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What are the Principles of Emergency Care for Acute Pneumonia?

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Abstract

A simple, rapid, accurate and sensitive spectrophotometric method for determination of Propranolol Hydrochloride has been developed. The proposed method is based on the reaction between chloride ion and mercuric thiocyanate, formation of a colored complex by the reaction between released thiocyanate and ferric ions to form red soluble product with maximum absorption at 454 nm. Beer's law is obeyed over the concentration range of $2.5-35\mu g/ml$, with molar absorptivity of 0.88444×104 l/mol.cm. The present method is considered to be simple because it does not need either heating or hydrolysis or solvent extraction steps. The method has been successfully applied for the determination of Propranolol Hydrochloride in environmental wastewater sample and in pure form, pharmaceutical preparations (Tablets).

Key words: propranolol hydrochloride; mercuric thiocyanate; pharmaceutical preparation; environmental wastewater sample

Introduction

Emergency care for acute diseases is an important part of medical care, largely determining its further outcome. The rapid development of the process dictates the use of such methods of care that allow slowing down or eliminating the action of the main mechanisms of the disease, preventing its further progression and the development of complications. In acute inflammatory processes, when swelling and tissue infiltration in the lesion site increase, timely and targeted efforts are required that can significantly slow down these processes and reduce the risk of further spread of the inflammation zone. The latter circumstance is of paramount importance in the case of localization of damage in vital systems, such as, for example, in acute pneumonia (AP).

The information of recent years on the results of medical care in AP is surprising and puzzling. Against the background of incredible successes and achievements of modern medicine in various fields and areas, one of the oldest topics - the problem of AP - slowly but steadily continues to strengthen its leading position among the causes of morbidity and mortality [1-3]. At the same time, recently the interest of clinicians and researchers has begun to be attracted by a group with a severe course of the disease, who, as a rule, are sent to intensive care units (ICU), and the results of treatment are particularly alarming [4,5]. In such situations with aggressive development of the inflammatory process in one of the vital systems of the body, the role of emergency care is of paramount importance. The results of treatment of this category of patients, which continue to discourage, despite, it would seem, all possible innovations, require a detailed analysis of the principles of formation and application of emergency care. Without such an analysis, it is impossible to hope for achieving stable success.

Discussion

The first description of AP is associated with the name of Hippocrates [6], however, for most of the time, this disease remained poorly studied and was interpreted as a severe inflammatory process with high mortality. At the

same time, one of the fundamental characteristics of this nosology throughout its history was the postulate that AP gets sick, not infected. Practical confirmation of this formulation is reflected in the established sanitary and hygienic traditions regarding this disease, which do not provide for the isolation of patients and compliance with quarantine conditions.

The first major breakthrough in the study of inflammatory processes occurred at the beginning of our era, when Celsus and Galen described five essential signs of this pathology - . In relation to AP, these classic manifestations of inflammatory changes in the lung tissue remained of little use until the 20th century, when the results of such developing disciplines as physiology and pathophysiology began to appear, as well as the emergence of new diagnostic capabilities with the discovery of X-rays [7]. However, the development of microbiology and the results of studies of the etiology of AP, the first results of which were obtained in the second half of the 19th century, outpaced the study of the functional characteristics of this pathology, which played a significant role in choosing the directions for solving this problem. Already at the end of the 19th century, the so-called microbial theory of the origin of diseases appeared, which in turn stimulated the emergence and development of immunology [8].

The noted period of emergency use of the indicated method of serotherapy turned out to be short-lived, since all these initiatives took place literally on the eve of the appearance of antibiotics, and soon after the beginning of their use and the first results, the relevance of the task began to disappear. Patients with AP began to be considered as patients who did not need the emergency support that had been carried out during the previous decade, and the attitude to this problem itself acquired features of calming and stress relief [8]. The erroneousness of such an assessment began to manifest itself soon after the beginning of widespread antibiotic therapy, since the qualitative characteristics of these drugs were known even before the beginning of their practical use, and its essence indicated the discrepancy between the

possibilities of this therapy and the role that was assigned to it in practice. In addition, before the beginning of the practical implementation of this antimicrobial care, it was known that its effectiveness could not be long-term [9, 10].

The atmosphere of confidence in the rapid effect of antibiotics and the convenience of using this therapy outweighed the measures of strict control over the targeted use of these drugs and a balanced assessment of the changes that began to appear and gradually increase. Such transformations of microflora as a change in AP pathogens and the emergence of microbial resistance did not find any other solution than the accelerated development and release of new generations of these drugs in the first decades [11]. Confidence in the exceptional role of antibiotics in the treatment of inflammatory diseases continued to grow, and the prescription of this therapy to many patients was of the nature of "antibiotics alone." This assessment of antibiotic therapy continued to grow and dominate professional views, despite the fact that these drugs are capable of acting only against certain types of pathogens, but do not have a direct effect on the mechanisms of inflammation.

It is now known that the initial idyll of antibiotic use could not long be maintained in the role it played in the early years. Over time, the number and diversity of resistant microflora increased, and early attempts at targeted antibiotic therapy were unsuccessful. The number of AP cases requiring additional and supportive therapy gradually increased. The etiology of AP became increasingly inaccessible to antibiotics, and viruses turned into a key problem of the disease [12-14]. In recent years, the number of patients with AP in whom the pathogen remains unidentified has consistently exceeded half of the cases [15]. However, by this time, antibiotics, paradoxically, despite all the inconsistencies and contradictions, have firmly entered the professional consciousness as a kind of panacea, without which modern medicine no longer sees other ways to treat patients with AP.

Currently, antibiotics are responsible for all the main tasks that need to be solved to achieve success in the treatment of patients with AP. These drugs are still considered by most clinicians as the main methods of treatment. Many specialists continue to consider this therapy not only as the main one, but also as the only option for specific action. At the same time, this therapy simultaneously continues to play the role of first emergency care for this group of patients. Since the effect of these drugs cannot be manifested by an immediate change in the measured parameters of the patient's condition, standard and widespread recommendations suggest a waiting period of up to 48-72 hours [16,17].

Such a wait-and-see period in patients with AP cannot be completely passive in all observations. Many of them require additional or auxiliary care. According to the dominant concept of the disease, in which the pathogen is considered as its main cause, such supportive therapy is aimed at eliminating the consequences of the infectious factor and today has a single standard for all inflammatory processes, regardless of localization. The most common method of supporting patients with inflammatory diseases is intravenous infusions of solutions, which in most observations begin from the moment of hospitalization of patients and are considered as a preventive measure for possible septic circulatory disorders. However, in this case, the fact that the circulatory systems of the small and large circles of blood circulation have a common interdependence and regulation, remaining with directly opposite blood flow indicators is not taken into account. In addition, it is in patients with AP that any intravenous infusion first reaches the inflammation zone with the increasing processes of edema and tissue infiltration at this time.

Unlike inflammatory processes localized in the systemic bloodstream, damage to lung tissue in severe cases of the disease is accompanied by generalized spasm of the pulmonary vessels, which causes signs of their overload [18-20]. The author of these lines has well felt and proven from his own experience what unpleasant surprises can await both the patient with AP and his attending staff in the event of early infusion therapy [18]. In this regard, the results of treatment of patients with AP, reflecting the state of medical care in modern medicine, are a natural consequence of the existing principles of emergency care for this category. Thus, as a result of the ineffectiveness of the initiated treatment, the deterioration of the condition

of up to 25% of patients with AP admitted to general departments requires their transfer to the ICU already in the first 2 days [21, 22]. The overall results show that the main problem is represented by patients with a severe course of the disease, for whom timely and adequate emergency care is of fundamental importance [1-3, 22,23].

It is surprising and inexplicable from the point of view of logic and critical analysis of the main facts related to the problem of AP that the search for its solution in modern conditions obsessively and stubbornly continues to focus on the diagnosis of pathogens and the search for the most optimal antibiotics [14,16,17,24,25]. Such a perception of approaches to those characteristics that today relate to the foundations of the problem of AP creates the impression that there were no 80 years of antibiotic use and everything is starting "from scratch", and the leading causative agent of the disease is still pneumococcus. But everyone is well aware of the data on the fundamental changes that have occurred in the etiology of the disease during this period. Such changes include a radical shake-up of the list of pathogens, in which viruses began to occupy a significant place [12-14]. At the same time, the percentage of AP observations is growing, already exceeding half of all cases in which the pathogen cannot be identified, and the question of the exact choice of etiotropic agents remains open [15,17,26].

All the initiatives described in the above publications do not bring the expected shifts in results, but the general conclusion is a persistent assertion about the need for further research in this direction. Such conclusions would be justified if we were talking about violations committed during the research process, but there are no such indications, and the fanatical commitment to the previous concept of the disease remains unshakable. To these features of firmly established ideas about the nature of the disease, which have the character of unshakable cliches, we should add a clearly exaggerated assessment of resistant microflora. This phenomenon, the development of which was predicted even before the beginning of the clinical use of antibiotics and accompanied this therapy with its appearance and development throughout the entire period, has recently begun to attract attention only because the effectiveness of the main therapeutic hope antibiotics - has significantly decreased. Without real proposals for a way out of this situation, the resistance of AP pathogens began to be indicated as the reason for the lack of success in the treatment of these patients.

A number of facts indicate that the frightening assessment of resistant microflora, which has now become the main symbol of lively discussions and one of the medical sections with suddenly increased attention and interest in it, is clearly and significantly exaggerated. Firstly, resistant strains have become familiar representatives of normal microflora, being symbionts of the microbiota of healthy people. For example, the prevalence of MRSA in the body of various categories of the population and professions fluctuates from 2% to 10%, without causing any problems to its carriers [27-31]. In the composition of the surrounding microflora, MRSA can make up to 35%, cephalosporin-resistant E. coli - up to 42% [32], resistant strains of pneumococcus - up to 20% [33, 34].

Secondly, at present, in the majority of patients with AP, as already noted, the pathogens remain unidentified, and the bacterial etiology of the disease accounts for less than half of the cases, where resistant strains represent an insignificant group [14]. Such a number of observations with resistant pathogens of inflammation cannot in any way serve as an explanation for the significantly greater number of failures that continue to occur in "simple" forms of the disease.

Thirdly, resistant strains of AP pathogens can pose a serious problem for the treatment of such patients only if etiotropic drugs continue to be considered the main means of providing medical care. This is exactly what is happening now, when antibiotics, despite the change in the etiology of the disease and the facts of direct ignoring of the pathogenetic side of the process, continue to determine the strategy for solving the entire problem.

Fourthly, modern medicine, continuing to focus on the leading role of the AP pathogen and hoping for the success of targeted antimicrobial therapy, has been trying for many years to find ways to differentially diagnose AP depending on the type of bacterial pathogen. Not only have repeated attempts at such efforts been in vain, but the desire to separate the bacterial and viral

forms of the disease has also been unsuccessful [35-37]. It would seem that such results clearly show that the causative agent of the inflammatory process does not have a specific effect on the manifestations of the disease, which retains its main features regardless of etiology. However, modern reality shows that all this rich experience, indicating the need to finally pay attention to the integral features of pathogenesis, has remained without critical analysis, and attempts to separate AP by the type of pathogen continue [24,38].

Finally, the narrow obsessive desire to revive the former activity of antibiotics that would continue to provide emergency assistance gives rise to ideas for developing new types of antimicrobial drugs, and such initiatives, supported by WHO experts, are acquiring a global character [39]. In fact, we are talking about the further development of the direction that caused the development of these severe side effects, which now require correction. In the context of significant changes in the foundations of the development of AP that have occurred during the period of antibiotic use, without a comprehensive analytical assessment of the observed transformations, continuing to blindly demonstrate an unwavering commitment to continuing and improving this direction is a very rash step. And here, plans to create completely new versions of such drugs at the level of molecules, nano- and genetic technologies are of particular concern [40,41]. It is difficult to even imagine what consequences the practical implementation of such plans can lead to, pursuing only the achievement of an antimicrobial effect, without imagining the natural protective reactions and their consequences.

Thus, emergency care for patients with AP remains focused on the expected success of etiotropic therapy, which requires, especially in conditions of aggressive development of the process, an unacceptably long wait. Practical implementation of the initial stage of treatment is accompanied by monitoring of integral indicators of vital functions of the body. At the same time, the conducted studies of the pathogenesis of the disease and attempts to apply the obtained results are based on the study of cellular and molecular deviations that form a virtual representation of the current process, but do not provide tangible practical advantages. Parallel appointment of auxiliary and additional means of assistance is used without taking into account the standard mechanisms of development of the disease panorama. Ultimately, patients with AP find themselves without real and effective emergency care, demonstrating a natural loss of timely opportunities for success.

The above picture of modern approaches to solving the problem of AP allows us to note that the main reason for the existing misconceptions in this matter is the negative didactic role of antibiotics in the formation of a professional worldview about the nature of this disease. In this regard, it seems completely unnecessary in this context to provide a description of already tested pathogenetic methods of providing first aid with objective evidence of their real effectiveness [18]. Such a description without a preliminary and radical revision of the foundations of the disease and a rethinking of all the accumulated facts in the light of the classical canons of medical science may turn out to be a completely ineffective presentation. Nevertheless, such a correction of views on the problem under discussion is an inevitable step, and its implementation today requires the same urgency as the use of the missing pathogenetic emergency care.

Conflict of interest: the author states that he has no conflict of interest **References**

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