ECRONICON OPEN ACCESS

EC PULMONOLOGY AND RESPIRATORY MEDICINE

Research Article

The Effect of Intravenous Infusion on the Dynamics of Acute Pneumonia

Igor Klepikov*

Pediatric Surgeon, USA

***Corresponding Author**: Igor Klepikov, Pediatric Surgeon, St. NE Renton, WA, USA.

Received: May 13, 2017; **Published:** July 12, 2017

Abstract

An experimental model of acute pneumonia on 44 rabbits was created. The use of intravenous infusions during the development of AP significantly influenced the occurrence of complications. Contrast studies of the vascular changes in the lung preparations (47 specimens) showed variants of this transformation at different stages of inflammation. The obtained results testify to the negative role of intravenous infusions in the initial period of acute inflammation of the lungs. The principles of treatment of acute pneumonia were revised and evidence of the advantages of a new strategy on clinical material was obtained. New treatment strategy was tested in 101 patients with the most severe forms of AP. The results of this work suggested the possibility of guaranteed prevention of suppurative complications of AP.

Keywords: *Acute Pneumonia; Intravenous Infusion; Pleural Complication*

Introduction

Everyone knows that if treatment does not bring the desired results, and even does not hinder the development of purulent complications, it means that such treatment is inadequate and does not correspond to the nature of the disease, isn't it? It is an axiom of clinical medicine.

Contemporary results of the treatment of acute pneumonia(AP) indicate the need for the revaluation of the help principles in this disease. Community-acquired pneumonia (CAP) is one of the 10 leading causes of death worldwide. Approximately 20% of CAP patients require hospitalisation, 25% of whom are admitted to an intensive care unit (ICU) and have a mortality rate of 30 - 50% [1]. Pneumonia is a leading cause of hospitalization among children in the United States, with medical costs estimated at almost $1 billion in 2009.

Despite this large burden of disease, critical gaps remain in our knowledge about pneumonia in children [2]. The rates of parapneumonic effusion have been increasing in the USA and Europe over recent years, and it is now encountered in approximately 40% of all patients with bacterial pneumonias [3]. Pediatric pleural empyema has increased substantially over the past 20 years and reasons for this rise remain not fully explained [4]. The above quotes indicate low efficiency of the modern treatment of AP and the absence of an explanation for this fact.

In this regard, the results, which the author of these lines has reached many years ago, should be of interest to professionals in this section of the medicine [5]. In this report, we are talking only about the impact of intravenous infusion on treatment of acute pneumonia. So here will be presented only the results of a large study that relate to the topic.

Material and Methods

The research was conducted in the clinic of pediatric surgery (Novokuznetsk, Russia) in 1976 - 1985. During this period in the hospital were treated 994 children aged from 4 months to 14 years with various forms of OP.199 patients from this number arrived at the clinic at the beginning of the disease, when the inflammatory process in the lung did not have signs of complications. At that time children with the

most aggressive forms of AP were selectively admitted to our department. The reason for the hospitalization was the fact that the surgical clinic was the only place in our area for intensive care. This group of patients differed high mortality and fast development of pleural complications. Unsatisfactory results of conventional treatment (massive doses of antibiotics, oxygen supply, intravenous infusion) forced us to find ways to solve the problem. To assess the role of intravenous infusions in the dynamics of AP following studies were performed.

Experimental research: 4 series of experiments were carried out on 44 rabbits. Model of AP was created. In the last AP series of experiments (15 animals) at the time of the development of inflammation in the lung is further carried out intravenous infusion (in 6 animals into solutions for the infusions added dye-methylene blue). In this series of experiments, all animals had pneumonia with pleurisy and was later obtained a patent for the invention [6].

Rentgenoanatomical studies of lung specimens: For this investigation 56 whole lung anatomical preparations were taken during an autopsy of children who died from pneumonia. Separate contrast lung vessels (arteries or veins) with subsequent x-ray was made on 47 specimens (in 9 cases contrasted bronchi).

Results of treatment of patients: Treatment results of children, who admitted to the clinic early in the disease without pleural complications, are of most interest to this theme. Such patients accounted for 2 groups. Patients in the first group (1976-1980;98 cases) were treated according to generally accepted standards (massive doses of antibiotics, oxygen supply, intravenous infusion). Patients in the second group (1981 - 1985; 101 cases) received treatment on the basis of new principles.

Results and Discussion

Specific treatment of each disease should focus on its unique characteristics of etiology and pathogenesis. So, the initial treatment of AP is recommended with antibiotics alone and this treatment proves as yet successful in most patients. But In this connection, it is necessary to recall the following facts.

This treatment is etiotropic only, but it is not aimed at correcting specific abnormalities in the body of the patient. The same antibiotic can be used to treat such different diseases (especially on their pathogenesis) as AP, acute pyelonephritis, tonsillitis, skin inflammation, etc. Nevertheless, the suppression of pathogens in the focus of inflammation turns in most patients sufficient to cope with the disease.

However, antibiotics alone does not solve the problem of cure all patients with AP. Part of the patients with aggressive onset or lack of effect of treatment begun is sent to the hospital. More intensive development of inflammation and progressive deterioration of these patients are forced to apply additional funds assistance. But, the results of such aid reveal her lack of efficiency: pleural effusion appears more than half of patients, and even pleural empyema develops contrary treatment [1-4]. Note that the above results characterize the work of the best hospitals and health systems. Such treatment failure is usually explained by the presence of very aggressive microflora. Then how can we explain such a fact that the bacteriological tests of pus does not reveal causative agents in many patients with empyema [7-9]? These data, from my point of view, bear witness of high reliability of modern antibiotics and indicate the need to search the causes of complications in the other direction.

Every specialist with medical education should be aware of the classical signs of acute inflammation, which centuries ago were described by Celsius (redness, swelling, heat, pain) and by Galen (loss of function). Acute pneumonia, as inflammation, has a whole set of these cardinal signs. The unique role of the lungs in the body is well known, including non-respiratory functions. Also, it is well known that the reaction of everyone to certain stimulus has individual character. Therefore, the speed of development of inflammation, the intensity of occurring disorders and the body's ability to adapt have a huge range of options. The defeat of a multifunctional organ as the lungs requires a correct view on the pathogenesis of the disease. This information helps determine the appropriate methods of therapeutic effects.

So, what is the root of the problem? In medical practice, the essence of any disease is determined by specific therapeutic actions. But in the case of diagnosis of acute pneumonia you won't find specific features in medical prescriptions. At the pre-hospital stage doctors decide only two tasks: an empirical (without establishing the etiology) recommendation of antibiotic and decision regarding the place of treatment (at home or in hospital). In the case of admission to hospital the patient is starting to get a standard (for many other diseases) intensive therapy in the form of syndromic and symptomatic relief. This approach to treatment does not take into account the specifics of the disease. The treatment in 1 group our patients was based on those principles. A typical example of the results of such treatment is one of our observations (Figure 1,2).

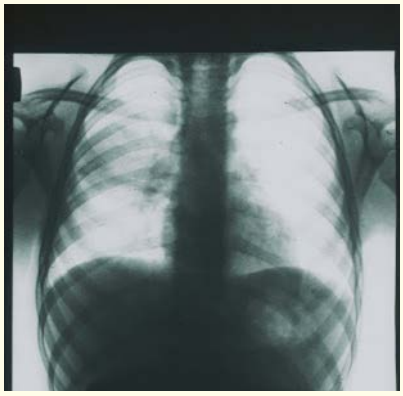

Figure 1: *X-ray photograph of 2 y.o. girl 12 hours after the first signs of AP with abdominal pain syndrome were discovered. There is homogeneous shading in a middle-right pulmonary field.*

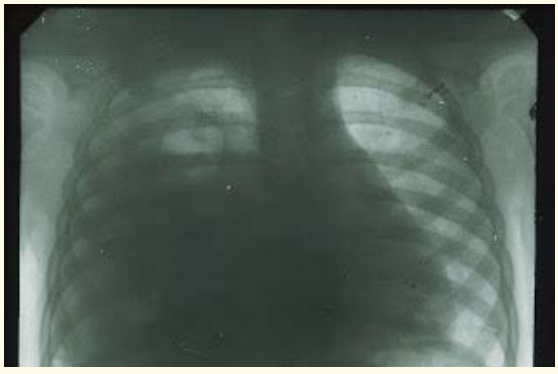

Figure 2: *X-ray photograph of the same patient (see Figure 1), 36 hours after starting the inpatient care (antibiotic therapy, intravenous infusion therapy with detoxification). There is an intensive homogenous shading of almost the whole right hemithorax with mediastinum, dislocation to the left, and a cavity with a fluid level in upper pulmonary field. (Bacteriological examination of pus from the pleural cavity revealed no microflora).*

Today is still customary to appoint infusion therapy in patients with acute pneumonia for "detoxication and replenish the fluid loss from perspiration". If inflammatory processes is localized in the system of large circulation with massive fluid loss (for example, diarrhea or peritonitis) input solutions pass through intact vessels of lesser circulation. Other conditions arise in the development of acute inflammation into the pulmonary circulation system. On the one hand, in the vessels of the pulmonary circulation unfolds vascular inflammatory response with increasing swelling and tissue infiltration, and, on the other hand, the area of inflammation turns out to be the first barrier to intravenously imposed solutions.

Our assumptions about the negative impact of intravenous infusions on the dynamics of the AP received additional confirmation in the experiment. We used the technique V Menkin who opened the permeability factor [8,9]. A dyestuff used in a solution for infusions colored the lung tissue on circumference of inflammatory zone identifying the areas where the inflammatory infiltration had spread (Figure 3). All animals with acute lung inflammation after intravenous infusions had parapneumonic pleuritis.

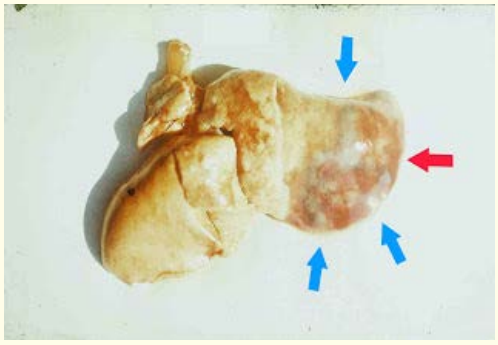

Figure 3: *Macro-preparation of the lung, experiment, series 4b. Massive focus of the inflammation in a pulmonary surface (red arrow), surrounded by the additional sections of infiltration with blue shading (blue arrows).*

Study of lung angioarchitectonics with AP showed additional reasons that reinforce the negative effect of intravenous infusions. Increased inflammatory infiltration first led to compression of more pliant venous vessels, impeding blood return from affected zone, with preserved (in initial stages of process) arterial afflux. Pathological vessel transformation in the center of AP created (in initial stages of the disease) "vessel trap" conditions, in which increased blood afflux toward inflammatory zone as a result of intravenous infusion contributed to rapid increase of alterations in pneumonic focus. Progress of these circulatory disruptions stopped the blood afflux toward inflammatory zone being the basic reason for the irreversible destructive alterations. An additional evidence of critical redistribution of the blood flow in lungs in AP was the discovery of shunting vessels on periphery of the non-afflicted parts received on 2 venograms of lung's specimens (Figure 4).

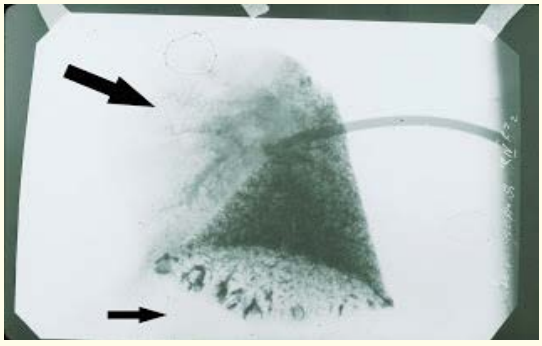

Figure 4: *Venogram of lung's preparation with a massive focus of the inflammation in the upper portion (histology which followed, did not show the features of the necrosis in inflammation's focus). Sharp depletion of the venous picture in the upper portion of the lung. Lower portion shows relatively large vascular formations of a half-ring shape with a well-contrasted venous vessels in the background.*

The false impression of the need for intravenous infusion in patients with AP create the following factors. The clinical picture of any inflammatory diseases is determined by its localization and, consequently, the value for the body of the affected organ. A direct anatomical-functional correlation between two blood circulation circles (great and lesser) are well known. Reflex influence of inflammation in the lung on the small circle of blood circulation causes the inevitable restructuring of the systemic circulation. Violation of metabolic processes is the next link after the changes in the systemic circulation. The above-described changes in the peripheral circulation correspond to the picture "shock". Therefore, homeostasis violations at AP, we were treated as "pulmonary shock". Treatment methods to eliminate those violations were named "anti-shock procedures" [5,10-13]. The characteristics and course of action of therapeutic activities allow both inhibition of the inflammatory process and its stimulation. With aggressive beginning AP, it is necessary to remove reflex spasm of pulmonary vessels and reduce blood flow to them. Intravenous infusion on the contrary increase blood flow to the lungs. That is why we have excluded from the treatment of patients with early forms of AP intravenous infusion.

The change of medical approach during the initial stage of so-called "toxic" forms of AP (use of vagosympathetic blockades, cups therapy and cold patients body wraps, escalation of bronchial drainage processes, complete abandonment of intravenous infusions and, of course, intravenous antibiotics with minimal volume of solution) highlighted the fact that once compared with an equivalent group, which had the first-aid approach based on intravenous detoxication therapy, the number of pleuro-pulmonary complications had reliably decreased (t = 8,65; P 0,001), the hospital stay declined three-fold, and there were no lethal cases [13]. To illustrate, we included roentgenograms from one of our patients (Figures 5 and 6).

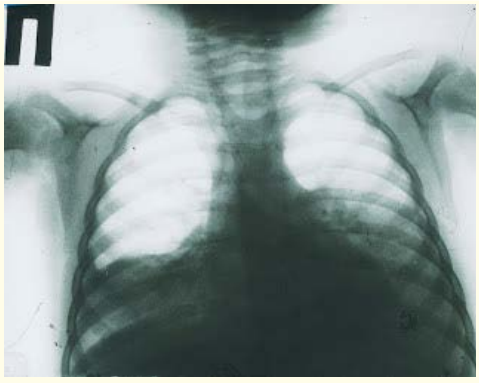

Figure 5: *X-ray photograph of a 22-month-old patient, 24 hours after starting ABP clinic. Intensive homogenous shading in basal sections of both lungs with moderate thickening of pleura in the left lung.*

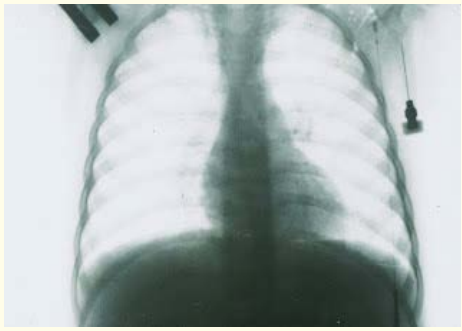

Figure 6: *X-ray photograph of the same patient (see Figure 5) five days following the clinical treatment (vagosympathetic blockades, antibiotic therapy, bronchus-draining therapy, including lavages through micro-tracheostoma). Full recovery of airiness in both lungs.*

These results are just one of the sections of the previous large studies. Currently, modern medical equipment offers a wide range of possibilities for a more detailed study of this problem.

Conclusion

Intravenous infusion during the initial period of AP are unproductive treatment. Increasing blood flow to the area of inflammation, they stimulate the processes of swelling and infiltration of tissues. The consequence of this approach to treatment is the growing number of suppurative and destructive complications of the disease.

Bibliography

1. Sibila., *et al*. "Corticosteroids in severe pneumonia". European Respiratory Journal 32.2 (2008): 259-264.
2. Seema Jain MD., *et al*. "Community-Acquired Pneumonia Requiring Hospitalization among U.S. Children". *New England Journal of Medicine* 372.9 (2015): 835-845.
3. Nicola Principi and Susanna Esposito. "Management of severe community-acquired pneumonia of children in developing and developed countries". Thorax 66.9 (2011): 815-822.
4. Mohamed A Elemraid., *et al*. "Risk factors for the development of pleural empyema in children". *Pediatric Pulmonology* 50.7 (2015): 721-726.
5. Klepikov I. "Acute pneumonia and its purulent and destructive complications in children in the midst of a major industrial centre of Western Siberia". Dissertation for the degree of doctor of medical sciences. Leningrad (1989).
6. Klepikov I and Rikov V. "Author's certificate for invention". SU, No 1631574, A1, 1 (1990).
7. Maskell NA., *et al*. "The bacteriology of pleural infection by genetic and standard methods and its mortality significance". *American Journal of Respiratory and Critical Care Medicine* 174.7 (2006): 817-823.
8. Rosenstenge A and Lee YCG. "Pleural infection-current diagnosis and management". *Journal of Thoracic Disease* 4.2 (2012): 186-193.
9. Menkin Valy. "Dinamics of inflammation". Macmillan (1940).
10. Klepikov I and Yudin J. "Shock in cases of acute bronchogenic pneumonia. A collection of articles "Clinical and anatomical aspects of disseminated intravascular blood coagulation and shock". Leningrad, 1986, Leningrad Institute for Advanced Training of Doctors (1986): 53-55.
11. Klepikov I., *et al*. "Characteristic of circulatory and metabolic lesion in cases of acute bronchogenic pneumonia in children. A collection of articles "Clinical and anatomical aspects of disseminated intravascular blood coagulation and shock". Leningrad, 1986, Leningrad Institute for Advanced Training of Doctors (1986): 68-69.
12. Klepikov I. "Anti-shocked therapy in cases of acute bronchogenic pneumonia". Medical science for practice (Heads of Scientific and Practical Congress)", Novokuznetsk (1988): 73.
13. Klepikov I. "The Meaning of Pulmonary Reflexes in the Pathogenesis of Acute Pneumonia". *Internal Medicine* 7 (2017): 232.

Volume 4 Issue 1 July 2017