of the animals, whereas in previous experiments this occurred in only 5 (23.8%) out of 21 animals.

A comparison of the results of the previous and current series of experiments—where, under identical methods of lung infection, the occurrence of APDLD increases sharply against the background of preliminary formation of an acute destructive focus—definitely confirms the hypothesis that the APDLD program is realized only in the presence of altered body reactivity.

With equal strength of the aggressive agent, the outcome of its conflict with the lungs is determined by the reactivity of the organism, and APDLD represents a mode of reaction that is initially predetermined by the presence of hyperergy.

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