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The Relation between Allergy and Covid-19 Pandemic

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Abstract :

The coexistence of food and airborne allergens poses significant health challenges to individuals worldwide, exceptionally during the COVID-19 pandemic. Food and respiratory allergies are common and affect millions of people worldwide. This review article aims to explore the complex relationship between diet and airway allergy and its relevance to the ongoing COVID-19 crisis. Food allergies, characterized by an allergic reaction to specific food proteins, range from mild to relentless, and can be life-threatening as well as pollen, dust of mosquitoes, pet dander, mold spores and other airborne allergens can trigger respiratory symptoms such as lung and nasal congestion CO-VID-19 outbreak for individuals with the den is greatly disturbed. Research suggests that allergic individuals may be at expanded risk for severe COVID-19 due to possible autoimmune diseases, respiratory infections including, the plague along with difficulty accessing health care, high levels of stress and changes in lifestyle and eating habits present additional challenges. Processes and lockdowns have changed daily routines, aka allergy exposure and diet. While reduced outdoor activity may relieve airway symptoms for some, increased indoor time can lead to increased indoor allergens and pollution a it's in the room has been great, especially for people with respiratory symptoms in food preparation areas - it can worsen unintentionally, like for months.

Keywords: Allergic diseases, Coronavirus -2019 Pandemic, (COVID-19), Food allergy, air allergy.

العلاقة بين الحساسية وجائحة كوفيد - 19

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مستخلص

يشكل التعايش بين المواد الغذائية والمواد المسببة للحساسية المحمولة جواً تحديات صحية كبيرة للأفراد في جميع أنحاء العالم، خاصة في أعقاب جائحة كوفيد- 19. تعد كل من الحساسية المنقولة بالغذاء والحساسية التنفسية شائعة وتؤثر على ملايين الأشخاص في جميع أنحاء العالم. الهدف من هذا المقال هو دراسة العلاقة المعقدة بين الحساسية الغذائية وحساسية مجرى الهواء وصلتها بأزمة كوفيد- 19 المستمرة. تتراوح الحساسية الغذائية، التي تتميز بتفاعلات المناعة الذاتية تجاه بروتينات غذائية معينة، من خفيفة إلى شديدة، ويمكن أن تهدد الحياة. الأسباب الشائعة للحساسية هي الفول السوداني وجوز الأشجار والمكسرات والبيض ومنتجات الألبان وفول الصويا. وبالمثل، يمكن أن تؤدي مسببات الحساسية المحمولة جواً، مثل حبوب اللقاح وعث الغبار ووبر الحيوانات الأليفة وجراثيم العفن، إلى ظهور أعراض تنفسية مثل العطاس واحتقان الأنف. وقد تسبب تفشي جائحة كوفيد- 19 في إثارة قلق كبير لدى الأفراد الذين يعانون من الحساسية. تظهر الأبحاث أن الأفراد الذين يعانون من الحساسية قد يكونون أكثر عرضة لخطر الإصابة بكوفيد- 19 الشديد بسبب احتمال الإصابة بأمراض المناعة الذاتية والأمراض التنفسية المصاحبة، علاوة على ذلك، يشمل التحسس صعوبة في الوصول إلى الرعاية الصحية، وارتفاع مستويات التوتر والتغيرات في نمط الحياة ونمط الحياة. عادات الأكل تم طرح تحديات جديدة، مما جعل إدارة الحساسية أكثر صعوبة. لقد غيرت إجراءات التباعد الاجتماعي وعمليات الإغلاق الروتين اليومي، مما أثر على التعرض لمسببات الحساسية والنظام الغذائي. في حين أن انخفاض النشاط الخارجي قد يخفف من أعراض تحسسس الهواء لدى البعض، فإن زيادة الوقت الداخلي يمكن أن يزيد من مسببات الحساسية الداخلية وتتفاقم الملوثات في الغرفة، خاصة في مناطق إعداد الطعام. يمكن أن تتفاقم أعراض الجهاز التنفسي لدى الأشخاص الذين يعانون من التحسس عن غير قصد، لعدة أشهر.

الكلمات الدالة (المفاتحية): الحساسية الغذائية والقصبات الهوائية، كوفيد- 19، عامل نمو بطانة الأوعية الدموية، الليبوكالين المرتبط بالجيلاتيناز، إنترلوكين 38 ونسبة الخلايا الليمفاوية المتعادلة.

1. Introduction

It is well known that the prevalence of allergic diseases in the industrialized world has increased in recent decades probably due to environmental and personal interactions because this increase in prevalence occurred over a relatively short period of time, so genetic variation cannot explain the phenomenon [1]. Thus, other factors external to the individual (i.e., the natural environment) appear to be important. Thus, lifestyle, certain allergens and environmental exposure to indoor (e.g., tobacco smoke) and outdoor (air pollution) pollutants or pollutants, or microorganisms interventions may account for the increases observed worldwide [2].

The concept of "allergy" covers a wide spectrum of symptoms, from life-threatening anaphylaxis to minor seasonal allergic rhinitis. Other allergic signs include eczema, active airway infections/asthma, and rashes [3]. Anaphylaxis need rigorous allergy avoidance and immediate treatment after exposure, whereas non-life-threatening responses and impulses can be handled in a number of ways. Avoiding allergies and focusing on exercise and diet can have a big influence on symptoms [4].

There are a few environmental factors to consider with allergies. It appears that exposure to allergens from infancy and early childhood may contribute to the development of the immune system to protect against allergic problems [5]. Lack or decrease (for example, higher usage of antibacterial soap and hand sanitizer) might increase vulnerability to allergens such as asthma, whooping cough, and allergic rhinitis. This is the foundation for the so-called "purity hypothesis." For example, keeping cats can prevent the development of asthma in atopic patients, whereas owning dogs protects against allergic asthma [6].

COVID-19, which is caused by the SARS-CoV-2 virus, was first detected in Wuhan, Hubei province, China, in December 2019. COVID-19 was originally known as 2019 Novel Coronavirus (2019-nCoV) respiratory illness until February 2020, when the World Health Organisation (WHO) officially named it. A new respiratory ailment, Coronavirus ailment 2019 (COVID-19), is spreading internationally, and there have been cases of COVID-19 community spread in the United

States [7]. Respiratory virus infection is a leading cause of worldwide pandemics due to its rapid spread through the respiratory tract. Coronaviruses and influenza viruses have attacked the world multiple times over the last two decades, causing substantial deaths, economic loss, and global worry. In 2002, the SARS outbreak killed 916 people out of nearly 8000 in 29 different nations. However, the introduction of MERS in 2012 resulted in at least 800 fatalities among 2254 people from 27 nationalities [8].

Allergy and immunology clinics quickly coordinated adjustments based on the guidelines' recommendations for the COVID-19 pandemic, while simultaneously trying to meet academic/health care demands and ensure the safety of medical staff and patients [9]. However, it is unclear if allergists in our nation follow specialty-specific guidelines for COVID-19. Understanding allergists' management strategies and being aware of latest guideline recommendations will aid in the implementation of a standardised protocol for allergy and clinical immunology clinics during pandemics [10].

2. Allergy and immune system

An allergy is a severe reaction involving immunological responses mediated by antibodies and/or cells. It often arises in reaction to non-pathogenic medications. The severity of an allergic reaction may differ based on the allergen dose involved, the allergen's route of entrance into the body, the presence of other outside microbes, and the individual's genetic profile [11]. IgE-mediated pathways are responsible for the great majority of allergy and inflammatory diseases. After multiple studies demonstrated that neutrophilic inflammation was associated with asthma severity, researchers began to look into neutrophil migration to the site of inflammation. A recent research found that the cytokine IL-17 produced by Th17 cells plays a key role in neutrophilic inflammation. Studies employing animal models demonstrated that Th17 cells, coupled with T cells, induced neutrophilic inflammation and improved airway sensitivity [12].

Neutrophils and lymphocytes are blood cells that play an important part in inflammatory responses. The number of these cells may change during inflammation. When an allergen is recognised, neutrophils are the first to

go to the site of inflammation. Neutrophils, which are produced in bone marrow and have a limited lifespan, move to the site of inflammation after entering the circulation in roughly an hour [12]. This increases the concentration of neutrophils in the circulation. The neutrophil-to-lymphocyte ratio (NLR) has been identified as a prognostic indicator and predictor of inflammation in a variety of conditions, including cardiovascular disease and cancer. However, the mechanism by which the NLR changes in allergies and other inflammatory disorders is unclear [13].

2.1 Allergic rhinitis

Allergic rhinitis is the most common kind of allergy in the world. Rhazes was the first to describe the disease, followed by Clemens Von Pirquet, who detailed the immunological response, and Charles Blakeley, who identified pollen as the source of hay fever. The diagnostic criteria for allergic rhinitis are ambiguous. The symptom score, which incorporates quality of life and sleep assessments, is used to calculate subjective severity. Acoustic rhinometry, rhinomanometry, and peak expiratory flow metres are all objective measurements. The skin prick test (48.7%) has a better positive predictive value in epidemiology than the RAST IgE test (38.6%). The former category of seasonal/perennial rhinitis has been replaced with intermittent/persistent rhinitis [14]. Cleanliness, vaccination, rural living, and infection all have a deleterious impact on allergic rhinitis. Hay fever is mostly caused by pollen from trees (Alder, Birch, Cider, Pinus), grasses, and weeds [14].

Rhinitis is an inflammation of the nasal epithelium that causes symptoms including sneezing, nasal blockage, and/or itching. Allergic rhinitis (AR) is the most prevalent kind of non-infectious rhinitis, characterised by an IgE-mediated immunological response to allergens. Allergic rhinitis is a worldwide health issue that causes major illness and damage. AR triggers nasal and systemic inflammation [15]. Few studies evaluate systemic inflammation in allergic rhinitis [16]. Previous studies, despite high sensitivity C-reactive protein (CRP), CRP, fibrinogen, inflammation in cardiovascular diseases, hypertension, diabetic mellitus, familial Mediterranean fever, hepatic cirrhosis, and cancer, suggested that NLR has a predictive value [17]. They understand a bit about NLR in allergic disorders.

These investigations were mostly conducted on asthmatic patients, and the results varied. There is also systemic inflammation in AR. As a result, we hypothesise that NLR, which signals systemic inflammation, is greater in children with allergic rhinitis and may serve as an inflammatory marker [18].

2.2 Food Allergy

Food allergies are serious health issues caused by an inappropriate immunological reaction to certain foods. When a person with a food allergy consumes or comes into contact with a specific food protein, their immune system incorrectly recognises it as hazardous and causes an allergic reaction. These responses can vary from moderate to severe, and in extreme circumstances, life-threatening [19]. Peanuts, tree nuts (such as almonds, walnuts, and cashews), milk, eggs, soy, wheat, fish, and shellfish are among the most common food allergies. These allergens can cause a wide range of symptoms, including skin reactions (like hives or eczema), gastrointestinal symptoms (including variables such as nausea, vomiting, or diarrhoea), respiratory symptoms (e.g. as coughing, wheezing, or difficulty breathing), and cardiovascular symptoms (such as low blood pressure or fainting) [20]. Anaphylaxis is one of the most severe forms of food allergies, a potentially fatal allergic reaction that need emergency medical intervention. Anaphylaxis can result in a quick and severe drop in blood pressure, trouble breathing owing to airway edoema, rapid pulse, dizziness or fainting, and loss of consciousness. Prompt delivery of epinephrine (adrenaline) via an auto-injector (such as an EpiPen) is critical to reverse the symptoms of anaphylaxis and avoid future problems [21] . Food allergies can be treated by rigorously avoiding trigger foods and paying close attention to ingredient labels while purchasing or consuming foods. Individuals with food allergies should also be prepared to respond rapidly to allergic responses by carrying an epinephrine auto-injector and having an emergency action plan in place[22] . It is critical that persons with food allergies educate themselves, their families, and their carers about their disease, recognise the signs and symptoms of an allergic response, and seek medical help as needed. Furthermore, continuing research is being performed to investigate new treatments and therapies

for food allergies, including oral immunotherapy and other desensitization techniques [23] .

3. Cumulative Incidence of COVID-19 and Allergy

According to studies, those with allergies may be more prone to COVID-19. For starters, people with inhalational allergies may suffer from persistent airway and skin irritation, making them more susceptible to infections [24]. Second, the administration of systemic immunosuppressive medicines may augment this vulnerability, as found in other patient groups utilizing immunosuppressive drugs [25]. Third, SARS-CoV-2 has been demonstrated to stimulate mast cells and eosinophils, potentially putting patients with mast cell illnesses at risk for severe COVID-19 [26]. Only a few researches with inconsistent findings have been published on the prevalence of COVID-19 among people with an atopic temperament. Individuals with allergic rhinitis and asthma are more sensitive to COVID-19 and have more serious clinical consequences (ICU hospitalization, invasive ventilation, or death) [27]. Allergic rhinitis doesn't raise the risk of Covid-19. However, these researches do not assess the possible impact of more stringent social isolation measures. Patients with allergic illnesses may be more cautious and hence adhere to isolation guidelines more rigorously[28].

4. Cytokine Dynamics and Progression

Cytokine properties play an important role in the pathophysiology and progression of allergic rhinitis (AR) and food allergy. Allergic rhinitis, commonly known as hay fever, is an inflammation of the nasal mucosa, triggered by allergens such as pollen, dust mites, pet dander, etc. and certain foods [29].

The inflammatory response is regulated by cytokines, which are tiny proteins that govern immune cell interactions from the outside [30]. In allergic rhinitis, exposure to allergens stimulates cytokines such as interleukins (IL-4, IL-5, IL-13), tumor necrosis factor- . alpha (TNF-α), and interferon-gamma (IFN-γ) These cytokines promote inflammation, eosinophils , also recruit mast cells, and stimulate IgE antibodies which are a major factor in allergy [31].

Similarly, in food allergies, inges-

tion of food allergens activates immune cells and releases cytokines such as IL-4, IL-5, IL-13 , IL-17 etc. release These cytokines induce allergic reactions in the gastrointestinal tract, skin, and other organs , ranging from mild diarrhea and ulcers to severe TB The signs are [32].

Cytokine production capacities and interactions with various immune cells contribute to the persistence and severity of allergic diseases. An imbalance in the cytokine profile is often observed, with Th2-type cytokines such as IL-4 and IL-5 being predominant in allergic conditions. In addition, the role of regulatory T cells (Tregs) and their anti-inflammatory cytokines such as IL-10 and transforming growth factor beta (TGF-β) in modulating the inflammatory response is gaining attention [33].

Understanding the complex cytokine interactions of allergic rhinitis and food allergy is important for the development of targeted therapeutic interventions New therapies aimed at modulating cytokine function, e.g monoclonal antibodies targeting specific cytokines or their receptors promise to improve symptom management and quality of life in individuals living with these allergic conditions [34]. In addition, elucidation of cytokine properties may pave the way for tailored therapeutic strategies tailored to the human immune system, leading to more effective and more precise oral management strategies allergy and food allergy management [35].

The development of cytokines is critical in the immunopathogenesis of COVID 19. The initial immune response occurs when viral antigens enter pulmonary epithelial cells, predominantly via the immune system's innate immune modules. The entrance of virus particles into lung epithelial cells induces apoptotic and necroptotic pathways, resulting in lung damage and the release of several chemokines [36].

5. Molecular Mechanisms of Inflammatory Cytokines in Diseases

Inflammatory cytokines play a significant role in the pathophysiology of allergic rhinitis (AR) and food allergy by coordinating the molecular cascade of inflammation and tissue destruction. These cytokines are triggered by a variety of immune cells as signalling molecules, and they facilitate communication between cells and tissues of the immune system [34]. Understanding

the molecular mechanisms underlying the actions of inflammatory cytokines in allergic diseases is important to unravel the complex pathology and identify potential therapeutic targets [37].

AR and food allergies involve abnormal immune responses to harmless environmental or dietary antigens, characterised by T helper 2 (Th2) cell activation and tumor-promoting factors such as IL-4, IL-5, IL-13, and TNF-α. These cytokines activate effector systems that contribute to the clinical symptoms of allergic disorders [38].

At the molecular level, IL-4 plays an important role in the IgE class switch in B cells, leading to the production of allergen-specific IgE antibodies These IgE antibodies bind to high-affinity receptors on mast cells and on basophils, sensitizing these cells to subsequent allergen exposure [39]. Engagement of IgE receptors following allergen re-exposure releases preformed mediators such as histamine and produces lipid mediators, prostaglandins and leukotrienes, which induce vasodilatation, pulmonary vasoconstriction and mucus which is the result of [40].

Another key cytokine in allergic inflammation, IL-5 is required for the recruitment, activation and survival of eosinophils, which are key effector cells in allergic diseases Eosinophils release cytotoxic granule proteins and pro-inflammatory cytokines, causing tissue damage and inflammation continues [31].

IL-13, which is closely linked to IL-4, contributes to allergic inflammation by increasing mucus production, airway hyperactivity, and muscle remodeling. It induces goblet cell hyperplasia and hypertrophy, which increases mucus secretion, and stimulates the expression of adhesion molecules and matrix metalloproteinases, facilitating tissue remodeling and fibrosis [41].

In addition, TNF-α, a pleiotropic cytokine produced by a variety of immune cells, contributes to inflammatory bowel disease by causing leukemia, permeability, and tissue damage by inducing the production of cytokines and other chemokines, as well as pro-inflammatory pathways [42].

Inflammatory cytokines and molecular mechanisms associated with food allergy in AR involve complex interactions between immune cells, endothelial cells, and epithelial cells a it is located between the involved tissues Failure to regulate these molecular pathways leads to chronic inflammation, tissue

damage, and continued symptoms of asthma [43]. Targeting these molecular pathways with novel therapeutic strategies, such as biologics targeting specific cytokines or their receptors, holds promise for better disease control and improved quality of life in individuals with epilepsy in allergies and food allergies [44].

Increased cytokine production is linked to severe pyrexia, blood leakage, the creation of numerous blood clots, and pleural effusions. Acute Respiratory Distress Syndrome (ARDS) is a frequent COVID-19 complication caused by increased cytokine production. During an episode of ARDS, proinflammatory cytokine and chemokine titers skyrocket, as do virus titers. COVID-19 patients exhibit elevated levels of IL-1B, IFN-γ, MCP-1, and IP-10 [45]. COVID-19 patients in the ICU showed higher levels of blood MCP-1, IP-10, TNF-α, macrophage inflammatory protein-1A, and granulocyte colony-activating factor compared to ordinary wards. The results of these research may imply a positive link between cytokine storm and illness severity [46].

6. The Impact of COVID-19 Inflammatory Cytokine Response on Different Organs

SARS-CoV-2 activates the innate immune response, causing neutrophils and other immune cells to quickly rise while T lymphocytes (CD4+ and CD8+) decline dramatically. However, the loss of T cells, together with an increase in IL-6 and IL-8, has been recognised as a critical characteristic of SARS-CoV-2 infection [47]. Patients with an inborn impairment in IFN type I immunity and compromised immunity produce an excess of inflammatory cytokines, culminating in a cytokine storm known as hypercytokinemia. Symptoms of dysregulated and excess cytokine production include severe pneumonia, acute lung injury, and acute respiratory distress syndrome (ARDS) [48].

The systemic inflammatory response, along with other co-morbid factors, may lead to a number of complications, such as heart failure, impaired renal function, liver damage, and various organ disruption; COVID-19 patients with a history of cardiovascular illness have an exceedingly poor prognosis [49]. According to a

comprehensive investigation of COVID-19 effects, people with a history of hypertension (17%), hyperglycemia (8%), and heart disease (5%) are more likely to experience severe COVID-19 consequences than those with no history of chronic medical conditions. Another study found that 7.2% of CVD patients with COVID-19 had elevated troponin levels and electrocardiogram (ECG) abnormalities, indicating probable heart injury [50].

7. Significance of Cytokine Storm

Hypercytokinemia is an uncontrolled hyperinflammatory response that occurs when a localised inflammatory reaction to a viral or bacterial infection spreads across the body. Elevated cytokine levels induce endothelial dysfunction, vascular system damage, and paracrine/metabolic dysregulation, all of which impact several organ systems [51]. Hypercytokinemia increases acute-response cytokines (TNF and IL-1β), chemotactic cytokines (IL-8 and MCP-1), and IL-6 levels over time. IL-6, like other pleiotropic cytokines, induces an acute phase response by increasing serum ferritin, complement, CRP, and procoagulant factors, all of which may be detected using commercially available blood tests. The acute phase response to a cytokine storm has improved. High serum cytokine levels are negatively related with the total lymphocyte count, therefore low numbers of cytotoxic T cells may lead to poor viral clearance [51]. Blocking upstream systems implicated in cytokine responses, such as macrophage JAK-STAT signalling to limit IL-1 and IL-6 synthesis, might work as a cytokine storm inhibitor [52].

The inflammatory response destroys lymphocytes in order to avoid SARS-CoV-2 infections. Lymphopenia develops, especially in patients who are seriously sick and require ICU care [53]. Cytokine storms can activate the NLRP-3 inflammasome while decreasing histone deacetylase 2 responsiveness to the NFκB complex [54].

8. Immunosenescence and Cytokine Storm

Immunosense, the progressive decline of the immune response associated with aging, cytokine storm, uncontrolled over-release of pro-inflammatory cytokines are two important aspects of the pathophysiology clinical pathway of allergic rhinitis.

(AR) and food allergies are most likely to be affected [55].

Changes in older adults include both genetic and metabolic changes, including decreased T-cell function, intact B-cell responses, and cytokine production So these age-related changes decrease immune surveillance and increase susceptibility to infections and inflammatory disorders including acne [56].

Immune deficiencies in individuals with allergic rhinitis and food allergies can exacerbate existing immune responses, resulting in delayed or severe allergic reactions may result in older adults having reduced tolerance to allergens and response to conventional treatment due to age-related changes in immune function because they have eaten [57]. A cytokine storm, characterized by impaired release of pro-inflammatory cytokines such as interleukin-6 (IL-6), interleukin-1β (IL-1β), and tumor necrosis factor-alpha (TNF-α). non-compliance may occur in response to allergen exposure or allergic reactions. This excessive immune response can lead to systemic inflammation, tissue damage and potentially life-threatening complications[58].

For allergies and food allergies, the cytokine storm can contribute to severe symptoms such as anaphylaxis, respiratory distress and cardiac arrest [59]. Immuno-sensitive older adults may be particularly vulnerable to complications related to cytokine storms, highlighting the importance of early detection and effective management of asthma in this population emphasize [60].

Understanding the interactions between the immune system, cytokine storms, and inflammatory diseases is essential for effective patient management and targeted therapeutic strategies [61]. Methods a aimed at balancing the immune response and controlling inflammation may help to reduce the impact of the immune system and cytokine storm on allergic rhinitis of temperature and food allergies , especially In older adults [62].

Elderly people, particularly older men with comorbidities, are more likely to have a bad prognosis, have severe illness, or die as a result of COVID-19. The loss in immune function known as “immunosenescence” is connected with ageing. The immune system may undergo a series of changes with age, involving immune senescence markers, a decrease in the generation of CD3+ T cells, a reversal of the CD4-

CD8 (CD4/CD8) T cell ratio resulting from a decline of CD8+ T cells (increased CD4/CD8 ratio), a rise in regulatory T cells (Treg), and a reduce in B lymphocytes [63]. It is suggested that a COVID-19-induced cytokine storm may result in poor outcomes in the elderly due to immunosenescence. The virus is capable of attacking T cells and causing apoptosis. It is still unknown if a viral infection of lymphocytes enhances the cytokine storm or not [64]. In a recent study of 7 patients with COVID-19 pneumonia, an immunomodulatory therapeutic strategy of intravenous transplantation of mesenchymal stem cells (MSCs) was revealed to be successful, particularly in really severe instances. Immunomodulatory treatments targeting the cytokine storm have the potential to improve outcomes and reduce death from COVID-19 in aged individuals [65][66].

9. COVID-19 and Cytokine Storm

The initial cohort of 41 COVID-19 patients in Wuhan, which prompted the discovery of the novel SARS-CoV-2 virus, showed a cytokine profile similar to secondary haem phagocytic lymphohistiocytosis (sHLH), a viral-induced hyper-inflammatory condition. Patients in the intensive care unit (ICU) had higher levels of GM-CSF, IP10, MCP-1, MIP1A, and TNFα than those who were not hospitalize 67]]. Observations of another 150 people with the condition in Wuhan revealed that those who died of COVID-19 complications had higher blood levels of CRP, IL-6, and ferritin, indicating an underlying hyper-inflammatory system. As a consequence, a combination of these markers can be used to estimate the severity of COVID-19 [68].

Individuals who die from severe COVID-19 have endothelial cell infection and endotheliitis, which impact several organs. The SARS-CoV-2 S protein binds to ACE2 and infects host cells [69]. In severe cases of COVID-19, disproportionate cytokine activity in the lungs causes substantial damage to the alveoli, development of hyaline membranes, formation of blood clots in small arteries attended during autopsies, the presence of fibrin deposits, and the baptism of fibrotic healing processes. These pathological alterations result in sudden lung damage, clinically known as intense respiratory distress syndrome (ARDS) [70]. Proteinuria and haematuria are report-

ed by 40% of COVID-19 patients, indicating kidney infection and damage. The presence of ACE2 receptors in the brush border of proximal tubular cells contributes to COVID-19-induced kidney damage. Although the kidneys of COVID-19 patients analysed postmortem exhibit SARS-CoV 2 antigens in the proximal tubules, the role of the cytokine storm in inducing renal impairment is questionable [71].

ACE2 receptors can also be found in heart tissue and the gastrointestinal system, which could explain why certain COVID-19 patients exhibit clinical symptoms. Existing research suggests that those with underlying cardiovascular illness, high blood pressure, severe dyslipidemia, obesity, or diabetes are more likely to develop severe COVID-19 [72].

Conclusion

Highlight the various issues faced by people with allergies in the complex interaction between food and airway allergies in the global COVID-19 pandemic. The pandemic has not only participate to respiratory health problems, but also poses extra challenges in the management of food allergens and allergens. assimilation these diseases requires comprehensive epilepsy interference that include patient education, access to health services, and community health efforts as we address the epidemic and meet the needs of people with epilepsy , their safety, welfare and priority of basic services and backing programs Equal access must be ensured . By addressing these argument comprehensively, we can work towards a healthy and comprehensive society in which people of color can thrive despite the challenges of modern life there is the.

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and approved the overall manuscript, and approved the final version of the manuscript.

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