LETTER TO EDITOR

Biochemical considerations between SARS-CoV-2 infection and Dengue: General aspects

Consideraciones bioquímicas entre la infección por SARS-CoV-2 y Dengue: Aspectos generales

Juan Serna-Trejos[1,3,4,5,6], Stefanya Bermudez-Moyano[2], Juan Lenis-Gonzalez[1], Diego Prado-Molina[1]

[1]Faculty of Health Sciences, Medicine Program, Universidad Libre – Seccional Cali. Cali, Colombia.
[2]Faculty of Health Sciences, Medicine Program, Universidad Santiago de Cali – Cali, Colombia.
[3]Department of Epidemiology, Universidad Libre – Seccional Cali. Cali, Colombia.
[4]Department of University Teaching, Universidad Piloto - Bogota, Colombia.
[5]Instituto Nacional de Salud. Bogota Colombia.
[6]Interdisciplinary research group in epidemiology and public health, Cali, Colombia.

To editor:

The clinical and global context associated with SARS-CoV-2 infection has generated a wide variety of clinical spectra and pathophysiological correlations that the clinician should suspect in the presence of this entity. Although most viral illnesses have similar prodromal symptoms, the appearance of symptoms specific to each viral infection should be individualized, which is a matter of great interest for global public health and especially in South America, where there is a high prevalence of tropical infectious diseases such as Dengue. An adequate approach to the most relevant biochemical aspects, such as their recognition, can lead to consider in both entities, the generation of a broader picture in the optimal development of diagnosis, treatment, prognosis and rehabilitation of the infected patient with the coexistence of these viruses.

Dengue is known for its endemic-epidemic potential, transmitted by the Aedes aegypti mosquito, whose magnitude can be extrapolated to the point of being an infection that generates considerable morbidity and mortality in Latin America. According to the latest epidemiological update of Dengue in the Context of SARS-CoV-2 generated by the World Health Organization (WHO), by the year 2021 in the region of the Americas about 2 million cases of Dengue were reported, giving an incidence of 221 cases per 100,000 inhabitants, generating about 872 deaths[(1)]. Parallel to the previous situation associated with Dengue, there were also worrying figures associated with SARS-CoV-2 in the same year, since 24 million cases were reported in the Americas region, generating an incidence of 2424 cases per 100,000 inhabitants, with a case fatality rate of 2.8%. The unusual behavior was observed given the persistence of dengue cases above what is usually expected, added to summer seasons, which represented a great challenge for the different health systems executing public health during that period. In the initial stages of Dengue infection it is typical to find phagocytic and dendritic mononuclear cells, which have been typified as targets of contact with the virus. In initial stages antiviral antibodies are formed, such formation induced by the presence of a serotype of the virus, then occurs the binding of the virus surface and its interaction with the Fc receptor (present in most of the cells of the immune system, such as macrophages, neutrophils, mast cells) then the NK lymphocytes "Natural Killers" are responsible for the induction of phagocytosis or cytotoxicity of the pathogen. Some biological models have been able to confirm the target of the virus such as peripheral blood mononuclear cells (PBMC); CD14+ monocytes, responsible for the production of TNF-α (Tumor necrosis factor alpha) and IL-6 (Interleukin-6), cytokines responsible for the expansion of the immune response and subsequent recruitment of adjuvant cells[(2)].

SARS-CoV-2 infection is generated by a massive immune response, mostly associated with severe pulmonary involvement. This infection has a tendency to evolve rapidly if the initial immune containment mechanisms against infection are not sufficiently adequate. A circuit of activation of the immune response in

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Correspondence to: Juan Serna; **E-mail**: juansantiagosernatrejos@gmail.com

Orcid: Serna-Trejos J.: https://orcid.org/0000-0002-3140-8995
Bermudez-Moyano S.: https://orcid.org/0000-0002-2259-6517
Lenis-Gonzalez J.: https://orcid.org/0000-0001-9004-475X
Prado-Molina D.: https://orcid.org/0000-0002-5402-8856

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patients admitted to intensive care units with respect to patients not admitted to such units has been described. The circuit starts with the activation of T helper lymphocytes (Th); CD4+ and CD8+. Increased levels of IL-6 and GM-CSF (Granulocyte-Monocyte Colony Stimulating Factor) have been observed in direct relation to increased CD4+ expression, which progresses around the clinical severity of the infected patient. When activation or hyperactivation (Cytokine Release Syndrome or "CRS") is insufficient to control the disease, a marked lymphocyte depletion or lymphopenia is consequently generated, leading to further tissue damage, inducing the characteristic Acute Respiratory Distress Syndrome (ARDS). The main cytokines involved in the pathogenesis and progression of the disease have been identified as; IL-6, IL-10(Interleukin-10), IFN(Interferon), MCP-1(monocyte chemoattractant protein-1), CM-CSF (granulocyte colony stimulating factor), TFN (tumor necrosis factor), IL-1(Interleukin-1), IL-2(Interleukin-2), IL-2-receptor (Interleukin-2-receptor complex) and IL-8(Interleukin-8)(3). Other models propose IL-6 as the responsible for the overactivation or initiator of the cytokine storm, passing through the hepatic synthesis of thrombopoietin and fibrinogen, which concludes in a balance of thrombin production, which at elevated levels can generate negative feedback or negative feed-back and induce thrombocytopenia by the cytokine storm[3].

The hematological and biochemical findings most commonly found in these states of conflation between SARS-Cov-2 and Dengue, conclude in states of depletion of immune system cells such as: lymphocytes and monocytes, in turn high levels of glycemia are found, explained by the state of acute infection. It has been documented that increased serum levels of IgG for Dengue may represent variations in the lymphocyte count and serum glucose levels. Other biochemical markers affected in the co-infection of these two entities were: Creatinine, ALT, AST and LDH[4]. Significant levels of IL-10 have been found in pneumonia associated with SARS-CoV-2 and Dengue hemorrhagic pneumonia, being higher in the latter group, unlike IL-6 and CD40L (CD40-Ligand) where significantly increased levels are expressed in both groups, but with greater predilection for the group in which pneumonia associated with SARS-CoV 2 occurred. However, it has been documented that regardless of the temporality of the infectious clinical picture and its duration, in some of these clinical pictures IL-6 levels are usually fully in normal ranges or even decreased[5,6].

The coexistence of these infectious entities presents a great similarity in relation to pathophysiological events, as well as signs and symptoms. More studies on molecular biology and evidence-based medicine in the treatment of the coexistence of these two pathologies are required to prevent unfavorable events or outcomes for patients with the infection, as well as the generation of training in the different actors of the health system in relation to these biochemical aspects little considered in the approach of these pathologies individually or simultaneously.

Authors' contributions

All authors contributed to the whole process of writing the letter.

References

1. Organización Mundial de la Salud. Actualización Epidemiológica: Dengue, chikunguña y Zika en el contexto de COVID-19. OMS [Internet]. 2022[cited 2022 aug 12];1(1):1–6. Available in: https://iris.paho.org/handle/10665.2/55639
2. Abduljalil JM, Abd Al Galil FM. Molecular pathogenesis of dengue virus infection in Aedes mosquitoes. J Insect Physiol [Internet]. 2022;138(April). doi: 10.1016/j.jinsphys.2022.104367
3. Wang Y, Perlman S. COVID-19: Inflammatory Profile. Annu Rev Med [Internet]. 2022;73:65–80. doi:10.1146/annurev-med-042220-012417
4. Soares Nogueria Teotonia IM. Clinical and biochemical parameters of COVID-19 patients with prior or active dengue fever. Acta Trop [Internet]. 2021;214(1):1. doi: 10.1016/j.actatropica.2020.105782
5. Dayarathna S, Jeewandara C, Gomes L, Somathilaka G, Jayathilaka D, Vimalachandran V, et al. Similarities and differences between the 'cytokine storms' in acute dengue and COVID-19. Sci Rep [Internet]. 2020;10(1):1–12. doi: 10.1038/s41598-020-76836-2
6. Tsheten T, Clements ACA, Gray DJ, Adhikary RK, Wangdi K. Clinical features and outcomes of COVID-19 and dengue co-infection: a systematic review. BMC Infect Dis [Internet]. 2021;21(1):1–9. doi:10.1038/s41598-020-76836-2