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Pathophysiology of skin disorders after COVID-19 infection and vaccination

Author

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Abstract

Skin is a common and important target for SARS-CoV-2 infection, and cutaneous manifestations and/or exacerbation of pre-existing diseases represent an early sign and a clue for the diagnosis. Also, several skin lesions have been reported after various COVID-19 vaccines.

SARS-CoV-2 RNA has been detected in some, but not all, tissue samples of COVID-19-related cutaneous lesions analyzed by real-time RT-PCR. So the direct invasion of the virus may be responsible for some of these lesions.

The absence of SARS-CoV-2 virus detection in many skin lesions indicates that these skin lesions related to COVID-19 may be attributed to a collateral effect of the activation of the immune system rather than being a direct effect of the virus or that these skin lesions are not related to the infection, and they are co-incidental.

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Neurological consequences of COVID-19 vaccination

Author

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Abstract

Nonetheless, all types of vaccines may be associated with the possible appearance of neurological complications, and COVID-19 vaccines are not free from neurological side effects. Neurological complications of COVID-19 vaccination are usually mild, short-duration, and self-limiting.
The most common neurological symptoms included dizziness, headache, pain, muscle spasms, myalgia, and paresthesias, which are anticipated to be acute, temporary effects of the vaccination. Though severe and unpredicted post-vaccination complications are rare, they are possible events. They include the Guillain-Barré syndrome, facial palsy, other neuropathies, encephalitis, meningitis, myelitis, autoimmune disorders, and cerebrovascular events. No neurological condition is an absolute contraindication to COVID-19 vaccination, but there are special considerations about COVID-19 vaccines for patients who take immunosuppressive medications. These patients might be at increased risk for severe COVID-19, making vaccination principally important. Yet some of these immunosuppressive drugs may reduce immune responses to vaccine antigens. So, the timing of the vaccine may play an essential role in balancing the immune response in these patients.
Overall, the benefits of COVID-19 vaccination far outweigh the risks of neurological complications. Future research will be needed to find any association between COVID-19 vaccines and neurological complications.
This review proposes to gather and discuss the frequency, management, and outcome of reported immune-mediated neurological complications of COVID-19 vaccines, including Guillain-Barré syndrome, myasthenia gravis, myositis, and neuropathies.

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Early and Late-Onset Dermatological Manifestations of COVID-19

Author

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Abstract

The most commonly reported COVID skin manifestations are morbilliform rash, pernio-like acral lesions, urticaria, macular erythema, vesicular eruption, papulosquamous eruption, and retiform purpura.

These skin lesions are early skin manifestations of COVID, and most of them concurrently occur with other symptoms.

In several case series, a morbilliform rash predominantly involving the trunk has been reported as the most common cutaneous manifestation of COVID-19. The rash has been noted either at the disease onset or, more frequently, after hospital discharge or recovery.

In patients with confirmed or suspected SARS-CoV-2 infection, pernio lesions started one to four weeks after the onset of COVID-19 symptoms, suggesting a post-viral or delayed-onset process.

Acute urticaria with or without concomitant fever has been reported as a presenting sign of COVID-19 infection.

There are several reports describing a vesicular-pustular, varicella-like eruption associated with COVID-19. In a series of 24 patients, an eruption of small papules, vesicles, and pustules appeared 4 to 30 days after the onset of COVID symptoms and resolved in a median of 10 days.

Less frequently reported early dermatologic manifestations to include papulosquamous eruptions, erythema multiforme-like lesions, dengue-like rashes, petechiae, and gangrene.

It appears that patients who have suffered from SARS-CoV-2 infection do not seem to show specific dermatological manifestations referable to the 'Long Covid syndrome' except for Telogen Effluvium.

Abstract

In addition, patients who experienced telogen effluvium had their peak hair loss approximately 3 months after infection, which would confirm that telogen effluvium is caused by an interruption of the anagen phase of the hair cycle (Late Onset Telogen effluvium). The prevalence of post-COVID telogen effluvium is more frequent in females.

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Inflammatory Mechanisms in MIS-C vs. Other Infectious or Autoimmune Diseases

Author

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Abstract

Multisystem inflammatory system in children usually occurs 2-4 weeks after COVID-19 infection in children—this phenomenon is known as the cytokine storm with multi-organ involvement, especially gastrointestinal and cardiac involvement. The usually negative COVID-19 PCR in MISC demonstrates the excessive immune system responses to previous virus infections. Macrophage activation syndrome in rheumatologic and infectious background disorders is an inflammatory process with activation of the immune system resulting in a critical situation caused by the cytokine storm.
In the acute phase of Kawasaki disease, cytokine waterfall is initiated by a Stimulator of interferon genes (STING) pathway inhibited by aspirin and intravenous immunoglobulins.
The same mechanism in COVID-19 happens by binding to ACE2 increases STING pathway activation.[1,2,3,3]
It is contrary to adult covid-19 pneumonia with respiratory failure with decreased type I IFN responses subsequent activation of pro-inflammatory cytokines (IL-1, IL-6, IL-8, TNF, etc.) and Small vessel thromboembolic disease.[1]
So, to respond to the question of why some children present COVID-19 as a multisystem inflammatory syndrome and some present it as a cold, the genetic signature may have an important role in the immune system response to SARS-COV2 infection.

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Post-COVID Depression and Its Challenges

Author

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Abstract

Due to the emergence of the COVID-19 pandemic and numerous research conducted in various centers around the world, it has been determined that there is a two-way relationship between contracting COVID-19 and short-term and long-term serious psychiatric consequences. Among these consequences, anxiety disorders and depressive disorders can be mentioned more than other disorders. The changes in the prevalence of depression reported in post-COVID research vary depending on the geographic location, time of research, and research tools used. However, in an international study of 204 regions and countries worldwide, the average increase in the prevalence of depression one year after COVID-19 was reported to be 6.27%.

In the Iranian National Study on the Evaluation of Mental Health, I also reported a prevalence of 9.24% for depression symptoms among the general population in 2020, which was significantly higher than previous numbers.

According to research and observations, the increased prevalence of depression, diagnostic skepticism, inaccuracy, rising costs, worsening course and prognosis of physical illnesses, and even increased suicide rates in communities is not unexpected in the post-COVID world. Therefore, the healthcare system should focus on a different and distinct approach to mental and social health in terms of structural and credibility aspects.

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Post covid Obsessive Compulsive Disorder (OCD)

Author

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Abstract

The emerging evidence clearly suggests that patients with severe acute respiratory syndrome coronavirus 2 show higher manifestations of various neuropsychiatric symptoms when compared to controls without COVID-19 infection.
The common psychiatric presentations among patients with COVID-19 are anxiety, depression, posttraumatic stress disorder, insomnia, and delirium.
However, obsessive-compulsive disorder (OCD) following COVID-19 infection is rarely reported in the literature; around 20% reported obsessive-compulsive symptoms 1 month after follow-up. Case reports reported new onset OCD after COVID-19 infection.
According to systematic reviews, people both with and without diagnosed OCD prior to the pandemic generally experienced a worsened landscape of symptoms of OCD during the COVID-19 pandemic. However, the responses are heterogeneous, and many factors other than the pandemic seemed to affect the development of OCD symptoms.
To prevent the impairment of symptoms and the development of new cases, close monitoring of patients with OCD and educating the general public is essential. Literature is still limited; thus, multinational and cross-cultural, longitudinal studies are warranted to gain further insights on this topic.

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The role of Antifibrotic Medications in the Treatment of Post-COVID Lung Disease

Author

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Abstract

Coronavirus disease 19 (COVID-19), caused by severe acute respiratory syndrome-virus 2 (SARS-CoV-2), is a multisystem disease, but the lungs are the primary target of infection and injury.
Pulmonary fibrosis is a recognized long-term sequel of several coronavirus lung infections.
The SARS-CoV-2 virus may induce lung fibrosis by at least four proposed mechanisms, namely: (1) direct stimulation of transforming growth factor-beta (TGF-b), a profibrotic cytokine; (2) COVID-19 ARDS causing lung fibrosis; (3) mechanical stretch of alveolar epithelial cells during mechanical ventilation causing a fibrotic response; and (4) presence of excess oxygen-free radicals due to prolonged use of high oxygen causing an oxidative stress-induced fibrogenic response.
Rapid onset of honeycombing fibrosis in a patient with COVID-19 identical to IPF is rare but has been reported.
Honeycombing, distorted lung architecture, and traction bronchiectasis are radiological features that may prompt considering empiric antifibrotic therapy, while ground-glass opacities, organizing pneumonia, and pleural tags are not features for starting antifibrotic drugs for post-COVID-19 ILD.
There is very little to choose between nintedanib and pirfenidone to treat post-COVID-19 ILD, although both drugs are not yet licensed for this condition. A few clinical trials are currently investigating the efficacy of these drugs in post-COVID-19 ILD. We will have to wait until then to make any recommendations.

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Psychological Consequences of COVID-19 in Children and Adolescents

Author

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Abstract

Although children and adolescents did not show severe pictures of COVID-19 as much as adults did, the direct and indirect psychological consequences of this event need to be considered in this age group. During the pandemic, children and adolescents experienced several lifestyle changes, including home-based education, social isolation, limited outdoor activity, and experiencing fear and stress. These changes have been shown to cause some psychological disturbances, including anxiety, depression, obsessive-compulsive disorder, inattention, irritability, and aggression. Moreover, since brain development during childhood has an important role in children's behaviors, emotions, and cognition, the acute and chronic stressors during the pandemic, apart from the inflammation consequences, can influence brain development and lead to psychopathology in this age group. In addition, youth with a history of psychiatric disorders, including autism spectrum disorder, ADHD, and anxiety, had an exacerbation in their symptoms during the pandemic.

In conclusion, the acute and especially chronic psychological consequences of the COVID-19 pandemic in children and adolescents must be considered.

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Covid-19 Vaccination Challenges in Iran & the World Author

Author

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Abstract

Immunization during childhood is a global investment to save the lives of about 2.6 million people worldwide every year. Despite the very good progress at the global level, unfortunately, according to the report of the World Health Organization in 2019, 19 million children still are deprived of receiving necessary vaccines; most of them live in developing countries.

The role of vaccines in eradicating, eliminating, and controlling childhood infectious diseases has been fully proven. However, vaccines are not only for childhood; many vaccines have been used in adults. In dealing with emerging and re-emerging diseases or pandemics, vaccination has played an important role among the other measures to control the extent and severity of disease complications.

Due to the rapid development of epidemics with high lethalities, such as bird flu, developing an effective and safe vaccine is one of the main challenges. Fortunately, with the advances made in medical science, the identification of the desired microorganism, the knowledge of the pathogenic mechanism, the body's immunological response, and finally, the acquisition of candidate antigens/antigens for vaccine development have gained significant speed.

In response to the pandemic, producing and providing an effective and safe vaccine is the first step. However, for the success of vaccination, the amount of vaccine production must be very high to reach all people and target groups in all countries, including high-income countries, with low income. This requires investing in the development of manufacturing factories and achieving new production methods in a shorter time and a larger amount. The experience of the Covid-19 pandemic and the production of various vaccines is very valuable. Despite the efforts of international health

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organizations led by the WHO for the fair access of all countries to the Covid-19 vaccines, unfortunately, in developing countries, especially in Africa, we saw the lowest vaccine use.
In addition to the need to change the regulatory rules for vaccine production to accelerate vaccine availability, creating a culture to accept the vaccine is also one of the most important points for the maximum use of the vaccine by the people and the target society. Despite the clear gains of immunization, anti-vaccine groups are still active. Although the activity of anti-vaccine groups began in the mid-19th century against the compulsory vaccination against smallpox, it continues. In 2021, the World Health Organization raised vaccine hesitancy as one of the most important health challenges in the world.

COVID-19, Hypertension, Potassium, ACE-2 and Inflammation

Author

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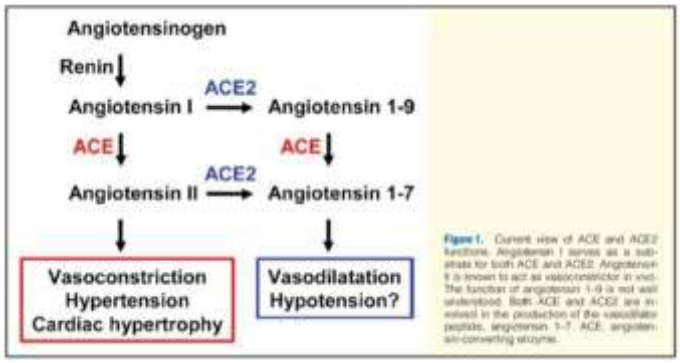
Abstract

Introduction

SARS-CoV, like the virus, is currently circulating in wildlife for a long time. It has a spike protein, which binds to the human ACE2 in lung epithelial cells & other tissues and enters host cells.
Different cells have different expression shapes and levels of ACE2. This polymorphism can describe the different susceptibility and outcomes of COVID-19.[1]



Hypertension, D.M. and CVD in Covid-19

AK Singh et al. believe there is a high prevalence of HTN, D.M. & CVD in COVID-19, and there is evidence of increased mortality with these comorbidities in COVID-19. They discovered a clinical equipoise on these patients' harm or benefit with RAS blockers. From the pooled data of all ten available Chinese studies (n = 2209) that have reported the characteristics of comorbidities in patients with COVID-19, HTN was present in 21%, followed by D.M. in 11%, & CVD in 7% of patients. Data hints at increased mortality in COVID-19 with HTN, D.M. & CVD. However, it was not adjusted for multiple confounding factors. [2]

Abstract

ACE2 in Covid-19

After that, attention was drawn to ACE2 & the role of RAAS & RASB as mechanistic equipoise. It remains unknown whether the treatment of HTN influences the mortality of COVID-19. A retrospective observational study of all patients admitted with COVID-19 to Huo Shen Shan Hospital was done (n=2877); 29.5% (850/2877) had a history of HTN. In this study, patients with HTN had a two-fold increase in the R.R. of mortality compared to patients without HTN [4.0% vs. 1.1%, adjusted HR 2.12, 95% CI 1.17–3.82, P = 0.013]. Patients with HTN and without antihypertensive treatment (n = 140) have a significantly higher risk of mortality compared to those with antihypertensive treatments (n = 730) (7.9% vs. 3.2%, adjusted HR 2.17, 95% CI 1.03–4.57, P = 0.041). In a few documents, the mortality rates were similar between the RAAS inhibitor (4/183) and non-RAAS inhibitor (19/527) cohorts (2.2% vs. 3.6%, adjusted HR 0.85, 95% CI 0.28–2.58, P = 0.774). However, a meta-analysis showed that patients with RAAS inhibitor use tend to have a lower risk of mortality (RR 0.65, 95% CI 0.45–0.94, P = 0.20). HTN & discontinuation of antihypertensive treatment is suspected to be related to increased mortality risk.[3]

After that, clinical & immune-inflammatory parameters were compared between non-severe & severe COVID-19 by Zhu, Z. et al. in a study in which 127 consecutive hospitalized patients with COVID-19 were enrolled in and classified into non-severe and severe groups. In this study, high-level IL-6, CRP & HTN were independent predictors for severity. IL-6 has the highest predictive value among other single immune-inflammatory parameters. The dynamic change of IL-6 in severe cases was parallel to the amelioration of this disease. Of 127 patients, 16 (12.60%) were severe. The baseline IL-6 was positively correlated with other immune-inflammatory parameters, and dynamic changes of IL-6 in severe cases were parallel to the amelioration of the disease.[4]

Yang, G., et al. show that by inducing elevated expression of ACE2, the cellular receptor for severe acute COVID, ARB or ACEi may have a controversial role in both facilitating virus infection & reducing pathogenic inflammation. In their study, 126 COVID-19 & pre-existing HTN patients were retrospectively allocated to ARB/ACEi group (n=43) & non-ARB/ACEi group (n=83); also 125 age- & sex-matched COVID-19 were randomly selected as non-hypertension controls. They show that ACE2 plays an important role in non-classical RAS pathway. The RAS blockade is known to increase ACE2 levels. Some reports have indicated a protective effect of RAS blockade on COVID-19. In contrast,

Abstract

others have reported an association of RAS blockade therapy with severe complications, such as AKI & admission to the ICU.

Electrolyte Disorders and Inflammation in COVID-19 in Covid-19

Electrolyte disorders are common in severe COVID-19 (hypokalemia, hyponatremia & hypocalcemia). They are caused by alteration of RAS, G.I. loss, effects of pro-inflammatory cytokines & tubular dysfunction.[5] ARB/ACEi group had significantly lower concentrations of hs-CRP (P=0.049) & procalcitonin (P=0.008), a lower proportion of critical patients (9.3% versus 22.9%; P=0.061), & a lower death rate (4.7% versus 13.3%; P=0.216) as well; although these differences failed to reach statistical significance. Our findings thus support ARB/ACEi in patients with COVID-19 and pre-existing HTN.[5]

The pro-inflammatory response is present in cardio-metabolic disease CMD (Obesity, T2DM, HTN & CVD) & affecting health during SARS-CoV2. Targeting inflammatory biomarkers could be promising for the early identification of patients at risk of adverse outcomes. About 50% of patients with CMD have an increased risk of morbidity & mortality in COVID-19. It has been reported in most countries affected by the SARS-CoV-2 virus.[6] Underlying HTN is likely associated with severe COVID-19, but the relationship is unclear due to confounding factors.[7] Hallmarks of COVID-19 are over-activation of the immune system with a prominent IL-6. Also, IL-2, IL-4, IL-8, and IL-10 interferon-gamma were strongly predictors of COVID-19 severity.[7]

in a study, Silhol F. et al. show that hypertension appears to be an independent factor for severity in COVID-19. The RAS is a hemodynamic and biological system that regulates Bp, plasma K+, and the stability of pulmonary epithelial membranes. They show that two antagonistic pathways are balanced; the first is the angiotensinogen pathway that transforms angiotensinogen into angiotensin I (by renin), and then converts it into angiotensin II by ACE. Angiotensin II attaches to angiotensin II type 1 receptor (AT1R) and activates the system to induce vasoconstriction, aldosterone secretion stimulation, hypokalemia, and pulmonary epithelium degradation. The second way in which the angiotensin system is balanced involves ACE2. This pathway transforms a part of angiotensin I & II before it attaches to its AT1R receptor. The AT I & II phosphorylation products are angiotensin 1–9 & 1–7. They attach to the AT II 2 receptor receiver, inducing antagonistic effects compared to AT1R. COVID-19 virus uses the ACE2 and penetrates the host cell. Coronavirus binding with ACE2 leads to

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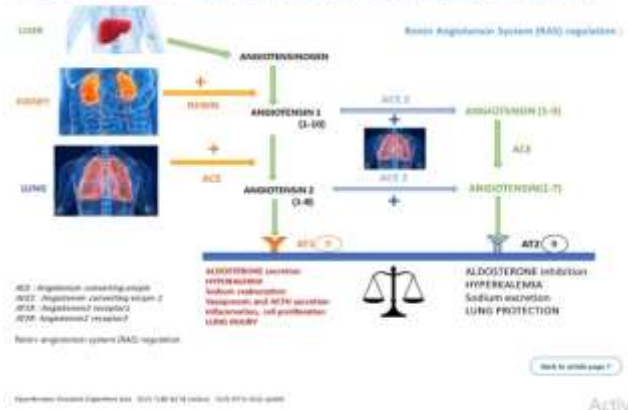
downregulation of ACE2, contributing to an increase in AT2 through ACE, as the decrease in ACE2 results in a lower conversion of AT to AT 1–7 vasodilator. The lower the level of ACE2, the lesser AT I & II will be degraded; thus, their plasmatic concentration gradually increases. A US ICU team demonstrated that an increase in AT 1–10 and a decrease in AT 1–9 (its ACE2 processing product) were correlated with a poor prognosis in ARDS. Other effects include increased secretion of aldosterone, hypokalemia induced by kaliuresis, and increased sodium reabsorption & inflammation. Hypokalemia is frequently found in patients with COVID-19 and is associated with poor outcome. Conversely, RAS blockers can increase ACE2 and potentially promote virus loading into the cell. Major imbalance in RAS induced by the downregulation of ACE2 is an essential element of unfavorable evolution in patients with COVID-19. The biological marker of this imbalance appears to be hypokalemia. Several studies in influenza & Ebola have shown the beneficial role of AT1R blockers on lung damage, with a decrease in inflammation & cytokines. In two animal studies, losartan demonstrated an increase in ACE2 expression. Clarification is needed to determine whether blockers of the angiotensin system have a protective or harmful effect in these patients.[8] Emerging evidence demonstrates that older individuals & people with underlying metabolic conditions of D.M., HTN, & HLP are at higher risk of morbidity & mortality. it is known that in metabolic disorders & in old age, there is an upregulation of ACE-Ang-II-AT1R axis with a Downregulation of ACE-2-Ang-(1–7)-Mas axis. The activated ACE-Ang-II-AT1R axis leads to pro-inflammatory and profibrotic effects in respiratory system, Vascular dysfunction, myocardial fibrosis, nephropathy & Insulin secretory defects with increased Insulin resistance. The ACE-2-Ang-(1–7)-Mas axis has anti-inflammatory & antifibrotic effects on the

Fig. 1



Abstract

respiratory system, antioxidative stress, and protective effects on vascular function. It protects it against myocardial fibrosis, nephropathy, pancreatitis & insulin resistance. [9]

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Screening and Treatment of Cancer During the COVID-19 Pandemic

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Abstract

Delays in cancer screening during the pandemic led to delayed diagnoses, a higher rate of patients diagnosed in an emergency setting, more diagnoses of later-stage cancers with higher tumor burden, and delays in effective treatment for patients with newly diagnosed malignancies.
Observational data suggest that cancer-specific mortality was higher during the COVID-19 pandemic when compared with prepandemi levels.
During the pandemic, specific recommendations about cancer screening and diagnostic/surveillance testing should be based on the extent of community transmission as well as the availability of resources.
Many screening programs have resumed in areas where the infection has been relatively controlled.
Hematologic malignancies, lung cancer, advanced or progressive cancer, active chemotherapy, older age, and comorbid conditions are risk factors for severe COVID-19. Prior cancer is also a risk factor, but the risk is lower compared with active cancer.
COVID-19 disease management is similar to the management used for the general population. However, cancer is considered a risk factor for progression to severe COVID-19 infection, influencing available treatment options.
For most cancer patients with COVID-19, chemotherapy or immunotherapy should be interrupted, whether patients are symptomatic from COVID-19.
Cancer treatment will be resumed once transmission-based precautions can be discontinued; the regional and institutional protocol often determines the duration of such precautions.

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Covid-19 Infection and Anxiety: A Bio-Psycho-Social Perspective

Author

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Abstract

Anxiety is a common feature in both usual and long-term Covid-19 infections. There is a kind of overlap between symptoms of anxiety and Covid-19 infection. For example, symptoms like dyspnea, tachycardia, and muscle tension may see in both conditions. We can consider a bio-psycho-social model for explaining the etiology of anxiety in Covid-19 infection. Inflammation, the release of cytokines, and hypoxia are common biological mechanisms that entail the activation of stress reactions. Furthermore, anxiety can emerge in certain conditions like delirium, pneumonia, and thromboembolic events, which are multifactorial in terms of etiology.

From a psychological stance, the coping styles of patients with Covid-19 are an important factor in the incidence of anxiety symptoms. Worrying about the exacerbation of the disease and media news about the increased mortality of the Covid-19 infection may have a role in faulty coping mechanisms. From the social perspective, stigma and social isolation of patients with Covid-19 infection may have an etiological role in the emergence of anxiety in these patients.

On the other hand, pharmacological interventions like corticosteroids and immune modulation in patients who suffer from severe Covid-19 infection may cause anxiety in some patients.

For the above reasons, the management of anxiety was considered an important section in most of the national guidelines for managing Covid-19 infection, including the Iranian National Guideline.

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Epidemiology and Future Perspectives of Covid-19: Asthma and Allergy standpoint

Author

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Abstract

In December 2019, a cluster of cases of severe viral pneumonia was identified in Wuhan, China, which was rapidly spreading to become a pandemic. The Coronavirus disease 2019 is commonly known as Covid-19. Globally, by February 3, 2023, more than 750 million cases (Iran: more than 7.5 million cases) and 6.8 million deaths (Iran: more than 144,000 deaths) have been reported to WHO. One of the most important questions to be addressed in the context of the Covid-19 pandemic is understanding the risk factors for disease severity. Risk factors for severe Covid-19 include older age, male gender, disability, and pre-existing comorbidities such as obesity, cardiovascular disease, respiratory disease, and high blood pressure. Asthma and allergic diseases are not associated with greater severity and mortality from Covid-19, apart from severe/poorly controlled asthma. Most experts recommend continuing maintenance therapy to prevent asthma exacerbations during the Covid-19 pandemic. Maintaining optimal asthma control should be able to reduce the risk of severe sequelae after covid-19 illness. Risk factors for developing long-term Covid-19 have received less attention. Asthma has not been reported as a risk factor for hospitalization; however, recent studies have shown that the preexistence of asthma is more common in people with prolonged covid-19 symptoms and worsening asthma control are common in people with asthma, while eosinophilic and T2 - asthma can protect against long-term complications of Covid-19.

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Post-Acute Neurological Consequences of COVID-19 in adults

Author

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Abstract

The first case of COVID-19 infection was reported three years ago in Iran. A short time after that this infection spread so fast that it resulted in Global Pandemic, causing millions of Deaths.
The respiratory system is the main target of COVID-19 infection, but the central and peripheral nervous systems could be involved directly or indirectly. Damage to neurons may cause long time deficits. For example, patients may suffer from anosmia after infection for a long duration.
Now after decreasing number of acute cases of infection, there are increasing reports of post-acute neurological consequences of COVID-19 infection.
In this presentation, first, I discuss the evidence of direct invasion of neurons by SARS-CoV-2.
There is more evidence of indirect damage to the nervous system by activating the immune system and thrombotic processes.
Considering the large number of people with a history of COVID-19 infection and the high prevalence of different neurologic diseases such as headaches, degenerative diseases such as Alzheimer's disease, stroke, we need definite epidemiological studies to find out any correlation between COVID-19 infection and any neurologic disease.
In one study, Evan Xu et al. (1) compared 154,068 individuals with COVID-19 one year after infection with 5,638,795 contemporary controls and 5,859,621 historical controls. They demonstrated that in the post-acute-phase of COVID-19, there was an increased risk of an array of incident neurologic sequelae, including ischemic and hemorrhagic stroke, cognition, and memory disorders, peripheral nervous system disorders, episodic disorders (for example, migraine and seizures), extrapyramidal and movement disorders, mental health disorders, musculoskeletal

Abstract

disorders, sensory disorders, Guillain–Barré syndrome, and encephalitis or encephalopathy. They estimated the hazard ratio of any neurologic sequela was 1.42. Other studies are going on and more correlations may be found in the future.

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Immunoediting in SARS-CoV-2: Mutual Relationship Between the Virus and the Host

Author

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Abstract

Immunoediting is a well-known concept that occurs in cancer through three steps of elimination, equilibrium, and escape (3Es), where the immune system first suppresses the growth of tumor cells and then promotes them toward the malignancy. This phenomenon has been conceptualized in some chronic viral infections such as HTLV-1 and HIV by obtaining resistance to elimination and making a persistent form of infected cells, especially in untreated patients. Coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), is a heterogeneous disease characterizing from mild/asymptomatic to severe/critical courses with some behavioral aspects in an immunoediting setting. In this context, a coordinated effort between innate and adaptive immune systems leads to detecting and destroying early infection, followed by an equilibrium between virus-specific responses and infected cells, which eventually ends up with an uncontrolled inflammatory response in severe/critical patients. Although the SARS-CoV-2 applies several escape strategies, such as mutations in viral epitopes, modulating the interferon response, and inhibiting the MHC I molecules similar to the cancer cells, the 3Es hallmark may not occur in all

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clinical conditions. Here, we discuss how the lesson learnt from cancer immunoediting and an accurate understanding of these pathophysiological mechanisms help to develop more effective therapeutic strategies for COVID-19.

Covid19 Renal Involvement in Pediatrics

Acute kidney injury (AKI) is an important factor affecting the outcome of hospitalized patients under any disease condition. While in previous infections such as SARS and MERS, acute kidney injury (AKI) was reported to be up to 15% with a mortality rate of approximately 60% to 90%, however, initial reports of COVID-19 suggest a lower rate in adults (3% to 9%).

Despite initial reports and background experiences of SARS and MERS cases which infection among children is rare, recent reports showed that all ages, even infants and neonates, can be infected with the virus. Renal complications are reported to be limited to severe cases in children and are mostly present as AKI in the context of multi-organ failure caused by direct virus invasion or acute inflammatory response. In a limited number of studies conducted among adults, proteinuria and hematuria were observed in patients. However, studies on children have not yet been conducted. More studies of renal complications in children with COVID-19 infections are recommended in the initial stages of this disease.

The risk of AKI (acute kidney injury) and decrease in GFR, and also abnormal urinalysis is high in children with COVID-19. So, more attention to the detection of kidney involvement is necessary, and more conservative management for the prevention of AKI and decrease of GFR is recommended.

Treatment

The management of AKI in children is based on the same principles as AKI in nonCOVID patients, however, there are important practical points to be considered. The fluid and hemodynamic assessment and exclusion of nephrotoxic medications is the first step of management. The role of antivirals, immunomodulatory agents, corticosteroids, renin–angiotensin inhibitors, statins, and anticoagulants in the prevention of AKI is not clear.

3rd International Congress on Inflammation

New Finding and Challenges of Non-Antiviral Drugs

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Abstract

The optimal approach to the treatment of COVID-19 is evolving. Trial data suggest a mortality benefit with dexamethasone as well as with adjunctive tocilizumab or baricitinib and a possible clinical benefit with remdesivir dexamethasone. Dexamethasone is recommended for severely ill patients with COVID-19 who are on supplemental oxygen or ventilatory support. Dexamethasone uses at a dose of 6 mg daily for 10 days or until discharge, whichever is shorter. Data from randomized trials support dexamethasone's role in severe COVID-19 IL-6 pathway inhibitors (tocilizumab) - Several agents that target the IL-6 pathway have been evaluated in randomized trials for the treatment of COVID-19; these include the IL-6 receptor blockers tocilizumab and sarilumab and the direct IL-6 inhibitor siltuximab. Tocilizumab (8 mg/kg as a single intravenous dose) is an option for individuals who require high-flow oxygen or more intensive respiratory support Baricitinib - is a Janus kinase (JAK) inhibitor used for the treatment of rheumatoid arthritis. In addition to immunomodulatory effects, it is thought to have potential antiviral effects through interference with viral entry. baricitinib as an option for patients requiring high-flow oxygen or noninvasive ventilation and for select patients who are on low-flow oxygen but are progressing toward needing higher levels of respiratory support despite initiation of dexamethasone. Baricitinib is reserved for those within 96 hours of hospitalization or 24 to 48 hours of initiating ICU-level care. Tofacitinib - another JAK inhibitor, may be an alternative if baricitinib is unavailable.

3rd International Congress on Inflammation

COVID-19 Anit-Viral Therapy

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Abstract

COVID-19 pandemic, is an ongoing pandemic of coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Most people with COVID-19 develop only mild (40%) or moderate (40%) disease, approximately 15% develop severe disease that requires oxygen support, and 5% have critical disease. The clinical manifestations of COVID-19 are similar in children and adults, but they are milder in children.

The people with underlying medical conditions are at risk for severe disease, and chronic pulmonary disease, obesity, neurologic and developmental conditions, cardiovascular disease, Diabetes, and immunosuppression conditions are the most frequently reported risk factors.

In this high- risk group of patients, the antiviral treatment is indicated. Several studies have been done to evaluate the effectiveness of antiviral agents in different stage of disease. According to these data, such therapy is effective for mild and moderate diseases to prevent severe COVID-19 illness.

Among different medications, remdesivir, Ritonavir-boosted nirmatrelvir, and Molnupiravir have been proven to be effective, and they must be initiated as soon as possible. The decision for the choosing one of the mentioned antivirals could be different, according to the age, underlying disease, and several other factors.