International Journal of Scientific Research in Engineering and Management (IJSREM)
Volume: 08 Issue: 07 | July - 2024 SJIF Rating: 8.448 ISSN: 2582-3930

A Review On: COVIED -19 TRANSMISSION, VARIOUS VARIANTS, CURRENT TREATMENT AND FUTURE STATERGIES

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ABSTRACT:

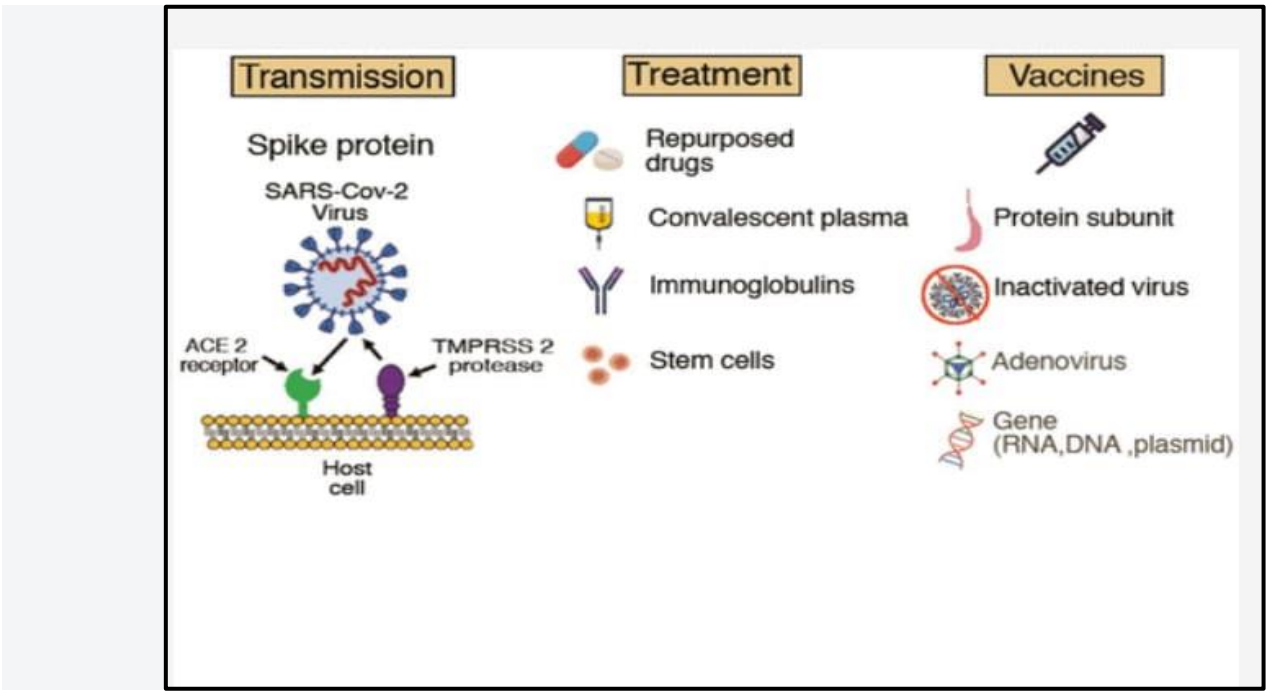
The Chinese city of Wuhan served as the focal point for the discovery of COVID-19, a zoonotic illness that would eventually spread around the world, in the start of 2020. Even though COVID-19 has a unique way of spreading and is quite strong, it shares resemblances to other zoonotic illnesses like SARS -CoV and MERS, among others, in that it produces acute respiratory distress and exhibits severe flu-like symptoms. The COVID-19 virus was dubbed SARS-CoV-2 due to a number of molecular similarities that have been found between it and SARS. These similarities have created a number of opportunities for COVID-19 patients to receive therapeutic treatments that have been proven successful in treating SARS. Potential for repurposing medications that have been effective in treating SARS has also been offered by the identification of commonalities between SARS-CoV and SARS-CoV-2 in terms of how they interact with the host, multiply, and cause life-threatening diseases. The article starts by providing a synopsis of COVID-19's genesis in relation to other zoonotic illnesses, namely SARS and MERS. Even at the molecular level, a number of similarities between SARS and COVID-19 have been found. These similarities have led to the COVID-19 virus being dubbed SARS-CoV-2. These similarities have opened up a number of options for treating COVID-19 patients with therapeutic techniques that have been successful in treating SARS.It is important to note that the discovery of parallels between SARS-CoV and SARS-CoV-2 in terms of how they infiltrate the host, multiply, and produce potentially fatal illnesses has made it possible to repurpose medications that have been shown to be successful against SARS.

KEYWORDS: SARS-CoV-2, COVID-19, vaccinations, treatment approaches, transmission

INTRODUCTION:

An overview of the existing knowledge on the transmission mechanism of the SARS-CoV-2 virus from patients to hosts is given in this article. It also looks into the cellular mechanisms involved, possible routes via which the virus enters the human body, and mathematical models used to estimate the probability of viral aerosol and droplet transmission. The paper also covers the clinical manifestations of COVID-19 and the efficacy of current diagnostic techniques in identifying the virus in people. In addition, it looks at novel treatment strategies for managing the virus, with an emphasis on creating a vaccine that works and repurposing existing medications to fight the infection. Owing to the substantial and ever-growing corpus of published research on COVID-19,

i. Successful tactics to stop SARS-CoV-2 from spreading from person to person.(1).
ii. COVID-19 clinical manifestations in those with symptoms (2-4) and people without symptoms.(5)
iii. A thorough examination of COVID-19's incubation and infectious times.(7)
iv. The immunological responses to SARS-CoV-2 in humans.
v. The relationship (9–12) between COVID-19 mortality and pre-existing comorbidities
vi. A variety of vaccination categories that are presently under clinical development (13).
vii. The body's immunological reaction to COVID-19 vaccinations.(8)
viii. A comparison between SARS-CoV-2 monoclonal antibody treatment and SARS-CoV and MERS-CoV(14)
ix. The effects of novel therapies on hospitalization length and death rates that are presently being investigated in clinical studies (15)



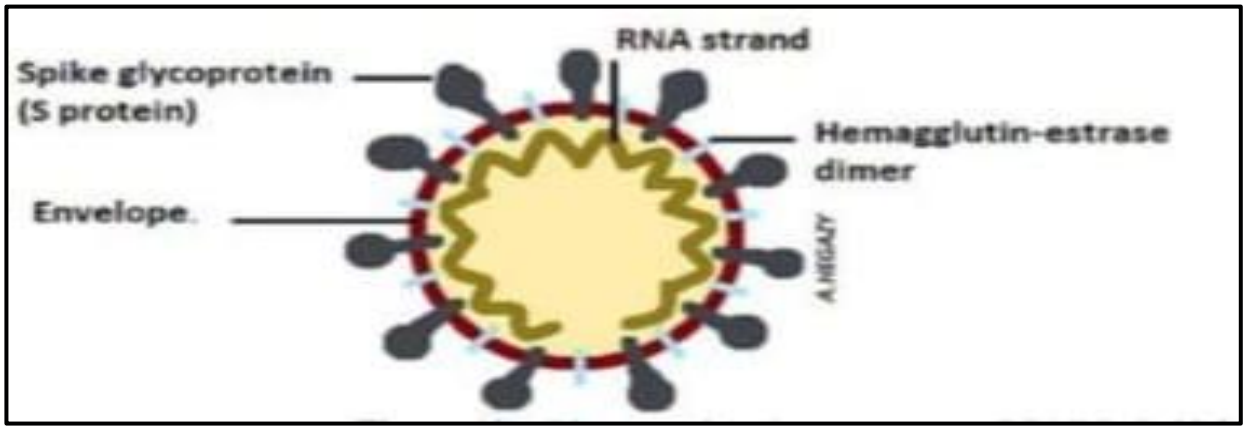
signs & Symptoms:

1) Cold or fever
2) Cough
3) Difficulty breathing or shortness of breath
4) Lethargy
5) Body or muscle discomfort
6) Headache
7) A recent loss of scent or flavor
8) Sore throat
9) Palpitations or chest aches
10) Pains and Aches

Adverse consequences:

1) Extreme Fatigue or Tiredness
2) Breathlessness
3) Memory and Concentration Issues
4) Papillary heartbeat
5) Lightheadedness
6) Muscle and joint discomfort

COVID-19 OVERVIEW:



Structure of SARS-COV-2.

A Historical View on Pandemics.

A sudden increase in illness within a particular area or population is indicative of a disease outbreak (16, 17). If the outbreak is not contained, it spreads to a large population, affecting a town or an entire area and leading to the emergence of an epidemic (18, 19). An epidemic develops into a pandemic when contaminated people and/or items carrying infectious materials spread over the world.(20)Pandemics such as smallpox, plagues, and cholera devastated many towns in Europe and Asia from the 16th to the 19th century. The Spanish Flu pandemic (1918–1919) in the 20th century was caused by the H1N1 influenza virus, which is thought to have been mostly spread by returning World War I troops.(21).Pandemic Origins: Zoonotic Sources. Most pandemics that humans have seen in recent memory

Pandemic Origins: Zoonotic Sources.

Most pandemics that humans have seen in recent memory are zoonoses, which are usually spread to humans by direct contact with animal body fluids or by vectors carrying zoonotic infections.(22).

For example, the HIV/AIDS pandemic is believed to have started in chimpanzees.(23)

The Dissimilarities and Similarities Between Other Coronaviruses and SARS-CoV-2

A capsid, like the sun corona in appearance, encloses a positive-sense single-stranded RNA (+ssRNA) in coronaviruses. When compared to other positive RNA viruses, coronaviruses are more sophisticated in their methods of invading host cells and possess a larger genome.(24) They

have the ability to infect people, traverse species boundaries, and use host cells as a means of replication and transmission. Unfortunately, coronaviruses have emerged as a major source of

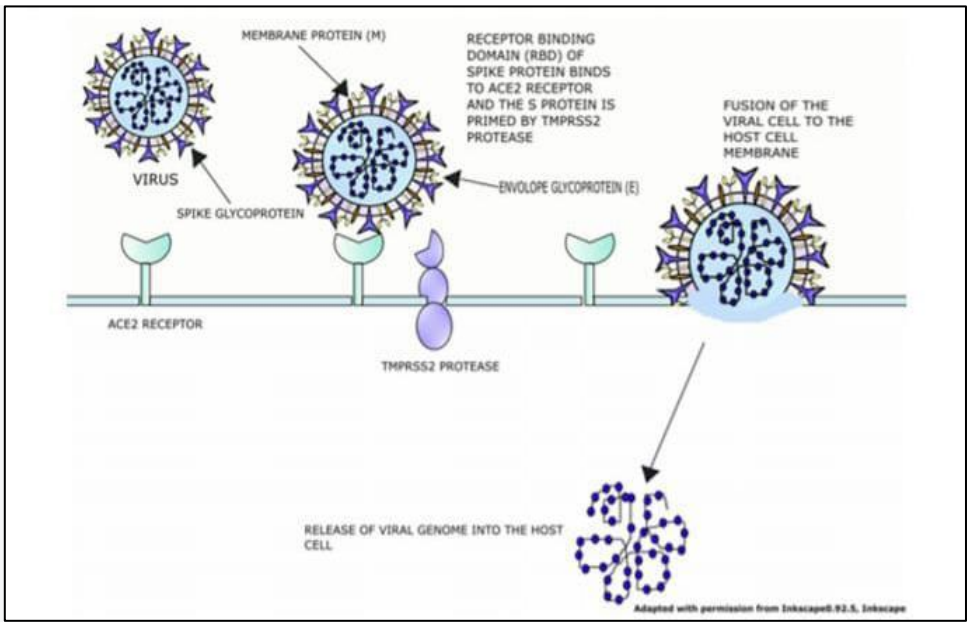
respiratory illness epidemics, and there are presently no effective medicines or preventative measures against them. Four of the six coronaviruses that have been identified as having previously infected humans are the cause of upper respiratory tract infections, intestinal diseases, and common colds. However, infections in the lower respiratory tract can be severe and frequently deadly when caused by beta coronaviruses, such as SARS-CoV and Middle-East respiratory syndrome coronavirus (MERS-CoV).(26) After being sequenced, the viral RNA from COVID-19-infected Wuhan patients was discovered to be a betacoronavirus with unique genetic traits. Interestingly, this betacoronavirus showed two recently identified putative short proteins that improve viral protein replication and transmission(27).

■ **TRANSMISSION OF SARS-COV-2**

Positive-sense RNA viruses known as coronaviruses can affect a variety of systems and have a broad spectrum of natural hosts (28). These viruses can cause a wide range of clinical disorders in people, from moderate colds to more serious respiratory conditions like SARS and

MERD(29).The advent of SARS-CoV-2 has caused extensive destruction in China and sparked a worldwide epidemic, despite continuous attempts to contain the outbreaks. According to the International Committee on Taxonomy of Viruses, the virus is known as SARS-CoV-2 (30). falling into the group of coronaviruses associated to severe acute respiratory syndrome and having a connection to SARS-CoVs(24) The order Nidovirales, family Coronaviridae, subfamily Orthocoronavirinae, and four genera—Alphacoronavirus, Betacoronavirus, Gammacoronavirus, and Deltacoronavirus—are where SARS-CoV-2 is categorized.(31). Bats are the original home of coronaviruses, including Gamma Coronavirus and 38% of the material. (34) A characteristic of CoVs that sets them apart from other positive-sense RNA viruses is their helical symmetry, which is encased in an envelope that contains viral nucleocapsids.SARS-CoV-2 electron micrographs exhibit a characteristic spherical form with variable virion sizes and spikes, like to that of a solar corona. 5'-leader-UTR-replicase-structural genes (S-E-M-N)-3' UTR-poly(A) is the linear organization of the CoV genome (35). The hemagglutinin-esterase gene (HE), 4a/b, and several accessory genes are scattered among the structural genes.Although SARS-CoV-2 codes for a number of auxiliary proteins, it does not have the HE gene that several other beta coronaviruses do. The mRNA, or positive-sense genome, of CoVs is translated into polyproteins 1a and 1ab. (36)The polyprotein gene-encoded nonstructural proteins (nsps) in double-membrane vesicles (DMVs) form a replication-transcription complex (RTC).(37). Afterwards, the RTC

Using molecular analysis, SARS-CoV-2, a new Betacoronavirus from the subgenus Sarbecovirus, has been discovered. Other zoonotic viruses of the same genus, including MERS-related CoV and SARS-related CoV, are comparable to it. 38.Nevertheless, with less than 90% similarity in the conserved open reading frame 1a/b (ORF1a/b), SARS-CoV-2 differs from other beta coronaviruses.Overall nucleotide identity with the original SARS-CoV is 80%, and with bat-related SARS-related CoVs ZC45 and ZXC21, it is 89%. Furthermore, SARS-CoV-2 and human SARS-CoV Tor2 and human SARS-CoV BJ01 2003 share 82% of their genetic makeup. In contrast, the recently discovered SARS-CoV-2 and the MERS-related CoV only share 51.8% of their genetic makeup.According to phylogenetic study of the structural genes, SARS-CoV-2 and bat SARS-related CoV are closely related.



Host receptor interaction with the SARS-COV-2 Spike protein & subsequent viral cell fusion with the host cell membrane.

Following this, virus-encoded proteases break these polyproteins into distinct non-structural proteins (nsps), such as papain-like proteases (PLPs), chymotrypsin-like protease (3CLpro), and the major protease (Mpro).These nsps are likewise encoded by SARS-CoV-2, and their roles have lately been determined.Notably, the discovery of a new short putative protein inside the ORF area represents a distinctive characteristic of SARS-CoV-2. This secreted protein, which is encoded by ORF8(42), has six strands in its beta-sheet and an alpha helix.

A few variations

1) Omicron

Omicron and its sub variants have been the most common SARS-CoV-2 strains in the US for about two years straight. The bulk of SARS-CoV-2 infections in the nation are today caused by different Omicron sub variants, despite the original Omicron strain (BA.1) no longer being in use. When Omicron was first discovered in late November 2021 in Botswana and South Africa, it quickly spread to neighboring countries and caused a spike in cases. Omicron led the number of daily cases in the US to soar to almost one million by December of the same year. It gave rise to many sub variants in 2022. A new Omicron strain called EG.5 (also known as "Eris") has emerged as the predominant strain as of 2023. the United States, and specialists are keeping a careful eye on another new strain known as BA.2.86, or "Pirola."

How infectious is it? Omicron's contagiousness is a serious worry. Omicron sub variants are well-known for their great transmission efficiency. The original Omicron strain was found to be

more transmissible than the Delta version. More than thirty changes in the virus's spike protein, which binds to human cells, are to blame for this increased transmissibility. It is thought that certain of these variations increase the risk of infection.

Intensity: Scientists are still working to determine if the most recent Omicron strains cause more severe infections than previous iterations. According to information, the first Omicron variety was usually less severe than later iterations, according to the CDC. Still, it is crucial.

Version 1: Researchers are still working to determine if the Omicron strains that are now in circulation cause more serious diseases than those that came before them. The CDC states that data suggests the initial Omicron strain was often less severe than previous versions. However, as was observed during the variant's early 2022 spread, when estimated death rates reached levels on par with or higher than those during the Delta variant surge the previous autumn, an increase in cases may lead to notable increases in hospitalizations and deaths.

Immunizations: they can aid in its prevention. The best defense against Omicron is to stay up to date on immunizations, however breakthrough infections in vaccinated persons may happen. This is according to the CDC. Researchers are now evaluating the effectiveness of a novel COVID-19 booster against EG.5 and BA.2.86 that is set for release in the autumn of 2023. According to the CDC, the It is expected that the improved vaccination would successfully reduce hospitalization and severe sickness brought on by these two new subvariants.

Version 1: It may be prevented by vaccination. The CDC notes that while it is possible for vaccinated persons to acquire breakthrough infections, adhering to vaccination regimens is essential for protection against Omicron. A novel COVID-19 booster that is scheduled for release in the fall of 2023 is now being studied for its efficacy against EG.5 and BA.2.86. According to the CDC, there should be a significant decrease in hospitalizations and severe illnesses brought on by these new subvariants due to the improved vaccination.

2) Delta: Towards the end of 2020, Delta (B.1.617.2) was first discovered in India. It then spread quickly around the world and became the dominant coronavirus strain. Nevertheless, Omicron's advent in December 2021 ultimately eclipsed its supremacy.It is very spreadable. The delta variation is thought to have caused more than twice as many illnesses than variants before it. It was expected to be 80–90% more transmissible in Connecticut than the Alpha version. The prior drop in COVID-19 cases and hospitalizations was abruptly reversed in June 2021 with the debut of Delta in the United States. There were spikes in the autumn of 2021 even in the states with the highest vaccination rates, leading specialists to advise additional injections.Compared to other variations, the Delta variant caused more severe sickness in those who had not gotten the vaccination. The Centers for Disease Control and Prevention (CDC) cited research from Scotland and Canada that showed Delta had a greater chance of hospitalizing unvaccinated individuals. According to a research in the Lancet, people in England who had Delta had double the chance of being hospitalized compared to those who had Alpha, the prior version that was more common in the area.It has been recognized that all three vaccinations are equally effective in protecting Americans against serious disease, hospital

stays, and death from Delta. All vaccinations do not guarantee 100% protection, though; some completely immunized people have reported developing breakthrough infections.Moreover, vaccinated people who contract the virus might still spread it to others. The CDC advised "layered prevention strategies" for vaccinated and unvaccinated people in response to the Delta variety. This entails becoming immunized and adhering to preventative measures including handwashing, mask use, and physical separation, especially in indoor environments where transmission rates are significant or elevated.

How spreadable is it?

In Connecticut, Delta was thought to have been 80–90% more transmissible than the Alpha form, accounting for more than twice as many infections as earlier variations**.**

After a consistent drop in COVID-19 instances and hospitalizations in the United States in June 2021, the entrance of Delta was accompanied by a sharp reversal of that trend. Even the states with the highest vaccination rates saw increases in the autumn of 2021, which is why experts are advising individuals to

Severity: In individuals who were not immunized, Delta produced a more severe sickness than other variations. According to early research from Canada and Scotland, which were both referenced by the CDC, people who are not vaccinated are more likely to get hospitalized after using Delta. People in England who used Delta had double the hospitalization risk compared to those who used Alpha, the prior main type in that nation, according to a Lancet analysis.

Is vaccination a preventive measure?

In the United States, all three vaccinations were thought to be quite successful in preventing serious sickness, hospital stays, and fatalities from Delta. While some individuals who were

completely vaccinated acquired breakthrough infections from Delta, no vaccination is 100% effective. Moreover, vaccinated individuals who contracted the virus may infect others, albeit it's probable that their infectious period was shortened.

Delta further initiated

3) Beta Version 1: This specific form, officially known as B.1.351, was discovered in South Africa toward the end of 2020 and then spread to other countries. Experts were concerned because of its many mutations and its ability to evade antibodies. It is important to remember, nevertheless, that the Beta version was uncommon in the US. The Centers for Disease Control an Prevention (CDC) estimates that Beta is around 50% more infectious than the initial coronavirus strain.

How spreadable is it?

According to the CDC, beta coronavirus was around 50% more infectious than the initial strain.

Severity: Compared to other variations, there were hints that Beta may have been more likely to cause problems.Is it feasible to have protection against it through vaccination? Clinical investigations show that the AstraZeneca-Oxford

vaccine, which is not available in the United States, does not provide considerable protection against mild and moderate instances of the Beta type, hence South Africa decided to stop using it in early 2021. Johnson & Johnson, Moderna, Pfizer-BioNTech, and others have similarly found decreased effectiveness against the Beta version.

Is vaccination a preventive measure?

Early in 2021, South Africa discontinued providing the AstraZeneca-Oxford vaccination (not available in the United States) due to clinical testing demonstrating its lack of efficacious protection against mild and severe sickness caused by the Beta

4) Alpha: Originally from Great Britain, Alpha (B.1.1.7) became the first widely reported variation. It was released in November 2020. By December of that year, the number of illnesses had greatly increased. Its popularity increased around the world and finally took the lead as the most common variant in the US, leading the CDC to label it as a variant of concern. But over time, the more forceful Delta variation became more popular, while Alpha gradually faded. Studies have shown that the B.1.1.7 lineage was more deadly than the original viral strain and

was more likely to cause hospitalization. Johnson & Johnson, Pfizer, and Moderna have all attested to the fact that their vaccinations are useful in instances where the Alpha version is involved in avoiding serious sickness and hospitalization. (43)

How spreadable is it?

It was believed that certain mutations in Alpha's spike protein increased its infectiousness. It was estimated that the B.1.1.7 lineage was 30 to 50% more infectious than the initial SARS-CoV-2 strain. Alpha accounted for 66% of cases in the U.S. in mid-April 2021, before Delta became the leading strain, according to a June CDC research.

Severity: Research indicates that the B.1.1.7 lineage was both deadlier than the original virus and more likely to cause hospitalization for infected individuals.

Is it anything that immunizations can stop?

Johnson & Johnson, Pfizer, and Moderna all claimed that their vaccinations helped keep Alpha patients out of the hospital and away from serious illness.

5) Omega:

The spike protein of the gamma variation shares several alterations with that of the beta and alpha strains.

Diagnosis:

1)RT-PCR is a diagnostic technique that uses samples from bronchoalveolar lavage (BAL), tracheal aspirate, or nose swab. Nasopharyngeal and or pharyngeal swabs are the major and recommended methods of obtaining upper respiratory samples for diagnosis. Bronchoscopy is not advised as a diagnostic technique for COVID-19 because of the serious risk that aerosolized particles provide to patients and medical personnel.

When upper respiratory samples produce negative results and using other diagnostic methods will substantially affect clinical treatment, bronchoscopy may be explored for intubated patients.

Nonetheless, when safety and clinical requirements are satisfied and the diagnosis is unclear, bronchoscopy could be the best course of action (44). As an alternative, respiratory specimens can be obtained via non-bronchoscopic BAL and tracheal aspirationrespiratory systems, upper and lower. BAL specimens and upper respiratory tract secretions have been used to cultivate the

virus. Unfortunately, there is a dearth of available RNA data. According to a research by Zou et al., samples taken from the upper respiratory tract had significantly greater amounts of SARS-CoV-2 RNA, especially in the first three days following the beginning of symptoms. Furthermore, samples taken from an asymptomatic patient's upper respiratory tract revealed elevated amounts of SARS-CoV-2 RNA (46).Studies have shown that SARS-CoV-2 RNA may be detected in feces and blood samples as well (47). It is still unknown how long SARS-CoV-2 RNA was present in extrapulmonary samples, the upper and lower respiratory tracts, and both. It Although the RT-PCR test has a high degree of specificity, swab contamination may result in false-positive results, especially in those who are asymptomatic.Although the exact sensitivity rate is unknown, estimates place it anywhere between 66 and 80% [59]. Even more ambiguous is the test's accuracy in asymptomatic people who have been in close proximity to symptomatic people; even in the absence of symptoms or a proven illness, the positive rate can reach 50% (49).When the test is done using a nasopharyngeal swab material early in the infection, a single negative test result may not rule out SARS-CoV-2 infection, especially in people with significant exposure. It can be advised to repeat the test or collect a larger respiratory sample.

2) Antibody: When an undesirable material enters your body, antibodies, which are proteins, shield you. Antibodies, which are generated by the immune system, attach themselves to these foreign chemicals and drive them out of your body. Antibody can also be referred to as immunoglobulin.

3) Antigen: An antigen is a sign that informs your immune system of the potential harm that certain substances in your body may pose. Antigens are present on bacteria, viruses, cancers, and healthy bodily cells.

Current COVID-19 Treatment:

Preventing transmission should be the first priority when it comes to treatment because the efficacy of currently available antiviral medications is unknown. This is especially true for people with moderate illness.Patients receiving

at-home care must be closely observed, and if their health worsens, therapy must be escalated right away.(50). There is conflicting evidence about corticosteroids' anti-inflammatory properties and their potential to increase viral proliferation.(51).Nonetheless, in situations of severe COPD or when other indicators are present, corticosteroids may be taken into consideration (52). Nebulized medicines are not as

effective as inhalers in reducing the risk of viral dissemination through aerosol-generating procedures (53). There is ongoing debate on the effects of nonsteroidal anti-inflammatory medicines (NSAIDs) on viral infection and ACE2 receptor levels.Although certain research indicates a The World Health Organization (WHO) and the European Medicines Agency (EMA) do not recommend against the use of NSAIDs. Acetaminophen is typically recommended over NSAIDs in hospital settings because of the increased risk of bleeding and kidney damage that comes with NSAIDs. Another contentious issue is the use of angiotensin receptor blockers and ACE medications. But as of right now, neither the American Society of Cardiology nor the European Society of Cardiology advise beginning or ceasing to use these drugs (56). It is best to make the option to treat COVID-19 patients with antiviral and anti-inflammatory medications individually, ideally in conjunction with infectious disease experts and as a part of a clinical study or registry. Oxygen treatment is frequently beneficial for patients with mild to severe illnesses. Adding more oxygen by All hospitalized patients should be evaluated for thromboembolism, unless there are particular contraindications because this patient population has a higher risk of venous thromboembolism.

Results of laboratory tests:

When hospitalized pneumonia patients were first admitted, the most common laboratory abnormalities found were low white blood cell counts (9–25%) or high counts (24–30%), low lymphocyte counts (63%), and elevated levels of aspartate and alanine aminotransferases (37%) (58). Of the 1099 patients with COVID-19, 83% had lymphocytopenia and 36% had.A considerable proportion of individuals had thrombocytopenia, and 34% also had leucopenia (59). Moreover, elevated lactate dehydrogenase, hypertransaminasemia, and moderate thrombocytopenia were seen (60).Increased inflammatory indices, which are generally defined by decreased procalcitonin levels and raised C-reactive protein (CRP) levels, were linked to clinical severity. According to Young et al., those with normal oxygen saturation had an average CRP of 1.1 mg/dL, but those who were hypoxemic had Radiographic findings: Ground-glass opacities were typically seen in the lower lobes and peripheral parts of the brain, along with bilateral numerous lobular and subsegmental areas of consolidation, especially in patients undergoing intensive care units (64).The number of lung segments damaged by the illness was shown to be connected with its severity. As the illness worsened, these opacities tended to

combine and get thicker. Unusual CT results include tumors, cavitations, pleural effusion (which rarely occurs in around 5% of cases), andlymphadenopathies may suggest other medical conditions (65).An illustration of the

common CT patterns observed in COVID-19 patients may be found in Figure 1.According to a research that measured the interval between the beginning of symptoms and the first CT scan, 56% of patients with symptoms within two days had normal CT scan results (66).There are very few cases when ultrasound has been used as a diagnostic technique. Its sensitivity is around 75%, and its low specificity is affected by factors such as patient weight, operator skill, and the severity of the disease (67).

Oxygen treatment is required in situations of hypoxia (SatO2 < 93%) or when there are indications of respiratory distress. Oxygen treatment is commonly administered by a face mask, a high-flow nasal cannula, or noninvasive breathing techniques such a full-facial interface or a continuous positive airway pressure helmet. Because of the potential for dispersing contaminated aerosols, it is crucial to refrain from wearing nasal masks and nasal pillows.(68). When receiving oxygen treatment, arterial SatO2 levels should be regularly checked.

Immunizations:

1) Covid-19
2) Covishield
3) Sputnik-V
4) Varzevaria
5) Nova Vax

Drugs:-

1) Colostomine
2) Baricitinib
3) Remedisiver
4) Dexamethasone
5) Tocilizumab

Potential future therapeutic targets:

Tocilizumab, a monoclonal antibody that targets IL-6R, is one such particular anti-inflammatory drug that is gaining popularity. In Wuhan, 272 COVID-19 patients received tocilizumab, which is presently being studied in a nationwide multicenter clinical study in Italy. Even if the statistics appear promising, there is still not enough information to draw firm judgments about how successful these therapies are. (69, 70).Other possible anti-inflammatory therapies include mesenchymal stem cell therapy, interferon, and anti-IL-17, which can lower inflammation and encourage tissue regeneration in ARDS patients (71, 72).

A potential substitute might be the growth of T cells specific to 2019nCoV (73). Currently being considered as purely hypothetical but potentially intriguing options are molecules that target the Th1-mediated inflammatory cascade, such as roflumilast (a selective, long-acting inhibitor of the enzyme phosphodiesterase-4, already used to manage neutrophilic inflammation in patients with chronic obstructive pulmonary disease) or canakinumab (a human monoclonal antibody that targets IL-1b) (74). Therapeutic antibody formation is progressively shifting to replicate the protective antibody responses that are normally elicited by the engagement of innate receptors, such as complement, Toll-like, and Fc receptors. Immunostimulants and various monoclonal antibodies are being investigated as treatments for neutralizing mutant strains, perhaps including SARS-CoV-2 (75).A small amount of study has concentrated on inhibitors that break the link between thePotential treatment approaches include furin (a serine endoprotease), pro-mazine (78), emodin, receptor 1 blockers (sartanics) (77), and monoclonal antibodies against the S1 domain of the S protein (79). Using short interfering RNAs to target the structural genes of the S protein, envelope, or membrane proteins is an intriguing strategy (80). It has been demonstrated that broad-spectrum antiviral drugs, such dsRNA-activated caspase oligomerizer, may specifically cause host cells harboring viruses to undergo apoptosis. These substances, however, are unable to stop viruses from entering cells or to damage the viral nucleic acid. As a result, they have to be assessed in conjunction with other treatments including zinc, mercury, thiopurine compounds, naphthalene inhibitors, and protease inhibitors (80). Furthermore, using antagonists of the bradykinin receptors B1 and B2 might be a uniquemethod for managing COVID-19-related local lung angioedema that is reliant on bradykinin. Additionally, these antagonists may react indirectly to anti-inflammatory drugs or neutralizing techniques for effects generated by anti-S antibodies. But it's doubtful that these antagonists will be able to completely correct all pulmonary edema (81). Using plasma from patients who are recovering for passive

immunotherapy is another intriguing technique that is now being investigated (82). However, patients should exercise caution because it has been discovered that those who produce anti-S-neutralizing antibodies early in the course of the disease are more likely to die from COVID-19. Additionally, pulmonary disease has previously been shown to deteriorate in cases of SARS-CoV infection (83). It is hoped that a vaccination would eventually be made accessible, despite the fact that its research and regulatory clearance will take time.

Conclusion:

COVID-19 has shown to be extremely infectious and lethal, while sharing clinical symptoms and molecular pathways with other illnesses brought on by beta coronaviruses. As a result, experts throughout the world have pledged to work together to save mankind against this amazing menace. Our understanding of the pathogenesis and clinical symptoms of COVID-19 has advanced thanks to this team effort, which has also improved hospitalized patients' prognosis. The creation of COVID-19 vaccines is moving quickly, with many candidates presently undergoing phase 3 trials. High-throughput drug discovery platforms are also being used to create COVID-19 treatment plans that work and repurpose current medications.

Currently, more than 300 clinical trials are being conducted on COVID-19 patients to evaluate the safety and effectiveness of several medication options. A ray of hope that a vaccine and/or efficacious therapy for COVID-19 may soon be accessible is provided by the recent explosion of publications in scholarly journals that address the etiology, pathophysiology, clinical therapies, drug development, and repurposing initiatives of this dreadful illness. This review article attempts to provide an overview of the quickly changing field of COVID-19 combat by summarizing the pathophysiology of the virus from the standpoint of pharmaceutical therapies that are now being researched.

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