

Tanishka S., Sheetalkumar D.C.

HERPETIC INFECTION AS A MANIFESTATION OF POST-COVID SYNDROME

Tutor: professor Dotsenko M.L.

Department of Infectious diseases

Belarusian State Medical University, Minsk

The global COVID-19 pandemic caused by the SARS-CoV-2 virus has led to a myriad of clinical manifestations, including the reactivation of the Varicella-Zoster virus (VZV) in some patients. This abstract provides an overview of the relationship between COVID-19 and VZV reactivation, highlighting the potential implications for patient care and management. The reactivation of VZV, known as herpes zoster (HZ), following COVID-19 infection or vaccination has raised questions about its clinical course and outcomes compared to HZ in the general population. Additionally, the long-term effects of COVID-19, including post-acute sequelae (PASC), present challenges in healthcare delivery, especially in managing functional mobility impairments, pulmonary issues, and mental health disorders. The co-infection of SARS-CoV-2 with other herpesviruses like Epstein–Barr virus (EBV) and human herpesvirus 6 (HHV6) may contribute to the severity of COVID-19 and post-COVID syndromes. This abstract underscores the importance of further research to elucidate the complex interplay between COVID-19 and VZV reactivation, as well as the broader implications for patient outcomes and healthcare systems worldwide.

SARS-CoV-2 infection triggers a hyperimmune response characterized by the release of pro-inflammatory cytokines, leading to a cytokine storm. The virus primarily affects T lymphocytes, impacting the balance between naive CD4+ T cells and memory T cells crucial for an effective immune response. In general, during coronavirus infection, the number of NK, T and B lymphocytes decreases while the number of monocytes and neutrophils increases and increasing concentration pro-inflammatory cytokines (IL-2, IL-6, IL-7, IL-8, TNF- α).

In immunocompetent patients with herpes zoster due to COVID-19 infection. Risk factors were age, lymphocytopenia and the severity of disorders in the T-cell immunity caused by coronavirus infection. This may be due to a decrease in the level of both the total number of lymphocytes and individual lymphocyte subpopulations in COVID-19: CD4+T-I, CD8+T-I, B-I, NK cells, and it is also known that in patients with herpes zoster the number of these cells is significantly lower than in the control group.

The reactivation of latent viruses like herpes zoster (HZ) has been reported in COVID-19 patients and those vaccinated against COVID-19, with studies showing associations between herpesvirus reactivations and COVID-19/COVID-19 vaccination. Furthermore, pharmaceutical interventions commonly used in these cases include steroids such as dexamethasone, antivirals like acyclovir and remdesivir, and immunomodulatory agents like tocilizumab, which may contribute to opportunistic infections or latent virus replications.

HZ after COVID-19 vaccination was more common in females, elderly and immunocompromised patients compared to those without a history, while HZ after COVID-19 infection tended to occur in younger patients. Most patients developed HZ after COVID-19 vaccination or infection within 5–10 days. Although the clinical manifestations, treatment outcomes, and complications were not significantly different from those of patients with HZ and without recent infection or vaccination, recurrent HZ and HZ dissemination were reported in a higher proportion of patients with HZ after COVID-19 vaccination or infection.

Herpes viral infections and SARS-Cov2 have common mechanisms of pathogenesis, disruption of the interferon system is a critical feature of SARS-CoV-2 infection, which leads to reactivation of herpes viruses in patients with coronavirus infection, Risk factors for reactivation of Herpes zoster infection in post-Covid are lymphopenia, decrease in CD4+ T-lymphocytes, CD8+ T-lymphocytes, B-lymphocytes, NK cells.