

Effect of Corona Virus on respiratory system: Review

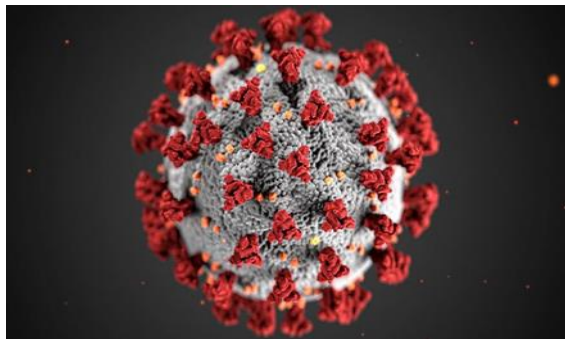
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Abstract—While Covid19 pandemic sweeps across the globe, it is important to understand the effect of the virus which has made it a pandemic. Covid19 belongs to the corona virus family, which also includes the SARS virus (severe acute respiratory syndrome) and MERS (Middle East Respiratory Symptoms) virus. The family of Corona virus includes virus strains that cause the cold and flu. It has been found that Covid19 is similar to SARS. SARS is a novel type of virus that was reported in 2007, and like SARS viruses, Covid19 affects the respiratory tract in humans. The infection starts with mild flu-like symptoms or no symptoms, and further progress to critical symptoms. Covid19 mainly infects the lungs in the affected individuals and in serious cases causes death due to ARDS (acute respiratory distress syndrome) and pneumonia. It is necessary to remember that it does not lead to ARDS and pneumonia in all the cases, which happens in most critical cases. In the majority of the cases i.e. 80% will show case mild signs, 14% will have pneumonia, 5% will suffer from septic shock and organ failure (mostly respiratory failure) and in 2% cases it will be lethal. Humanity needs leadership and solidarity to defeat the corona virus.



I. CHARACTERISTICS

According to a report published on 24 Jan 2020, corona virus infected patient have many similar attribute such as fever, dry cough, tiredness and fatigue while diarrhea sore throat, conjunctivitis and loss of taste and smell were found to be as less

common symptoms. Many of them patient reported bilateral irregularity. Corona virus was separated from bronchoalveolar lavage fluid in china in 2020. It is also found in blood samples. Till now, corona virus was not verified in faces and urine sample of patient. To explain the distinct procedures that happen within the body, we have break the disease into four different phases that roughly match the different levels of seriousness: mild, moderate, severe, and critical.

II. PROCEDURE FOR PAPER SUBMISSION

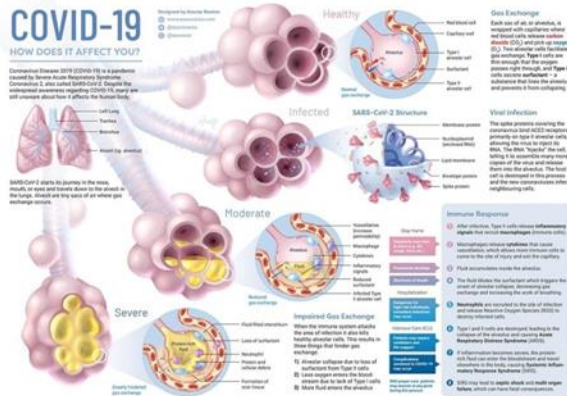
Phase 1: Cell invasion and viral replication within the nose

Both SARS-CoV-2 and SARS-CoV get entry through a receptor called ACE2. Recently, an international team of researchers has found the ACE2 receptors on goblet (secretory) cells in and on ciliated (hairy) cells in the nose. Researcher's also obtained an ample supply of a protease called TMPRSS2, which chemically splits off the top of the coronavirus spike to allow the SARS-CoV-2 RNA to enter into the nasal cells. As a result, SARS-CoV-2 can adhere 10 times more strongly to insert its RNA into the cell, which give us a side why COVID-19 spreads so quickly.

Phase 2: Replication in the lung and immune system alerted

In the lung, the ACE2 receptor sits on top of lung cells called pneumocytes. These have an significant part in producing surfactant — a compound that covers the air sacs (alveoli), thus helps in maintaining sufficient surface tension to keep the sacs open for the exchange of oxygen and carbon dioxide. As soon as the body finds an unfamiliar protein, it exhibit the first response. One part of the body's immune response — the lymphocytes, starts to produce the first defence IgM-type antibodies and then the longer term particular neutralizing antibodies (the IgG type). 80%

of population with COVID-19 will have mild or asymptomatic disease, with common signs including fever, dry cough, and tiredness. Many people will only have phase 1 or 2 physiological responses to SARS-CoV-2 infection.



Figures

Phase 3: Pneumonia

Nearly 13.8% of people with COVID-19 will have serious disease and will need hospitalization as they have difficulty in breathing. Of these people, around 75% will encounter bilateral pneumonia. Pneumonia in COVID-19 occurs when parts of the lung consolidate and collapse. Lowered surfactant in the alveoli from the viral destruction of pneumocytes makes it hard for the lungs to keep the alveoli open.

Because of the immune response, white blood cells, such as neutrophils and macrophages, runs into the alveoli. Meanwhile, blood vessels surrounding the air sacs start leaking because of inflammatory chemicals that the white blood cells secrete. This fluid exerts pressure on the alveoli from outside and, in combination with the deficiency of surfactant, makes them collapse. Because of it breathing becomes hard, and the surface area in the lung where oxygen transfer usually happens becomes less, which causes breathlessness. The body try to heal itself by promoting inflammatory and immune responses. The World Health Organization (WHO) advise again the utilization of gluco corticosteroids during this phase, as they could stop the natural healing response. The evidence seems to disapprove this position, but this is a quickly growing area, and findings are subject to change. Most patients will recover at this stage with supportive intravenous fluids and oxygen through a mask Oran external positive pressure mask.

Phase 4: Acute respiratory distress syndrome, the cytokine storm, and multiple organ failure

The most four-four time for the onset of critical disease is 10 days, and it can come on suddenly during a small proportion of individuals with mild or moderate disease. In severe acute respiratory distress syndrome (ARDS), the inflammation stage gives way to the fibrosis stage. Fibrin clots form into the alveoli, and fibrin-platelet micro thrombi (small blood clots) pepper the tiny blood vessels within the lung that are liable for gas exchange with the alveoli. There is hope that drugs already licensed for anti-clotting action in strokes might be helpful at this stage. Cytokines are chemical mediators that white blood cells such as macrophages release, and the ycan engulf infected cells. These cytokines — which have names like IL1, IL6, and TNF α — have actions that include dilating the vessel walls and making them more permeable. In extreme circumstances, this can lead to a collapse of the cardiovascular system. Estrogen in mouse cells suppresses the release of cytokines from macrophages. Although animal studies often fail to translate into important findings in humans, this could be one explanation for worse outcomes from COVID-19 in males. While smaller numbers of ACE2 receptors are protective in phase 1, as there are fewer landing sites for the virus, by the time we reach phase 4, these receptors may become protective. ACE2 receptors in health play an important regulating role for the activities of angiotensin converting enzyme 1 (ACE1). In response to infection, ACE1 creates excess angiotensin2 from angiotensin 1. Angiotensin 2 directly damages the lungs, causes blood vessel constriction, and makes the blood vessels leaky. Drugs that doctors typically use in the treatment of hypertension (ACE inhibitors and ARBs) may be helpful at this stage. The role of ACE2 inhibitors in treating COVID-19 are a complex one. As some authors note, on the one hand, using them may lead to a higher risk of SARS-CoV-2 infection. On the other hand, ACE inhibitors may reduce the lung damage that this infection causes.

CONCLUSION

Corona virus was spreading human to human to transmission by close contact via air borne droplets generating by coughing, sneezing. So avoid these

activities with infected partners and family members. Corona virus may transmit through pet animals such as dog, cat, pig, cow, turkeys. So avoid contact and separate them if observed any infection activities like diarrhea, cold, fever. As per WHO and ECDC guideline avoid the contact with sick person and also avoid the market or public place as per possible. There is no anti corona virus vaccine to prevent or treatment but some supporting therapy work. Future research needed to fight with corona virus. Till only 'Distance is rescue'

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