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Association between Guillain-Barré Syndrome and Application of the Janssen Vaccine

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Abstract:
This update was carried out in order to establish an association between the application of the jhanssen vaccine and the development of Guillan-Barré syndrome. Materials and methods: A detailed bibliographic search of information published since 2020 is carried out, in the databases Pubmed, Elsevier, Scielo, Update, Medline, national and international libraries. Results: the FDA who notified the detection of 100 possible cases of the syndrome among the 12.8 million people who have received the Janssen vaccine in the United States, that is, it affected 0.0007% of the total. Discussion: immune stimulation induced by vaccination could theoretically result in GBS through a variety of possible mechanisms. Conclusion: the inflammatory response that develops after a COVID-19 infection would have a negative effect on activated T cells, which in turn release pro-inflammatory cytokines, fix complement, damage Schwann cells and finally produce myelin dissolution and as a potential result we have the development of Guillian-Barre syndrome. Key words: JANSSEN vaccine, Guillain-Barré syndrome, covid-19, peripheral nerves.

Introduction

Guillain-Barré syndrome (GBS) or so-called immune-mediated polyradiculoneuropathy is an acute inflammatory disease characterized by the presence of tingling, progressive weakness, autonomic dysfunction, and pain resulting from a lesion that occurs specifically in the myelin sheath and related components. Schwann cells (1).

GBS is of the idiopathic type, although in most cases it is suspected that the nonspecific activation of the immune system against its own tissues in Guillian-Barre is associated with infections by certain pathogens that carry molecules with a high analogy to their own pain (mimicry molecular) (2).

One of the pathogens most frequently related to GBS is Clostridium jejuni (C. jejuni), however, other agents such as Zika virus (ZIKV), Epstein Barr Virus, other Herpes viruses, Influenza, Hepatitis, etc., And recently evidence has been shown of keeping a relationship with this syndrome (3,4).

Infection-related GBS patients produce frequently obtained against gangliosides (Lipid Type) of human peripheral nerves through molecular mimicry. These results are linked to both the gangliosides of the nerves and the microbial durings, an immune response mechanism that has been observed in the "cytokine storm" that develops in those patients who are struggling with covid-19 infection (5 ,6).

Materials and methods:

A detailed bibliographic search of information published since 2020 is carried out in the databases pubmed, Elsevier, scielo, Update, medline, national and international libraries. We use the following descriptors: covid-19, JANSSEN vaccine, Guillian-Barre, association between Guillian-Barre and covid-19. The data obtained range between 5 and 15 records after the use of the different keywords. The search for articles was conducted in Spanish and English, limited by year of publication, and studies published since 2020 were used.

Results:

Several cases have been reported in different health centers, of admission of patients who present Guillain-Barré syndrome (GBS) and are active positive COVID-19 or had the disease, for which the association between both pathologies has been proposed. Some authors state that the presentation of GBS associated with COVID-19 generates more acute onset symptoms, so it seems to be a form of pre-pandemic GBS (7,8).

If we analyze the clinical manifestations reported in the studies analyzed, among the most frequent we find muscle weakness of the lower limbs, areflexia, cranial nerve involvement (facial paralysis, facial diplegia, dysphagia, among others), paraparesis and quadriparesis, followed by slightly less frequent signs such as cough, fever, diarrhea, respiratory distress, anosmia, ageusia, among others. Regarding the feverish picture as symptomatology, it was present in most of the reported cases; however, it must be considered that several of the patients had active COVID-19 at the time of admission and, in the case of patients who did not present fever, it was due to GBS presenting on 10, 14 and up to 21 days after the diagnosis of COVID-19 (9,10).

In various pathological studies, including that of Hamming et al., it was shown that ACE2 (angiotensin-converting enzyme 2) acts as a functional receptor for SARS-CoV in human tissues. Due to the sequence similarity of the S proteins (in English, spike proteins) of SARS-CoV and SARS-CoV-2, the prediction was made that SARS-CoV-2 also uses ACE2 as a functional receptor, information that was confirmed by other studies during the first months of 2020 (11, 12,13).

They have also been proposed as possible mechanisms by which SARS-CoV-2 can cause neurological damage, the anchoring of the virus to ACE2 in the blood-brain barrier, facilitating its entry into the central nervous system; as well as it has been proposed that there are retrograde, transcribal and hematogenous neural dissemination pathways (14,15)

Given the alarming mortality figures resulting from COVID-19, it was necessary to create a vaccine that could strengthen the immune system and achieve an acute immune response that does not compromise the different systems and target organs, which is when international companies, like many laboratories, set out to to the task of developing an effective bilogixo, as is the case of Johnson & Johnson, said with its JANSSEN vaccine, which obtained approval from the FDA to be applied (16,17). However, days ago it was the FDA who notified the detection of 100 possible cases of the syndrome among the 12.8 million people who have received the Janssen vaccine in the United States, that is, it affected 0.0007% of the total (18, 19).

According to the FDA, 95% of cases were serious and patients had to be hospitalized. One of them died (20).

He also pointed out that the data is "insufficient to establish a causal relationship", that is, that the vaccine is directly responsible for the development of the symptom. However, it is worth considering that; 12.5 million vaccinated people were needed to find 100 cases of GBS, which means that it remains an extremely rare reaction and whose presentation is more

associated with individual characteristics. In other words, even without vaccination, it is likely that in the course of his life he would have developed GBS when encountering a different stimulus such as natural infection by SARS-CoV-2 (21,22).

Discussion:

Active immunization by a vaccine stimulates the immune system to produce antigen-specific humoral and/or cellular immunity. Therefore, vaccination-induced immune stimulation could theoretically result in GBS through a variety of possible mechanisms (23).

SARS-CoV-2 can cause neurological damage, due to the capacity for neuro-invasion that it acquires by anchoring the virus to ACE2 in the blood-brain barrier, facilitating its entry into the central nervous system; as well as it has been proposed that there are retrograde, transcribal and hematogenous neural dissemination pathways. Which leads to the exposure of antigens directly in the CNS (24). Factor that is additional to the levels of autoimmunity that are generated during severe infections and that also provide the ideal environment for the development of GBS. In the case of the Janssen (J&J) vaccine, it is possible that GBS is associated with the fact that a viral vector (Adenovirus Ad26) is used that could add peptides on cell membranes when fusing to deliver the genetic load of the protein S and thereby induce mimicry, antigen modification, or nonspecific immune response could be the GBS primer (25,26).

Conclusion:

Patients with infection-related GBS frequently produce antibodies against the gangliosides (Lipid Type) of human peripheral nerves through molecular mimicry. These antibodies bind to both nerve gangliosides and microbial antigens. Activated macrophages and microglia within the CNS release cytokines and free radicals (eg, nitric oxide), invade compact myelin, the periaxonal space, and occasionally block nerve conduction or cause axonal degeneration. Based on the studies that have been carried out, it can be inferred that the inflammatory response that develops after a COVID-19 infection would have a negative effect on activated T cells, which in turn release proinflammatory cytokines, fix complement, damage Schwann cells and finally produce the dissolution of myelin and as a potential result we have the development of Guillian-Barre syndrome. This would explain the exact relationship between the pathogenesis of COVID-19, GBS and the application of the JANSSEN vaccine.

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