International Journal for Multidisciplinary Research (IJFMR)
E-ISSN: 2582-2160 ● Website: www.ijfmr.com ● Email: editor@ijfmr.com

COVID-19: A Comprehensive Review of Epidemiology, Pathogenesis, and Clinical Management

Priyanka Sawant[1], Bushara Shaikh[2]

[1]Assistant Professor, Department of Pharmacy, M.S. College of Pharmacy, Devghar, Maharashtra
[2]B. Pharmacy Student, Department of Pharmacy, M.S. College of Pharmacy, Devghar, Maharashtra

Abstract
By 2024, COVID-19 had mostly become an endemic virus, and like seasonal flu, its control is now integrated into routine public health monitoring. Although the severity has decreased due to vaccination, booster shots, and better treatments, WHO and CDC are keeping a careful eye on the emergence of novel Omicron-derived variants (such as JN.1, XEC, and NB.1.8.1/Nimbus). About 6–7% of individuals worldwide suffer with long-term COVID, which is still a serious problem. Its symptoms include exhaustion, mental fog, and dyspnoea, and it disproportionately affects women, those with disabilities, and those with lower incomes. In terms of the economy, the pandemic revealed disparities in health and vaccination availability while speeding up digital transformation, remote labour, and supply-chain shifts. Despite ongoing global efforts in pandemic preparedness, future health disasters are always a possibility.

Keywords: Variants, Endemic transition, Vaccination & boosters, COVID.

1. Introduction:

SARS-CoV-2 begins infection when its spike (S) protein's receptor-binding domain (RBD) in the S1 subunit binds to ACE2 on respiratory and intestinal epithelial cells. The spike is synthesized as an inactive precursor and requires two proteolytic cleavages first by furin at the S1/S2 boundary during viral maturation, then by host proteases like TMPRSS2 at the cell surface or cathepsins B/L in endosomes to expose the S2 fusion peptide. These cleavages trigger conformational rearrangements that enable direct membrane fusion or endocytosis-based entry, releasing viral RNA into the cytoplasm to kickstart replication. Besides TMPRSS2, other proteases such as trypsin, plasmin, and factor Xa may also contribute [1,2]. RT-PCR remains the gold standard for COVID-19 diagnosis due to its high sensitivity and specificity, though it requires sufficient viral RNA and technical expertise. Insights from SARS, MERS, and influenza have guided immune-monitoring strategies [3]. Highlight peripheral T-lymphocyte subsection dynamics that correlate with disease progression and outcomes. SARS-CoV-2 likely originated from insectivorous bats (Rhinolophus sinicus) and spilt over into humans through intermediate hosts like raccoon dogs (Nyctereutes procyonoides) or masked palm civets (Paguma larvata). The rapid, prolific output of preprints and peer-reviewed studies has significantly deepened understanding of viral pathogenesis and spurred exploration of therapeutic and diagnostic innovations. A notable portion of COVID-19 patients develop long COVID, experiencing persistent symptoms like extreme fatigue, shortness of breath, joint pain, brain fog, and mood swings—often lasting months or even years post-

infection [4,5]. Long COVID spans over 200 different symptoms across multiple organs, commonly involving cognitive dysfunction, respiratory issues, cardiovascular irregularities, and musculoskeletal pain [6]. Cases have been documented in teens, adults, and the severely ill alike, requiring multidisciplinary care due to their varied and often overlapping nature. The virus responsible for COVID-19, SARS-CoV-2 (initially named 2019-nCoV), emerged in Wuhan, China, in late 2019. Genetic analysis established that Chinese researchers sequenced and shared the full genome in early January 2020 accelerating diagnostics and epidemiological tracking [7,8]. The outbreak was linked to a cluster of pneumonia cases at the Huanan Seafood Market, prompting the WHO to declare a Public Health Emergency of International Concern on January 30, 2020, and a pandemic by March 2020; early cases quickly spread beyond China to countries like Thailand, Japan, and the United States [9,10]. Epidemiologic tracing supports zoonotic transmission with bats (Rhinolophus species) as the reservoir and possible intermediate hosts (e.g., raccoon dogs), although investigations continue into the exact pathway of spillover. SARS-CoV-2 is a betacoronavirus with a pleomorphic, ~80–160 nm encased virion and a ~27–32 kb positive-sense RNA genome. It is the third highly deadly coronavirus to emerge in barely 20 years, following SARS-CoV (2002–04) and MERS-CoV (2012) [11,12]. The WHO declared a global public health emergency in January 2020 and a pandemic by March due to the high rate of human-to-human transmission, which reached over a million cases globally in a matter of months. It attaches itself to ACE2 on human cells and enters by protease-activated fusion or endocytosis [13]. Its zoonotic spillover is still being studied by scientists, although it most likely originated in bats, with potential intermediate hosts like civets or raccoon dogs. From minor respiratory symptoms to severe pneumonia, organ failure, and death, COVID-19 exhibits a wide range of clinical manifestations [14,15]. Multi-organ dysfunction and hyperinflammatory cytokine storms are common features of severe patients. Although there are still issues with fair vaccination and medication distribution worldwide, treatment has progressed from repurposed medications to targeted antivirals like molnupiravir, paxlovid (nirmatrelvir-ritonavir), and remdesivir, in addition to immunomodulators such as dexamethasone and tocilizumab. Furthermore, many patients have long-lasting COVID, which causes symptoms including joint pain, exhaustion, and fogginess to persist for a long time after infection [16,17,18].

1.1 What Are Coronaviruses?

Coronaviruses are enveloped, positive-sense RNA viruses (~80–160 nm, ~27–32 kb) classified into four genera—α, β, γ, and δ. SARS-CoV-2, a β-coronavirus, encodes four main structural proteins: Spike (S), Envelope (E), Membrane (M), and Nucleocapsid (N) [19]. The trimeric S protein split into S1 (binding) and S2 (fusion) mediates host entry via ACE2 receptors, aided by proteolytic activation by host enzymes like furin, TMPRSS2, and cathepsins B/L, enabling either direct membrane fusion or low-pH endosomal entry [20,21]. This process can generate pathological syncytia multinucleated cells that enhance viral spread and evade immune detection. Coronaviruses also form double-membrane vesicles and deploy non-structural proteins and accessory factors to disrupt host pattern-recognition receptors, interferon responses, and antigen presentation, effectively evading innate immunity [22,23].

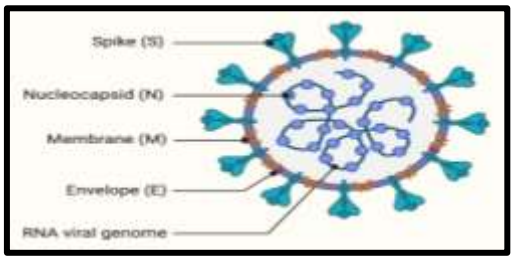

Figure. 1 Schematic Structure of Coronaviruses

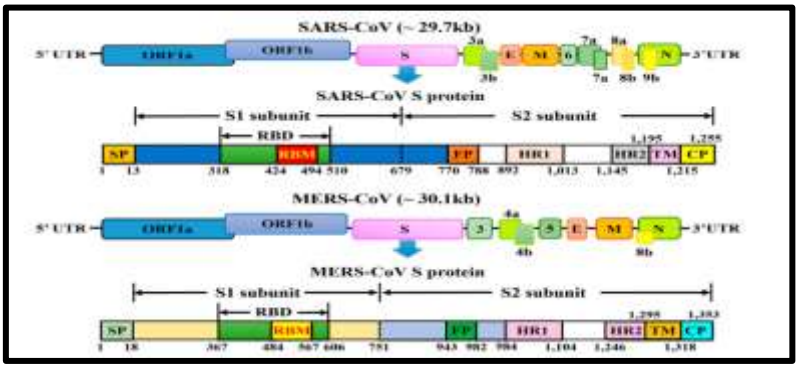

Figure. 2 Schematic representation of the genome organization and functional domains of S protein

Coronaviruses, including SARS-CoV and MERS-CoV, have large (~27–32 kb) positive-sense ssRNA genomes. Two overlapping ORFs at the 5′ end ORF1a and ORF1b encode polyproteins that are proteolytically cleaved into 16 non-structural proteins (nsp1–nsp16), essential for replication and transcription. The remaining 3′ third of the genome encodes four structural proteins Spike (S), Envelope (E), Membrane (M), and Nucleocapsid (N) plus accessory proteins whose number and arrangement vary by virus [24]. The S protein consists of S1 (with signal peptide, N-terminal domain, and receptor-binding domain) and S2 (with fusion peptide, heptad repeats, transmembrane, and cytoplasmic tail); the S1/S2 cleavage sites mark critical activation points. This genomic layout and proteolytic processing highlighted in schematics of SARS-CoV and MERS-CoV S proteins demonstrate how conserved non-structural proteins support viral replication, while structural and accessory proteins define entry, assembly, immune evasion, and pathogenicity [25,26].

1.2 TYPES OF CORONA VIRUSES:

Coronaviruses are enveloped, positive-sense single-stranded RNA viruses (~26–32 kb) classified into four genera: alpha, beta, gamma, and delta. Seven coronaviruses infect humans: four relatively benign ones HCoV-229E and HCoV-NL63 (alphacoronaviruses), along with HCoV-OC43 and HCoV-HKU1 (beta coronaviruses) which together cause 15–30% of common colds and typically result in upper-respiratory infections, with occasional mild pneumonia in vulnerable individuals. The remaining three SARS-CoV, MERS-CoV, and SARS-CoV-2 are more pathogenic beta coronaviruses associated with severe respiratory illness and global outbreaks: SARS (2002), MERS (2012), and the ongoing COVID-19 pandemic.

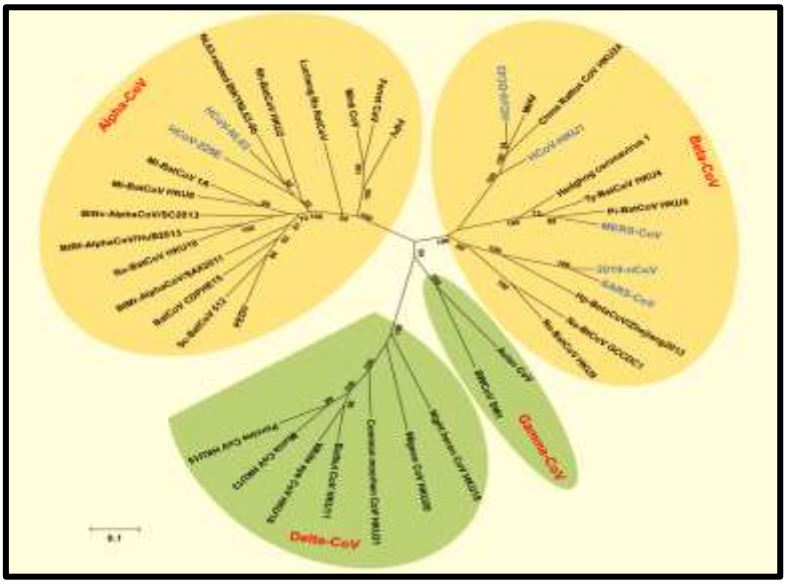

Figure. 3 Phylogenetic tree of coronaviruses (CoVs) based on the nucleotide sequences of RNA dependent RNA polymerase (RdRp)

Coronaviruses belong to the subfamily Orthocoronavirinae and are classified into four genera Alpha, Beta, Gamma, and Delta as identified through maximum likelihood phylogenetic analyses (e.g., using MEGA 6 with 1,000 bootstrap replicates). Each genus comprises various subgenera, with human-infecting coronaviruses including SARS-CoV, MERS-CoV, and SARS-CoV-2 falling within the Beta coronavirus group. The phylogenetic tree clearly delineates these clusters, with human-only coronaviruses highlighted in blue and high bootstrap values confirming their evolutionary relationships [19,27].

2. EPIDEMIOLOGY:

COVID-19 initially spread from a Wuhan seafood market through animal-to-human transmission but quickly evolved into widespread human-to-human transmission, primarily via large respiratory droplets (exhaled when coughing, sneezing, and talking), which typically travel up to 1–2 m and settle on surfaces. Viral load peaks around symptom onset in the nasal passages higher than in the throat and infectious virus can be transmitted shortly before symptoms appear. Around 78–85% of transmission clusters occurred within households or close contact settings. Although surface (fomite) transmission is possible, research indicates close and prolonged contact remains the dominant route. Zoonotic analysis shows bats as the original reservoir (96% genomic similarity), while the intermediate host remains unconfirmed [28,29].

3. VIROLOGY- PATHOGENESIS:

SARS-CoV-2 belongs to the Beta coronavirus subgenus, sharing ~95% genome homology with bat coronaviruses and ~70% similarity in the spike protein's receptor-binding region to SARS-CoV, which earns it the name SARS-CoV-2. Its spike protein binds the ACE2 receptor on respiratory epithelial cells, initiating entry via fusion or endocytosis. Following entry, the viral RNA along with ~two-thirds of the genome encoding ORF1a/ORF1b and associated non-structural proteins replicates, while the remaining third encodes structural proteins (S, E, M, N) necessary for assembling new virions. The disease typically begins with an asymptomatic phase (~3 days), followed by a symptomatic phase (~3 days). Transmission can occur before symptoms appear, making early containment challenging. SARS-CoV-2 can infect not

only lungs but also kidneys, liver, intestines, and T lymphocytes. Early estimates of case-fatality rates (CFR) varied widely from 0.5% to 5% globally but more recent meta-analyses indicate ~1% across the general population and up to ~13% in hospitalised patients, with higher mortality among those over 50 or in intensive care [30].

4. LIFE CYCLE OF CORONA VIRUS:

These are the steps involved in the life cycle of corona virus:

1. Attachment and entry.
2. Replicase protein expression
3. Replication and transcription
4. Assembly and release. [17,31]

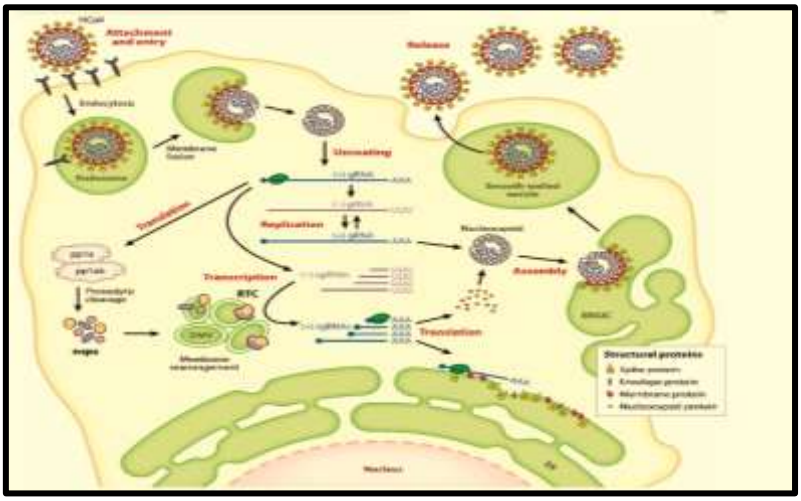

Figure.4 Life Cycle of Corona Virus

1. Attachment And Entry:

By causing conformational changes in the spike that promote membrane fusion, coronavirus spike (S) binding to ACE2 starts infection. Direct fusion at the plasma membrane (with TMPRSS2 priming) or endocytosis via low-pH endosomes, where cathepsins B/L activate the spike, are the two ways that entry happens. In order to prevent viral fusion, host cells use interferon-induced transmembrane proteins (IFITMs), primarily IFITM1 at the surface and IFITM2/3 in endo-lysosomal membranes. This prevents alpha-CoVs (such as HCoV-229E/NL63) and pathogenic SARS-CoV and MERS-CoV from entering the cell. Notably, in low-pH compartments, IFITM3 (and other IFITMs) can prevent spike-mediated entrance by halting fusion at intermediate stages. IFITM3's function in blocking early viral entry is further supported by cell-to-cell fusion tests, which show that it inhibits spike-driven syncytia formation.

2. Replicase Protein Expression:

Coronavirus replication begins with the translation of ORF 1a and 1b into polyproteins pp1a and pp1ab. ORF1b is translated through a ribosomal frameshifting mechanism, cleaving polyproteins into 15 nsps. This assembly forms the replication-transcription complex, which transcribes the full-length positive strand of genomic RNA into a negative strand template for synthesis of different genomic RNAs and sub genomic negative-strand templates. Sub genomic mRNAs are then translated to form structural and

accessory proteins. Several RNA-binding proteins, such as m-aconitase, poly-A-binding protein (PABP), DDX1, PCBP1/2, and MADP1, play a role in efficient RNA replication.

3. Replication And Transcription:

The cytoplasm receives the discharge of the viral RNA genome. Complementary RNA strands are created by the viral RNA-dependent RNA polymerase (RdRp). Both structural and non-structural proteins are made from sub genomic RNAs.draws attention to the creation of new RNA strands and the cytoplasmic replication complex.

4. Assembly And Release:

The assembly of virions occurs during the infection life cycle, with the helical nucleocapsid containing genomic RNA intermingling with other viral structural proteins to form the assembled virus. Coronavirus particles are assembled through budding of the helical nucleocapsid through membranes. The M protein orchestrates the assembly process by organizing viral envelope components and facilitating interactions with the nucleocapsid. The M protein intermingles with E protein to assemble into a mature virus, generating the scaffolding of the virion envelope and causing budding and release of the M protein-modified membrane. After assembly, virions are transported in vesicles and released by exocytosis.

5. CLINICAL SYMPTOMS COMPLICATIONS:

SARS-CoV-2 infections are thought to incubate for 14 days after exposure, with the majority of cases occurring four to five days later. Although most SARS-CoV-2 infections occur in middle aged and older people, they can occur in people of any age. The median age ranged from 49 to 56 years in certain groups of hospitalized patients with a confirmed COVID-19 infection [15,32].

Clinical Features, Course and Complications of COVID-19 Disease.

COVID-19 typically begins with non-specific symptoms such as fever, dry cough, fatigue, sore throat, rhinorrhoea, headache, myalgia, nausea, vomiting, diarrhoea, and occasionally conjunctivitis. These symptoms overlap significantly with other respiratory viral infections, making early differentiation challenging. In about 15–20% of cases, typically by the end of the first week, the disease progresses to pneumonia, characterised by persistent fever, worsening dry cough, dyspnoea, and bilateral infiltrates on chest imaging. Severe cases may evolve into acute respiratory distress syndrome and multi-organ failure, which can be fatal. 81% of the 44,000 patients in a large cohort that China's CDC reported had mild to severe disease, usually exhibiting symptoms as light as pneumonia. Severe sickness, defined as dyspnoea, hypoxia, or lung imaging involvement greater than 50%, occurred in 14% of cases. The bulk of COVID-19 fatalities occurred in this category, with an overall case fatality rate of about 2.3%. Of these, 5% developed critical disease, with respiratory failure, shock, or multi-organ dysfunction to follow [33,34].

6. DIAGNOSIS:

According to China's National Health Commission, the diagnosis is made by combining epidemiological risk and clinical indicators (fever, cough, sore throat), which are verified by finding SARS-CoV-2 using RT-PCR, genome sequencing, or IgM/IgG serology [35]. Although nasopharyngeal swabs are the preferred method for RT-PCR, viral RNA can also be detected in anal swabs, saliva, sputum, blood, and stool. In some cases, anal swabs may remain positive after respiratory samples turn negative, indicating faecal shedding and necessitating careful discharge decisions. Lab findings frequently reveal lymphopenia, thrombocytopenia, delayed APTT, and normal or reduced white blood cell levels. When

PCR results are falsely negative and IgM and IgG may be found after the first week of sickness, serology becomes useful [36].

7. TREATMENT:

Supportive care is the cornerstone of treatment, with isolation as the first and most critical step to prevent transmission. Patients with mild illness (fever, cough, pharyngitis) should recover at home maintaining hydration, nutrition, and rest, and using symptomatic treatments like antipyretics and cough suppressants. Antibiotics and antivirals (e.g., oseltamivir) are not routinely recommended unless a co-infection is suspected. In moderate to severe cases involving hypoxia, supplemental oxygen is administered via nasal cannula, face mask, high-flow nasal cannula, or non-invasive ventilation. Patients with critical illness, including ARDS, may require mechanical ventilation or extracorporeal membrane oxygenation (ECMO). Renal replacement therapy is provided if necessary, and co-infections are treated with antibiotics or antifungals. Corticosteroids (e.g., dexamethasone) are strongly recommended for patients requiring supplemental oxygen or ventilation they reduce mortality by up to one-third in ventilated patients and by one-fifth in those on oxygen only. Steroids are *not* advised for non-hypoxic patients. Additionally, baricitinib or tocilizumab, in combination with steroids, is suggested for patients with severe disease and signs of systemic inflammation. For antiviral therapy, remdesivir is the only approved option for hospitalised patients requiring oxygen it modestly shortens time to recovery and may reduce progression and mortality, though its benefit is limited in ventilated patients. WHO previously issued a conditional recommendation, but recent data support its use in severe disease. Oral antivirals nirmatrelvir-ritonavir (Paxlovid) and molnupiravir are strongly recommended for non-hospitalised high-risk patients early in the disease to prevent progression. Other therapies like hydroxychloroquine, lopinavir/ritonavir, ivermectin, and convalescent plasma have been shown to have no significant benefit and are not recommended. Inhaled interferon-α nebulisation has been used in some countries, but it remains investigational and is not part of standard WHO/IDSA treatment protocols [37,38].

8. PREVENTION:

Traditional Chinese Medicine (TCM), particularly Yupingfeng San (Astragalus, Fangfeng, Atractylodes), has been used to bolster immune function and support lung health. Early studies during SARS and COVID-19 suggest TCM combined with modern medicine may reduce symptoms, accelerate recovery, and potentially lower mortality, though large-scale clinical evidence remains limited. Formulas like Sang Ju Yin, Yin Qiao San, Lianhua Qingwen, and others have been widely used in China and even among Chinese communities abroad with reported benefits in symptom relief and immunoregulation. Recognising the virus's stealthy infectiousness including asymptomatic transmission, prolonged incubation, and "wet-heat" respiratory pathology TCM emphasises prevention by "protecting lung Qi", as seen with Yupingfeng San. Public health strategies, in parallel, focus on home isolation, mask use, hand and respiratory hygiene, ventilation, and physical distancing, consistent with WHO and CDC guidelines. Together, this integrative approach marrying TCM's preventive and symptom-relief traditions with evidence-driven infection control aims to reduce disease spread and safeguard patient health in communities worldwide [39,40].

9. FUTURE PROSPECT:

COVID-19, caused by SARS-CoV-2, remains a high-priority target for diagnostics and therapeutics. Rap

id bioanalytical methods like RT-PCR, antigen tests, and serology have been favoured for their accuracy, cost-effectiveness, and sensitivity. A notable variant, VUI-202012/01 (later called Alpha, B.1.1.7), emerged in the UK in late 2020 with 17 spike mutations, including N501Y, which enhances ACE2 binding and increases transmissibility by an estimated 40–80% compared to earlier strains; though more contagious, it has not been definitively linked to higher severity. This prompted global genomic surveillance and public health measures. Vaccine development surged early in the pandemic: the Pfizer–BioNTech Comirnaty mRNA vaccine was the first to receive WHO Emergency Use Listing (EUL) in December 2020 and continues to be updated and authorised (e.g., 2024–25 formula for children aged ≥6 months) under emergency use frameworks. Other major vaccines Moderna, AstraZeneca, Novavax, and others also hold WHO EULs and are used globally. Meanwhile, antiviral drug research remains ongoing: older drugs like chloroquine have shown limited efficacy, while natural-product screens and in silico modelling highlight promising compounds that require further experimental validation [41].

Clinical features of COVID-19:

More than 80% of COVID-19 cases are asymptomatic or present with mild symptoms like fever, cough, respiratory discomfort, and shortness of breath. However, in severe cases, the illness can lead to serious complications, including severe pneumonia, acute respiratory distress syndrome (ARDS), respiratory failure, pulmonary oedema, sepsis, septic shock, multiple organ failure, and even death [42].

10. CONCLUSION:

COVID-19 has reshaped global health, economies, and societies. Health systems were stretched to breaking point, with over 282 million reported cases and 5.4 million deaths by 2021. Even now, the aftermath endures economic losses are estimated at US $13.8 trillion by 2024, widening inequalities, and uneven recoveries echo across nations. Long COVID affects roughly 400 million people globally and has trimmed labour forces by an estimated 3 million across OECD countries, with an annual economic toll exceeding US$ 1 trillion. Health systems continue to evolve rethinking funding, workforce well-being, digital infrastructure, and public health surveillance to enhance resilience. Major lessons trust-building, international collaboration, robust primary care, and proactive planning have emerged to guide future pandemic preparedness.

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