LETTER TO EDITOR

Main endocrine alterations generated in the POST-COVID-19 syndrome

Principales alteraciones endocrinas generadas en el síndrome POST-COVID-19

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To Editor:

The global health contingency generated by SARS-CoV-2 has produced multiple changes and sequelae in the healthcare system. On the one hand, it has led to a large deployment in terms of immunization, generation of new biological agents and mechanisms for the latter. It has also brought about the creation of new health policies in different health systems. In addition to the previously described effects, there has also been a significant impact on the main actors involved in the pandemic, more specifically in the health-disease process and the biological sphere. These sequelae have been attributed to "POST-COVID-19 syndrome".

Cite as: Serna-Trejos JS, Bermudez-Moyano SG, Lenis-Gonzalez JP, Altman-Salcedo D. Principales alteraciones endocrinas generadas en el sindrome Post-COVID-19. Rev. Peru. Investig. Salud. [Internet]; 2023; 7(1): 41-43. https://doi.org/10.35839/repis.17.1.1597

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Conflicts of interest: the authors declare no conflicts of interest.

Funding: self-funded.

Editor: Jarviz Raraz, UNHEVAL

Received: december 08, 2022
Approved: february 7, 2023
Online: march 03, 2023

Endocrinological sequelae constitute a target of great interest in POST-COVID-19 syndrome research since highly sensitive and susceptible organs are located at this level, secondary to the expression of angiotensin-2 receptors. This is the reason why the virus and its virulence capacity have a significant impact at this level[(1)]. High levels of angiotensin-2 receptors have been identified in the pancreas, making this organ susceptible to changes associated with SARS-CoV-2 infection. Elevated levels of pancreatic amylase and lipase have been documented in approximately 20% of patients infected with SARS-CoV-2, either in one or both, which can affect glycemic control. In addition, the coexistence or development of type 1 diabetes mellitus may occur due to damage generated by the virus at the beta-pancreatic cell level, as well as worsening insulin resistance in the case of type 2 diabetes mellitus secondary to overexpression of fetuin A levels. However, these repercussions in the latter entity have not been widely documented[(2)].

Other alterations secondary to SARS-CoV-2 infection are related to adrenal level; the previously described phenomenon is related to the production of a protein similar to adrenocorticotropic hormone (ACTH), generating the production of antibody against endogenous ACTH, producing hormonal suppression effects, ultimately leading to hypocortisolism. Hypothetically, molecular mimicry employed by SARS-CoV-2 could be associated with this phenomenon. In some severe cases, central hypocortisolism is generated due to extensive hypophyseal damage[(3)]. In patients with previous obesity, it could induce more pro-inflammatory states due to overexpression of angiotensin-2 receptors, which favor virus entry into the adipocyte and consequently generate a more extensive inflammatory cascade[(4)].

Regarding the gonadal level, the sequelae have been mostly observed in males. A study conducted by Ma L, Xie W, Li D et al, in which total testosterone levels were measured in 81 men infected with SARS-CoV, showed that serum levels were lower (without statistical significance, p>0.05), while serum luteinizing

hormone (LH) levels were significantly elevated compared to 100 healthy men of the same age. The latter was associated with direct injury to the Leydig cells (responsible for testosterone production)[5]. The most described clinical manifestation in the literature and more common at the endocrine level regarding the hypothalamic-pituitary axis is related to neurological alterations in the organs of the senses, more directly in olfactory symptoms. Hyposmia has been described as secondary to the damage generated by SARS-CoV-2 to the cribriform plate, where access to different preganglionic cells is located at this level, with a high content of angiotensin-2 receptors, generating edema and neuronal degeneration. Sequelae associated with central hypocortisolism have been described, of which approximately 90% express symptoms associated with their underlying condition, such as fatigue and postural vertigo[6,7].

Thyroid dysfunction has been poorly explained; however, a post-mortem study conducted by Wei L, Sun S, Xu C, Zhang J et al, showed marked destruction of the follicular and parafollicular cells of the thyroid, generating lower levels of T3 and T4L than corpses that did not suffer from the infection. Acute stages of the disease have also been described as leading to hyperthyroidism or subclinical hypothyroidism secondary to dysfunction of the thyroid follicles[8].

In perspective, the endocrine sequelae generated by SARS-CoV-2 infection may have non-specific symptoms mostly related to constitutional symptoms. The extrapulmonary sequelae, in this case of an endocrine type generated in POST-COVID-19 syndrome, are illustrated in Figure 1 according to the affected organ and/or gland.

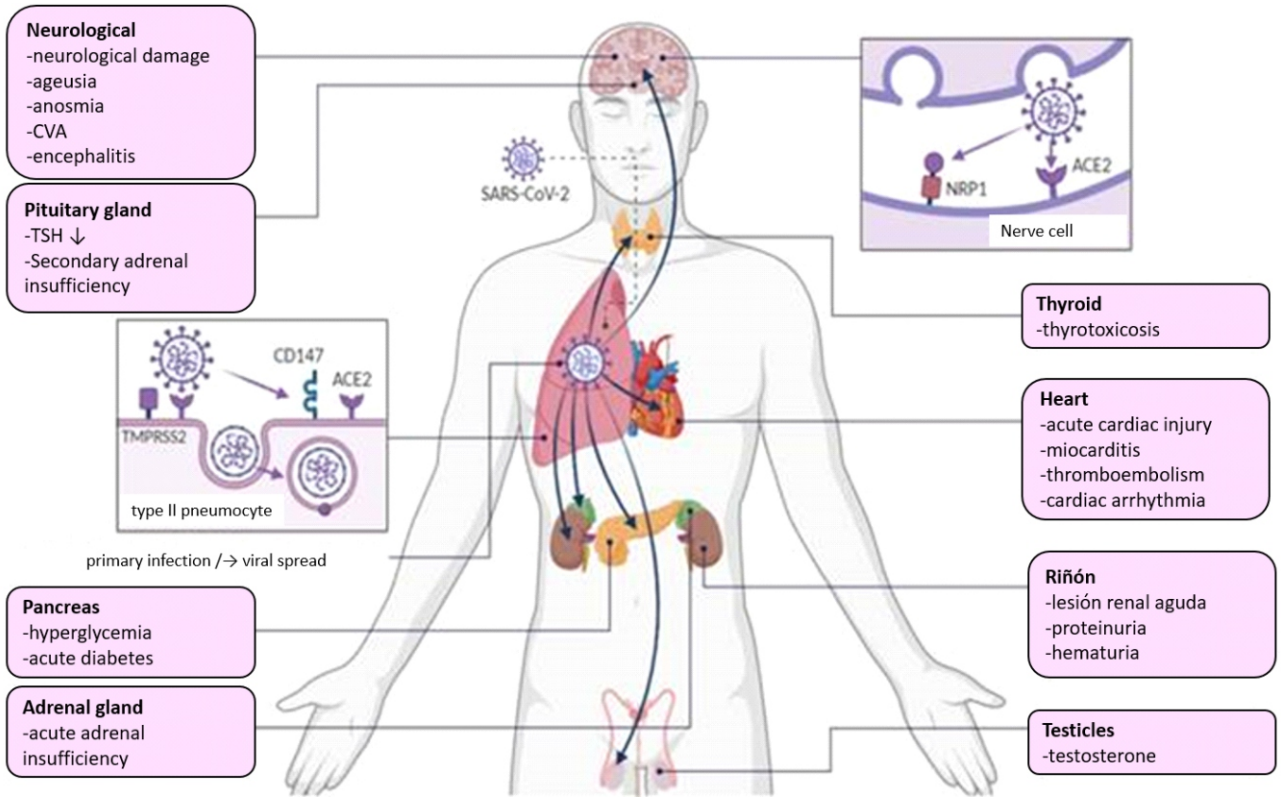

Figure 1. Main extrapulmonary manifestations in POST-COVID-19 syndrome

Some pictures may simulate underlying pathologies or pre-existing conditions of the patient, multicenter studies have been executed in this area in search of new clinical correlations of SARS-CoV-2 infection with clinical sequelae, it is necessary to individualize each case of "POST-COVID-19 Syndrome" and perform the relevant clinical interventions, as well as the strengthening of new searches and research related to this topic.

Description: The POST-COVID-19 syndrome has direct affectation at glandular level such as: pituitary gland, pancreas, adrenal gland, Thyroid and testicles, the manifestations vary according to the function exerted by each of the affected organs and its severity will depend according to the suppression or over activation of each one[9].

Author contribution

All authors have contributed to the conception, drafting of the final manuscript, revision and approval of the manuscript.

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