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Review Article

The Importance of the Pneumonia Guiding Concept in the Fight against Sepsis

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Abstract

Sepsis (SS) is currently one of the leading problems of global health. Being a complication of various inflammatory diseases, SS is diagnosed in more than half of cases as a consequence of severe acute inflammation of the lung tissue, often at the very beginning of the disease. Despite such an obvious connection with pneumonia, SS has recently been considered and studied as a separate syndrome. The experience of previous studies and clinical trials indicates that a radical change in conceptual ideas about the uniqueness of the pathogenesis of inflammation in the lungs and the priority of pathogenetic approaches in justifying its treatment opens up opportunities for the prevention of such complications.

Keywords: *Sepsis; Acute Pneumonia; Pathogenesis; Pulmonogenic Shock; Pseudosepsis*

Abbreviations

AP: Acute Pneumonia; ARDS: Acute Respiratory Distress Syndrome; COVID-19: Coronavirus Disease 2019; MERS: Middle East Respiratory Syndrome; RAAS: Renin-Angiotensin-Aldosterone System; SARS: Severe Acute Respiratory Syndrome; SARS-CoV-2: Severe Acute Respiratory Syndrome Coronavirus 2; SC: Septic Shock; SS: Sepsis; WHO: World Health Organization

Introduction

Despite numerous studies and attempts at various practical innovations, the problem of sepsis (SS) remains one of the global problems of world health [1], very far from a successful solution. Against the background of a widespread monolithic consensus on the leading role of antibiotics in the treatment of this contingent of patients, voices of doubt have recently begun to be heard that the increasing course of septic conditions can be stopped with the help of antibiotics and supportive measures [2]. Unfortunately, this is only an expression of doubt without any specific proposals, and even more so without targeted actions.

The source of this report is an analysis of well-known facts on the problem under discussion, accumulated over the past decades, as well as the author's previous experience in this area. One of the turning points in the career of the author of these lines was the forced decision of the local administration of that time to refer and hospitalize all severe forms of acute pneumonia (AP) to our surgical department immediately after diagnosis. The experience accumulated over a long period of work with this non-surgical contingent of patients (in parallel with the work of a surgeon), the results of special studies and their clinical trials [3] provide grounds for considering the prospects for further study and solution of the AP problem from an angle different from the priorities generally accepted today.

Discussion

Acute pneumonia and sepsis

The following suggested ways of further studying and solving the problem of SS concern only those cases in which the primary cause of septic complications is considered to be only AP. Nevertheless, a detailed analysis of the pathogenesis of acute inflammatory transformation of lung tissue is necessary and very indicative, and can also be instructive for understanding the essence of the problems being solved in parallel.

Firstly, over the past decades, AP has been the leader among other diseases in terms of the number of septic complications. Moreover, in recent years, this disease accounts for more than half of all cases of SS and septic shock (SK) [4,5], but the reasons for such an absolute leadership of AP in the development of SS throughout this time remain without convincing explanations. Therefore, the phenomenal expansion of this disease as a source of SS simply obliges us to subject this problem to targeted study.

Secondly, AP differs fundamentally from all other inflammatory diseases by its localization, being the only nosology from this list that occurs in the pulmonary circulation. The result of such location of the inflammation focus, first of all, is the disruption of pulmonary blood flow, which has indicators opposite to the systemic one, and the ability for general regulation [6,7]. The disruption of blood flow in the pulmonary circulation is accompanied by secondary restructuring of the systemic circulation. In aggressive forms of development of inflammatory transformation in the lungs, compensatory transformations corresponding to the picture of shock are observed on the periphery. However, such a reaction has an exclusively pulmonogenic origin and is not septic in nature [3].

Thirdly, in recent decades sepsis has been diagnosed using uniform schemes, regardless of the localization of the primary process and the features of the pathogenesis of these diseases [8,9]. These schemes include such characteristic signs of severe AP as dyspnea and hypotension. With AP, dyspnea appears already at the time of primary diagnosis, and the tendency to hypotension is observed somewhat later, but both signs are a reflection of the initial compensatory efforts of the body. However, as a result of using these schemes, almost every patient with aggressive development of AP in the first days and even hours of the disease is diagnosed with so-called SS [4,5,10].

Finally, errors leading to the diagnosis of pseudosepsis entail the use of treatment methods that dampen the compensatory efforts of the body, stimulating the mechanisms of development of the process [3]. First of all, this remark applies to the prescription of intravenous infusions, especially in the initial period of the disease, when their negative effect is most pronounced [3].

Modern principles of forming research objectives in solving the problem of pneumonia and its complications

The importance and frequency of various diseases in the development of SS have practically ceased to be a subject of discussion. Today, the role of the microbial factor dominates in the development of inflammatory processes of non-specific etiology and their septic complications, which is a didactic result of many years of unconditional faith in the therapeutic effect and indispensability of antibiotics. This view of the problem has concentrated research and various tests at the cellular-molecular level, which corresponds to the scale of the pathogen and attempts to obtain the necessary information about the mechanisms of its aggressiveness. However, such virtual details at the micro level, realizing some scientific interests, do not open up levers of influence on the clinical manifestations of the process, and attempts to influence the identified deviations do not bring positive dividends in everyday practice.

Unfortunately, in recent decades, attention and interests have not only shifted to the level of microstructures. Research and study of AP manifestations are increasingly losing cause-and-effect relationships and increasingly represent an isolated study of the observed phenomena. The logically consistent development of the disease (from the primary focus to the occurrence of complications and critical

conditions) has become unjustifiably divided into separate syndromes and phenomena, each of which, being one of the links in a single pathogenetic chain, is considered and studied independently at the level of microstructures [11-13].

An isolated study of disease manifestations with an emphasis on virtual molecular-cellular links of the process and in isolation from its pathogenetic root causes, in my opinion, clearly cannot provide the necessary conditions for success. Thus, today reactions and interactions between individual types of cellular and molecular structures in such syndromes as ARDS (acute respiratory distress syndrome), SS, multiple organ failure or in such phenomena as peripheral microcirculation disorders are actively discussed. Instead of taking measures to quickly eliminate the root causes of such development of the main process and, first of all, to prevent its progression, today enormous resources and efforts are spent on attempts to correct the secondary situation, which at this stage of the disease is already becoming uncontrollable and unmanageable.

In the context of numerous contradictions and inconsistencies in the modern concept of AP and oblivion of the cause-and-effect relationships of the process, the pathogen is still considered as a factor determining the development of critical conditions. However, what is truly surprising is not so much the interpretation of SS as a separate symptom complex, but the revision of the interpretation of AP. Thus, in recent years, one can find reports in which AP is no longer considered an independent disease, but is assessed along with SS as a global burden of severe pathophysiological and biochemical deviations depending on the type (!?) of the pathogen [14-16]. This point of view reflects the depth and tenacity of misconceptions that have developed today and dominate in professional consciousness, despite refuting facts.

Discrepancies between modern ideas and real facts on the problem of AP and SS

If several decades ago such a view of the problem of AP at least had such mitigating facts as the almost total prevalence of bacterial forms of the disease and the validity of free empirical choice of antibiotics, by now these conditions have changed dramatically. Firstly, in more than half of the cases of AP, according to a review of such statistics, the nature of the pathogen remains unknown [17], and among the remaining observations in which it was possible to determine the microbiology of the process, viruses predominate [18,19]. Even among patients with SS, at least in a third of cases the question of the pathogen remains unanswered [20].

Secondly, what bacterial dependence of clinical manifestations of AP and SS can we talk about if it was not possible to obtain convincing differences in differential diagnostics not only between various bacterial forms of inflammation, but also to differentiate bacterial and viral lesions [21,22]. At the same time, the etiology of AP has undergone significant changes over a long period of antibiotic use, but this did not have a fundamental effect on the classic signs of the disease, which persist regardless of the pathogen.

Thirdly, specialists have not yet explained the fact that almost 40% of patients with acute inflammation of the lung tissue already at the time of hospitalization have signs of the so-called secondary SS, and among very elderly patients this figure reaches 71% [23,24]. In such cases, SS cannot be an independent pathology, playing the role of a consequence and continuation of severe AP. The widespread explanation of such a threatening development of the disease by the aggressiveness of the pathogen over many years is declarative in nature, being nothing more than an assumption and lacking convincing evidence. Moreover, it should be noted that soon after hospitalization and despite the treatment, already in the first two days up to a quarter of patients with AP are admitted from general wards to the intensive care unit due to a critical deterioration in their condition [10,25,26].

In this regard, adherents of the so-called germ theory of AP need to pay attention to the unprecedented experience of the SARS-CoV-2 pandemic, the results of which convincingly showed us all that neither the nature of the pathogen, which in this case was the same for all infected, nor the lack of etiotropic therapy against the coronavirus were decisive factors for the final results. Now, as is known, the vast

majority of infected people on the planet survived this event without specific medical care, and many learned about this "surprise" only from test results. Only about 5% of the infected population with COVID-19 pneumonia who required hospitalization in the intensive care unit posed a serious problem for the medical service [27].

Fourth, the statistics of the recent coronavirus pandemic provide compelling reasons for analysis and reflection [28], but unfortunately, the unique material of this phenomenon remains insufficiently studied and understood. Therefore, one of the most common retrospective assessments of the coronavirus invasion remains a spectrum of opinions about difficult-to-explain triggers for the emergence of the pandemic - from deliberate (conspiracy theories) to natural (global warming) causes [29,30]. Such views largely reflect the mental effect of the concept of the dominant role of the microbiological factor.

However, this overlooks the fact that under the influence of long-term antimicrobial aggression, there have been shifts in the etiology of AP towards viruses, which have competed with bacteria in this list for many years. In addition, over the past two decades, there have been at least two coronavirus epidemics (SARS and MERS), demonstrating the severity of the pneumonias they cause, which, unfortunately, did not affect the principles of their treatment. At the same time, it is not advertised that coronavirus pneumonia continued to be registered quite often in the period between epidemics and the pandemic [31,32].

There is no doubt that the etiology of AP has changed dramatically over the long period of the antibiotic era. However, with the proven and already widely recognized prevalence of viral AP, not only does the active development of diagnostic systems for bacterial pathogens continue [31,33], but great hopes continue to be placed on a breakthrough in solving the entire problem through the development and use of new generations of antibiotics without a detailed analysis and assessment of the fruits of this therapy already reaped [34,35].

Fifth, currently, 60% or more of AP are caused by viruses [36]. It is known that viral inflammations are most typical for respiratory diseases, and the pathogens themselves are characterized by frequent mutations. As a result, we observe an annual wave of a new variant of respiratory epidemics and are forced to update vaccines to protect against them. If in the case of bacterial AP, the concept of SS was formed on the basis of old ideas, when bacteremia was important for diagnosis, now this syndrome is determined by counting points when assessing mainly functional indicators [8,9]. No one has presented convincing arguments in favor of the fact that the observed clinical picture in patients with severe AP is a consequence of a truly generalized viral infection. In such cases, the diagnosis of viral SK is based only on the analogy with the picture of bacterial SK, especially since they have indistinguishable similarities [37].

Sixthly, the attention of scientific and practical medicine is currently focused on severe AP, the rapid development of which requires prompt emergency care for such patients. The choice of such a goal is absolutely fair, but in this regard, another long-standing problematic situation is revealed. In the modern official arsenal of treatment for AP, there are no methods whose action could be attributed to first and emergency care. The role of such care is traditionally performed by etiotropic drugs, and the main place among them is still given to antibiotics. The effect of antimicrobial therapy can be assessed by attending physicians only after 48-72 hours [38,39]. For seriously ill patients with AP, the first 2 - 3 days of the disease are critical, when, despite the start of inpatient treatment, the condition of many of them worsens. Therefore, such an approach to providing emergency care is a waste of time and hopelessly missed opportunities. However, this is only the tip of the iceberg.

The broad discussion of the prospects for treating patients with AP constantly revolves around antibiotics and options for increasing their effectiveness, but no one focuses on the fact that these drugs cannot perform functions that are not characteristic of them. Each drug is designed to suppress certain types of bacteria, but none of them has a direct, and even more so immediate, effect on the mechanisms of the disease. They can act only indirectly and, having achieved their goal, help neutralize one of the factors of the intensity of the inflammatory process, but leave further elimination of its consequences to the discretion of the patient's body.

In this regard, the question naturally arises: why do antibiotics continue to be considered the main method of treating AP, while simultaneously having a reputation as an emergency aid? Yes, there was a period when bacterial forms of AP prevailed and antibiotics were considered the only treatment, used according to the principle of "antibiotics alone." At the same time, one drug could be the main and only method of treating completely different diseases. But if a drug turns into a panacea for incomparable ailments, then, in my opinion, it is impossible to give a scientifically sound answer to the question posed above.

Seventh, many specialists currently come to the conclusion about the leading role of circulatory disorders among general shifts in the body of patients with SS [40,41]. These data confirm our previous conclusion that AP, oddly enough, is primarily a circulatory, not a respiratory problem [3]. However, a misconception in modern interpretations of circulatory disorders in AP is their assessment and subsequent correction based on systemic blood flow indicators, whereas the main events begin and occur in the pulmonary circulation. In such a situation, the parameters of peripheral circulation in AP reflect the degree of secondary compensatory transformations, which in the modern understanding are perceived as manifestations of SS and are subject to therapeutic action regardless of the pathogenesis of the underlying disease.

Recent studies have shown that changes in systemic blood flow in patients with severe AP are initially observed at the level of microcirculation, and only then do signs of hypotension appear [42,43]. The main method of correcting such disturbances according to general treatment regimens, regardless of the pathogenesis of the underlying cause, remains intravenous infusions, which in the most severe situations are of a bolus nature. The role of intravenous infusions has increased in recent years, taking into account the initial and less pronounced shifts in microcirculation. If until recently the immediate use of antibiotics was considered first-line therapy, now many specialists consider the immediate administration of solutions to be extremely important, and some even prefer them to antibiotics as emergency care [41,43,44].

The described approaches to providing first aid to patients with severe AP, especially with frequent diagnosis of SS, have an effect opposite to that expected. The pulmonogenic shock that occurs in these patients with its unique, exceptional pathogenesis is a contraindication to intravenous infusions, which only aggravate further disorders [3]. In this regard, the explanation for the current failures of inpatient treatment of AP should be sought primarily in the false diagnosis of SS and in the professional commitment to the early use of infusion therapy.

Finally, it is necessary to pay attention to one more important detail. Starting to comprehend the intricacies of medicine on the university bench, we get an idea that each disease as a separate nosology has its own characteristics, expressed in their etiology and pathogenesis. Etiology describes the triggers that contribute to the occurrence of the disease, while pathogenesis gives an idea of the cause-and-effect relationship of those mechanisms that form the uniqueness of its clinical picture. This is one of those sections of education that turns our general ideas about diseases, their occurrence and development into professional views. The more such information a specialist has and the ability to quickly apply it, the more his skills and capabilities are valued. However, these sections, presented in modern literature on the problem of AP, including in educational and methodological and training materials, are distinguished, in my opinion, by a clear exaggeration of the role of etiological factors.

The unforeseen course of events for medicine during the SARS-CoV-2 pandemic significantly accelerated the possibility of understanding the obvious fact that the etiology of pneumonia does not play a significant role in the severity of its development. However, this natural message remained outside the scope of professional perception, and the obvious loss of the main therapeutic hope - antibiotics - did not have a significant impact on the usual stereotypes of treatment. Patients with COVID-19 pneumonia in more than 70 - 80% of cases continued to receive antibiotics, despite the fact that bacterial coinfection was detected only in a small number of observations [45,46].

During the pandemic, numerous active studies were conducted to find means and methods for treating coronavirus infection at the cellular and molecular level, and their results were discussed as the pathogenesis of the disease [47,48]. Some reports even used terminology corresponding to the stated research goals - "microbial pathogenesis", "microbiological pathogenesis". At present, it is known that new frontiers in the treatment of pneumonia in general and viral pneumonia in particular have not been reached, and after the official statement by WHO about the end of the pandemic, interest in antibiotics and the use of their new generations remains in high demand [34,35].

The subject of research in COVID-19 pneumonia has been individual manifestations of the disease, which have been considered and discussed as the main causes of the severity of the disease. For example, such as the level of cytokines and the so-called cytokine storm [49], the reaction of the renin-angiotensin-aldosterone system - RAAS [50] or ventilation and perfusion disorders due to inflammatory edema and atelectasis [10]. Finally, heterogeneous reactions of the body against one pathogen have intensified the study of the immunological status of patients during the pandemic, highlighting this characteristic as an important section for the provision of emergency medical care [49,51].

It would seem that the wide scope of the studied sections of the problem should give rise to hopes for achieving success. However, from my point of view, this is only an appearance that is unable to justify expectations. All the mentioned studies consider the observed deviations as the main influencing factors of the disease development and focus on their dependence on the virulence of the pathogen. The latter circumstance explains the further development of SS, ARDS, multiple organ failure, as well as attempts to reduce the aggressiveness of inflammation by neutralizing or correcting the identified deviations in the condition of patients. For example, attempts to neutralize the "cytokine storm" [49] or to use RAAS blockers [50] did not bring the expected results.

The above-listed research options are aimed only at small segments of the general pathogenesis of the disease at the cellular and molecular level. Such mechanisms occupy a certain place in a strict temporal and cause-and-effect progression, forming a common chain of pathogenesis with other phenomena. Unfortunately, in recent years there has been an inexplicable tendency to focus attention on the study of such complications of AP as SS, ARDS, etc., considering them as a separate syndrome and not attaching importance to the primary source of their occurrence. A sign of this tendency is the absence of information in recent years in patients with SS about the frequency of nosologies that served as its source.

The description of the essence of these syndromes begins with the moment of their occurrence, and not with those prerequisites and triggers in the pathogenesis of AP, by influencing which it would be possible to prevent their development. In this regard, it has become customary to concentrate efforts on eliminating complications that have already arisen and attempts to bring patients out of critical conditions. Since the goals of such efforts often remain unattainable, another pattern has arisen - predicting the probability of negative results using various tests. If we evaluate this situation in other words, we can state the formation of a losing expectant psychology in approaches to treatment in this group of patients.

For example, instead of searching for opportunities to quickly neutralize the mechanisms of the inflammatory process in the lung, efforts are being made to determine the potential timing of the development of the so-called SS in these patients [52]. That is, complications of the process are expected, not prevented. Or, for example, to predict the results of treatment of patients with AP, they began to use the definition of probable indicators of delayed mortality at different time intervals from the beginning of treatment [53]. In recent years, one of the most frequently used tests is the mortality rate on the 28 - 30th day. And this is a whole month of treatment! This means that doctors are not sure of the treatment they are conducting, and the wait-and-see attitude of observers requires at least a prognosis of fatal outcomes, right?

The examples provided show that despite the lack of success in solving the problems under discussion, previous ideas about the prospects for solving them remain unshakable. There are no signs of a radical revision of the concept of AP and its complications, although such an inevitable need is increasingly caused by new facts and circumstances every year. Today, the situation has become completely obvious when, with severe development of AP, generally accepted principles of treatment often do not bring the expected result and the process progresses, reaching the stage of complications. However, the results of such escalation began to be interpreted and treated as separate syndromes. At the same time, additional medical care as a result of the emergence of a new pathology begins to be used with its appearance, and not as a way to prevent such complications. Attempts to foresee such a course of events with the aim of early referral of patients to the intensive care unit do not change anything, since the basis of medical care remains the same, and the intensity of its implementation in patients with AP can only accelerate the negative dynamics [3].

When analyzing and discussing the problem of SS, it is necessary to note the results of studies that have shown the similarity of the body's response to "infected" and "sterile" inflammation [54]. A suddenly emerging powerful trigger, regardless of the presence of infectious factors, is accompanied by a uniform shock-like reaction of the body. Such materials should be used as food for thought before continuing to declare trends in which the diagnosis of AP pathogens is presented as a priority task. The results of these studies are further indirect evidence that to eliminate such reactions, one should rely more on pathogenetic rather than etiotropic therapy, especially when providing first aid.

The essence of the presented analysis is that the same information can be assessed from different positions, which leads to different conclusions. However, the implementation of such an approach is possible only if the potential analyst can impartially assess new facts and phenomena that do not coincide with generally accepted and unshakable attitudes. Such examinations help to promptly and consistently correct our ideas, moving forward in solving the problems facing us.

Prospects for research into the problem of AP and SS

The results of treatment of severe AP have already been discussed above, which quite fully reflect the unresolved tasks of the problem under discussion. Not only the figures, but, above all, the dynamics of clinical observations make us recall one of the fundamental rules of medicine, on which its main principles are based and supported: "if after the start of treatment the patient does not feel better, then such treatment does not correspond to the nature of the disease".

Currently, an endless stream of reports on the results of studies and clinical trials on the problem under discussion is published, which report on the achievement of some positive dynamics of various tests and therapeutic maneuvers and which end with standard reservations that this requires further study and verification. Such additions are a sign of the authors' own uncertainty in the significance of the trials being conducted and their future prospects. Such materials concern only tactical actions, while the strategy of the problem remains without the necessary revision.

In addition, another version of the conclusions that the results of the studies conducted "promise us improvement, achievement... etc." has become quite widespread. I think that we should not mislead ourselves and create the appearance that the set goal has almost been achieved. No one and nothing promises us anything. Such promises are only the fruit of our desires and fantasies, while the situation requires from us a balanced and realistic assessment of the current situation.

The prospects of the conducted research for solving the problem under discussion are largely determined not only by the possibility of applying the obtained results in clinical practice, but also by monitoring their effect. Today, there are very few such works. Many studies are conducted at the cellular and molecular level, presenting a virtual picture of what is happening. With an ever-deepening interest in research at the micro level, medical science does not attach importance to the fact that examination of patients and further monitoring

of the dynamics of their condition over many years continue to be based on integral indicators of the function of the main systems of the body (respiratory, cardiovascular, central nervous). Each attending physician expects to receive positive changes in these very signs as a result of his actions, doesn't he?

It is difficult to imagine that the general clinical condition of a patient is assessed only by a set of qualitative changes in the composition and quantity of certain cells or molecular compounds. Therefore, the choice of methods for providing first aid to patients with AP should correspond to the integral level of their monitoring and give preference in an objective verification of the effect to those methods that are capable of influencing the main functional indicators of the patient immediately after their use.

Below are the main tasks that need to be implemented to achieve success in solving the problem of AP and the syndrome of its progressive development, which is currently interpreted as SS, but the first steps in this direction were made by the author of these lines several decades ago [3]. The results of clinical and experimental trials were confirmed by objective evidence at the level of the capabilities that were available at that time. The effectiveness of the approaches used to solve such problems is limited by the level of research and monitoring methods available at that time. At present, medicine has significant potential for monitoring, ongoing diagnostics and various types of testing, which opens up great prospects for obtaining objective criteria for the necessary work. The main tasks of such work can be presented as follows:

1. The first step, without which all subsequent undertakings will be deprived of meaningful and purposeful actions, is the correction of professional ideas about the essence of the problem under discussion, taking into account the peculiarities of the pathogenesis of pulmonary inflammations and the reassessment of the exaggerated role of their etiology and etiotropic treatment. Such work requires bringing the established idea of the disease into line with the basic canons and laws of medical and biological science.
2. The implementation of the first step will allow us to note the directions and nature of the body's compensatory-adaptive reactions when AP occurs, to understand the depth and consequences of such a reaction. At the same time, it is necessary to solve the complex problem of establishing a boundary when the positive adaptation of the body begins to take the form of negative transformations that require external assistance.
3. The fate of a patient with AP directly depends on the timeliness of the application and the nature of first aid. The sooner pathogenetically substantiated methods of such aid are applied, the more indicative and convincing the achieved result will be. Among the already proven methods of first aid with an immediate integral effect, we can recommend such methods as cupping therapy, cervical vagosympathetic blockade and general cooling of the patient's body. Cooling, which gives the most noticeable result, can now be reproduced using the devices for whole-body cryotherapy produced today.
4. It is necessary to completely revise the criteria for diagnosing SS in patients with AP based on a single scheme and continue studying the pulmonogenic variant of shock reaction in patients of this category. This step will significantly reduce, and possibly eliminate, the diagnosis of SS as a consequence of AP.
5. A separate analysis of patients with AP, for whom infusion therapy is an undesirable and aggressive method, especially in the early stages of the disease, will allow a more detailed study of the problem of SS with peripheral sources of inflammation and, in particular, when assessing the effect of the same infusions. Etiotropic therapy should be considered as auxiliary, but in no case the main and urgent.

The presented set of goals for implementation concerns only the reassessment and rethinking of the probability of developing septic conditions in patients with AP and does not extend to the entire problem of SS. However, if we take into account the fact that AP is currently the absolutely dominant cause of SS, the diagnosis of which as a result of this disease already stably exceeds half of all observations, then the implementation of these tasks on a large scale can yield colossal results.

Conclusion

Rethinking the causes and mechanisms of the clinical picture in patients with AP, imitating the signs of SS, and reassessing the principles of diagnosing this condition will allow us to exclude the verdict on mythical septic complications and radically revise the principles of treating this category of patients. Such a restructuring of the system of professional ideas about the features and leading role of the pathogenesis of the disease, as well as a shift in emphasis to pathogenetically substantiated approaches to treatment and the earliest possible use of first aid methods capable of providing a rapid integral effect, will finally allow us to see real successes in this section of medicine.

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Conflict of Interest

The author states that he has no conflict of interest.

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