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MODERN ASPECTS OF ETIOLOGY AND PATHOGENESIS OF HERPES ZOSTER

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Annatation
Herpesvirus infections are one of the most important problems of modern medicine. Last 20th anniversary there has been a steady increase in diseases due toherpes viruses. New herpesviruses have been identified that can cause disease in humans. Given the exceptional medical and social severity of the problem herpetic infection, the huge economic costs of its treatment, currently in the leading clinics of the world, intensive scientific searches are underway for the causes and key links in the pathogenesis of the disease, since without this, it is impossible to develop adequate methods therapy.

Keywords: Herpes zoster, etiology, shingles, belt of roses from hell

Introduction

Among newly discovered infections in recent decades with proven nosological and etiological independence, herpesviruses are the most relevant [1]. It is known that about 90% of the world's population is infected with herpesviruses, 2-12% suffer from recurrent forms of these diseases [3]. In 2002, WHO declared a pandemic of herpes infections (HI) in the world. One of the most common GI is herpes zoster (SH), which manifests itself as a general infectious syndrome, the occurrence of limited or generalized rashes, damage to the nervous system, and often other systems and organs [2]. Rashes are usually preceded by severe pain and hyperesthesia, in connection with which in the English literature herpes zoster is figuratively called “belt of roses from Hell” (“belt of roses from hell”) [9,11]. Herpes zoster has been known for a long time. Even the ancient Greek scientists Celsus (1st century BC) and Pliny (23-79) gave its first descriptions in literature. The disease got its name from the appearance of the rash. It reflected two significant features of skin manifestations: 1) the peculiar location of the rash - one-sided, with a different number of foci, which, merging, form a continuous tape (zoster - belt); 2) the spread of the rash in the dermatome has a multi-temporal character (herpes - creeping). It should be noted that the most common name is "shingles" [5, 8]. The term "lichen", used until the

middle of the XIX century. to refer to dermatoses of various nature, essentially limits the clinical concept of the disease as a local skin disease and, moreover, does not provide an adequate translation of the Latin term "herpes" into Russian ("lichen" comes from the Greek word lichen - lichen). In countries with an alphabet that arose on the Greek-Latin basis, as a rule, the Latin term "Herpes zoster" is used. In Anglo-American medical literature one can find the name of the disease "Shingles", in French - "Zona". In recent years, Russian authors have used the terms "zoster", "herpes zoster" in Russian or Latin transcription. The evolution of terminology naturally led to the emergence of a new nomenclature - "herpes zoster", which eliminates the noted shortcomings, and most importantly, takes into account the historical experience of studying the etiological aspects of this nosological form, which radically changed the content of the concept of "herpes". The causative agent of OH is the varicella-zoster virus (VZV), belonging to the Herpesviridae family. It was discovered in 1911 by the Brazilian scientist X. Aragao, who discovered small formations - elementary bodies - in the contents of the vesicles of patients. It was first isolated in tissue culture in 1953 by Weller. VVZ has a large virion (visible under light microscopy) containing linear double-stranded DNA. The capsid (shell containing the nucleic acid) contains 162 capsomeres. There is an additional glycoprotein shell - super-capsid. The virus quickly dies when exposed to ultraviolet rays, disinfectants, heating (at 50-60 degrees, inactivation occurs after 30 minutes). However, it can be stored for a long time at low temperatures, withstands repeated freezing. It has a close antigenic relationship to the herpes simplex virus. For animals it is not pathogenic. There is no own metabolism. Metabolism is associated with the cell in which VVZ parasitizes. A characteristic feature is a short reproduction cycle, as well as a cytopathic effect. In this case, due to the destruction of 131 infected cells, multinuclear symplasts are formed. VVZ infection in most cases occurs in childhood and proceeds with the clinical picture of chicken pox (VO). After that, the pathogen is not eliminated from the body; a latent phase begins, characterized by the persistence (from English persist - persist) of viruses in the cranial and spinal sensory ganglia [6,7]. Since the manifestations of VO are disseminated, the virus occupies the ganglia of all levels of the spinal cord, as well as the nodes of the cranial nerves - trigeminal, facial. OH arises as a result of reactivation of VVZ, which persists in the human body in satellite cells of the spinal and cranial nodes. Perhaps this is due to the presence of severe pain in OH. Thus, children who recovered from VO are potential patients with OH. According to this, OH is a secondary endogenous infection that develops as a result of virus reactivation against the background of immunological failure [4]. The first reports on the relationship between VO and OG appeared in the second half of the 19th century (Moor, 1852,

Bokey, 1888). Further, in 1926, at the Brussels Congress of Dermatologists, Flandin substantiated the point of view that these diseases are caused by the same virus. In the historical aspect, these are the main stages in the presentation of VVZ - infection as a kind of two-stage process. The identity of the pathogen VO and EG was experimentally established only in 1953 by T. Weller and H. Witton. The latent phase of infection (the interval between the primary infection and the manifestation of OH) usually lasts for decades, but in some cases it can be very short - only a few months [10,14]. The persistence and reproduction of the virus in infected cells does not always lead to the death of the latter, as is noted in acute infections caused by many DNA and RNA viruses. This was facilitated by the evolution of views on this disease, its recognition as an acute generalized neuroinfectious process, in contrast to the previously existing ideas about it as a purely local phenomenon [15]. The presence of this disease indicates a premorbid decrease in immunity. Numerous studies point to the relationship between the occurrence of OH and various immunological disorders. The most common disease occurs among the elderly and senile age. In addition, an increase in the frequency of its occurrence and the severity of the course in patients with malignant neoplasms was established. And, finally, in the literature there are indications of VZV activation in patients with secondary immunodeficiency due to the use of corticosteroids and immunosuppressants after organ and tissue transplantation [13]. Studies on GI have shown that the nature of immunopathogenetic changes in patients is largely due to the possibility of integrating the virus genome into the genome of the host cell, as well as the affinity of herpesviruses to blood cells and immunocytes. This causes the inferiority of the immune (predominantly cell-mediated) response in these patients. It is known that antiviral protection includes two types of reactions to infection: 1 - natural (nonspecific) or congenital; 2 - adaptive (specific), or acquired. Both types are extremely important and complement each other. The immune response always begins with non-specific reactions. Thus, the body's first line of defense against viruses is represented by natural immunity. Conventionally, this process is divided into three active phases. In the first of these, the virus is attacked by proteins such as complement, preexisting natural antibodies of class M (Ig M) and antimicrobial peptides. In the second phase, interferons come into play. Finally, in the third phase, antiviral activity is shown by effector cells: neutrophils, macrophages, and natural killers. The above factors exist in the body even before infection or are activated immediately after it. They do not have a clear specificity and protect the body in the early stages of the fight against the pathogen, when the acquired immunity reactions have not yet formed. This is a separate population of cytotoxic lymphocytes that have

the ability to destroy cells infected with viruses. The interferon system exists in every cell, is comparable in weight to the immune system, and surpasses it in versatility. Two types of interferons are currently known. Interferons alpha and beta belong to type I, gamma - to type II. They differ in origin and properties. Interferons are secreted by all infected cells, are formed quickly, and serve as an express defense. Ultimately, they prevent the reproduction of virions in neighboring cells, thereby fulfilling their paracrine function. Moreover, type G interferons enhance the cytotoxicity of natural killers. The latter, together with activated T-lymphocytes, produce interferon. An important role in natural defense also belongs to macrophage cells, which provide phagocytosis and presentation of viral antigens [3,8]. Due to the activation of the innate immune system, viruses are rebuffed from the moment of infection, and the body prepares the information ground for the subsequent implementation of the acquired immune response [2]. In addition, this dermatosis in severe cases can be considered an HIV-indicator, as well as a marker of oncoprocesses. In 40% of patients with OH, the problem of chronic pain remains unresolved. Ophthalmic OH is the cause of damage to the organ of vision with possible clouding of transparent media, and the development of irreversible blindness. In recent years, there have been indications of the development of cerebral angiitis with delayed contralateral hemiparesis, Guillain-Barré demyelinating polyradiculopathy, and vasculopathy.

Conclusion

And, finally, at present, along with other herpesvirus infections, OH is considered to be associated with diseases of "civilization", in particular.

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