ISSN: 2577 - 8005

Opinion Article

Medical & Clinical Research

Neurological Complications of COVID 19

Shimon Shatzmiller

Department of chemistry, Arile University, Ariel, Israel

*Corresponding author
Shimon Shatzmiller, Department of chemistry, Arile University, Ariel, Israel

Submitted:27 July 2020; **Accepted**: 04 Aug 2020; **Published**: 12 Aug 2020

Foreword

The current coronavirus is hitting the world. Like other outbreaks of epidemics, it hits waves. Today the world, after the experience of a brief recovery from the first wave, is experiencing the second wave. The past teaches us that the second wave can be more destructive than the first. But we are in the midst of this, East and West all over the world infected with the virus.

Modern medicine knows very well and very effectively how to treat and even cure the lesion as it passes through the body systems. From infection running through the body. The eyes and nose to the intestines to the lungs, kidneys, heart, and more. But its penetration into the human brain is a challenge that is difficult to deal with [1]. This is what causes the penetration to rest and the "cytokine storm" human immune system to destroy the nerve cells of the brain and cause the activity of all life systems in the body to stop. This is a stage that brings with it the bitter death of the person infected with the virus.

And the exaltation of God in the throats of the experts will not help, because like Alzheimer's disease, there is no cure for it yet.

The long-awaited vaccine will probably be able to treat mainly during the infection phase. But preventing brain damage and cytokines still poses a huge challenge to science. And right now, there is no savior. Hence probably the great anxiety.

The world is trying by closure and cessation of activity, a measure taken back in the times of the Black Death or the Black Death was a plague that struck Asia and Europe in several waves for about five years (1347–1351) and killed, according to various estimates, between 75 and 200 million people in Europe and Asia. Estimates range from 60% of Europe's population, to about 35 million people in China alone. Outbreaks of the disease, on a smaller scale, continued to strike in various places in Europe until the 17th century.

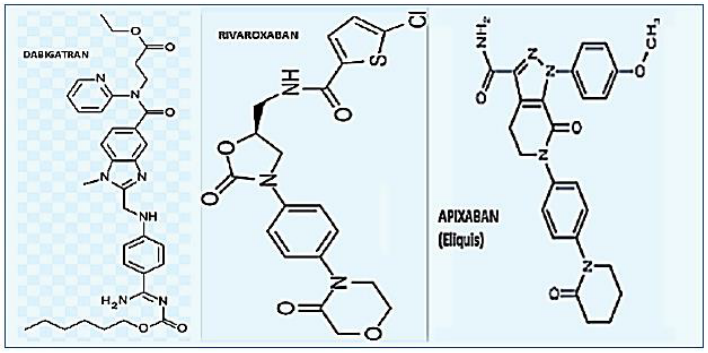
The public health crisis COVID-19 has led to general recommendations from officials to contain its spread. Anxiety about "contracting" the virus is prominent among the public. Specific pathways for anxiety due to contractile disease examined. Expected that a contributing characteristic of fear of being infected with COVID-19 involves a tendency to tenderness and sensitivity, emotional responses that are part of a broader behavioural immune system. The results support a moderate relationship between a tendency to disgust and sensitivity in the relationship between physical concerns related to anxiety sensitivity and fear of infection in COVID-19. These results provide support for individual differences in BIS activation. Mental health consequences of epidemics.

Since December 2019 almost 10 million cases and 500,000 deaths as a result of the coronavirus virus. Severe acute respiratory distress syndrome (SARS-CoV-2) has been reported worldwide. Although coronary artery disease (COVID-19) respiratory complications have been the most common and life threatening, there are more and more reports of central and peripheral nervous system (PNS) involvement. In people with pre-existing cardiovascular obstruction, infection, fever and inflammation can impair the stability of the asymptomatic lipid layer that previously existed in the cardiovascular system. Fever and inflammation also make the blood more prone to blood clotting, interfering with the body's ability to dissolve blood clots - a punch of two similar to injecting gasoline into the coals [2].

Doctors report on a mysterious neurological syndrome known as encephalitis for tragedy appeared around the end of World War I and continued to affect more than a million people worldwide. There is limited evidence for its causes, and whether the trigger was influenza or an autoimmune disorder after infection [3].

In addition to a sleepy coma, some patients suffered from movement disorders that is renenissant to the Parkinson's disease, which affected them for the rest of their lives. In people with pre-existing cardiovascular obstruction, infection, fever and inflammation can impair the stability of the asymptomatic lipid layer that previously existed in the cardiovascular system. Fever and inflammation also make the blood more prone to blood clotting, interfering with the body's ability to dissolve blood clots - a punch of two similar to injecting gasoline into the coals [4].

These neurological complications included encephalopathy, ischemic stroke, acute nodular encephalopathy, and Guilin-Barra syndrome (GBS). Coronavirus 2019 (COVID-19) is an epidemic. Neurological complications of COVID-19 Not reported. Encephalopathy has not yet been described as a presenting symptom, either complication of COVID-19. There is a report, a case of a 74-year-old patient, traveling from Europe to the United States and served with encephalopathy and COVID-19. Categories [5].



Blood Thinners

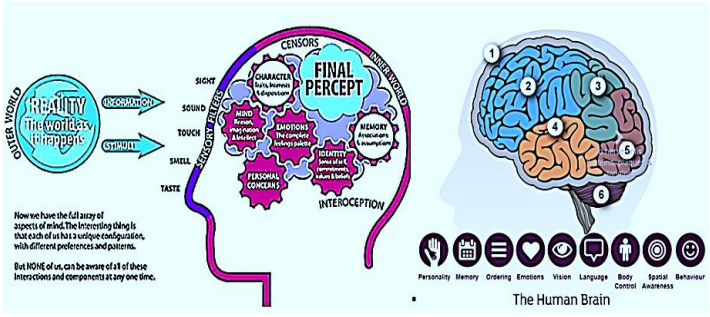
Brain damage could also take place in such circumstances. The doctors thought they thought the blood thinners were causing bleeding in the brain, but what was happening was weird and different. Some people suffer from an acute stroke as a result of a clot, and exclude vital areas of the brain from the blood supply. Tests show that there are remarkably high levels of a marker for the amount of clotting in the blood known as D-dim
er. It is usually less than 300 and in stroke, patients can rise to 1,000. Patient's levels were over 80,000. This high level of coagulation shows something about his body's response to the infection that caused the blood to become incredibly sticky. The body's response to infection causes the blood to become so sticky. During the lock, there was a decrease in the number of surgeries for emergency stroke. But within two weeks, neurologists had treated six 19 Cubid patients who had suffered a stroke. These were not related to the common risk factors for stroke, such as high blood pressure or diabetes. In both cases, they saw very high coagulation levels.

Part of the trigger for the beatings was a massive overreaction of the immune system, causing inflammation in the body and brain. Doctors examined the patient's brain images projected on a wall. They can highlight the vast areas of damage, presented as white blur, that affect his vision, memory, coordination, and speech. The stroke was so severe that doctors thought he was unlikely to survive or be severely disabled [2].

The sudden odor loss in COVID-19 may help us understand how SARS-CoV-2 works. However, several early studies show that the anosmia seen with COVID-19 exists in 30-98 % of infected people seen in hospitals, far more than it does in other respiratory infections. (Examining odor function is standardly difficult to standardize across locations, cultures, and scenarios, so this leads to high variability in the estimated prevalence of hyposemia). Another international study, presented in large numbers, asked people to check themselves with household items and report odor intensity. There is something special about the coronavirus that attacks the sense of smell in particular, which may help us understand how the virus works.

Losing the sense of smell

Lots of senses like sight, smell, taste, and more are reminiscent of neurodegeneration like in Alzheimer's disease.

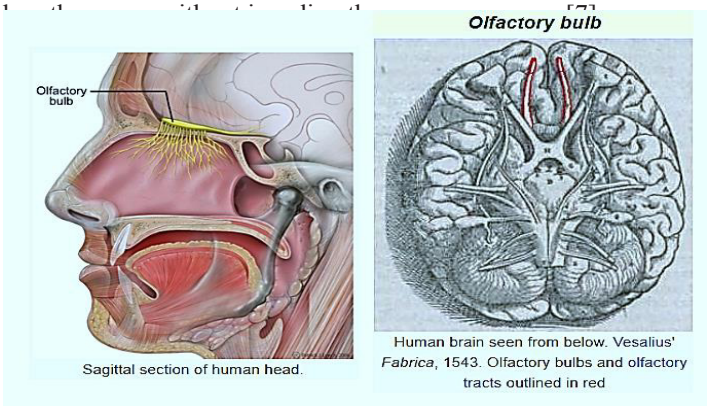


Progression of neurodegeneration brain destruction in Alzheimer's

How does the coronavirus attack the sense of smell? The research comes out quickly, and we have access to early reports, usually using pre-print servers that have not yet gone through the peer review process and should be interpreted carefully (as happens in many of the studies discussed above). Mechanical studies indicate which factors are relevant to hypoxemia and anosmia. Most scientists agree that SARS-CoV-2, like the previously known SARS-CoV, uses the angiotensin-converting enzyme 2 receptors (ACE2) to achieve cell entry through contact with a spike protein. SARS-CoV-2 appears to need PRSS2TM, a protease, to help deliver a prickly protein in the process of entering cells and some other proteins. This shows that cells must express all of these proteins in order for the virus to penetrate them and hijack their machines in order to replicate [6].

ACE2 and TMPRSS2 are expressed in many cell types, and are abundant in the nose, throat, and upper respiratory tract. In the nose, the expression is seen in both the respiratory epithelium (RE) and the sensory epithelium (OSE) but at much higher levels in OSE. In OSE, the proteins are expressed in the supporting stem cells (suppositories) and the smell of pregnancy, as well as in the cell glands (mucus) and the stem cells at lower levels.

There is accumulating evidence that the virus can migrate to the central nervous system through the nose and olfactory bulbs and as



The olfactory bulbs (credit Wikipedia)

All of these tissues help maintain the health of the sensory nerve cells and the mucous layer so that odors can activate the sensory neurons. To date, olfactory sensory neurons themselves do not appear to have the correct expression patterns for SARS-CoV-2

binding, which means that the virus cannot invade directly into these neurons that infiltrate the olfactory cortex directly into the cortex.

There is proof that the virus can migrate to the central nervous system through the bulbs and the odor of life as well as in other ways without invading the sensory neurons. However, the expression patterns of ACE2 and PRSS2TM and the sudden onset and relatively rapid recovery suggest that COVID-19 anemia is not caused by damage to the central nervous system but by loss of olfactory information before it reaches the brain. If the symptoms were by the inner brain driven, we would expect a slower recovery process and more complex range of symptoms, including perhaps prosemia or pantosemia (distorted odors or hallucinations) that have not been reported.

Sensory epithelial inflammation may restrict airflow to the relatively small, high-up nasal odor cleft without causing a stuffy nose or breathing disorder, as demonstrated in a published report. Damage to the pale cells in SE can also affect the function of sensory nerve cells in many ways (like Metabolic, structural, inflammatory), so that even if odors can reach neurons, they may not be able to transmit signals.

There are still many questions open to this mystery, but unprecedented international collaborations and early data sharing will undoubtedly propel research forward faster than usual. The odor relationship is an important clue to the virus mechanisms and leverage many decades of basic research by chemosensory scientists that took place in relative obscurity and put a spotlight on anosmia as a sensory disability.

Acute Respiratory Syndrome (SARS-CoV-2) causes COVID-19, a pandemic respiratory disease that presents with fever, cough, and often pneumonia. Furthermore, thromboembolic events throughout the body including the central nervous system (CNS). Given the first indication of the presence of viral RNA in the brain and cerebrospinal fluid and in light of the neurological symptoms in a large majority of COVID-19 patients, it is likely that SARS-CoV-2 infiltration of the central nervous system. By accurately examining and mapping the aerobic and pharyngeal regions and brain of patients dying from COVID-19 accurately, we not only describe a central nervous system infarction as a result of the cerebral blood drug, but also demonstrate SARS-CoV-2 neurotropism. SARS-CoV-2 enters the nervous system by trampling the border of the olfactory-mucosal interface by utilizing the immediate vicinity of the olfactory mucosa and healing the nerves, including sensitive and sensitive nerve endings. Subsequently, SARS-CoV-2 monitors defined neuroanatomical structures, penetrating defined neuroanatomical regions, including the primary respiratory control and cardiovascular control center in the *medulla oberonga [7].*

References

1. Shimon Shatzmiller, Galina Zats, Inbal Lapidot, Ludmila Buzhansky, Rami Krieger (2020) Cytokine Storm: A Coronavirus Complication. CPQ Microbiology 3: 01-07.
2. https://news.harvard.edu/gazette/story/2020/04/covid-19s-consequences-for-the-heart/
3. Fergus Walsh (2020) "Coronavirus: What does Covid-19 do to the brain? https://www.bbc.com/news/health-53081022
4. https://news.harvard.edu/gazette/story/2020/04/covid-19s-consequences-for-the-heart/
5. Asia Filatov, Pamraj Sharma, Fawzi Hindi, Patricio S Espinosa (2020) "Neurological Complications of Coronavirus Disease (COVID-19): Encephalopathy". Cureus 12: e7352.
6. Leslie Kay (2020) Why COVID-19 Makes People Lose Their Sense of Smell. https://www.scientificamerican.com/article/why-covid-19-makes-people-lose-their-sense-of-smell1/
7. Jenny Meinhardt, Josefine Radke, Carsten Dittmayer, Ronja Mothes, Jonas Franz, et al. (2020) "Olfactory trans mucosal SARS-CoV-2 invasion as port of Central Nervous System entry in COVID-19 patients". https://www.biorxiv.org/content/10.1101/2020.06.04.135012v1