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Harmful Effect Of SARS-Cov-2 Virus On The Body

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Abstract.
A severe acute respiratory condition called Coronavirus Disease 2019 (COVID-19) is brought on by the SARS-CoV-2 virus. Following the discovery of the first case in Wuhan, China, the number of cases quickly grew and spread throughout the entire world [1]. The coronavirus genus and family of zoonotic viruses include beta-coronaviruses (-CoVs). Following the occurrences of the Middle East respiratory syndrome coronavirus (MERS-CoV) and the severe acute respiratory syndrome coronavirus (SARS-CoV) in 2003 and 2012, respectively, the most recent -CoV, known as SARS-CoV-2, first occurred in December 2019 in Wuhan, China [18]. Coronavirus disease 2019 (Covid-19), a severe inflammatory viral disease, has been linked to SARS-CoV-2 [19].

Keywords: *SARS-CoV-2, autoimmune disease, immune system*

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I. Introduction.

There have been over 80 million confirmed instances of Covid-19 worldwide, with a 2.2% case fatality rate that primarily affects the 15-20% of the disease's severe cases with bilateral interstitial pneumonia [20]. Acute respiratory distress syndrome (ARDS), which is characterised by a sudden onset of extensive lung inflammation, is thought to be the main cause of death in these situations [21]. Regarding the defence against SARS-CoV-2 and the development of the illness, both innate and acquired immunity are important [22].

Covid-19 has been proven to weaken the immune system's protective immunity, especially in people with co-morbid conditions, older age, and certain genetic makeup. It causes lymphopenia, which is a decrease in the number of CD4+, CD8+, NK-, and B-cells in the body. It also reduces the number of regulatory T-cells (Tregs), overactivates T cells, overproduces cytokines (IL-2R, IL-1, IL-6, IL-8, IL-10, IL-17, and TNF-α), exhausts T cells, and increases antibodies [23]. Defects in immunological tolerance and homeostasis processes, according to studies, result in incorrect activation of the interferon pathway and autoinflammation [24].

The most frequent clinical signs are similar to other viral infections, and may result in the impairment of the functions of multiple organs [2]. Once in the respiratory tract, SARS coV-2 attaches to the Angiotensin Converting Enzyme (ACE) Receptors, which are found in a variety of human cells, including pancreatic cells, thyroid cells, testicular cells, testis cells, ovaries, adrenals, and pituitaries [3]. The thyroid gland may be affected either directly by the virus infecting the target cells, or indirectly through abnormal immune regulation [4]. Thyroiditis (Destructive or Inflammatory) is a prevalent form of thyroiditis that may be accompanied by an increase in cytokines, commonly referred to as a cytokine storm. A retrospective study demonstrated a correlation between thyroidoxicosis due to systemic immune activation and an increase in IL-6 during the hospitalization period in 287 non-Immediate Care Units (ICUs) of COVID-19-infected patients [5]. Sub-acute thyroiditis has been strongly linked to viral infections [6]. Since the beginning of the COVID-19 pandemic, there have been numerous case reports in which it is thought to be a possible trigger of SARS-Copy-2 [7, 8]. Additionally, recent reports have indicated that Graves' disease and Hashimoto's thyroiditis may occur following infection with COVID-19. Recent research has indicated that the COVID-19 pandemic may be a contributing factor to the development of Graves' disease and Hashimoto's thyroiditis, either as a novel flare-up or in remission in reported cases. Graves' disease is an auto-immune thyroid disorder that is caused by a variety of environmental factors, such as viruses, in susceptible individuals. Molecular mimicry may be one of the potential mechanisms, as the disease is triggered by a breakdown of immune tolerance to the TSH-Rs, TPOs and TGs. Numerous infectious agents have been studied for their potential role in the formation of Graves' disease, including foamy viruses, the Parvoviruses, the Epstein-Barr viruses and the hepatitis C virus [9]. Similarly, various viruses, including hepatitis C and the B19 virus, are thought to be involved in the development of Hashimoto's thyroiditis, though not fully elucidated, as in Graves' disease [10]. Therefore, COVID-19 could be another contributing factor to the formation of autoimmune thyroiditis, as in these reported cases. However, while case studies may raise this concern, they do not provide evidence of a causal relationship. Since the pandemic outbreak in December 2019, many autoimmune disorders other than autoimmune thyroid disease have been linked to COVID 19, including

autoimmune hemolytic anaemia, Guillain-Barre syndrome, and systemic lupus erythematosus. Increased production of pro-inflammatory cytokines in COVID-19, like in autoimmune illnesses, can cause organ damage in some patients [10]. SARS-CoV-2 antibodies have been demonstrated to react with numerous human tissues, including the thyroid [11].

They also discovered similarities and homology between the human tissue antigens mitochondria M2, F-actin, and TPO and spike, nucleoprotein, and many other SARS-CoV-2 proteins. This immunological cross-reactivity may cause the onset of a new autoimmune disease as well as aggravate autoimmunity in susceptible individuals, such as the patients in this study [11].

During severe acute or chronic sickness (trauma, sepsis, starvation, hepatic disorders, major systemic illness), altered thyroid functions (also called as "non thyroidal illness syndrome" or "low T3 syndrome, or euthyroid sick syndrome) can be noticed. Free T3, TSH, and FT3/FT4 levels were considerably lower in COVID-19 patients who were severely or critically sick compared to non-critical patients [12].

Another study that looked at thyroid function in 287 patients in a non-intensive care unit found that COVID 19 had overt (10.8%) and subclinical (14.6%) thyrotoxicosis. Because of systemic immunological activation caused by SARS-Cov-2 infection, serum IL-6 levels were found to be substantially associated with TSH levels [5]. With both negative TRab, anti-TG, and anti-TPO levels in nine patients and spontaneous improvement during follow-up, it is possible to speculate that a possible mechanism was destructive thyroiditis in these patients. However, the research indicates that associated thyroid dysfunctions were severe in some patients, and two patients died: one with Graves' disease and thyroid storm as a result of adult respiratory distress syndrome, and another with hypothyroidism as a result of myxedema coma and sudden cardiac arrest. Graves' disease and Hashimoto's thyroiditis can develop a few months following subacute thyroiditis, which is assumed to be a type of virally induced destructive thyroiditis [13]. It may be beneficial to conduct long-term follow-up of COVID-19-related thyroid-related patients in order to assess the progression of an autoimmune thyroid disorder. In the present cases, the majority of cases occurred after a period of time following a possible increase in cytokines. It is unknown if a destructive thyroiditis was present in the initial phase of the infection. The autoimmunity of 120 hospitalized COVID-19 subjects was assessed through the use of a panel of thyroid and rheumatic antibodies, as well as anti-nuclear antibodies and cyclic Citrullinated Peptide 3 antibodies, in comparison to pre-existing healthy controls. The presence of TPO antibodies, β2- glycopotein 1 antibodies, and cardiolipin-related antibodies were more commonly observed in the COVID-19 individuals compared to those in the pre-existing controls. The higher titers of cardiolipin antibody were associated with a poorer outcome (meanetric ventilation and death) [14].

This may indicate that the more severe the disease, the greater the risk of autoimmunity due to an inflammatory response.

One study looked at the effects of the COVID-19 pandemic on thyroid function tests and thyroid autoimmune responses in COVID survivors [15]. The study included 122 non-critical ill patients who were assessed at baseline and at a median 90-day follow-up. The study found that 20 patients (16.4% of the total) had abnormal thyroid function tests at baseline (5 of subclinical thyroidrotoxicosis, 15 of non-thyroid disease) and all returned to normal range with the exception of 2 patients (1 had T3-Toxicosis, 1 had NTIS). The anti-TPO titer and the anti-TG titer significantly increased on reassessment and 4 patients became positive for Anti-TPO. In 2 of these patients, the reason for hospitalization was due to a thyroid storm, not COVID-19, and these two studies were conducted on hospitalized patients and most of the patients were male. . Patients with COVID-19-associated autoimmune thyroid disease who have been described to date are predominantly women with moderate illness, as is typical of this disease's history. Further research in a broader group is required to determine the cause of thyroid dysfunction in COVID-19 survivors. COVID-19 vaccinations may cause the onset of autoimmune thyroiditis in addition to the disease itself. A nanoparticle encoding a modified SARS-CoV-2 spike protein is included in the BNT162b2 mRNA vaccine [16]. The vaccination mimics the virus, and identical mechanisms in the infection may elicit autoimmune reactions. Furthermore, adjuvants in vaccinations have been implicated in the development of thyroiditis, adding to the notion "autoimmune/inflammatory syndrome induced by adjuvants" [17].

It is widely accepted that autoimmune illnesses are associated with autoinflammatory states [25]. Although the exact cause of such complex diseases is unknown, several variables such as genetic predisposition [26], epigenetic effects, and environmental triggers such as microbial, fungal, parasitic, and viral infections can all predispose a person to autoimmune disorders [27].

Viral infections can cause intolerance through a variety of mechanisms, including molecular mimicry (cross-reacting epitope between pathogen-derived and self-antigens), bystander killing (virus specific CTLs migrating to target tissues and exerting cytotoxicity), epitope spreading (polyclonal activation due to the constant presence of viral antigens driving immune-mediated injury), clearance deficiency, and viral persistence, which can increase the risk of autoreactivity. These factors contribute to autoinflammatory reactions and autoimmune disease exacerbations [28].

SARS-CoV-2, like many viral infections [29], can cause a variety of autoimmune signs [30]. The virus replicates in the respiratory mucosa's epithelial cells after entering the upper respiratory tract. The virus is destroyed by the immune system.

Otherwise, the virus enters the lungs, where it may cause overactivation of the innate and acquired immune systems, followed by antibody entrance into the bloodstream. Antibodies generated in response to the virus can react with proteins expressed on human cells, resulting in systemic symptoms [31]. Several autoimmune illnesses, including Immune thrombocytopenic purpura (ITP), Guillian Barre syndrome (GBS), Miller Fisher syndrome (MFS), Kawasaki-like disease in children, and others, have been linked to COVID-19 [32].

Hyperstimulation of the immune system: cytokine storm and hyperferritinemia

Severe COVID-19 disease is characterised by fever, hyperferritinemia, and a huge production of pro-inflammatory cytokines ('cytokine storm,' which can result in a high mortality rate. The phenomena of cytokine storms in severe SARS-CoV-2 infected patients has been thoroughly investigated and described in COVID-19 critically unwell patients [33]. Macrophages, one of the main immune populations found in the lung parenchyma, have been implicated in the pathophysiology of SARS-CoV-2-induced acute respiratory distress syndrome (ARDS) and life-threatening manifestations in critically ill patients.

SARS-CoV-2 may cause immune system hyperstimulation in genetically predisposed individuals, leading to overactivation of local macrophages and the production of high levels of inflammatory mediators such as cytokines, chemokines, and ferritin. Overproduction of cytokines by macrophages has been demonstrated to increase the inflammatory process and to result in abnormally high levels of ferritin in the blood ('hyperferritenemia'). Importantly, it was recently discovered that SARS-CoV-2 infected individuals have elevated ferritin levels when admitted to hospitals. In light of this, it is worth noting that cytokine storm and hyperferitenemia have previously been linked to pathogenic viruses such as influenza and dengue [33].

Infection with COVID-19 in genetically susceptible humans: relationship with HLA gene polymorphism.

The human leukocyte antigen (HLA) gene and its polymorphism have been linked to the development of several autoimmune diseases/disorders. Recently, researchers have been attempting to determine how human genetics may influence the transmission and infection of the current SARS-Cov-2 virus. Regarding the evidence for a link between the SARS-Cov-2 virus and autoimmune mentioned above, it is not surprising that scientists investigated a strong link between covid- 19 and HLA genetic variants [33].

Sharing peptides between SARS-CoV-2 virus and Human antigens: implication for the upcoming vaccine against COVID-19.

Molecular mimicry between pathogenic viruses and human proteins has already been studied and considered to play a significant role in the aetiology of numerous inflammatory and autoimmune illnesses. It has been quantified the hexa and heptapeptide sharing of SARS-CoV-2 spike glycoprotein with mammalian proteomes and discovered that there is a significant heptapeptide sharing between SARS-CoV-2 spike glycoprotein and human proteins [33].

This study emphasises the possibility of molecular mimicry-induced adverse autoimmune-related manifestations, which have already been reported in SARSCoV-2-infected patients, and raises concerns about the upcoming desired vaccine, indicating the need for vaccines based on pathogen-specific immune determinants that are absent in the human proteome . Concerning future post-COVID-19 vaccine-related autoimmune manifestations, it is worth noting recent reports regarding participants in the AstraZeneca company's trial assessing the safety and efficacy of COVID-19 vaccine who developed symptoms of transverse myelitis, an inflammation of the spinal cord (already reported secondary to COVID-19 infection [33]).

The emergence of autoimmune disorders as a result of COVID-19 infection.

The authors investigated and reported extensively on the relationship between various common pathogenic viruses, including Parvovirus B19, Epstein-Barr virus (EBV), Cytomegalovirus (CMV), Herpes virus-6, HTLV-1, Hepatitis A and C virus, and Rubella virus, and the development of chronic inflammatory and autoimmune diseases . In light of these findings, there has recently been a study of the emergence of autoimmune diseases/disorders reported to be induced by SARS-CoV-2 infection [33]. Patients with COVID-19 infection have been documented to develop autoimmune illnesses such as Guillain-Barre syndrome, Miler Fisher Syndrome (MFS), Antiphospholipid syndrome, Immune thrombocytopaenic purpura, systemic lupus erythematosus (SLE), and Kawasaki disease.

It has been shown previously shown olfactory dysfunction in a number of autoimmune conditions such as SLE, Multiple Sclerosis and Myasthenia Gravis (MG). As mentioned above, there is a clear correlation between autoimmune conditions and COVID-19. Therefore, the recent observation of a high prevalence of Olfactory

Dysfunctionation, particularly in the early presentation of COVID-19 Patients, is not unexpected. [33].

A classic example of COVID-19 infection is ASIA syndrome.

All in all, the above-mentioned suggested relationship between SARS -CoV-2 infection and autoimmune disease can be supported by the concept of Autoimmune/Inflammatory Syndrome induced by Adjuvants (ASIA syndrome), first introduced in 2011 by researchers to collect all autoimmune events that occurred after exposure to an external stimulus (infection, adjuvants, vaccine and silicone) [50].

II. Concluson.

In this aspect, a virus infection such as SARS-CoV-2 can be harmful.

i) a significant activation of the immune system; ii) the emergence of includes 'typical' clinical symptoms such as myalgia, myositis, arthralgia, and fibromyalgia

ii) Chronic fatigue, sleep problems, neurological symptoms, cognitive impairment, memory loss, and pyrexia have all been described in SARS-CoV-2-infected individuals;

iii) the emergence of antibodies, which may result in the development of autoimmune diseases disorders in genetically predisposed individuals (e.g., HLA-DRB1, etc.). As a result, the current COVID-19 pandemic meets practically all of the criteria.

Following COVID-19 disease, main and minor criteria for ASIA syndrome [34] progresses in autoimmune disorders.