

Pathology and Pathogenesis of SARS CoV-2

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Description

The current pandemic of Corona Virus Disease-2019 (COVID-19) caused by Severe Acute Respiratory Syndrome Corona Virus-2 (SARS CoV-2) led to complete lockdown in many countries contributing to major socio-economic crisis and irreparable recession, globally. SARS CoV-2, a novel β CoV was first identified in adults presenting with acute lower respiratory tract infection of unexplained etiologic in China. Though no age group is spared, severe forms occur in patients older than 60 years specifically with co-morbidities. The majority of the infected individuals are asymptomatic or with mild form of disease and are potential transmitters.

This disease is highly contagious and mainly spread through respiratory droplets, close contact with infected areas or materials (fomites) and other patients and health care workers in the hospitals. COVID-19 has a much lower case fatality ratio and significantly greater transmission rate than 2003 SARS pandemic. Currently RT-PCR of upper and lower respiratory swabs or samples is the gold standard diagnostic test. Asymptomatic state (starting 1-2 days of disease).

The infection SARS-CoV-2 probably links to epithelial cells in the nasal pit and starts repeating the process. ACE2 is the basic fundamental receptor for both SARS-CoV-2 and SARS-CoV. *In vitro* information with SARS-CoV demonstrates that the ciliated cells are essential cells contaminated in the directed routes. At this stage the infection can be distinguished by nasal swabs. Albeit the viral weight might be low, these people are irresistible. For the RT-PCR cycle number to be helpful, the example assortment system would need to be normalized. Nasal swabs may be more responsive than throat swabs.

The infection spreads and relocates down the respiratory plot along the directing aviation routes, and a more powerful intrinsic safe reaction is set off. Nasal swabs or sputum should yield the infection (SARS-CoV-2) just as early markers of the intrinsic safe reaction. For about 80% of the contaminated patients, the sickness will be gentle and generally limited to the upper and directing aviation routes. These people might be checked at home with moderate indicative treatment.

Hypoxia

About 20% of the affected patients will advance to organize illness and will create aspiratory affects and a portion of these will tend to extremely serious infection. Beginning assessments of the casualty rate are around 2%, yet this fluctuates especially with age. The casualty and bleakness rates might be re-examined once the predominance of gentle and asymptomatic cases is better characterized.

Pathogenesis

SARS CoV-2 is a contamination type II pneumocytes (liable for tissue fix and surfactant bio-blend) are causing expanded surface strain bringing about dyspnoea. What's more, these harmful type-II pneumocytes compromise the alveolar immunologic equilibrium work by improperly setting off a course of nearby and fundamental provocative reaction because of extreme cytokine combination and delivery (cytokine storm) by the fluctuated fiery cells inferable from aggregation of un-cleaved angiotensin II.

This cytokine overabundance when extreme, causes wide spread tissue harm because of Systemic Inflammatory Response Syndrome (SIRS). Likewise, there is inescapable enactment of supportive coagulant factors prompting micro thrombi in different tissues/organs bringing about ARDS, Multiple Organ Dysfunction (MODS), ischemia and high mortality. This marvel is proven by the presence of essentially expanded favourable to incendiary cytokines (Interleukins 1- β , 1RA, 7, 8, 9, 10, b FGF2, GCSF, GMCSF, IFN γ , IP10, MCP1, MIP1 α , MIP1 β , PDGF β , TNF α , and VEGFA.) in extreme COVID-19 patients.

Conclusion

The pandemic by COVID-19 is a live issue influencing individuals around the world. Without basic helpful intercessions, momentum the executives is to lessen the infection spread and give steady consideration to ailing patients. There is a dire need to foster designated treatments. Understanding the distinction in paediatric and grown-up reactions to this infection might assist with coordinating insusceptible based therapeutics.

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