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A Review on Covid -19

Bongirwar Nidhi*[1], TalluriSritanya[2], Sadiya Fatima[3], Salekula Keerthi[4], Dr. Chandrasekhara Rao Baru[6]

1 Assistant Professor Of Chilkur Balaji College Of Pharmacy ,Aziz Nagar RR District .
2,3,4 Students OfChilkur Balaji College Of Pharmacy, Aziz Nagar RR District.
6 Principal OfChilkur Balaji College Of Pharmacy, Aziz Nagar RR District.

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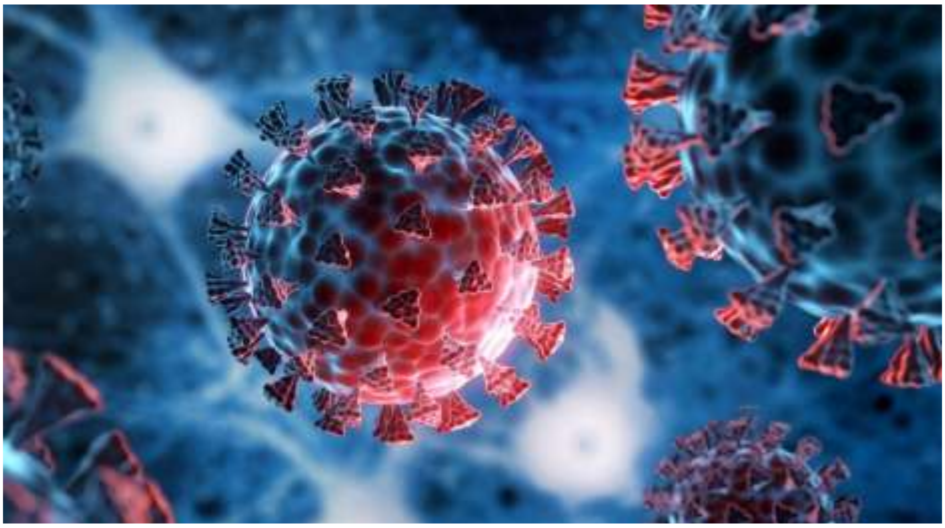
ABSTRACT:The coronavirus disease COVID-2019, which was termed 2019-nCoV (2019 novel coronavirus) until February 11, 2020, is presented in this study. COVID-2019 is caused by the SARS-CoV-2 coronavirus. An unidentified etiology pneumonia outbreak in Wuhan, China's Hubei province, was initially reported in a World Health Organization paper in Chinese. The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) that causes coronavirus disease 2019 (COVID-19) was first discovered in Wuhan, the provincial capital of Hubei, China, in December 2019. Since then, it has quickly spread throughout the world. On January 30, 2020, the World Health Organization (WHO) designated the epidemic as a Public Health Emergency of International Concern. On March 11, 2020, the WHO classified the outbreak as a pandemic.
Additionally, the diagnosis of the disease starts by gathering the samples of the upper and lower respiratory tracts of the infected person. Also, chest x-rays and CT scan are used in the diagnosis stage. Basically, there is no precise treatment for the aliment and this calls for the need to prevent the disease from spreading. Notable prevention strategies are isolation of the infected persons, proper ventilation, hand hygiene and use of personal protective equipment.
KEY WORD :SAR ,SMERS 2019-nCoV ,COVID-19,SARS-CoV-2



I. INTRODUCTION TO COVID-19

It includes a brief explanation of epidemiology,pathogenesis,diagnosis,therapy,prevention of covid-19.

The corona virus identified in patients with unknown cause of acute respiratory disease in wuhan,china at the end of 2019 has caused a global out break.

In this ,we summarized the knowledge about the causative pathogenesis of covid-19 and various diagnostic methods in the pandemic for better understanding of the limitations of virus testing for covid-19.

The purpose of the study was to investigate the opportunities and challenges of covid-19 disturbances.

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EPIDEMIOLOGY OF COVID-19

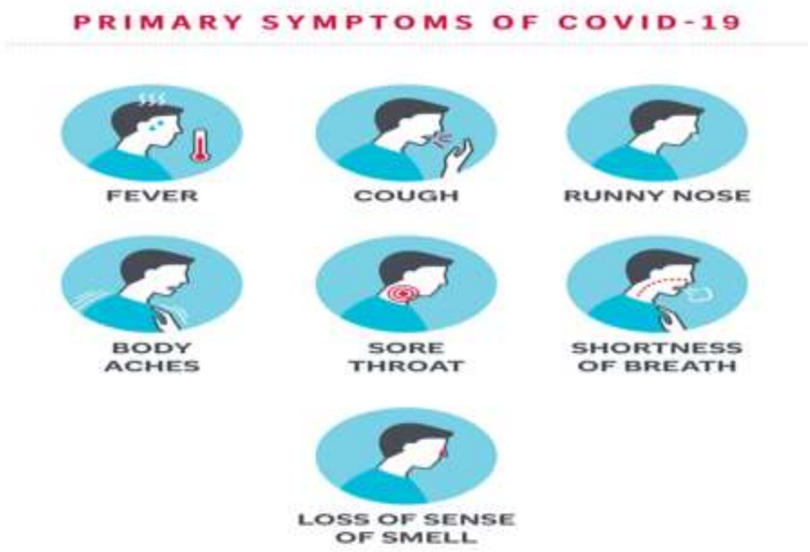
According to preliminary research, between 49 and 66 percent of patients had previously interacted with the Huanan seafood market, which sold a variety of live wild animals, such as chickens, bats, and marmots.[1-3].There is currently conjecture that wild animals are connected to the COVID-19 epidemic in Wuhan. WHO reports that SARS-CoV-2 was detected in environmental samples collected from the Huanan seafood market[4].SARS-COV and MERS-COV naturally occur in bats and are transmitted to humans by palm civets and dromedary camels, respectively[5].

Spectrum Of Infection.

The majority of COVID-19 cases with minor symptoms will recover in 1-2 weeks, and the virus has been described as a sort of infectious sickness that can self-limit. The five possible consequences of SARS-CoV-2 infection are: 1.2% of infected individuals may not show any symptoms; 80.9% of mild to medium cases; 13.8% of severe cases; 4.7% of critical cases; and 2.3% of all reported cases result in death[6].

Clinical Features

The most frequent symptoms in the first 41 patients were fever (98%), cough (76%), and myalgia or weariness (44%). Sputum production (28%) headaches (8%), hemoptysis (5%), and diarrhea (3%), on the other hand, were less frequent symptoms. In excess of 50% of the patients had dyspnea. The estimated values for the fundamental reproduction number (R0) and average incubation period were 2.2 (95% CI: 1.4–3.9) and 5.2 d (95% CI: 4.1–7.0), respectively[7,6].A blood test revealed lymphopenia (65%) and a white blood cell count that was normal or decreased (25%)(9). Under chest CT, 98% of patients had bilateral involvement. When ICU patients were first admitted, their chest CT scans typically revealed bilateral numerous lobular and subsegmental regions of consolidation. Both subsegmental regions of consolidation and bilateral ground-glass opacity were visible on the representative chest CT scans of non-ICU patients[8,9].



Epidemiological Characteristics In Mainland China.

Only Wuhan and the surrounding areas of Hebei Province saw the initial outbreak (8 December 2020), and on January 19, 2020, an imported case was first detected in Guangdong Province[6,7].44,672 cases were reported in all 31 provinces of mainland China as of February 11, 2020 (74.7% in Hubei). Of these, the percentage of cases that began before December 31, 2019, January 10, 2020, January 20, 2020, and January 31, 2020 were, respectively, 0.2% (100% in Hubei), 1.7% (88.5% in Hubei), 13.8% (77.6% in Hubei), and 73.1% (74.7% in Hubei) (Figure 1). After January 10, 2020, the number of instances recorded increased quickly, peaking on February

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12, 2020[10].214 cases (12.7%) nationwide, excluding Hubei, 394 cases (23.3%) in Hubei, and 1080 cases in Wuhan, accounting for 64.0% of the total incidence, were found through the study of 1688 medically confirmed cases with severe symptoms. The case-fatality rate for all COVID-19 patients over 80 years of age was as high as 14.8%. Male case fatality rates were 2.8% and female case fatality rates were 1.7% [6]. The prognosis was bad for patients with underlying fundamental illnesses. Cases without fundamental disorders had a case-fatality rate as low as 0.9%, but cases with cancer, diabetes, hypertension, cardiovascular disease, and chronic respiratory diseases had case-fatality rates of 10.5%, 7.3%, 6.3%, 6.0%, and 5.6%, respectively. Notably, the case-fatality rate for critical cases was the highest at 49%[6].

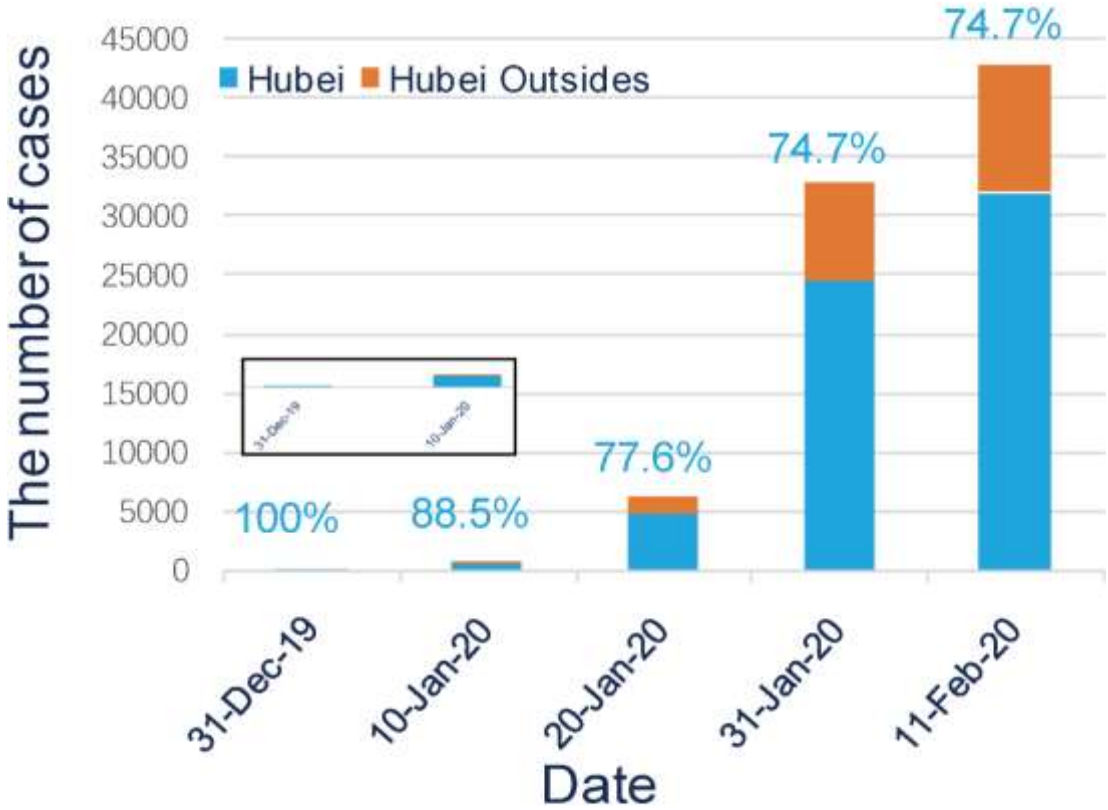

Fig.1.The number of cases in wuhan and surrounding areas.

Routes Of Transmission.

As of right now, respiratory droplets and touch transmission are the primary modes of transmission. SARS-CoV-2 has been found in the urine and stool of laboratory-confirmed patients, according to recent findings, suggesting a potential of fecal–oral transmission[11].

COVID-19PATHOGENESIS.

The SARS-CoV2 virus is the cause of COVID-19, a potentially deadly illness that is a major public health problem worldwide. Human pneumonia is caused by the SARS-CoV2 virus, which affects the lower respiratory tract. Although the symptoms of pneumonia are not as severe as those of SARS or MERS infection, hyperinflammation and respiratory failure eventually result in death[12].An asymptomatic phase with or without detectable virus, a non-severe symptomatic phase involving upper airway involvement, and a severe, potentially fatal disease with hypoxia, "ground glass" infiltrates in the lung, and progression to acute respiratory distress syndrome (ARDS) with a high viral load are the three phases that the bySARS-CoV2 infection and disease can be roughly divided (Fig. 2) [13].

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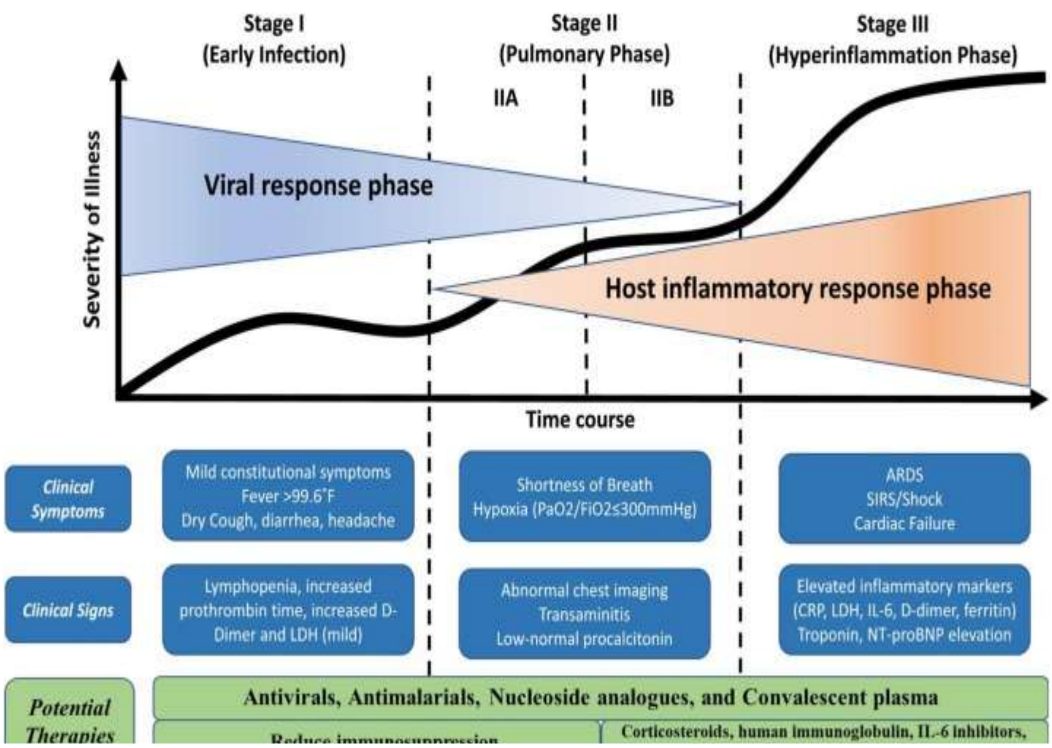

Fig. 2. COVID-19 pathogenic phases and potential therapeutic targets.

The four primary proteins that the coronavirus genome encodes are spike (S), nucleocapsid (N), membrane (M), and envelope (E). Viral entrance into target ACEII-expressing body cells is mediated by the S protein. The SARS-CoV2 and SARS-CoV genomes are nearly 75% identical, and both viruses use the angiotensin converting enzyme 2 (ACE-2) receptor to infect endothelial and airway epithelial cells. These two viruses also share the same amino acid residues needed for receptor binding. [14].ARDS is the primary cause of mortality in COVID-19 cases, and it seems that MERS-CoV and SARS-CoV infections share immunopathogenic characteristics [15].The cytokine storm, an uncontrollably high level of systemic inflammation caused by immune effector cells releasing pro-inflammatory cytokines and chemokines, is one of the key characteristics of acute respiratory distress syndrome (ARDS) [16]. Patients infected with COVID-19 have been found to have elevated blood levels of various cytokines and chemokines, such as: IL1-β, IL1RA, IL7, IL8, IL9, IL10, basic FGF2, GCSF, GMCSF, IP10, MCP1, MIP1α, MIP1β, PDGFB, TNFα, and VEGFA [17].Similar to SARS-CoV and MERS-CoV infections, the resulting cytokine storm sets off a powerful inflammatory immune response that leads to ARDS, multiple organ failure, and ultimately death in severe cases of SARS-CoV-2 infection [16].Higher leukocyte counts, aberrant respiratory symptoms, and elevated plasma pro-inflammatory cytokine levels were all observed in COVID-19-infected patients [15] (Fig. 3) [18].Acute COVID-19 causes damage to the lungs and several other organs, including the liver, kidneys, and heart, which results in multiple organ depletion and is the direct cause of mortality [19-22].

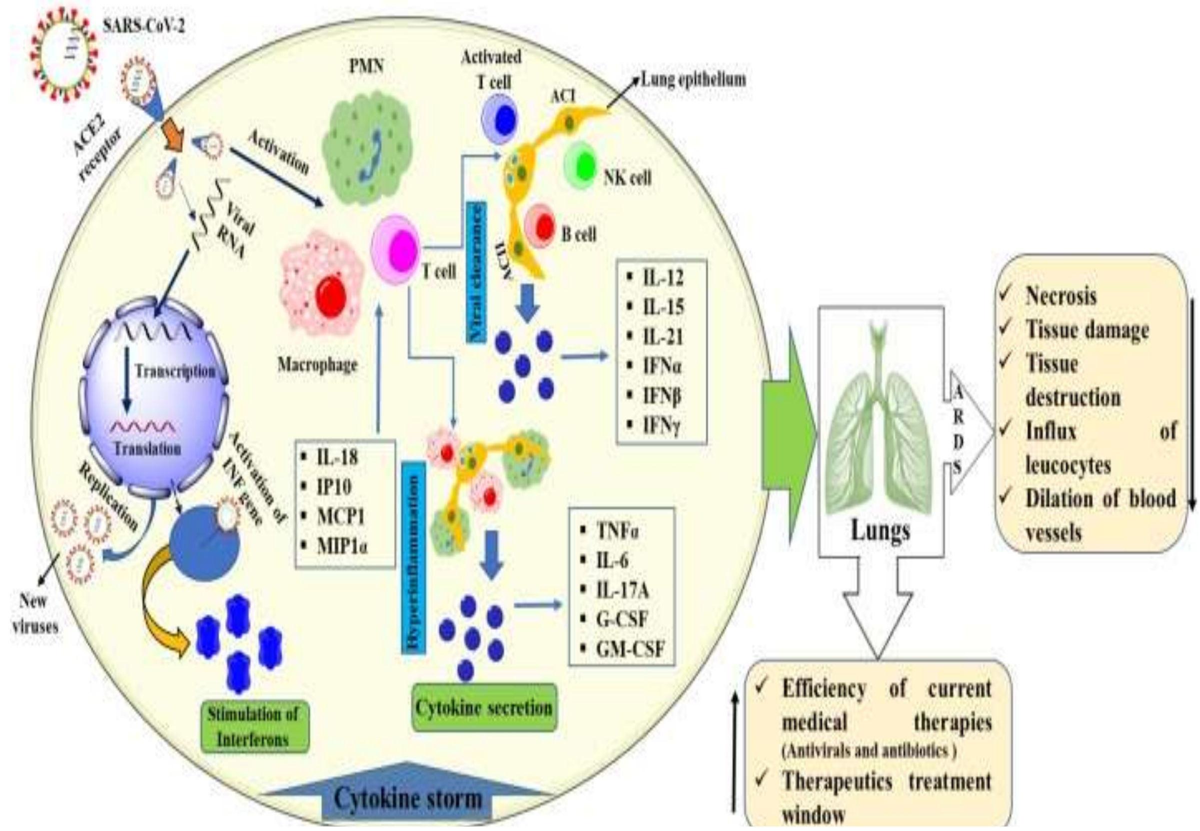

Fig. 3. Schematic representation of COVID-19 pathogenesis and cytokine storm with possible effects.

Diagnosis for the COVID-19

An precise line of diagnosis is necessary to determine the presence of COVID-19 in order to control it. It's interesting to note that there are several methods for diagnosing COVID-19. Isolating specific individuals from the population must come after confirmation. Therefore, early prevention is restricted to the diagnostic stage. It is very recommended that travel history be properly tracked during the COVID-19 outbreak, as evidenced by several previous cases from China (Wuhan city)[23].In accordance with regulatory criteria, the suspected instances must be verified by a preliminary thermal screening, molecular testing, and CT imaging[24].The following lists the suggested diagnostic methods for COVID-19 screening.

Infrared sensors and thermal scanning

Isolating the impacted population from the general population is a dependable method. It is also necessary for preliminary auxiliary diagnosis screening. Initially, a thermal camera was installed in the emergency room and at hospital gates to identify patients with varying body temperatures. For these cameras, a scanning distance of 10 m is necessary. Infrared energy is converted into a visual image by thermal cameras, which also detect and record it as heat. Installing these cameras in public locations such as airports, train stations, universities, and research facilities can be helpful in monitoring the body temperature of large crowds with an accuracy of ±0.5 °C. Thermal cameras often function at greater wavelengths of infrared radiation[25].In contrast, IR scanners are utilized for individual scanning; nevertheless, screening a huge population requires more time. Therefore, it can be said that thermal cameras are a better option than infrared scanners for large-scale screening.

Nucleic acid amplification test (NAAT)

According to the WHO-established procedure, NAAT is used to confirm COVID-19 disease using a nose swab or blood sample utilizing a real-time fluorescence polymerase chain reaction (RT-PCR)[26].According to current research, low viral load levels result in poor sensitivity because virus nucleic acid detection is difficult. This low detection could result in findings that are falsely negative[27].The US-FDA and other authorities

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have recently approved the emergency use of diagnostic kits that use RT-PCR technology to identify COVID-19[28].The Clinical Laboratory Improvement Amendments of 1988 (CLIA) mandated that emergency medical kits perform tests with moderate to high complexity. Cobas® SARS-CoV-2 was the first commercial diagnostic kit to meet these requirements[29].

Computerized tomography scan

Because NAAT is currently limited, Chinese researchers have suggested that CT imaging be used as the main foundation for the diagnosis of COVID-19[30].But this was a case study after the report by Huang et al., which said that the patient had just a history of fever (37.8 °C). The patient's report indicated that they were experiencing weariness and painful throat symptoms. Initially, a fluorescent RT-PCR test of the sputum produced negative COVID-19 results for six days. The patient's past travels included visits to Wuhan, China. Chest CT scan of the suspected patient revealed numerous peripheral ground-glass opacities in both lungs on the first day of the evaluation, with the left upper lobe and liner segment showing the most involvement. This group documented the development of ground-glass opacities in the lungs, which became more noticeable following three days of COVID-19 hospitalization[31].Consequently, the researchers strongly advise against using chest CT imaging as it has a low detection probability of viral pneumonia. Nevertheless, further findings, such as patchy consolidation of ground-glass opacities (GGOs) and a tendency for posterior or lower lobe involvement in COVID-19 cases [32].discussed the creation and application in clinical settings of a quick IgM-IgG mixed antibody test for the detection of SARS-CoV-2 infection. This team created a quick—less than 15 minutes—and easy point-of-care lateral flow immunoassay. This newly discovered immunoassay may concurrently identify IgM and IgG antibodies against the SARS-CoV-2 virus at various COVID-19 stages. This research team passed the specificity tests and validated the test kit using clinical samples for quick screening[33]

Covid-19 therapeutic options.

Acute respiratory distress syndrome (ARDS) was the most frequent consequence in COVID-19 patients, according to Huang et al. Acute heart damage, anemia, and secondary infections were the next most prevalent complications. Therefore, systemic corticosteroids, empirical antibiotics, and antiviral medications are employed as therapies . Furthermore, for patients with uncontrollable hypoxemia, Holshue et al. recommended the use of invasive mechanical ventilation. Therefore, the majority of COVID-19 infection patients' therapies are symptomatic[34-37].

Virally targeted agents

In the clinic, antiviral medications and systemic corticosteroids are used to treat viral infections. These medications include neuraminidase inhibitors (Oseltamivir, PeraImivir, and Zanamivir), ganciclovir, acyclovir, and ribavirin, as well as methylprednisolone, which decreased the risk of death in patients with acute respiratory distress syndrome. Nevertheless, they are not advised because they have not been demonstrated to have any impact on COVID-19[38-40].

Favipiravir (T-705)

In the clinic, antiviral medications and systemic corticosteroids are used to treat viral infections. These medications include neuraminidase inhibitors (Oseltamivir, PeraIA guanine analog that is authorized for use in treating influenza is favipiravir. It has the ability to suppress the RNA-dependent RNA polymerase of RNA viruses, including enterovirus, norovirus, influenza, yellow fever, chikungunya, and Ebola [68]. Additionally, researchers have confirmed its efficacy against 2019-nCoV in a recent investigation. Randomized clinical trials are being conducted to evaluate the effectiveness of Favipiravir plus Interferon-α (ChiCTR2000029600) and Favipiravir plus Baloxavirmarboxil (an authorized influenza inhibitor that targets the cap-dependent endonuclease) in treating COVID-19 patients (ChiCTR2000029544)[41-43].

Ribavirin.

A guanine derivative called ribavirin is authorized for the treatment of hepatitis C virus (HCV) and respiratory syncytial virus (RSV); it has also demonstrated promising outcomes in patients with SARS and MERS. However, at high dosages, ribavirin's adverse effects, including anemia, can be quite serious . Furthermore, its effectiveness against SARS-CoV-2 remains uncertain .Remdesevir, Sofosbuvir, and Ribavirin may be able to block SARS-CoV-2 polymerase, according to one in-silico investigation . But as previously indicated, another study disqualified it as a COVID-19 treatment that worked. So, further research is required as it is currently unclear if it is effective against COVID-19[44-47].

Remdesivir (GS-5734)

Remdesivir is a phosphoramidite pro-drug of an adenine derivative that shares structural similarities with the authorized HIV reverse transcriptase inhibitor Tenofovir alafenamide. In both cell culture and animal models, Remdesivir shown broad-spectrum action against Ebola as well as a variety of other coronaviruses, such as human, zoonotic bat, and pre-pandemic zoonotic CoVs [. Recent research findings indicate that Remdesivir may be able to inhibit 2019-nCoV . Additionally, on February 4, 2020, a phase III clinical trial of Remdesivir against COVID-19 was initiated in Wuhan. It is not anticipated that remdesivir would be extensively accessible as an experimental medication to treat a large number of individuals . Remdesivir, on the other hand, is a COVID-19 medication that has been licensed by the Food and Drug Administration (FDA)[48-51,43].

Galidesivir (BCX4430)

Adenosine analoggalidesivir was first created to treat HCV. Preclinical investigations against several RNA viruses, such as SARS-CoV and MERS-CoV, demonstrated the antiviral activity of galidesivir when it came to treating Yellow fever . Primary clinical trials are now being conducted to evaluate its safety in healthy individuals. It could therefore be helpful against SARS-CoV-2[44].

Protease inhibitors.

Lopinavir.

The main intended function of lopinavir was to block the 3-chymotrypsin-like protease seen in MERS and SARS. While a non-randomized open-label experiment showed improved clinical outcomes for SARS patients, it remains unclear if HIV protease inhibitors could effectively inhibit the 3-chymotrypsin-like and papain-like proteases of SARS-CoV-2.This is due to the fact that coronavirus proteases lack the C2-symmetric pocket that the HIV protease inhibitors' molecular design specifically tailored to meet the C2 symmetry in the catalytic site of the HIV protease dimer. It has been demonstrated that the combination of Lopinavir and Ritonavir is ineffective in treating patients with severe COVID-19, in contrast to earlier studies that demonstrated after administration of Lopinavir/Ritonavir, β-coronavirus the viral loads were significantly reduced and coronavirus titers were low or absent[44,52-54].

Disulfiram

Disulfiram is a medication that causes an acute intolerance to ethanol, or alcohol consumption, in order to cure persistent alcoholism. Although there isn't enough clinical data to support the claim, disulfiram has been shown to inhibit the papain-like protease of MERS and SARS in cell culture [55]. More research is need to determine whether it works against SARS-CoV-2.

Danoprevir

Danoprevir is an anti-HCV medication that inhibits the NS3/4A protease. It prevents the virus from replicating and lessens its impact on the host's defense against viral infection. After using Danoprevir for four to twelve days, all eleven COVID-19 patients in a limited clinical trial recovered [56]. Therefore, it is possible that danoprevir will work against SARS-CoV-2.

Chloroquine and hydroxychloroquine.

Chloroquine is a commonly used medication for autoimmune diseases and malaria that may also have broad-spectrum antiviral properties[57].By raising the endosomal pH necessary for virus/cell fusion and interfering with the glycosylation of SARS-CoV cellular receptors, chloroquine is known to prevent virus infection[58].According to the initial data gathered from over 100 patients, chloroquine appears to be effective in reducing pneumonia exacerbations, symptom duration, and viral clearance delay, all without causing serious adverse effects. The guidelines for the treatment and prevention of COVID-19 pneumonia included chloroquine. It will be necessary to evaluate the ideal chloroquine dosage for SARS-CoV-2 in further trials[59-61].An analog of chloroquine for which there are less worries about drug-drug interactions is hydroxychloroquine[62]. It was found that hydroxychloroquine has anti-SARS-CoV action in vitro during the earlier SARS outbreak[63].In SARS-CoV-2-infected Vero cells, hydroxychloroquine was found to be more effective than chloroquine using physiologically-based pharmacokinetic (PBPK) models[64].There have been reports of an increase in the cytokines IL-6 and IL-10 in response to SARS-CoV-2 infection . This could worsen into a cytokine storm, which would then lead to multiple organ failure and death. Chloroquine and hydroxychloroquine have the ability to decrease the immunological response and have immunomodulatory effects[65-68].

Corticosteroids.

In a study of 41 COVID-19 patients, 21% received corticosteroids, which could suppress lung inflammation [65].Methylprednisolone dosages were adjusted based on the severity of the illness. As of January 28, 2020, the World Health Organization's interim guidelines for the clinical management of severe acute respiratory infection in cases where SARS-CoV-2 infection is suspected prohibit the use of corticosteroids unless there is an alternative indication. The use of corticosteroids is not justified by the clinical results of coronavirus and related outbreaks. Among 309 critically sick people with MERS in a retrospective observational research, patients using corticosteroids had a higher need for mechanical ventilation, vasopressors, and renal replacement treatment[69].Corticosteroid therapy for the treatment of SARS was more closely linked to avascular necrosis, diabetes, and psychosis [70,71]. Overall, there is no special reason to believe that corticosteroids will help individuals who have COVID-19 infection, and these medications may even be harmful [72].

Convalescent plasma transfusion

When compared to placebo or no medication, the pooled chances of mortality after treatment for SARS were lower when convalescent plasma was given soon after symptoms began.Nevertheless, there was no discernible increase in survival in Ebola virus disease when 84 patients had transfusions of up to 500 mL of convalescent plasma The COVID-19 virus was isolated from a severely ill patient's bronchoalveolar lavage fluid in a laboratory test, and sera from many patients were able to destroy it[73-75].In order to combat COVID-19 infection, the National Health Commission of China urged recuperating patients to provide blood. Within two weeks after recuperation, convalescent plasma should be obtained in order to guarantee a high neutralizing antibody titer. The clinical utility of plasma is limited by the challenge of getting it during convalescence. To further assess the safety and effectiveness of convalescent plasma therapy in patients infected with COVID-19, carefully planned clinical trials are required.

Oxygen therapy

The majority of patients receive oxygen therapy, and extracorporeal membrane oxygenation (ECMO) has been suggested by the WHO for those with hypoxemia that is recalcitrant[76].

Antibiotics

In vitro research revealed Teicoplanin could stop SARS-CoV-2 from entering the cytoplasm, and antibiotics have been proposed as a treatment for pneumonitis symptoms [77]. Azithromycin has additionally demonstrated some efficacy against COVID-19.

Vitamins

Numerous studies have shown that vitamin B3 (nicotinamide or niacin) effectively reduced lung damage in animal models of bleomycin-induced lung damage. As vitamin C can lessen the severity of lower respiratory tract infections, it may also be useful in preventing COVID-19. Furthermore, it was proposed that taking vitamin D and E supplements could strengthen resistance to SARS-CoV-2. For patients with COVID-19, vitamin supplements may therefore be worth combining with other therapy[78-80].

Vaccine

Ten million doses of the COVID-19 vaccine were donated to the Nepalese population by the Indian government. The Nepali government made the decision to begin widespread vaccinations, beginning with security and medical personnel[81].The day before the immunization, all security and health personnel received short messaging service (SMS) notifications regarding their vaccine time slot. They awaited their turn to get their identity card verified on the day of the immunization. Frontline healthcare personnel were requested to complete a printed form with all of their personal information, including past allergies and cases of Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2). The Oxford (Covishield) vaccine, which contains the non-replicating chimpanzee adenovirus with SARS-CoV-2 spike protein in it, was given to the patient in the deltoid region by skilled nurses.

Following immunization, participants were instructed to wait in the observation room for thirty minutes in order to see if they might develop lightheadedness, dizziness, or minor headaches. Health professionals reported feeling irritable four hours after the vaccinations, and six hours later, several reported myalgia, nausea, injection site soreness, and a hot feeling. Twelve hours later, a fever and chills set in, which could only be relieved with paracetamol.Given that the body will require

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some time to adjust to the COVID-19 vaccine dose and for the immune system to produce protective antibodies, it appears that such minor side effects are tolerable. Therefore, the general public should be made aware of these mild side effects, which can be treated with paracetamol to relieve symptoms quickly, or such medications can be taken as prophylaxis to prevent developing post-vaccination symptoms. This will increase public acceptance of the COVID-19 vaccine while lowering psychological fear of any SARS-CoV-2 vaccination side effects, which will undoubtedly aid in the ongoing vaccination program's effort to combat the pandemic diseaseeffectively.It is intended to deliver the second dose of the vaccination as a booster after 28 days. Even though frontline health workers conducted mass vaccinations and offered Nepalese society hope, there is still a problem. The effectiveness and safety of vaccines are among the difficulties. An additional difficulty may be vaccine hesitancy and literacy[82].Another issue is that Nepal has discovered the new, more contagious coronavirus strains, which were initially identified in the UK and pose a threat to the vaccination campaign[83].surveillance by pharmacovigilance centers within a nation is urgently needed, as is long-term surveillance of adverse medication reactions[84].It is necessary to conduct randomized controlled clinical trials to verify the effectiveness of the vaccination in Nepalese communities. A pharmacogenomics investigation can validate the variations in the genetic sequence linked to the vaccination

Prevention.
Hygiene Maintenance

Respiratory infections are effectively prevented by good health and hygiene. Thus, to reduce the risk of respiratory droplets or aerosols, personal protective measures such as frequent hand washing and face mask use have been widely implemented[85].Distinctive ratios of alcohol-based hand sanitizers and disinfectants, such bleach, have been crucial in rendering SARS-CoV-2 inactive. But using a good N95 or FFP3 mask can help avoid respiratory tract infections. Using the proper sanitizers to disinfect surfaces and potentially hazardous materials is also advised for the environment[85-86].

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Use of Biocidal Agents

Around the world, a variety of biocidal chemicals, mostly in hospital settings, were employed for disinfection. These agents included alcohols, hydrogen peroxide, sodium hypochlorite, and benzalkonium chloride[87-88]Out of all the biocidal agents, 95% ethanol was shown to be extremely successful in COVID-19 disinfection after 30 seconds of treatment[88].The WHO published a guidance on local manufacture of alcohol-based hand rub formulations for patient safety in response to the overwhelming need for biocidal standards[89].

Social Distancing

Since social gatherings are the primary means of preventing COVID-19 from spreading throughout the community, it is imperative that community mitigation strategies to stop local transmission be properly thought out and implemented wherever practicable. It covers things like closing public spaces like schools, colleges, or private businesses, as well as transportation. Its expanded form, which forbids mass gatherings and mandates mask use, the segregation of sick individuals, and the use of suitable sanitation and hygiene practices, could lower the death rate.Curfews and extended national lockdowns will be useful measures to slow the spread of the epidemic and give it more time to spread. Therefore, by lowering both the total number of infected individuals and the overall health burden, the overall effect of social distance controls the height of the epidemic peak[90].

Quarantine People

Patients with confirmed COVID-19 cases or those with early signs should be quarantined in hospitals. Such quarantine populations need to have their close relationships closely monitored, tracked, and quarantined at home or in facilities designated for that purpose. These measures are used by the majority of nations (Hong Kong, Singapore, Taiwan, etc.) to stop the disease from spreading.These containment strategies seem to have been successful in stopping persistent local transmission thus far. Quarantine regulations, however, can be expensive to implement and present unique ethical and legal issues for each site, which could impede control attempts[86].

Mentality

Any preventive or precautionary strategy is behavioral, and a good mindset within oneself or the community has led to a fighting spirit and a synergistic effect against lethal viral illnesses.

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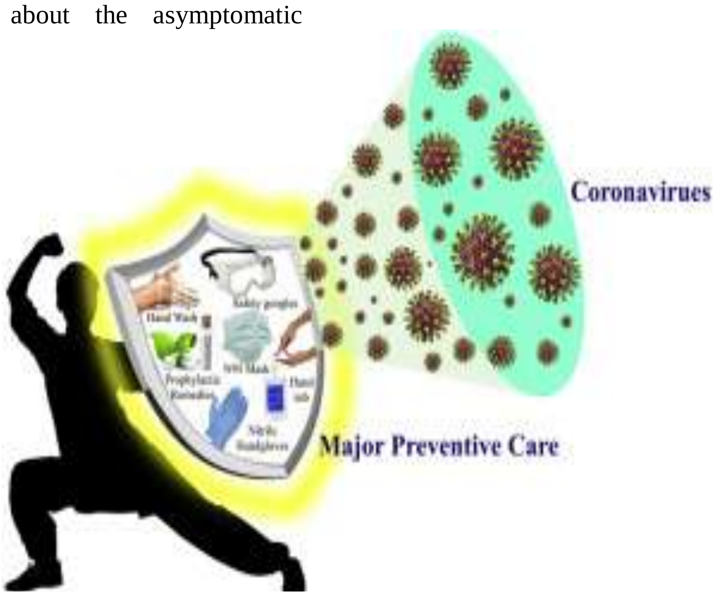
Therefore, the COVID-19 mentality paradigm will be one of selfishness, with the community coming second to yourself. Therefore, it is crucial to self-disclose information about the asymptomatic patient and its isolation endeavor, as well as to accept decisions made by the government and health department[86].



II. CONCLUSION

Since there is now no effective treatment for COVID-19, as is well known to all people on the planet, it is crucial to stop the disease from spreading throughout society.RNA viruses like COVID 19 are very dangerous to the general public's health.As of right now, the illness is responsible for thousands of infections and fatalities.Hand cleanliness, social distancing, and quarantine are the key components in stopping the spread in society.

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