COVID FACTS

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(COVID-19 is a disease, caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), a Novel Coronavirus, which emerged in the city of Wuhan, Hubei, China, in early December 2019 and which is transmitted as a Droplet Infection and has spread worldwide as a pandemic. Genetic sequencing of the virus suggests that SARS-CoV-2 is a betacoronavirus closely linked to the SARS virus.)

PATHOGENESIS

(There are significant knowledge gaps in the pathogenesis of COVID-19, so update this periodically)

1 Port Of Entry: Mucous Membrane in Nose, Mouth and Eyes

2 Mode of person to person Infection.

- 2.1 Primary Mode: Droplet Infection at the time of Coughing, Sneezing, Talking, Singing, etc where mucous discharge from the respiratory tract is thrown out.
- 2.2 Other Modes: Person to Person Contact, Self-Contact from infected skin to one's port of entry, Aerosols formed at the time of Oxygen Therapy Initiation or at the time of providing Ventilation Support.

3 Pathogenesis Stage 1

- 3.1 The inhaled virus binds to ciliated epithelial cells in the mucous membrane of entry post, primarily nasal cavity, and starts replicating.
- 3.2 Patient is Asymptomatic but infectious.
- 3.3 Period 1–2 days after infection
- 3.4 Virus Burden is Low (<100,000 copies per millilitre of Blood Plasma)
- 3.5 It does not activate body's immune response
- 3.6 Testing is RT-PCR of Nasal swabs (more sensitive) or Throat swabs

4 Pathogenesis Stage 2

- 4.1 The virus propagates and migrates down the respiratory tract along the airways, and triggers body's immune response. It may travel up the Olfactory Nerve to brain. It may enter blood stream and travel to multiple organs, specifically to the kidneys.
- 4.2 Patient is symptomatic and infectious, has mild disease and a more robust innate immune response is triggered.
- 4.3 Period 3 – 15 days after infection.
- 4.4 Virus Burden is medium (>100,000 but < 1,000,000 copies per millilitre of Blood Plasma)

4.5 Early markers of the immune response are visible in patients' body and can be measured. (By measuring CXCL10 interferon responsive gene, induced proteins) Determining the host innate immune response might improve predictions on the subsequent course of the disease and need for more aggressive monitoring.

4.6 For about 80% of the infected patients, the disease will be mild and mostly restricted to the upper and conducting airways. These individuals may be monitored at home with conservative symptomatic therapy.

4.7 After a Diagnostic RT-PCR test, patient may be monitored using multiple immune antibody tests.

4.8 Confirmation of cure is RT-PCR test. (The test is negative when Virus Burden is negligent < 200 copies per millilitre of Blood Plasma)

5 Pathogenesis Stage 3

5.1 The virus propagates and reaches the gas exchange units of the lung and infects alveolar type II cells there. The virus propagates within type II cells, and the cells undergo apoptosis and die. It leads to release of large number of viral particles which infect type II cells in adjacent gas exchange units of the lung.

5.2 The result is a self-replicating pulmonary toxin as the released viral particles infect most of the type II cells which leads to pulmonary infiltrates and progressing the disease to Acute Respiratory Distress Syndrome (ARDS).

5.3 Patient is usually Hypoxic – Hungry for Oxygen, and highly infectious and a robust innate immune response is triggered.

5.4 About 2% - 5% cases develop Severe Respiratory Distress Syndrome and require Ventilator Support, due to diffuse alveolar damage with fibrin rich hyaline membranes and a few multinucleated giant cells. Full recovery requires a vigorous innate and acquired immune response and epithelial regeneration. Healing leads to severe scarring and fibrosis in the Lungs.

5.5 Elderly patients and Patients with COPD (due to smoking) are particularly at risk because of their diminished immune response and reduced ability to repair the damaged epithelium. They also have reduced muco-ciliary clearance, and this may allow the virus to spread to the gas exchange units of the lung more readily.

5.6 Period 10 – 17 days after infection.

5.7 Virus Burden is high (>1,000,000 copies per millilitre of Blood Plasma) and it is associated with high virus shedding.

5.8 All these patients require Institutional care.

5.8.1 All COVID-19 symptomatic patients, especially ARDS patients are treated in COVID Hospitals with facilities for Oxygen Therapy and adequate isolation measures to prevent further infections.

5.8.2 All COVID-19 patients, with diffuse disease and high virus shedding, and all high risk patients, and all patients with co-morbid diseases, should be treated in “Biocontainment Patient Care Units” (BPCU) which provide multiple layers of protection for Healthcare Providers.

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