ОСОБЕННОСТИ ЛЕЧЕНИЯ БРОНХИАЛЬНОЙ АСТМЫ У ДЕТЕЙ ШКОЛЬНОГО ВОЗРАСТА С РАЗНЫМИ ГРУППАМИ КРОВИ

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Резюме. Проведение комплексного обследования 56 детей, больных бронхиальной астмой, показало, что тяжелое течение астмы и интенсивность использования комбинации "ингаляционные глюкокортикостероиды + β_2-агонисты короткого действия" характерны для больных с А(II), Rh(-),MN эритроцитарными антигенами. У детей с В(III), M, Rh(+) группами крови наиболее эффективным при наличии тяжелого приступа астмы оказался комплекс: системные глюкокортикостероиды, β_2-агонисты короткого действия (сальбутамол) и эуфиллин.

Ключевые слова: бронхиальная астма, дети, группы крови.

SPECIFIC CHARACTERISTICS OF TREATING BRONCHIAL ASTHMA IN SCHOOL AGE CHILDREN WITH DIFFERENT BLOOD GROUPS

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Abstract. The performance of a complex examination of 56 children afflicted with bronchial asthma has shown a severer course of asthma and the intensity of using the combination of "inhalation glucocorticosteroids + β_2-agonists of a short-term action" inherent to persons with A (II), Rh (-), MN erythrocytic antigens. In children with B (III), M, Rh (+) blood groups the following complex turned out to be the most effective in the presence of a severe attack of asthma: systemic glucocorticosteroids, β_2 - agonists of a short-term action (salbutamol) and aminophylline.

Key words: children, bronchial asthma, blood groups

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CLINICAL CHARACTERISTICS OF THE COURSE OF HIV INFECTION WITH CONCOMITANT CRYOGLOBULINEMIA

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Abstracts. This paper describes clinical cases of HIV-infection with concomitant cryopathy at different clinical stages. The peculiarities of HIV/AIDS according to comorbidity and the effect of different modes of treatment on the phenomenon of cryoglobulinemia have been characterized.

Key words: HIV/AIDS, cryopathy, cryoglobulins, antiretroviral therapy, dipiridamol.

Introduction. Cryoglobulinemia refers to the widespread and insufficiently studied phenomena that may manifest clinically by the development of cold urticaria and angioedema, cryoglobulinemic arthrites, vasculites, etc. [5]. Until recently, its occurrence associated with a variety of factors and diseases: malignant tumors, connective tissue diseases, lymphoproliferative disease, hypothermia, dyshormonoses [11].

Even with the use of the most modern methods of examination about a third of all patients remain with cryoglobulinemia in whom the trigger factor of cryolabile proteins cannot be established. These cases are identified as "idiopathic mixed cryoglobulinemia" (IMC) or "essential mixed cryoglobulinemia" [9]. In these patients, there are no tumors, systemic connective tissue diseases, lymphoproliferative syndromes or active viral infections. As a rule, even in the case of repeated examinations, no markers of infectious processes are detected. IMC syndrome first described by M. Melzer back in 1966 with the clinical characteristics, later named Meltzer's triad: skin purpura, arthralgias and weakness. With time, the liver, kidneys, nervous system (usually peripheral) are involved in the process, relatively rarely Raynaud's and Sjogren's syndromes, diffuse vasculitis are detected [9, 13].

In some patients the symptoms of IMC may precede for several years the debut lymphoproliferative [15] or autoimmune disease [14], and this period may range from 1 to 20 years. Quite often in patients with IMC markers or specific histologic manifestations of infectious diseases, most often – viral ones, are detected upon a deeper and most detailed examination,

especially an amplificated morphologic one [9]. However, even in such cases, IGC has certain peculiarities of the course that distinguish it from typical, secondary mixed cryoglobulinemia. Typically, the symptoms of the infection process itself are obliterated, faintly marked or absent at all. The clinical presentation of the disease is dominated by organic lesions at that caused by vasculitis. The latter is formed as a result of the effect on the vascular endothelium of cryoprecipitating immune complexes [7, 8].

In recent years, a heated debate over the nature and origin of mixed cryoglobulinemia, including the idiopathic one have flared up. From the point of view of researchers, the cause of the latter may be congenital or hereditary defects in the synthesis of immunoglobulins, deficiency of trace elements, dyshormonoses or yet unknown factors nowdays. If all the above mentioned factors play a role of a background against which mixed cryopathy is formed, the main trigger factors, most researchers still believe to be microorganisms, first and foremost - viruses [10]. In cases of "an idiopathic" process the releaser role is apparently played by yet unknown viruses or pathogens whose replication at present is so weak, that it is not determined by standard methods.

In HIV-infected patients a development of a variety of clinical manifestations and laboratory phenomena is possible that occur in case of systemic in systemic rheumatic diseases. The presence of cell-specific antibodies, heat-labile proteins – cryoglobulins (CGs) (more often – with concomitant hepatitis C), increasing concentrations of acid-labile interferon-α is described[12]

So it is assumed that CGs have an important pathogenetic role in the progression of HIV infection [13]. They differentiate 3 main types of CGs: monoclonal (type 1) complexes of mono-and polyclonal (2^{nd}) and polyclonal ones (3^{rd}). The last two types are often called mixed. CGs of type-1 are predominantly detected in some versions of malignant tumors and lymphoproliferative diseases, and most cases of CGs of the 2^{nd} and 3^{rd} types (mixed) are explained by the influence of viruses – primarily lymphotropic and hepatotropic [4]. The frequent causes of the formation of thermolabile proteins are often believed to be the viruses of human immunodeficiency, hepatites (especially C), herpes, cytomegalovirus, Epstein-Barr virus and others. It is believed that the appearance of the body CG is the result of an antigenic stimulation by direct or indirect effects of an infectious agent. In addition, these proteins may appear in the body at the secondary phase of the immune response in response to infecting or to an inflammatory process that occur in the body [6].

The aim of the study was to investigate the specific characteristics of cryopathies of different types in different clinical periods of HIV-infection and cryoglobulinemia during a course of antiretroviral therapy.

Material and methods. The study involved 127 patients with HIV infection who were on an ambulatory treatment during 2008-2011 in the Regional Center for Prevention and Fight against AIDS of Chernivtsi. Among these patients there were 66 men and 61 women aged from 19 to 44 years.

Study results and discussion. We have found out that during the I^{st} clinical stage of HIV-infection in almost half of patients cryoglobulins are detected, most of which are mono-and polyclonal (the 2^{nd} type).

With the progression of HIV infection the cryoprecipitation phenomenon becomes more characteristic: we found CGs of type-2 in an absolute majority of HIV-infected persons of the II clinical stage, and CGs of the 3-type – in almost every third patient at clinical stage III CGs of type 2 and 3 were found approximately equally frequently in all the patients.

Our data are consistent with bibliographical published reports, according to which patients with HIV-infection/AIDS have cryoglobulins which consist of polyclonal IgM, IgG or IgA (IgM possesses the rheumatoid factor activity) [2]. Such a composition of cold IgGs is typical of cryoglobulinemia of the 2^{nd} and 3^{d} type [3].

Cryoglobulinemia syndrome characterized by symptomatology polymorphism, has much in common with the clinical manifestations of HIV-infection. In particular, it concerns a prolonged subfebrile temperature, meteorological dependence, poor perception of cold, myalgias and arthralgias, lymphadenopathies, vegetative-vascular crises and others [1].

So we noticed that patients with HIV-infection with cryoglobulins in the blood developed more frequently acrocyanosis, poor perception of cold, lymphadenopathy and myalgias, i.e., the symptoms that are common manifestations of cryoglobulinemia.

It is logical to assume that cryopathies can influence on the course of HIV-infection first and foremost, due to the ability of CGs reversibly or irreversibly bind virions. Thus, cryoprecipitates of type 2, because of their stability, in the peripheral vessels do not dissolve in case of a subsequent rise of temperature. They partially bind HIV, and bringing it to the "periphery", probably, slightly reduce its replicative activity. At the IV^{th} clinical stage of HIV-infection, apparently, with the onset of a decompensation of many organs and body systems the frequency of the registration of CG decreases almost by half. Thus, one can recognize an adaptive, protective reaction, at the bases of CG of type 2, attempts to "withdraw" the virus from replication sites and block it in practically insoluble cryoprecipitates.

In view of this one can explain a fact revealed by us of significantly rarer detection of cryoglobulinemic cryopathy (only in isolated patients) at the stage of wasting syndrome due to HIV-infection (the IV^{th} clinical stage). We assume that at the terminal stage of HIV-infection due to the development of systemic decompensation of organs and systems, cryoglobulinemia loses its adaptive mechanism.

It is likely that the same phenomenon explains a direct correlation of the mean force established by us (r=0,58) between the level of CD4 +-lymphocytes in the range of "500 – 200 in 1 mm^3 of blood" and the frequency of detecting CG and average force inverse correlation (r=-0,53) between the level of CD4 +-lymphocytes <200 in 1 mm3 of blood" and the frequency of detecting CG.

So cryoglobulinemia is a frequent complication of HIV infection that must be considered in the algorithm of treatment of such patients.

It has been found out that as a result of a 3-month course of HAART, the number of patients with CG decreased from 96 (75,6±3,8 %) to 63 (49,6±4,4 %) patients (p<0,001). Thus, with concomitant cryoglobulinemia of type-2, the standard first-line HAART not only increases the level of CD4 +-lymphocytes and, obviously, reduces the replication of the virus, but also eliminates cryoglobulinemia in a half of the cured patients (59 – 46,5±4,4 % to 30 – 23,6±3,8 %) (p<0,001). A combination of standard first-line HAART with an additional use of antiplatelet agent-dipiridamol allows to slightly decrease the percentage of patients with the phenomenon of cryoprecipitation.

However, in AIDS patients with cryoglobulinemia of the 3rd type the use of antiretroviral drugs (zidovudine + lamivudine + Efavirenz) and dipiridamol was less effective (before the start of treatment CG was in 33 persons – 26,0±3,9 %, and after 3 months of treatment – in 31 patients – 24,4±3,8 %) (p>0,05) and is accompanied with a frequent apposition of AIDS-associated infections.

We present the findings of our clinical observations. Patient S., 34 years old, sought a medical advice at the Chernivtsi Regional Center for Prevention and Control of AIDS, complaining of general weakness, drowsiness, fatigue, weight loss by 4 kg per month, fever up to 37,5ºC, liquid stools more than 2 times per diem for two months, loss of memory, cooling and numbing extremities. She had to spend in bed up to 50 % of the daytime because of weakness.

From the past medical history it is known that the diagnosis of HIV infection confirmed in 2005 simultaneously with ex-husband patient – an active intravenous drug abuser.

On a physical examination: the nutritional state is diminished, shadows under the eyes, the skin is pale, dry – seborrheic dermatitis, T – 37,5ºC, cold urticaria on both hands. Candidiasis of the mucous membranes of the mouth (thrush) onychomycosis of the feet. The submandibular, cervical, occipital lymph nodes are enlarged, tender on palpation, dense. The cardiovascular system: P - 80/min, muffled heart sounds. BP – 110/70 mm Hg. Auscultation of the lungs – vesicular breathing, no rales. The abdomen is tympanic, diffuse moderate tenderness on palpation. No hepatosplenomegaly. Fluid excrements, 2-4 times per diem during last 2 months. Clinical diagnosis – HIV, III clinical stage. Clinical blood count: erythrocytes – $3,85\times10^{12}$ /L-1, hem. – 118 g/l, CI – 1.0; Leuk. – $3,9\times10^{9}$ L-1, eos. – 2 %, b. – 8 %, s. – 65 %, lymph. – 17 %, mono. – 8 %, ESR – 19 mm/h. Immunological studies: lymphocytes – $0,30\times10^{9}$ L-1: 0 lymphocytes – 44,9 %, T total – 39,3 %, incomplete T – 19,5 %, T "active" – 12,2 %, helpers – 23,4 % suppressors – 13,4 %, D-cells – 0,8 %, B cells – 4,2 %, IgA – 0,66 g/l, IgM – 3,5 g/l, IgG – 4,9 g/l, CIC – 126 units., lysozyme – 2,3 mg/l. The number of CD4+-lymphocytes measured by flow cytometry made up 260 cells in 1 mm^3 of blood.

On a coagulogram investigation: a prolongation of the prothrombin time, recalcification time. CG of type 2 was diagnosed.

The patient was prescribed first-line antiretroviral drugs. In 3 months of this therapy her general condition essentially improved, the appetite restored, she put 3 kg on weight. But she keeps on complaining of rapid fatigability, cooling and numbing of limbs, dizziness, and cold urticaria.

On a physical examination: lymphadenopathy retains, but the body temperature during a 24-hours period does not rise above 36,9º C. Pulse – 72/min, muffled heart sounds. BP is 120/70 mm Hg. Auscultation of the lungs – vesicular breathing, no rales. The abdomen is soft, not tender; the liver and spleen are not enlarged on percussion and palpation. CG of type 2 are continue to be detected. She is receiving previous treatment.

Patient P., 29 years old, appealed for medical advice to the Regional Center for the Prevention and Control of AIDS, complaining of a significant general weakness, rapid fatigue, progressive haggardness, poor appetite, intermittent diarrhea, hands going blue on their least cooling, cooling and numbing limbs, apathy.

From the past medical history we learn that the diagnosis of HIV-infection was confirmed in 1999. He was an active intravenous drug abuser.

On an objective examination: diminished well-nourished condition, sunken eyes, blue shadows under the eyes, acrocyanosis, extensive onychomycosis. The skin of the hands is cyanotic, cutis marmonata on both thighs and legs. The body temperature – 36,6 ºC. The submandibular, cervical, occipital lymph nodes are enlarged, tender on palpation, dense. The cardiovascular system: P – 80/min, muffled heart sounds. BP – 110/70 mm Hg. Auscultation of the lungs – vesicular breathing, no rales. The abdomen is soft, tender on palpation. The liver on palpation bulges out at a distance of 2 cm from under the edge of the costal margin, tender. The splin is not palpated. The excrements are fluid, 4-5 times per diem during 3 months. Clinical diagnosis – HIV, III clinical stage.

CBC: erythrocytes – $3,6\times10^{12}$ /L-1, hem. – 92 g/l, CI – 0.8; leuk. – $4,8\times10^{9}$ L – 1, eos. – 1 %, b. – 3 %, s. – 70 %, lymph. – 15 %, mono. – 11 %, ESR – 8 mm/h. Biochemistry: bilirubin 23 – μmol/L, ALT – 0,84 mmol/(L×h), AST – 0,78 mmol/(l×h). Immunological investigation: lymphocytes – $0,27\times10^{9}$ L-1: 0 lymphocytes – 46,4 %, T total – 40,2 %, incomplete T – 19,8 % T "active" – 12,6 %,

helpers – 22,8 %, suppressors – 12,9 %, D-cells – 0,8 %, B cells – 4,3 %, IgA – 0,60 g/l, IgM – 3,2 g/l, IgG – 4,3 g/l, CIC – 115 units., lysozyme – 2,9 mg/l. The number of CD4+-lymphocytes, measured by flow cytometry, made up 240 cells in 1 mm^3 of blood.

On a coagulogram examination: a considerable prolongation of the prothrombin time and recalcification time. CG of type 2 was diagnosed.

The patient was administered first-line antiretroviral drugs and dipiridamol 75 mg in a single dose at night once every 2 circadian periods for 3 months. In 3 months of such therapy his condition significantly improved: his appetite restored, cold urticaria disappeared, numbness of the extremities did not trouble, diarrhea almost calmed down. As a result of improved appetite and a normalization of evacuation the body weight increased by 3 kg.

The objective status is indicative of a normal body weight, the preservation of general lymphadenopathy, the disappearance of acrocyanosis, cold urticaria. The pulse – 74/min. The cardiac sounds are muffled heart tones. The arterial pressure is 110/70 mm Hg. On auscultation of the lungs – vesicular respiration, no rales. The abdomen is soft, not tender. The inferior border of the liver is palpated 1 cm below the costal margin, elastic and not tender. The spleen is not palpated.

CBC: erythrocytes – $4,0\times10^{12}$ /L-1, hem. – 105 g/l, CI – 0,9; leuk. – $5,5\times10^{9}$ L-1, eos. – 0 %, b. – 6 %, s. – 56 %, lymph. – 21 %, mono. – 17 %, ESR – 11 mm/h. The biochemical blood analysis did not practically change: bilirubin 21 – μmol/L, ALT – 0,78 mmol/(l×h), AST – 0,75 mmol/(l×h). Immunological studies are indicative of an increase of the number of lymphocytes – up to $0,81\times10^{9}$ L-1: 0 lymphocytes – 28,2 %, T total – 45,6 %, incomplete T – 18,0 %, T "active" – 18,4 %, helpers – 24,7 %, suppressors – 12,5 %, D-cells – 2,2 %, B cells – 7,3 %, IgA – 0,76 g/l, IgM – 3,4 g/l, IgG – 4,2 g/l, CIC – 100 units, lysozyme – 3,2 mg/l. The number of CD4 +-lymphocytes, measured by flow cytometry, increased to 410 cells in 1 mm^3 of blood.

Conclusions

It is known that type 3 cryoprecipitates are more heat-labile, compared with type 2. In an insoluble form, they are not able to consistently block virions, but only protect them from the effects of the immune system and etiotropic medications. In case of a rise of temperature type 3 cryoprecipitates partially dissolve. A significant part of virions released, at that and undessolved components of a cryoprecipitate flow back into the systemic circulation and block the blood vessels of the microcirculation, causing stable rheological and microcirculatory disorders, thereby, obviously, provoking the development of vasculites, Raynaud's syndrome or arthropathies.

Thus, the phenomenon of cryoglobulinemia, which is the result of the HIV action, has an opposite effect on the course of viral infection and determines its characteristics.

Thus, a significant impact on the course of HIV-infection is, to a considerable extent, exerted by concomitant cryoglobulinemia that must be considered, when diagnosing illnesses and treating such patients. In particular, a combination of standard HAART based on the first line regimen (zidovudine + lamivudine + Efavirenz) with an additional use of the antiplatelet agent-dipiridamol makes it possible to lower somewhat the percentage of patients with the phenomenon of cryoprecipitation.

Prospects for further research. To study the role of the endothelium in the pathogenesis of HIV-infection.

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КЛИНИЧЕСКАЯ ХАРАКТЕРИСТИКА ТЕЧЕНИЯ ВИЧ-ИНФЕКЦИИ С СОПУТСТВУЮЩЕЙ КРИОГЛОБУЛИНЕМИЕЙ

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Резюме. В статье описаны клинические случаи ВИЧ-инфекции с сопутствующей криопатией в различных клинических стадиях. Охарактеризованы особенности течения ВИЧ/СПИДа в зависимости от сопутствующей патологии и влияния различных методов лечения на феномен криоглобулинемии.

Ключевые слова: ВИЧ-инфекция/СПИД, криопатия, криоглобулины, антиретровирусная терапия, дипиридамол.

КЛІНІЧНА ХАРАКТЕРИСТИКА ПЕРЕБІГУ ВІЛ-ІНФЕКЦІЇ ІЗ СУПУТНЬОЮ КРІОГЛОБУЛІНЕМІЄЮ

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Резюме. У статті описано клінічні випадки ВІЛ-інфекції із супутньою кріопатією у різних клінічних стадіях. Охарактеризовано особливості перебігу ВІЛ/СНІДу залежно від супутньої патології та впливу різних методів лікування на феномен кріоглобулінемії.

Ключові слова: ВІЛ-інфекція/СНІД, кріопатія, кріоглобуліни, антиретровірусна терапія, дипіридамол.

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PHARMACOLOGICAL CORRECTION OF ENDOTHELIAL DYSFUNCTION IN PATIENTS WITH HIV-INFECTION/AIDS

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Abstract. On the basis of inspection 127 patients with HIV-infection/AIDS it has been established that in case of this pathology the concentration of thrombomodulin, E-selectin and von Willebrand's factor substantially grows, which is indicative of a HIV-induced affection of the vascular wall. As immunodeficiency progresses, the concentration of all the mentioned indices grows significantly.

3-month symptomatic therapy does not influence on the state of the endothelium. The use of the dipiridamol, aggregant, and also a 3-month antiretroviral treatment of the first row provide only a partial decline of the thrombomodulin, E-selectin and von Willebrand's factor levels.

However, the inclusion to the antiretroviral therapy (ART) of dipiridamol maximally optimizes the endothelial state of the endothelium: the level of thrombomodulin and von Willebrand's factor at the IInd clinical stage of HIV-infection normalizes and at the III^{d}-IVth stages – significantly lowers, although it does not reach the values of healthy persons. The same ponderable difference is established in relation to the content of E-selectin.

Key words: HIV/AIDS, endothelial dysfunction, treatment.

Introduction. Special attention of scientists has been focused on the role of the endothelium in the pathogenesis of HIV infection lately. Locally, the endothelium forms the wall of the hepatic hemocapil-