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Opinion

Why is Pain not Characteristic of Inflammation of the Lung Tissue?

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Abstract

The article presents materials that make it possible to understand the reason for the absence of one of the classic signs of inflammatory processes in patients with acute pneumonia. The peculiarities of the functional significance of the lungs for the body are the reason that in the case of inflammation in the tissues of the organ, nature has provided for the presence of a more important adaptive mechanism instead of pain as a signal sign. Understanding the causes of the absence of pain in pneumonia in the initial period, which is most responsible for timely and effective care for these patients, allows us to look at the pathogenesis of the disease from a new point of view, which is of fundamental importance for the correction and selection of pathogenetic means of care.

More Information

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Acute pneumonia (AP) has been known to medicine as a separate nosology with one familiar name for more than two millennia, but in recent decades it has been overgrown with a large number of new terms that have been proposed to reflect primarily its etiological features: community-acquired, nosocomial, atypical, COVID-19 pneumonia, etc. However, it is unlikely that anyone will have objections to the fact that regardless of the change in the etiology and new designations of this disease, as well as the fact that it began to be classified as infectious, it was and remains based on the inflammatory process.

Inflammation as a biological phenomenon observed in many diseases has also been known to medicine since ancient times, and its classic signs, described almost a couple of millennia ago by Celsus and Galen, have been tested by time and constitute the golden fund of fundamental medical science. One of the five classic signs of inflammation is pain, which serves as a signal to the body about the occurrence of an unexpected problem in its structures. This signal, as a rule, causes compensatory reactions of the body and the patient himself to spare the affected organ, which is accompanied by another classic manifestation in the form of a violation or loss of function.

The pain accompanying inflammatory diseases can be so pronounced that it is one of the causes of shock reactions. Severe and debilitating pain in various forms of inflammation forces the use of various ways to eliminate it. In connection with the latter, it may not be entirely clear that there is no pain with such a serious localization of the lesion as acute inflammation of the lung tissue. Even with extensive lesions of the pulmonary parenchyma, pain syndrome occurs only when the inflammatory process spreads to the pleural leaflets [1]. The illogicality of such a situation is emphasized by the fact that nature provided for the occurrence of pain during inflammation as a warning and protective sign, but in the case of one of the most severe forms of inflammation, this signal turned out to be unforeseen in the initial most important period of the disease. A typical paradox, isn't it?

When we classify any features of the biological world as paradoxes, it only means that in such cases we do not understand the mysteries of nature, the derivative subjects of which we ourselves are. Moreover, such a verbal definition as a paradox was invented by us to denote phenomena that have not yet been explained. If we analyze the above-mentioned feature of pneumonia from the point of view of the anatomical and functional features of the lung tissue, which are already well known to us, then the absence of pain in the initial period of the disease does not conceal any contradictions or absurdity, but on the contrary, has a completely natural meaning.

If, for example, the inflammatory process affects the limbs, pain and its intensification during movements sharply reduce their functional activity, which is a logical consequence of the disease, allowing the body not to aggravate the further development of the process, avoiding damage to easily vulnerable inflamed tissues. At the same time, restriction or

temporary loss of mobility does not pose a direct danger to the vital activity of the body. A completely different picture arises in the case of inflammatory transformation in such a vital organ as the lungs. If the onset of inflammation in the lung tissue was always accompanied by pain, then this sign could not be useful, since in this anatomical and physiological situation, the pain syndrome would only be an additional obstacle that hinders the work of the pulmonary-cardiac apparatus, but by itself is not able to provide the necessary adaptation and protection.

Nature, having deprived the lung tissue of pain receptors as an impractical element in the initial stages of inflammation of this localization, made sure that this vital organ had fast and reliable protection. Inflammatory tissue changes that cause irritation of nerve-sensitive elements can affect any other receptors. The change in blood flow and pressure in the pulmonary vessels, which occurs as a result of their inflammatory reaction, affects the baroreceptors of this zone. This phenomenon saves the life of the body because it instantly and autonomously activates reflex defense mechanisms. One of these basic mechanisms of restructuring is the so-called discharge reflex, first described almost a century ago [2]. This reflex is accompanied by a restructuring of the systemic circulation with a delay in part of the circulating blood at the periphery and unloading of the pulmonary zone of increasing edema and infiltration.

However, replacing pain in the case of AP with such an important adaptive response is not always able to protect the lung tissue from a sudden relative excess of venous return. In the case of rapid development of an inflammatory reaction, which is an individual feature of the body, it does not have time to adequately redistribute the circulating blood. In such situations, the autonomous realization of this reflex proceeds in parallel with the rapid dynamics of inflammation, acquiring the features of an uncoordinated reaction and creating an additional problem. The mechanism of protecting the lung tissue from the increasing phenomena of inflammatory edema goes beyond its initial limits, trying to reduce blood flow to this area, which is accompanied by a reflex generalized spasm of the vessels of the small circle. Consequently, patients with AP at the onset of the disease may experience typical shock instead of typical pain. The presence of such a mechanism of changes in blood circulation in this cohort of patients was proved using objective criteria for evaluating the results after applying methods to eliminate the described disorders [3].

Currently, such a generalized rearrangement of blood flow in the pulmonary vessels has been further confirmed by evaluating the blood supply to lung tissue in patients with COVID-19 pneumonia. The study of tomograms of this category of patients, due to the lack of parallels between the volume of inflammatory transformation of lung tissue and the severity of clinical manifestations, made it possible to establish a spasm of the vascular bed of a small circle at the level of small vessels with a diameter of 2 mm or less [4,5]. In addition to these findings, W. Dierckx, et al. [5] it was noted that the smaller the volume of blood in the lungs, the more patients need oxygen supply. The latter circumstance once again confirms that the main cause of ventilation and gas exchange disorders in AP is due not to the volume of damage to the lung tissue, but to a total violation of blood circulation in the small circulatory circle [3].

As already noted, pain in AP disease appears in the late stages of the development of the focus of inflammation, when the process reaches the pleural membrane containing an abundance of pain receptors. This phenomenon reflects the ongoing expansion of the inflammatory zone, and the appearance of pain is a signal addition to these shifts, indicating the approach of even deeper violations of the respiratory cycle [3]. If there are no other reasons for the appearance of pain syndrome, then the pain with AP, which increases with breathing, is a serious sign that indicates to the attending physician the need for urgent targeted assistance to the patient to eliminate the root cause of the disorders that have arisen. According to our experience and test results, of all pathogenetic first aid methods, the most effective remedy in the case of pain syndrome in AP is cervical vagosympathetic blockade, which not only instantly eliminates pain, but also eliminates generalized spasms of pulmonary vessels, acting as an anti-shock measure [3]. In addition, this method allows for a clear differential diagnosis diagnosis between pain syndrome in AP and possible intra-abdominal causes.

Conclusion

The analysis of materials on compensatory and adaptive reactions of the body allows us to note the following. Such a classic sign of inflammation as pain is mainly of a signaling nature, but its appearance is not accompanied by autonomous adaptive actions of the body itself. Such a signal basically forces the patient to observe protective measures in relation to the affected area. The absence of pain in most cases of AP development was initially replaced by nature with a more important and effective reaction, which allows us to avoid an imbalance in the work of the two halves of the cardiovascular system, automatically ridding us of conditions incompatible with life in the case of this disease. Understanding the essence of these mechanisms and situations where their development goes beyond adaptive shifts is of paramount importance for substantiating a therapeutic strategy and applying timely pathogenetic care.

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