16/MHS06/065

MLS 406 (VIROLOGY) ASSIGNMENT.

COVID-19

INTRODUCTION AND ETIOLOGY

Covid-19 a short term for Corona virus disease 2019 and formerly known as the Novel Coronavirus is an infectious disease caused by a newly discovered coronavirus (SARS-CoV-2). Corona virus comprises of a large family of viruses that are common in human beings as well animals (camels, cattle, cats, and bats). There are seven different strains of corona virus infectious to man.

- 229E which is an alpha coronavirus
- NL63 which is an alpha coronavirus
- OC43 which is a beta coronavirus
- HKU1 also a beta coronavirus
- MERS-CoV a beta coronavirus that causes Middle East Respiratory Syndrome, or MERS
- SARS-CoV a beta coronavirus that causes severe acute respiratory syndrome, or SARS
- SARS-CoV-2 a beta coronavirus that causes coronavirus disease 2019, or COVID-19

The virus that causes COVID-19 is designated severe acute respiratory syndrome corona virus 2 (SARS-CoV-2); previously, referred to as 2019-nCoV. Coronavirus disease 2019, or COVID-19, is a disease that can a respiratory tract infection. It can affect the upper respiratory tract (sinuses, nose, and throat) or lower respiratory tract (windpipe and lungs).

ORIGIN OF COVID-19

COVID-19 emerged in Wuhan, a city in the Hubei Province of China and spread around the world, this virus was identified as the cause of upper and lower tract infection in China. Genomic analysis revealed that SARS-CoV-2 is phylogenetically related to severe acute respiratory syndrome-like (SARS-like) bat viruses, therefore bats could be the possible primary reservoir, with the theory being that an individual became infected having made contact with a pangolin (an intermediate reservoir of COVID-19 which became infected from bats as the primary reservoir) in a market at Wuhan where meat gotten from different animals are sold and from there began the spread of the virus from human to human contact through droplets from sneezing or coughing of an infected to a healthy person. Also the virus spreads from touching a surface or object the virus is on, then touching the mouth, nose, or possibly the eyes with that same infected hands. Most viruses can live for several hours on a surface that they land on. The virus can also spread via fecal oral route.

STRUCTURE OF COVID-19

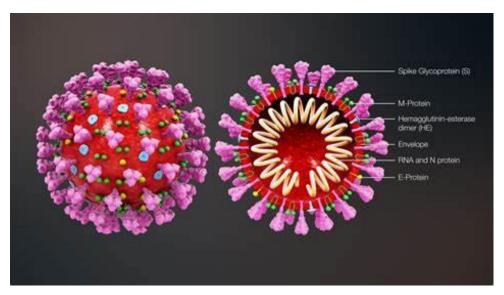


IMAGE OF THE STRUTURE OF THE COVID-19

Corona virus derives its name from its appearance in electron micrographs that creates a spike like projection on the surface of the virus hence the name corona which means "crown" in Latin.

The SARS-CoV-2 is a large, enveloped single stranded RNA virus and that is positive sensed, linear and has a helical capsid. From its family of *CORONAVIRIDAE*, it consists of Peplomers or spike proteins on the surface of the virus. Theses spikes aids in receptor binding of the virus to the host cell thus gaining entry into the cell. It is these spike proteins that show diversity in SARS-CoV-2.

They have the largest genome among all RNA viruses, typically ranging from 27 to 32 kb. The genome is packed inside a helical capsid formed by the nucleocapsid protein (N) and further surrounded by an envelope. Associated with the viral envelope are at least three structural proteins:

• The membrane protein (M) and the envelope protein (E) are involved in virus assembly, whereas the spike protein (S) mediates virus entry into host cells. Some coronaviruses also encode an envelope-associated hemagglutinin-esterase protein (HE).

Among these structural proteins, the spike forms large protrusions from the virus surface, giving coronaviruses the appearance of having crowns. In addition to mediating virus entry, the spike is a critical determinant of viral host range and tissue tropism and a major inducer of host immune responses.

The coronavirus spike contains three segments:

a large ectodomain, a single-pass transmembrane anchor, and a short intracellular tail. The ectodomain consists of a receptor-binding subunit S1 and a membrane-fusion subunit S2. Electron microscopy studies revealed that the spike is a clove-shaped trimer with three S1 heads and a trimeric S2 stalk. During virus entry, S1 binds to a receptor on the host cell surface for viral attachment, and S2 fuses the host and viral membranes, allowing viral genomes to enter host cells. Receptor binding and membrane fusion are the initial and critical steps in the coronavirus infection cycle; they also serve as primary targets for human inventions.

PATHPHYSIOLOGY

Once the virus gains access into the body through fecal oral route or respiratory droplets, the virus goes into the lungs and attacks the alveoli.

In the alveoli,

The virus attaches to the TYPE II pneumocytes which is responsible for the production of surfactants that functions as a surface tension reducer within the alveoli. The S-spike proteins bind to specific receptors sites on the TYPE II pneumocyte (ACE-2). The virus then becomes engulfed into the cell and releases its RNA for replication to occur. The RNA is transcribed using the ribosome of the host cell and then translated which produces polyproteins. These polyproteins are converted into key components of the virus by the enzyme proteineses.

As replication occurs, the cells are being destroyed and begin to malfunction. The polyproteins cause an inflammatory response and this stimulates the macrophages. The macrophages activate interleukin 1, 6 and alpha which cause the blood vessel to dilate increasing capillary permeability causing the plasma to leak into the interstitial layer, compressing the alveoli surrounding while also trying to gain access into the alveoli.

During this process, the concentration of the surfactant begins to reduce by drawing out the surfactant thus increasing surface tension which leads to alveoli collapse leading to a decrease in gaseous exchange causing shortness of breath.

The inflammatory mediators (Interleukins 1, 6 and alpha) attracts neutrophils to the site of inflammation trying to destroy the virus by releasing specific substances. In the process of destroying the virus, the pnemocytes (TYPE I AND II) are also destroyed leading to a poor gaseous exchange and a reduced surfactant production increasing surface tension and eventually, collapsing the alveoli. As the cells are getting destroyed, the begin to move to the center accumulating and forming a consolidation which alters the gaseous exchange leading to hypoxia, the accumulation also results in coughing of the mucous fluid.

When the interleukins are released in large amounts, they can travel as far as the central nervous system through the blood and when this occurs, the hypothalamus is affected. The interleukins cause the hypothalamus to release specific prostaglandins which affects the thermostat causing a rise in temperature which then leads to fever.

Because of the poor gaseous exchange in the alveoli, the oxygen partial pressure is low and when this happens, the chemoreceptors are stimulated and in reflex, stimulates the Sympathetic Nervous System which increases the heart rate. Also in attempt of the SNS to resolve the low oxygen level by increasing the oxygen level, there would be an increased respiratory rate.

The inflammation becomes severe if the immune system cannot fight the virus and hence the inflammation begins to spread leading to systemic inflammatory response syndrome which leads to septic shock. The blood vessels begin to increase its capillary permeability causing leakage of fluids and accumulating in tissue spaces decreasing the blood volume, the total peripheral resistance is reduced also due to vasodilation and this leads to low blood pressure (hypotension) leading to perfusion of organs and then multiple organ failure. Some of these organs include the kidney where urine cannot be eliminated as a result of perfusion and insufficient blood flow with this leading to elevated levels of BUN and creatinine.

The liver also is an organ that is affected and as a result of poor blood supply, there is an increased level of ALT, AST and bilirubin.

Without subsequent resolution of the inflammation, multiple organ failure would occur leading to the death of the patient.

REFERENCES

- 1. <u>www.sciencedirect.com</u>
- 2. <u>http://nbcnews.com</u>
- 3. https://osms.it/covid-19.com
- 4. <u>https://youtu.be/PWzbArPgo-o</u>
- 5. INTERNATIONAL PULMONOLOGIST'S CONSENSUS ON COVID-19

Chief Editors Dr. Tinku Joseph (India), Dr. Mohammed Ashkan Moslehi (Iran)

- 6. <u>www.who.it</u>
- 7. https://www.webmd.com/lung/coronavirus