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COVID-19 Related Organ Damage

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Abstract

Coronavirus particles contain four main structural proteins. These are the spike (S), membrane (M), envelope (E), and nucleocapsid (N) proteins, all of which are encoded within the 3' end of the viral genome. The S protein (~150 kDa) utilizes an N-terminal signal sequence to gain access to the ER, and is heavily N-linked glycosylated. In view of the increase in death due to COVID-19 (coronavirus disease), it is important to investigate the potential effects the coronavirus on different organs. A literature search was performed from Elsevier, Pubmed, Springer, and Hindawi, and literature is reviewed using customized search strategies. The search strategy included the following terms Cardiovascular effect, Neurological effect, and kidney. Myocardial damage is a common occurrence in patients with COVID-19 disease hospitalisation. This is characterized by a rise in troponin. Vascular endothelial damage in both small and mid-sized pulmonary vessels was noted together with Disseminated intravascular coagulation (DIC), Deep vein thrombosis (DVT), and Pulmonary embolism (PE), resulting in pulmonary infarction. Liver damage in patients with coronavirus infections might be directly caused by the viral infection of liver cells. Neuronal pathway is an important vehicle for neurotropic viruses to enter the CNS (Central Nervous system). Recent research studies show that apoptosis is implicated in a variety of ocular disorders, including glaucoma, retinitis pigmentosa, cataract development, retinoblastoma, retinal ischemia, diabetic retinopathy, and ocular murine glaucoma. The more the understanding about this new virus and its occurrence, the better the ability of people to cope with it. It's far hoped that we will conquer COVID-19 soon with the invention of powerful vaccines, pills, and remedies.

Keywords

Myocardial Damage, Corona Virus, Liver, Kidney

1. Introduction

Coronaviruses area united an outsized family of single positive-stranded and swallowed RNA viruses that may infect humans and lots of animal species. Human coronaviruses are divided supported by their pathogenicity. The kinds with high pathogenicity as well as SARS-CoV (Severe Acute metabolic process Syndrome-coronavirus), MERS-CoV (Middle East metabolic process Syndrome), and current novel SARS-CoV-21, though diffuse alveolar harm and acute metabolic process failure, were the most options of COVID-19, the involvement of different organs required to be explored. Coronaviruses (CoVs) area units the most important cluster of viruses happiness to the Nidovirales order, which incorporates Coronaviridae, Arteriviridae, Mesoniviridae, and Roniviridae families. The Coronavirinae comprise one among 2 subfamilies within the Coronaviridae family, with the opposite being the Torovirinae. The Coronavirinae is divided into four genera, the alpha, beta, gamma, and delta coronaviruses. The viruses were at the start sorted into these genera supported medical science however area unit is currently divided by phyletic cluster.

Coronavirus particles contain four main structural proteins. These are the spike (S), membrane (M), envelope (E), and nucleocapsid (N) proteins, all of that are encoded within the 3' end of the viral genome. The S macromolecule (~150 kDa) utilizes AN N-terminal signal sequence to realize access to the ER, and is heavily N-linked glycosylated. Homotrimers of the virus-encoded S macromolecule structure the distinctive spike structure on the surface of the virus. The trimeric S {glycoprotein|conjugated macromolecule|compound protein} may be a category I fusion protein and mediates attachment to the host receptor. In most, coronaviruses S is cleaved by a bunch cell furin-like proteolytic enzyme into 2 separate polypeptides noted S1 and S2. S1 makes up the big receptor-binding domain of the S macromolecule, whereas S2 forms the stalk of the spike molecule.

The M supermolecule is the most plenteous structural supermolecule within the particle. It's a little (~25 - 30 kDa) supermolecule with 3 transmembrane domains and is believed to relinquish the particle its form. It's a little N-terminal glycosylated ectodomain and a far larger C-terminal endodomain that extends 6 - 8 nm into the microorganism particle. Despite being co-translationally inserted within the ER membrane, most M proteins don't contain a proof sequence. Recent studies counsel that the M supermolecule exists as a chemical compound within the particle, and will adopt 2 totally different conformations, permitting it to push membrane curvature similarly on bind to the nucleocapsid [1] [2].

2. Effect of Coronavirus on Heart

2.1. Myocardial Damage

Myocardial harm could be a common prevalence in patients with COVID-19 unwellness medical care. This can be characterised by an increase in troponin [3]. It is illustrious from the prevailing literature that twelve-tone music of

COVID-19 patients have raised troponin to higher than zero.028 nanogram/mil. As delineated earlier, within the presence of a protein storm that elucidates extreme COVID nineteen this can be presumably thanks to small chemical element provide relative to associate degree accumulated would like. Specific vas institutions have given various advisories; even so, it's wide in agreement that troponin will solely be assessed if acute infarct is suspected [4]. ACE-2 (Angiotensin changing Enzyme-2) was 1st discovered as a homologue of ACE-1 in 2000, that converts angiotensin II to vasoconstrictor vasoconstrictor. ACE-2 may be a kind I transmembrane supermolecule, that is especially anchored at the top surface of the cell. Its chemical process domain is found at the living thing aspect of the cell, which might be cleaved and discharged into blood by ADAM17 (a disintegrin and metalloproteinase domain-containing supermolecule seventeen. The recombinant human ACE2 (rhACE2), that is refined from the supernatant of ACE2 transfected cells, will generate vasoconstrictor vasoconstrictor from angiotensin II and shows the power to stop vasoconstrictor II–induced heart muscle hypertrophy, pulse pathology, and heart muscle pathology [5]. Written and anecdotal studies indicate that early-onset coronary heart condition, arrhythmias, early internal organ injury like MI, carditis, and asystole area unit the varied viable vessel complications of COVID-19. A number of the declared internal organ complications area unit altogether probability to square up from the pro-inflammatory scenario ensuing from this infection, and later on from higher metabolic demand.

2.2. Cardiac Arrhythmias

In one in all the earliest retrospective reviews of COVID-19 patients treated in China, Wang *et al.* found that of the 138 patients analyzed, twenty three (17%) had internal organ arrhythmias and therefore the incidence was considerably higher among those requiring ICU care (44.4% versus vi.9%, $p < 0.001$), compared with those treated in non-ICU beds [6]. Furthermore, in 2 totally different studies 5.9% and 6.7% of COVID-19 patients developed malignant arrhythmias. Accumulating proof suggests that COVID-19 patients might have a hyperinflammatory state-3 with arrhythmogenic protein profile. Acute arrhythmogenic hypercytokinemia may well be one in every of the explanations for prime cardiac-related mortality in these patients. However, the particular figure on the incidence of new-onset heart condition in COVID-19 patients continues to be lacking. Managing the hyperinflammatory state with approved therapies in these patients may well be of potential profit in preventing deadly arrhythmias and pertinent mortality. Viral infections will trigger a hyperinflammatory state with fatal hypercytokinemia and arrhythmogenic potential. COVID-19 patients have options of protein storm syndrome with inflated lymphokine one (IL-1), lymphokine two (IL-2), lymphokine 1β (IL-1β), IL-7, white cell colony-stimulating issue, interferon-γ, inducible macromolecule ten, white blood corpuscle chemoattractant macromolecule one, macrophage, inflammatory macromolecule

1-α, and neoplasm gangrene gangrene. lymphokine one (IL-1), lymphokine two (IL-2), lymphokine lymphokine (IL-1β), white blood corpuscle chemoattractant macromolecule one,6 and neoplasm gangrene issue (TNF-α) are related to deadly arrhythmias [4] [5] [6] [7].

3. Thromboembolic Complications

During the relative recent occurrence of respiratory disease CoV in 2003, that was related to even higher morbidity and mortality than COVID-19, tube epithelium harm in each tiny and mid-sized pneumonic vessels was noted along with Disseminated intravascular clotting (DIC), Deep vein occlusion (DVT), embolism (PE), ensuing pneumonic infarction [8] [9] [10]. A case study of Associate in Nursing autopsy describes occlusion in multiple organs during a patient with verified SARS-CoV infection. High plasma levels of pro-inflammatory cytokines (interleukin-2, interleukin-7, white blood corpuscle colony-stimulating issue, IP10, MCP1, MIP1A and tumour sphacelus factor-α) are ascertained in COVID-19 patients admitted to medical care units. this is often in keeping with a "cytokine storm" with the secondary development of a hemophagocytic lymphohistiocytosis. SARS-CoV-2 is probably going to push large protein formation and deposition which might conjointly account for the terribly high D-dimer levels found in these patients. Protein deposition in alveolar and opening respiratory organ areas, additionally to microcirculation occlusion, could contribute to worsen metabolism failure leading to prolonged mechanical ventilation, poor prognosis, and death. What is more, alternative major blood vessel thromboembolic events and blood vessel complications (e.g., acute cardiac muscle infarction) are rumored and square measure probably to be mostly underestimated [11].

4. Liver Complication

SARS-CoV-2 shares 82% ordination sequence similarity to SARS-CoV and 50% ordination sequence similarity to Middle East respiratory syndrome coronavirus (MERS-CoV)—all 3 coronaviruses are well-known to cause severe respiratory symptoms. Liver impairment has been rumored in up to hr of patients with respiratory disorder and has additionally been rumored in patients infected with MERS-CoV. Liver harm in patients with coronavirus infections could be directly caused by the infection of liver cells. Roughly 2% - 10% of patients with COVID-19 gift with diarrhea, and SARS-CoV-2 ribonucleic acid has been detected in stool and blood samples. This proof implicates the likelihood of infectious agent exposure within the liver. In patients with MERS, infectious agent particles weren't detectable in liver tissue. Gamma-glutamyl enzyme (GGT), a diagnostic biomarker for cholangiocyte injury, has not been rumored within the existing COVID-19 case studies; we have a tendency to found that it absolutely was elevated in thirty (54%) of fifty-six patients with COVID-19 throughout hospitalization in our centre [12]. The liver diagnostic assay specimens of pa-

tients deceased because of severe COVID-19 showed moderate microvascular steatosis and gentle lobe and portal activity, indicating the injury might are caused by either SARS-CoV-2 infection or drug-induced liver injury. It should ensue to the subsequent:

A. Immune mediate harm because of the severe inflammatory response following COVID-19 infection. The inflammation biomarkers as well as C reactive macromolecule (CRP), serum protein, LDH (Lactate Dehydrogenase), D-dimer, IL-6, IL-2, were vital elevated in severe patients with COVID-19.

B. Direct toxicity because of active microorganism replication in hepatic cells: SARS-CoV-2 binds to focus on cells through ACE-2. As a result of ACE-2 is expressed profusely within the liver and especially on biliary animal tissue cells, the liver may be a potential target for direct infection, that was but not nonetheless incontestable [13].

5. Effect on Kidney

After respiratory organ infection, the virus could enter the blood, accumulate in urinary organ, and cause injury to resident nephritic cells. In one study, a total of 701 patients were included which gives clinical alternatives to COVID-19 patients. Median period from sickness onset to admission was 10 days. Of the overall patients, 42.6% were reported as having ≥ 1 comorbidity: 2.0%, 1.9%, 33.4%, 14.3%, and 4.6% reported having, severally, chronic nephrosis, chronic clogging pneumonic illness, cardiovascular disease, diabetes, and tumor. Mean lymph cell count was $0.9 \pm 0.5 \times 10^9$/l below the lower limit of traditional. Most patients had elevated levels of high-sensitivity CRP (83.0%) and blood cell rate (81.6%), however elevated levels of procalcitonin were rare (9.8%). Coagulopathies were common in patients with COVID-19. Additionally, mean serum lactose dehydrogenase (377 ± 195 U/l) was exaggerated, particularly in those with high baseline serum creatinine levels. It's all over that, the prevalence of renal disorder in patients with COVID-19 is high [14]. Conventional knowledge suggests that a nonadaptive general inflammatory reaction, within the face of a protein storm, contributes to hypoperfusion-related injury of the urinary organ tubules. The angiotensin converting enzyme a pair of receptor and members of the amino alkanoic acid enzyme family, essential for infective agent uptake by host cells, area unit extremely expressed on podocytes and tube-shaped structure animal tissue cells. Reports of proteinuria and other COVID-19 infection-related symptoms, along with the isolation of infectious agent ribonucleic acid from excreta, all suggest the possibility of an infection-related reaction affecting the excretory organ [15].

6. Effect on Nervous System

6.1. Direct Infection Injury

The genetic material and even proteins of varied viruses will typically be detected in system tissue samples (such as spinal fluid or brain), Suggesting that

viruses will directly invade the system and cause nerve harm.

6.2. Neuronal Pathway

The neuronal pathway is vital vehicles for tropism viruses to enter the central nervous system. Viruses will migrate by infecting sensory or nerve endings, achieving retrograde or anterograde neural transport through the motor proteins, dynein and kinesins. A Neural pathway is that of sensory system nerve cell transport. The distinctive anatomical organization of sensory system nerves, and also the neural structure within the nasal cavity neural structure effectively makes it a channel between the nasals animal tissue and also the central nervous system. As a consequence, CoV will enter the brain through the sensory system tract within the early stages of infection or nasal vaccination. When CoV infects nasal cells, it will reach the whole brain and humor through the first cranial nerve and neural structure inside seven days and cause inflammation and demyelinating reaction (Figure 1).

7. Angiotensin Converting Enzyme

Angiotensin-converting enzyme 2 (ACE2) may be a cardio-cerebral tube protection issue existing during a type of organs, as well as the system and skeletal muscles, enjoying a serious role in control vital sign and anti-atherosclerosis

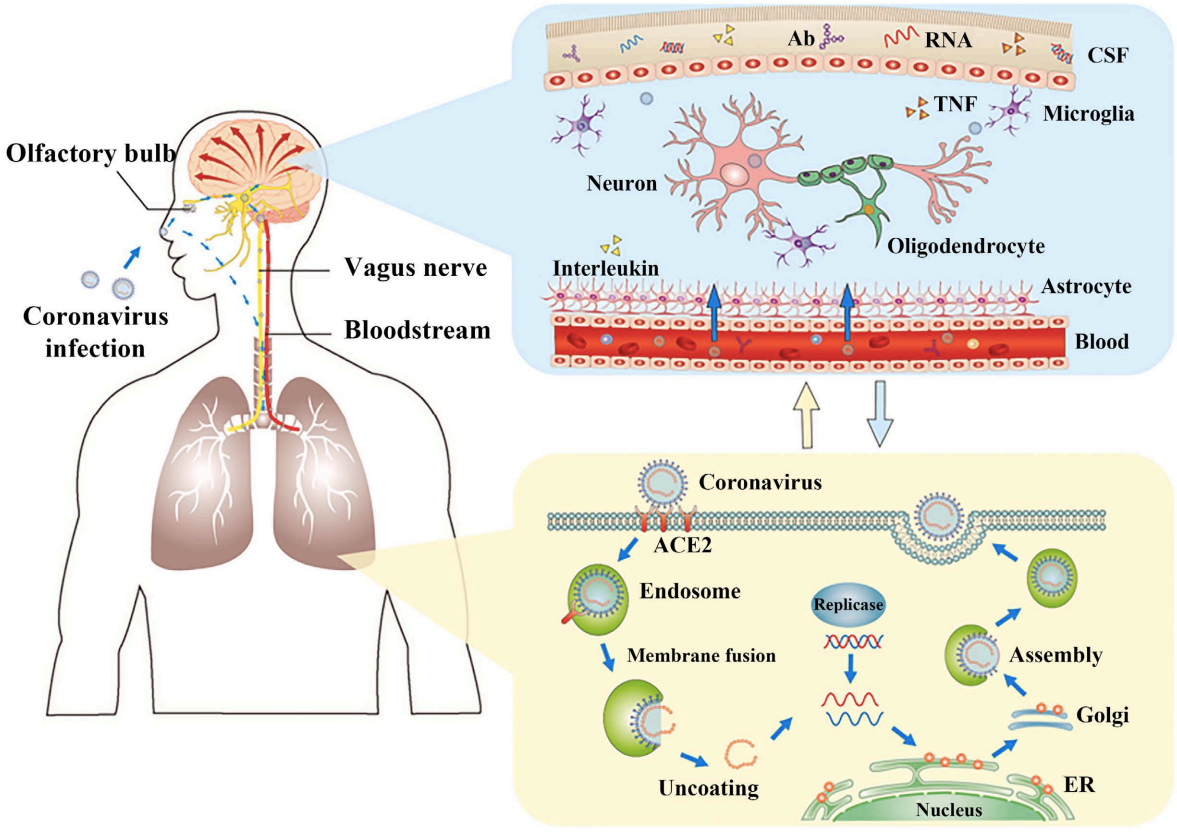

Figure 1. Neurological damage caused by coronavirus.

mechanisms. Meanwhile, ACE2 is additionally a very important target for numerous CoV and respiratory illness viruses. Binding to ACE-2 receptors, the preceding viruses could cause abnormally elevated vital signs and increase the danger of hemorrhage. Additionally, as long as SARS-CoV-2 spike macromolecule might act with ACE-2 expressed within the capillary epithelial tissue, the virus may injury the blood-brain barrier and enter the central nervous system by assaultive the system [16]. The brain has been reported to specific ACE-2 receptors that are detected over interstitial tissue cells and neurons, which makes them a possible target of COVID-19. The COVID-19 virus, like SARS-CoV, uses a spike supermolecule S1 that allows the attachment of the particle to the cell wall by interacting with the host ACE-2 receptor. In some studies, it had been shown that the ACE2 binding affinity of the 2019-nCoV spike supermolecule ectodomain was 10 - 20-fold on top of that of the SARS-CoV spike supermolecule. A BLASTp search of the COVID-19 virus (SARS-CoV-2) receptor-binding domain (RBD) subdomain-1 (319th to 591st aa) fetched a spike {glycoprotein|conjugated supermolecule|compound protein} [bat coronavirus RaTG [13] and S1 protein partial [SARS coronavirus GD322] as homologs [17].

8. Effect of COVID-19 on Eye

It has been reported that COVID-19 linked to ocular signs and symptoms [18]. The entry of the virus through the exposure of airborne droplets or hand contact. The ACE-2 receptor, in conjunction with the cell surface protease enzyme (TMPRSS2), is the central factor responsible for viral binding and virus entry into the host cell. It is still unknown whether these receptors exist on the ocular surface. Some studies reported that the presence of these receptor on the conductive, limbus and cornea [19]. COVID-19 was discovered in the conjunctival discharge of COVID-19 positive patients suffering from conjunctivitis. The researchers have isolated virus from the tears of coronavirus infected patients by reverse transcription polymerase chain reaction. Itching and photophobia were among the other signs [20]. ACE 2 has also been found in the human retina, like the vascularised retinal pigment epithelium, choroid, and conjunctival epithelia. The virus lipid shell can be emulsified by surfactants present in basic soap, which destroys the virus [21]. Recent research studies show that apoptosis is implicated in a variety of ocular disorders, including glaucoma, retinitis pigmentosa, cataract development, retinoblastoma, retinal ischemia, diabetic retinopathy, and ocular murine glaucoma, infection of the herpes simplex virus [22].

9. Effect of COVID-19 on Smell

Impairment of smell is a frequent symptom after a patients experiences Covid-19 infection. The class of virus that caused the odor loss had not been determined previously, nor had the process that caused this loss and how it is related to COVID-19 infection. The olfactory system is made up of three major parts: the brain, nerves, and receptors. Each part of this system can develop pa-

thology that inhibits smell; the most common way pathology develops by pathogenesis in the olfactory receptors [23]. Various studies demonstrated that COVID-19 attack on the central nervous system through the neuroepithelium and spread inside the olfactory bulb. Recent studies have been established to find the facts related to COVID-19 infection causes dysfunctions of olfactory and gustatory system. The sense of olfaction is an important chemical alarming mechanism that controls food consumption and plays a role in interpersonal relationships [24]. The ability to taste is critical for evaluating food content in the oral cavity. It identifies nutrients including sugars or amino acids, electrolytes including sodium or protons, and highly harmful compounds that should be prevented. Sweet, salty, acidic, bitter, and umami are the five essential flavor characteristics that can be detected by the gustatory organ. G-protein coupled receptors are responsible for the recognition of sweet, salty, and umami compounds. Taste buds are controlled by three nerves: the chorda tympani nerve (facial nerve CN-VII), the glossopharyngeal nerve (CN-IX), and the vagus nerve (CN-X), which transmit taste information to the nucleus tractus solitarius (NTS) in the central nervous system. It was suggested that COVID-19 is a neurotropic and neuro-invasive virus that infect peripheral neurone and then by spreading into the central nervous system. The viral infection and inflammatory reaction can disturb saliva composition, taste signalling, and impair the continuous renewal of taste buds [25].

10. Effect on Psychological Behaviour

Recent studies found a consistently negative impact of COVID-19 on mental health. It has been found that people with poor sleep quality are at an increased risk for mental health [26]. After all, during this current health menace, people suffering from mental, behavioural, and psychological problems outnumber those suffering from COVID-19. Evidently, the mental tension associated with the present situation has the ability to aggravate pre-existing psychological problems or to precipitate their symptomatology. There has been a substantial rise in feelings of physical disability, boredom, stigma, worry, phobia, annoyance, and indignation. Individuals with nervous, data compression, and/or depressive temperaments are expected to experience greater mental effect as a consequence of the present situation, according to the findings. In some studies, women appeared to be more susceptible to stress than men [27]. Individuals with COVID-19 can exhibit a variety of neuropsychiatric symptoms as a consequence of systemic inflammation, cytokine effects in the CNS, SARS-COV-2 contamination of neural cells, neuroinflammation, glial dysfunction, or abnormal epigenetic modifications of stress-related genes [28].

11. Conclusion

Hence, taken along with the medical specialty consequences of COVID-19 unconcealed over the previous few days of the natural event, patients reportage al-

tered mental state or associate degree in an inability to style or smell will now not be neglected and should be tested for COVID-19 while not fail. CoV infections will have an effect on the nervous system, and it's presently believed that CoV collectively with host immune mechanisms could flip these infections into persistent infections which will cause medical specialty diseases. The prevalence of COVID-19 infection has become a clinical threat to the final population and aid employees around the world. Therefore, patients with CoV infections ought to be evaluated early for medical specialty symptoms, as well as headache, consciousness disorder, symptoms, and alternative pathological signs. The additional information regards to this new virus, and its prevalence, higher the flexibility of individual. Its miles hoped that we are going to conquer COVID-19 shortly with the invention of powerful vaccines, pills, and remedies. Medical treatment has undoubtedly improved, even from an ophthalmological aspect. Several reports of COVID-19 patients experiencing eye redness and pain have been published. Olfaction impairment has a huge effect on the lives of patients. It is difficult for the normosmic person to consider life without the sense of smell. There has been a substantial rise in feelings of physical disability, boredom, stigma, worry, phobia, annoyance, and indignation.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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