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LATENT VIRAL INFECTIONS IN BURNED PATIENTS WITH DEEP BURNS, STATE OF THE PROBLEM

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Abstract. This review presents an analysis of the treatment of patients with extensive deep skin burns, since it is the severity of the burn injury that is the main condition for the development of immunosuppression, the activation of latent viral infections, which leads to severe complications of burn disease. Over the past 3 years, all medical institutions have encountered a new coronavirus infection SARS-CoV-2. In the burn departments, it was characterized by specific changes in the immune status. The purpose of this review was to summarize data on the effect of various types of latent viruses on the course of burn disease in patients with extensive deep burns. In the course of the work, the features of the impact on the course of the wound process and the results of skin plasty of the human herpes virus family, varicella-zoster virus, cytomegalovirus, and Epstein-Barr viruses were analyzed. It has been established that the manifestations of a viral infection are diverse, the effect on the body of a severely burned person is multifaceted, and the information received from various authors is contradictory. Given the annual increase in the number of such victims being treated in intensive care units, active screening studies are required to identify viral infections and determine their impact on the course of burn disease.

Key words: burns; deep burns; immunosuppression; viruses; herpes virus; chickenpox; cytomegalovirus; Epstein–Barr virus.

ЛАТЕНТНЫЕ ВИРУСНЫЕ ИНФЕКЦИИ У ОБОЖЖЕННЫХ С ГЛУБОКИМИ ОЖОГАМИ, СОСТОЯНИЕ ПРОБЛЕМЫ

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Резюме. В данном обзоре представлен анализ особенностей лечения пострадавших с обширными глубокими ожогами кожи, так как именно тяжесть ожоговой травмы является основным условием развития иммуносупрессии, активации латентных вирусных инфекций, что приводит к тяжелым осложнениям ожоговой болезни. За последние 3 года с новой коронавирусной инфекцией SARS-CoV-2 столкнулись все медицинские учреждения. В ожоговых отделениях она характеризовалась специфическими изменениями иммунного статуса. Цель настоящего обзора — обобщить данные о влиянии различных видов латентных вирусов на течение ожоговой болезни у пострадавших с обширными глубокими ожогами. В ходе работы проанализированы особенности влияния на течение раневого процесса и результаты кожной пластики семейства вирусов герпеса человека, вируса ветряной оспы, цитомегаловируса, вирусов Эпштейна–Барр. Установлено, что проявления вирусной инфекции многообразны, влияние на организм тяжелообожженного многогранно, а сведения, поступающие от различных авторов, противоречивы. Учитывая ежегодный рост количества таких пострадавших, находящихся на лечении в отделениях интенсивной терапии, требуется активное проведение скрининговых исследований для выявления вирусных инфекций и определения их влияния на течение ожоговой болезни.

Ключевые слова: ожоги; глубокие ожоги; иммуносупрессия; вирусы; вирус герпеса; ветряная оспа; цитомегаловирус; вирус Эпштейна–Барр.

According to the World Health Organisation (WHO), 2.9 million burn victims are hospitalised annually worldwide and 176,000 of them die [22]. In this review, the main emphasis is placed on analysing the peculiarities of treatment of victims with extensive deep skin burns, since the severity of burn injury is the main condition for the development of immunosuppression, activation of latent viral infections, which leads to severe complications of burn disease [12, 27]. In our opinion, viral infections in severely burned patients should be divided into three groups: the first group is latent viral infections (human herpes virus family); the second is blood-borne infections; the third is a new coronavirus infection COVID-19. Data on the course of burn disease in combination with blood-borne infections (hepatitis B and C, immunodeficiency virus) have not been studied. Over the past 3 years, the novel SARS-CoV-2 coronavirus infection has been a real challenge for all healthcare settings, including burn units. It has been characterised by specific changes in immune status, but the data require analysis and synthesis.

This review analyses data on the influence of different types of latent viruses on the course of burn disease in burn victims with extensive deep burns.

The weakened organism of burn victims is susceptible to various infections — bacterial, viral and fungal, which may be exogenous, endogenous or opportunistic [27]. Burn disease is a complex set of symptoms that develops in thermal trauma accompanied by burn shock, affecting the immune status of the victim. The main changes in immunity are manifested by a decrease in neutrophil activity and excessive release of cytokines and growth factors, which leads to the development of a pronounced uncontrolled systemic inflammatory response. The authors note that along with bacterial infections, viral infections are also one of the causes of fatal outcome among victims with extensive deep burns [8, 12, 24, 27, 51]. Bacterial and viral infections are the most common cause of burn disease complications such as pneumonia and septicemia [26, 71]. According to authors, phenotypic changes in T-cells develop under the influence of burn injury, which causes post-burn immunosuppression [8, 36, 75]. Researches have shown that suffered complications of burn disease in the form of sepsis and pneumonia enhance the hypermetabolic response, leading to profound impairment of immune responses within two years after burn injury [24]. Prevention of viral infections is a way to reduce the number of both early and late complications of burn disease.

The human herpes virus (HHV) family, which includes herpes simplex virus type 1 (HSV-1), herpes simplex virus type 2 (HSV-2), varicella zoster virus (VZV), cytomegalovirus (CMV), Epstein-Barr viruses (EBV) and human herpes virus types 6 to 8, is the most common in the human population. Initial contact with infection leads to the production of immunoglobulin G (IgG) against the virus [51, 56]. The overall seroprevalence rate for herpes simplex virus type 2 in the United States is 21.9 %, according to an analysis of more than 13,000 serum samples [19]. In Australia, a study showed seroprevalence of 75 % for HSV-1 and 12 % for HSV-2 in the adult population [11]. The seroprevalence of CMV was found to be 50.4 % [3]. About 95 % of adults acquired immunity against varicella-zoster virus infections in their childhood [15, 21].

Fever and blistering of skin grafts have been reported in patients with herpes simplex virus infections [7]. Other authors have reported a longer duration of respiratory support in burned patients infected with herpes simplex virus and a

high risk of bacterial infections [56]. A correlation between the duration of hospitalisation and the occurrence of viral infections has been described [34], suggesting that a more severe burn injury with a longer length of hospital stay increases the risk of viral infection in burned patients. Most studies suggest that there may be a causal relationship between herpes simplex virus infection during hospitalisation and prolonged hospital stay. The correlation between mortality and viral infection in patients with extensive deep burns was studied based on data from a pathological study of 54 patients with severe thermal trauma. According to the authors, herpes virus infection of lung tissue was closely associated with acute respiratory distress syndrome and subsequent mortality [7, 18, 44, 47].

Herpes simplex viruses types 1 and 2 (HSV-1 and HSV-2) are a group of enveloped viruses containing a relatively large genome of double-stranded DNA and having a short reproductive cycle [39]. The prevalence of HSV-1 and HSV-2 infections increases with patient age [60]. The prevalence of HSV-1 varies from 28 to 60 % depending on age. The prevalence of HSV-2 ranges from 0.8 % in the young to 21 % by age 50 years [49]. Herpes simplex viruses persist asymptomatically in the sensory ganglia of the autonomic nervous system for long periods of time [69]. Recurrence of HSV infection can be triggered by severe trauma or other strong stimulus [4].

HSV reactivation mainly occurs in adult patients who have been previously infected with herpes, whereas primary infections are more common in children and usually have a more aggressive course and longer duration. Typically, HSV-1 mainly affects the oral cavity, whereas HSV-2 mainly affects skin sites in burned patients with reduced immunity [52, 61].

According to the authors' observations, clinical manifestations of herpes simplex virus infection are most often observed on donor sites in the form of blisters with serous contents rather than directly in the burn wound [46]. Clinical manifestations of HSV in the skin can also occur in superficial recently healed burns. In this case, the wound process in burn wounds is significantly prolonged, the treatment period increases, and the functional and aesthetic outcome deteriorates [56]. HSV infection manifestations occur in burns of the upper extremities and trunk [7], and most herpetic infections associated with burn wounds are more common in males [23].

The disorders associated with HSV infection are accompanied by impairment predominantly within the suppressor T-lymphocyte subpopulation, characterised by reduced T-lymphocyte population, decreased lymphocyte sensitivity to mitogenic and antigenic stimuli, as well as reduced interleukin-2 release and abnormal antibody production [72]. The most common clinical manifestations of herpes simplex virus infection occur in extensive deep burn wounds, while small wounds are less susceptible to it. In 2017, it was found that the manifestations of herpes simplex virus infection are quite common in cases where the total burn wound area is more than 53 % of the body surface area [70]. The severity of herpes simplex virus infection is also unpredictable as it can range from mild symptoms such as vesicular skin rash to even severe necrotising hepatitis or encephalitis with fatal outcome [17, 50, 54, 64].

Morphologically, HSV infection is manifested by a group of vesicles or vesicopustules in the burn area and usually occurs in the first three weeks after injury. Herpes simplex virus reactivation can occur either in the burn area or manifest systematically. In addition, asymptomatic reactivation is also quite common and should be tested and confirmed by appropriate laboratory tests. Burns of the face or neck usually result in reactivation of HSV infection located in the trigeminal ganglion [48]. Reports of HSV-associated disease in burn patients in the form of tracheobronchitis and pneumonia have been reported in the literature [58]. It has been shown that 52 % of patients with extensive deep burns had a significant increase in anti-HSV immunoglobulin G (IgG) titres to herpes viruses in general [34]. Approximately 2 weeks after deep burns in the head and neck region, 15 % of ventilated burn victims developed a facial rash due to herpes simplex virus infection [18]. In 2017, a case of a fatality in a burned woman due to diffuse hepatitis associated with herpes simplex virus that ended in massive liver necrosis was reported [10]. Such complications are relatively rare and require liver biopsy for confirmation [55]. A case has also been described in which HSV-2 infection was associated with severe pneumonitis, tracheitis and focal necrotising hepatitis in a severely burned patient [51]. In 2008, a diagnosis of HSV-1-induced encephalitis (laboratory-confirmed) was described in a 43-year-old man with extensive deep flame burns over 65 % of the body surface area [6]. According to the literature, quite often rash elements such as vesicles or vesiculopustules may be diagnosed wrong and treated as impetigo, eventually leading to much more serious complications of HSV infection [67]. As early as 1996, it was demonstrated that 50 % of burn victims were found to have the presence of herpes simplex virus in lung tissue, and 16 patients developed acute respiratory distress syndrome, among whom 13 were infected with herpes simplex virus [8, 32]. The most severe complications of herpes simplex virus infection, such as hepatitis, liver necrosis, pneumonitis, tracheitis, encephalitis or acute respiratory distress syndrome, are associated with reactivation of latent HSV infection, which is caused by burns [66]. According to other authors, there is no statistical correlation between

active HSV infection and increased morbidity and mortality in burn patients [71].

Clinical diagnosis of HSV infection is difficult because of the lack of specific symptoms. In severely burned patients, laboratory diagnostic methods should be used even if viral infection is suspected because of the possibility of asymptomatic infection with *Herpes simplex* virus. There are many methods for its detection [56]. PCR is currently the "gold standard" for the diagnosis of *Herpes simplex* virus infection [9, 63]. Fluorescence *in situ* hybridisation (FISH) and next generation sequencing (NGS) are also used to diagnose HSV infection [30, 42, 45]. The detection of intranuclear eosinophilic inclusions (Cowdry bodies type A) allows rapid confirmation of the diagnosis. The presence of type A Cowdry bodies is also characteristic of varicella zoster virus (VZV) infection [73]. The fastest and most accurate diagnosis of *Herpes simplex* virus allows timely initiation of treatment, which reduces the duration of hospitalisation and the development of complications. Treatment of HSV infection is started with Acyclovir; Ganciclovir or Foscarnet are also used [74].

Cytomegalovirus is an enveloped virus that causes mononucleosis and pneumonia in humans [28]. Infection with this virus occurs during childhood [13, 25, 62, 68]. The severity of cytomegalovirus infection varies considerably from mild symptoms to severe manifestations including pneumonia, encephalitis, hepatitis, colitis or retinitis [1, 41]. However, no statistically significant effect of cytomegalovirus infection on mortality in severely burned patients has been found, according to the authors [43, 53]. In adult burns, cytomegalovirus infection occurs as a result of reactivation of latent infection or as a result of primary infection. The latent period occurs in the bone marrow, in monocyte/granulocyte progenitor cells [5]. Viral reactivation and further replication occur in hematopoietic cells [20]. The incidence of reactivation of cytomegalovirus infection in burn victims ranges from 55 to 71 % [59]. Primary and reactivated cytomegalovirus infection can be differentiated by anti-CMV-IgG and microneutralisation [2]. Due to impaired immunological response including hyperactivation of T-helper cells and defective T-cell response, severely burned individuals are the most vulnerable population for reactivation of a dormant infection [29]. The frequency of cytomegalovirus seroconversion in burned patients ranges from 18 to 22 %. Moreover, about 50 % of patients with latent period may develop reactivation of infection against the background of immunosuppression with glucocorticoids and cytostatics [37]. Solid-phase enzyme-linked immunosorbent assay (ELISA) is used in the diagnostics of cytomegalovirus infection [44]. Ganciclovir and Valganciclovir are the most commonly used for treatment [31].

Infections with several different viruses have been reported in severe burned patients. This usually leads to more severe clinical outcomes [16]. According to D'Avignon et al, in a study conducted in 2009, out of 97 autopsies performed, viral infections were fatal in 5 patients in the study group; unfortunately, the diagnosis of viral infection was not made prior to autopsy [12]. Due to the development of high-tech techniques in various fields of medicine, including the emergence of new improved diagnostic methods, the literature reports a significant decrease in morbidity and mortality rates among burn victims [71].

Varicella zoster virus (VZV) is a human alpha-herpesvirus. In children, primary infection is manifested by varicella zoster, in adults it is more common manifested with shingles or latent infection is established in trigeminal ganglion neurons or posterior roots of the spinal cord [14, 40]. VZV reactivation leads to herpes zoster and post-herpetic neuralgia [35]. VZV reactivation in burn victims is caused by severe trauma and subsequent immune decline [65]. Despite immunosuppression, VZV reactivation is relatively rare in burn victims. There have been postinfectious cases of varicella and pneumonitis that developed in burned children with active VZV infection [57]. Immunity to VZV, acquired as a result of infection or vaccination, significantly reduces the incidence of VZV infection among burn victims [38].

As early as 1981, an observation of Epstein-Barr virus (EBV) infection in 27 severely burned children was presented [44]. According to the authors data, only 3 of them were found to have elevated titres of antibodies to EBV without significant clinical manifestations. No other studies have shown the presence of EBV infection specifically in burn patients.

Thus, the manifestations of viral infection are manifold, the impact on the organism of a severely burned person is multifaceted, and the information from different authors is contradictory [33]. Considering the annual increase in the number of such victims treated in intensive care units, active screening studies are required to detect viral infections and to determine their impact on the course of burn disease.

ADDITIONAL INFORMATION

Author contribution. Thereby, all authors made a substantial contribution to the conception of the study, acquisition, analysis, interpretation of data for the work, drafting and revising the article, final approval of the version to be published and agree to be accountable for all aspects of the study.

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