Decoding Omicron: Characterization and Global Impact of the SARS-CoV-2 (B.1.1.529) Variant

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Abstract

The SARS-CoV-2 B.1.1.529 variant, known as Omicron, is characterized by unique mutations in the spike protein that significantly enhance its transmissibility. First identified in late 2021, Omicron's rapid global spread has intensified public health responses and led to surges in COVID-19 cases. Epidemiological data indicate that Omicron can evade immunity from vaccines and prior infections, resulting in a higher incidence of breakthrough infections. The variant's impact on vaccine efficacy underscores the need for updated vaccines and booster strategies. Additionally, Omicron has caused significant socioeconomic disruptions, including strain on healthcare systems and daily life interruptions. The importance of global cooperation, genomic surveillance, and adaptable public health strategies is emphasized to manage and mitigate the effects of emerging variants. The review also discusses the implications for vaccine efficacy, emphasizing the necessity for updated vaccines and booster strategies. Additionally, it examines the socioeconomic repercussions of Omicron, including the strain on healthcare systems and disruptions to daily life. From a critical standpoint, the review highlights the importance of global collaboration and genomic surveillance in understanding and mitigating the effects of emerging variants. It underscores the need for continuous research and adaptable public health strategies to effectively combat COVID-19.

Keywords: Corona, SARS-CoV, Omicron variant, transmissibility, global spread, clinical.

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1. Introduction

The word "corona" means "crown," and the spherical virus contains a "crown" of peplomers, or proteins, protruding from its center in each direction, as demonstrated by scientific testing. Not only is this not the first time a coronavirus has made headlines, but a coronavirus was also responsible for the deadly 2003 SARS pandemic. Similar to the 2019 virus, the SARS virus was discovered in animals before it manifested in people. It is believed that the SARS virus originated in bats and then spread to other animals before reaching humans. After being transferred to humans, the SARS virus quickly began to spread among individuals.[1]

A serious threat to international health worldwide was represented by the coronavirus disease 2019 (COVID-19), which was brought on by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) that surfaced from Wuhan, China in December 2019. [/2] A variety of symptoms are brought on by COVID-19, such as fever, sore throat, anosmia, muscle soreness, dry cough, and shortness of breath. After COVID-19 first appears, the virus undergoes a number of mutations that lead to the formation of different mutant strains, including delta, beta, and alpha forms. On Nov. 24, 2021, a new SARS-CoV-2 variation of concern (VoC), omicron, was identified in South Africa, almost 23 months after the first case of COVID-19 was diagnosed. Specimens obtained in Botswana on November 11, 2021, and in South Africa on November 14, 2021, revealed B.1.1.529.[/3]

On November 26, 2021, the World Health Organization designated strain B.1.1.529 as a variation of concern (VOC) and called it Omicron, based on the suggestion of the WHO's Technical Advisory Group on Virus Evolution. The Omicron variant is thought to be the most mutated strain of SARS-CoV2, including VOCs and VOIs. It is a highly divergent strain with many mutations, including 26–32 in the spike protein. These mutations are concerning and may be linked to humoral immune escape potential and increased transmission rates. The Omicron variant's amino acid changes are present in four structural proteins: spike (S), envelope (E), membrane (M), nucleocapsid (N) proteins, and non-structural proteins (NSPs) (NSP3, NSP4, NSP5, NSP6, NSP12, NSP1).[4] Around the 16th of December in 2021, 89 nations in all six WHO regions were aware of the presence of the Omicron variant. January 10, 2021, saw the propagation of B.1.1.529 to 105 nations. On November 28, 2021, the World Health Organization announced that there was no proof indicating the manifestations connected to Omicron are different from those connected to other variations. Such specific manifestations of the illness and its severity are still unknown (WHO, 2021). With 99.7% of all alleles recorded between February 23 and March 24, 2022, the Omicron variation—identified in 188 countries up to March 31—has firmly established itself as the predominant strain globally.[5] BA.1, BA.1.1, BA.2, and BA.3 are the four sub-lineages of the Omicron type that have surfaced. Omicron versions BA.1, BA.1.1, and BA.2 are the most common ones in use today. When identifying the Omicron BA.1 variant—also known as the original form—S-gene target failure might be utilized. With an R346K mutation in the spike protein, BA.1.1 is a sub-lineage of BA.1. It is noteworthy that a greater proportion of BA.2 is not producing SGTF, and that Omicron BA.2 variations are becoming more common in several countries, such as Denmark, India, Norway, and Singapore, suggesting that it may have a selection advantage over Omicron BA.1 variant. The effective reproduction number of BA.2 was about 1.26 times more than that of BA.1, based on epidemiological studies done in Denmark. [6] Even though BA.1 is growing faster than BA.2, starting in January 2022, BA.2 has gained ground in several nations. Since the genetic sequence of the spike protein of the

BA.2 lineage differs from that of the BA.1 lineage, it may confer a greater immunological tolerance to antibodies. [7] Our knowledge of the Omicron version is still developing as new information becomes accessible. There's compelling evidence to suggest that Omicron is much outpacing Delta in terms of growth. Considering a doubled incidence period of 1.5–3 days, it spreads far quicker than the Delta variation in nations where community transmission has been established. In nations with high levels of population immunity, omicron is spreading quickly. It is unknown how much of this rapid development can be attributable to immune evasion, intrinsically higher transmissibility, or a combination of the two. On the other hand, according to available evidence, Omicron is probably going to beat Delta in the event of communal propagation.[8]

SARS-CoV-2's Omicron version infects cells that rely on its required receptor, angiotensin-converting enzyme 2. (ACE2). The spike protein of SARS-CoV2 contains furin protease cleavage sites and the S1 and S2 subunits. The S1 subunit (RBD) is composed of the N-terminal domain (NTD) and the receptor-binding domain. The angiotensin-converting enzyme-2 (ACE2) receptor on the surface of human cells and the receptor-binding motif (RBM) interact directly to mediate the invasion of viruses and determine the dissemination of the virus.[9] Additionally, vaccines, convalescent plasma, and monoclonal antibodies (mAbs) also target the spike protein for removal. The SARSCoV2 genome can undergo adaptive mutation, which can change the virus's phenotypic characteristics, immune evasion, and infectivity.10 Serious worries about increased infectivity, immunological escape potential, and reinfection have been raised by the finding of the Omicron variant.11] Starting in April 2022, the two new Omicron lineages (BA.4 and BA.5), which together account for over half of all sequenced cases, rapidly initiated the fifth wave in South Africa. Samples collected in South Africa in January and February of 2022 provided the first confirmation of the BA.4 and BA.5 lineages. Since then, these lineages have also been discovered in other parts of the globe, and numerous countries have come to acknowledge them. In the meantime, a number of new Omicron sub-variants/sub-lineages have emerged; these include BA.2.11 (France), BA.2.12.1 (USA), and BA.4/5 (South Africa), which are presently replacing BA.2 in several countries.[12] Globally, there are more incidences of BA.4 and BA.5, according to GISAID. In South Africa, by the end of April 2022, the sequence percentages for BA.4 and BA.5 had risen to 35% and 20%, respectively. The ECDC reports that as of May 8, 2022, 37% of positive cases in Portugal were linked to BA.5. The Omicron lineages BA.4 and BA.5 may be more transmissible than other Omicron lineages based on their growth rates. Most of these cases are reported from South Africa; however, BA.4 has also been reported from Austria, the UK, the USA, and Denmark; BA.5 has been reported from Germany, Portugal, the UK, and the USA. It is anticipated that these mutations will be more transmissible, which could lead to a significant increase in COVID-19 cases overall in the future. The ECDC has reclassified these two sub-lineages (BA.4 and BA.5) as variants of concern (VOC) after they were previously categorized as variants of interest (VOI).[13]

Beyond this, three further SARS-CoV-2 recombinant/hybrid forms (XD, XE, and XF) have been identified; of these, XE (BA.1/BA.2 recombinant form) has posed the largest global health risk during the ongoing COVID-19 pandemic. The genome sequence has been transmitted to GISAID, and the XD variant (AY.4/BA.1) has also been recognized and confirmed by the Institute Pasteur in France. [14] Considered a recombinant lineage, the XD lineage was formed by recombining the VOCs Delta (AY.4) and Omicron BA.1. This recombinant variation (XD) was confirmed together with two more SARS-CoV-2 recombinant variations, XE and XF. The two Omicron sister variations—the recombinant

genomic elements Omicron BA.1 and Omicron BA.2—were the source of the recombinant XE variant. The recombinant genomic components of two variants—the Delta variant and the Omicron BA.1 variation—were combined to create the recombinant XF variant.[15]

This review article on SARS-CoV-2 aims to compile and synthesize the extensive and rapidly evolving body of research on the virus, offering a comprehensive overview of its characteristics, transmission dynamics, clinical impacts, and treatment strategies. Such a review serves to critically assess the current state of knowledge, identify key findings and research gaps, and integrate diverse studies to provide a cohesive understanding of the virus. By organizing complex information into a clear and accessible format, the review aids researchers, clinicians, and policymakers in grasping the nuances of SARS-CoV-2, informs evidence-based decision-making, and highlights future research directions and potential areas for intervention. Ultimately, the review contributes to a more informed and coordinated response to the ongoing pandemic and helps advance the collective understanding of SARS-CoV-2 and its implications.

1.1 Pathogenicity of Omicron

Human respiratory system cells, including vascular endothelial cells, alveolar epithelial cells, and airway and alveolar epithelium cells, are all susceptible to infection by SARS-CoV-2 when it binds to the ACE2 receptor. As we have just seen, the spike protein facilitates this binding. Research has also revealed that cells with a large number of ACE2 receptors exhibit elevated Omicron infection. In [16] Omicron, on the other hand, has been observed to take an alternative path. Instead of entering through the plasma membrane as other versions do, omicron penetrates through the endosomal route. It is cathepsins, not TMPRSS2, that boost this route. An investigation revealed that the cells with reduced TMPRSS2 expression had a more substantial Omicron infection. This demonstrated even further that Omicron entered the cell via the endosomal route. [17] In addition to having a less effective splitting efficiency compared to previously emerged variants like the Gamma and Alpha variant—mutations like the N679K and H655Y are the main cause of the same—Omicron has been shown to exhibit a higher rate of success for the virus's replication in the upper respiratory tract.[18]

The variant's increased cellular permeability has been connected to further mutations in the N and S proteins. Additionally, these alterations contribute to three times more efficient capsid assembly as compared to the Delta form.[19] Because of the several alterations in the S protein, the sub-variants of Omicron (BA.1 and BA.2) do not create syncytia, which are typically produced at the boundary of S1 and S2 during the processing of the S protein. These modifications give rise to distinct cellular tropisms in conjunction with the modified entry channel.[20]

The high transmissibility and growth rate of Omicron may potentially be linked to the viral load. An increased viral load was noted in the upper respiratory tract, which includes the throat, trachea, and nose. larger growth and a larger viral load may be the cause of this agglomeration of viral particles in the upper respiratory tract.[21]

Moreover, omicron can lead to reinfection. Re-infection rates rose dramatically during the Omicron wave compared to the Beta and Delta waves, according to data from South Africa. [22] According to reports, the Delta variety of Omicron carries a six-fold increased risk of reinfection in the UK. Eighty percent protection against the Delta variant and just nineteen

percent protection against the Omicron variant was seen after a previous COVID-19 infection. The good news is that hospitalization was not necessary in as many Omicron infections; nevertheless, Alpha and Delta infections were more likely to result in severe cases.[23]

1.2 Structure of Omicron

Its structure distinguishes Omicron from all other varieties that are now available. Researchers have been concerned by some of the 60 mutations in the variant compared to the original Wuhan strain (8 synonymous, 2 non-coding, and 50 non-synonymous). The 24th There are a tonne of unique mutations in this variety, which is incredibly numerous. Omicron has at least 50 mutations affecting amino acids and roughly ten modifications that do not affect amino acids; some of these mutations may be in regulatory areas.[25] A significant fraction of these impacted the spike protein that most COVID-19 vaccines targeted at the time of the Omicron variant's discovery. The spike protein contains 32 mutations and is the main target of antibodies produced by infections and several widely used vaccines.[26] A large number of the changes were not present in any other strain. The variation of the virus contains thirty amino acid alterations, three tiny deletions, one short insertion, and fifteen amino acid modifications in the receptor-binding domain (residues 319–541) compared to the original virus. It also has many deletions and modifications in other genomic regions. In this version, the Furin cleavage site likewise has three mutations.[27] The following are the key amino acid substitutions found in spike protein: A67V, del69–70, T95I, del142–144, Y145D, del211, L212I, ins214EPE, G339D, S371L, S373P, S375F, K417N, N440K, G446S, S477N, T478K, E484A, Q493R, G496S, Q498R, N501Y, Y505H, T547K, D614G, H655Y, N679K, P681H, N764K, D796Y, N856K, Q954H, N969K, and L981F. (28] The researchers have identified five Omicron sub-lineages. The latest sub-lineages are termed BA.4 and BA.5, and the standard sub-lineage is now known as BA.1 /B.1.1.529.1. The other two are known as BA.2 /B.1.1.529.2 and BA.3 /B.1.1.529.3. Computational modeling suggests that the variation might not be susceptible to cell-mediated immunity.[29] Omicron is derived from the first main variant of SARS-CoV-2, which we call The Triad and others call D614G, except a few East African strains (A.30 and A.23.1). Omicron has been shown to contain numerous novel mutations in both the S and non-S proteins, in addition to many of the most worrisome alterations previously documented in variations of concern. [30] Omicron has several additional proteins, like NSP12 and NSP14, that are essential for viral replication in addition to a significant number of previously identified alterations in various VOCs.[31] It is believed that changes in the Spike proteins have enhanced Spike's capacity to attach to the host cell's ACE2 receptor. A change in amino acid composition is not observed in Omicron due to ten synonymous mutations. All ten of Omicron's synonymous changes are distinct, except C241U. Yet, these alterations may interfere with what is known as cis-acting regulatory sequences by altering the structures and critical recognition sequences in viral RNA that are necessary for transcription, translation, and replication. One study result indicates that the changed spike protein of the Omicron variety will still bind to current neutralizing antibodies. Omicron's RBD, however, seems to have a lower propensity for neutralizing antibodies when compared to the reference RBD structures. According to this discovery, immunity against Omicron may be partially provided by antibodies generated after a previous infection or vaccination. 33] Immunological regulators make up the majority of accessory proteins. Oddly, Omicron has no mutations in this domain, even though proteins like Orf8 and Orf6 are typically significantly changed, which may point to Omicron's selection pressures.[33] Comparing the Omicron variation to previous variants, the sequence of nucleotide modifications was as follows: SARS-CoV-2 USA isolate > Mut variant > Beta variant > Delta variant > Gamma variant >

Alpha variant > Omicron variant, with 141, 140, 138, 132, 130, and 109 mutations, respectively. Comparing the many versions, the Alpha variant has the highest identity percentage (99.63%) with the Omicron version, followed by the Gamma and Mu variants (99.56%). [34]

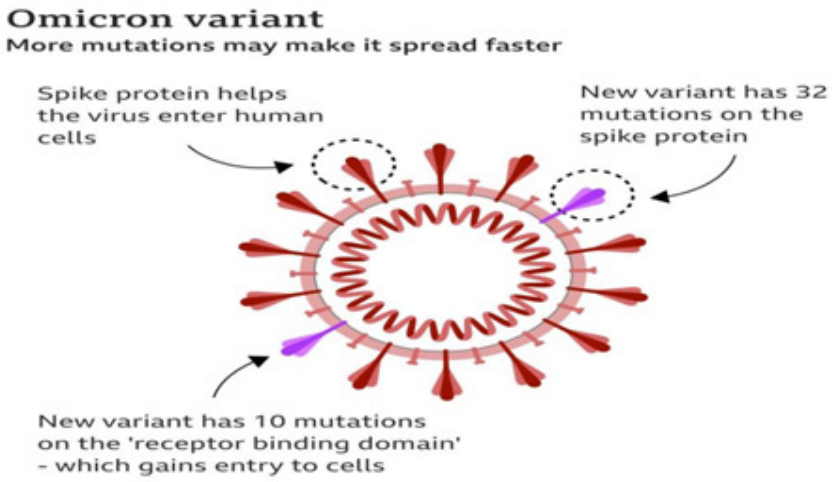

Fig 1 . Diagrammatic representation of Omicron Variant

1.3 Transmission of Omicron

The two main ways that SARS-CoV-2 spreads are by droplet infection and direct viral transmission. Omicron infects the host cells in a manner akin to the other variations. Its rate of infectivity is higher, though. The number 35 Many studies have documented its strong transmissibility; for example, one from South Africa recorded doubling times of 3.38 days, while another from the UK showed doubling times of 2-3.5 days, with a basic reproduction number greater than 3. When it comes to the Delta type, Omicron is said to have a 3.2-fold higher transmission rate, with an average doubling time of 3 days. It's been suggested that Omicron's high contagiousness is partly caused by mutations in the spike protein.[36]

The aforementioned claim has been supported by a variety of evidence presented by various studies. A Chinese study conducted in vitro and employed an artificial intelligence model to demonstrate that the mutations at positions N440K, N501Y, and T478K directly led to elevated infectivity. Specifically, there was a ten-fold rise in Omicron for the original SARS-CoV-2 variation and a two-fold increase for the Delta variant. A different study discovered that the N501Y and Q498R mutations together greatly increased the binding affinity of the S protein to the ACE2 receptors. There is also evidence linking further variants including N679K, D614G, and P681H to increased infectivity [37]

2. Clinical manifestations of Omicron

Compared to the previous iterations, the Omicron infection presented distinct clinical signs. Cough, nasal congestion, sore throat, and exhaustion were the most common symptoms. The Delta variety was less likely to have symptoms like fever, headaches, disorientation, and loss of taste or smell. The virus also affects the organs that express ACE2 in large amounts, such as the gastric, duodenal, and rectal epithelial cells. 38] According to a study, the average

duration of acute symptoms during the Omicron outbreak was 6.87 days, and during the Delta wave, it was 8.89 days. [39]. According to others, omicron infections also have less severe symptoms; in fact, a large number of cases are asymptomatic and don't require hospitalization. Of the patients infected with the Omicron virus, just 1.9% needed to be hospitalized, compared to 2.6% of individuals infected with the Delta variety. 40] 36.1% of Omicron-infected people did not show any antibody response, whereas 62.7% produced IgG and just 1.2% developed IgM in addition to IgG, according to a study. Haematological problems like lymphocytopenia, neutrophilia, anemia, erythrocytopenia, thrombocytopenia, etc. were seen in patients infected with omicron. When Omicron infections occur, these can be utilized as indicators to improve prognosis [41].

3. Diagnosis and Management

The molecular examination and the immunological evaluation are the two components of the diagnostic laboratory of Omicron. NGS and real-time PCR are the two primary subfields of molecular diagnosis. Checking for antigens and antibodies is a necessary step in immunological diagnostics. The identification of viral infection is possible through the use of molecular tests to amplify viral RNA.42] These tests (NAATs) are also referred to as "nucleic acid amplification testing."[43] Obtaining a sample from the mouth or nose of a person who may be sick is the first step in determining whether or not the virus is there [44. Assuming SARS-CoV-2 be detected in the specimen, a molecular diagnostic method can detect millions of copies of viral genetic material. Nasopharyngeal surfaces are most likely to harbor the virus, hence secretions from these surfaces must be gathered to do molecular testing. To facilitate testing, the majority of COVID-19 assays that are now available or being developed employ oral or nasopharyngeal materials.[45] Viral RNA will be found in the case of an infection. Existing molecular diagnostics yield results more quickly than those obtained using conventional PCR-based methods. Because they enhance the genetic material in the patient sample, rapid molecular diagnostics like LAMP are very sensitive but selective. The essential process used by all of these methods is the same: identify pathogens that are DNA or RNA based on their identification, then amplify a chosen area of their genome. Next, give a measurement of the amount of amplified genetic material in the sample, if any. In [46] A minor deletion mutation, identified as deletions 69–70 in BA.1, modifies the spike protein's structure and makes it incapable of being detected by the PCR assay. This makes Omicron's test result questionable.[47] In the BA.2 sub-variant, this omission mutation does not occur, therefore although though the test is Omicron, the results will be incorrect when used with other strains, particularly the Delta variation in regular PCRs. Nevertheless, other specialist tests can identify the strain. In the United States, type BA.2 is referred to as the Stealth Omicron due to its rapid proliferation and mutations that make it challenging to distinguish this subtype from the Delta type using standard PCR testing [48].

In a short amount of time, NGS sequencing methods have become the method of choice for many virological applications, including the identification of novel viruses.[49] This technique is essential for figuring out where SARS-CoV-2 originated. Scholars may already use the majority of coronavirus and SARS-CoV-2 genomes discovered using next-generation sequencing (NGS) to research the origins of SARS-CoV-2. Both second and third-generation NGS technologies as well as a range of proprietary library preparation methods developed by different firms have proven beneficial for SARS-CoV-2. One of the most important benefits of NGS is the ability to reconstruct whole-length viral genomes using NGS-based methods, even for viruses that have not been previously identified. Moreover, Omicron subvariants and

likely new SARS-CoV-2 mutations can be found using NGS. Sequencing of additional specimens is therefore required [50].

Diagnostic tests that detect antigens in saliva or upper respiratory samples can detect viral proteins and screen for SARS-CoV-2 infection. It may be more practicable to screen for SARS-CoV-2 using antigen-detecting rapid diagnostic tests (Ag-RDTs) as opposed to NAATs. Ag-RDTs are most effective in patients with a high viral load early in the course of their disease, and they would be most accurate in regions with a prevalence of less than 5% of SARS-CoV-2. When there is little transmission, Ag-RDTs have a low positive predictive value. Therefore, in these circumstances, NAATs are advised for initial testing or verifying Ag-RDT positive results. Ag-RDTs are less accurate than NAAT, particularly in those who don't exhibit any symptoms. Nevertheless, this can be lessened by choosing test cohorts wisely. Ag-RDTs with 80% sensitivity and 97% specificity minimal performance requirements are advised by the WHO. [51] The aforementioned tactics may enhance prompt patient diagnosis and, consequently, successful disruption of the transmission cycle.

Since spike glycoprotein attachment to ACE2 receptors is the primary pathway by which SARS-CoV-2 enters host cells, any alteration in the virus's genome can lessen the efficacy of therapies intended to block viral attachment. Consequently, it has been anticipated that this variation will be resistant to modern treatments, such as monoclonal antibodies (mAb) [52]. Stated differently, a recent study found that seven of the nine monoclonal antibodies tested (Bamlanvimab, Etesevimab, Casirivimab (REGN10933), Imdevimab (REGN10987), sotrovimab (S309), DZIF-10c, P2B-2F6, C102, and Fab2-36) were unable to exhibit effective neutralizing activity against the Omicron variant. Nonetheless, the Wu01 strain and the Alpha variant were successfully neutralized by them. Fascinatingly, 7 out of 9 and 5 out of 9 of the monoclonal antibodies demonstrated enough neutralizing activity against the Beta and Delta variations, respectively, indicating that the variants were somewhat resistant to these antibodies [53]. It is anticipated that numerous medicines will yield therapeutic benefits for the Omicron variety. TriSb92, for instance, is a trimeric human nephrocystin SH3 domain-derived antibody that, when given intraperitoneally, is thought to block the novel variation. [54]

4. Treatment

Treatment options for SARS-CoV-2 infection now include supportive care, symptomatic therapy, and repurposed medications. These therapies' primary objective is to prevent the virus from entering cells. Human recombinant soluble ACE2 (hrsACE2) imported extrinsically can kill the virus in serum, and small-molecule medicines or antibodies that have a higher binding affinity for the virus's RBD can prevent the virus from attaching itself to cells.[55]. The RBD is not well conserved among the coronaviruses, though. SARS-CoV-2's S2 protein subunit may be a therapeutic target since it has been shown that peptides targeting other human coronaviruses have fusion inhibitory activity against membrane fusion domains. This suggests that targeting membrane fusion, one of the most conserved regions of the S protein, may one day be used to treat broad-spectrum pan-CoV disease. [56] Single-stranded DNA or RNA molecules called aptamers can bind to particular targets by assuming a special three-dimensional structure. The creation of aptamers that specifically target the S protein of SARS-CoV-2 is now underway. Research has shown that MSA52 is a universal aptamer that can bind to the trimeric S proteins present in a variety of potentially dangerous SARS-CoV-2 subtypes.

[57] This broad-spectrum binding ability is crucial for creating antiviral medications that effectively combat the virus. Since endosome acidification is necessary for SARS-CoV-2 entrance, reducing endosome acidity may hinder viral entry routes. It was discovered that the antiviral peptide 8P9R may decrease endosomal acidification and cross-link viruses at the same time to prevent viral entry. As an alternate strategy, blocking viral entrance into cells may target TMPRSS2 and lysosomal cathepsins. SARS-CoV-2 is susceptible to inhibition by conventional antivirals, such as remdesivir, which block the production of viral RNA in vitro [58]. Remdesivir cannot be widely used during the pandemic, though, because it has be injected intravenously. With acceptable safety, tolerability, and pharmacokinetic characteristics in healthy individuals, the oral remdesivir analog VV116 has demonstrated enhanced in vitro antiviral activity and selectivity. In [59] In patients infected with the SARSCoV-2 omicron form, who received VV116 within 5 days of the first positive test, a real-world investigation showed that treatment significantly increased viral shedding.[60] In contrast to paxlovid, a novel oral medication that combines the pharmaceutical enhancer ritonavir with the second-generation protease inhibitor nirmatrelvir, which has been recommended for use in WHO guidelines, VV116 demonstrated a comparable time to sustained clinical recovery with fewer safety concerns in symptomatic adults with mild to moderate COVID-19 at risk of progression.(61) Molnupiravir, an oral nucleoside analog that targets the RdRp, is an isopropyl ester prodrug of β-D-N4-hydroxycytidine with broad-spectrum antiviral action and resistance to drug resistance. Molnupiravir works against viruses by causing lethal mutagenesis, which raises the frequencies of G→A and C→U transitions during virus replication. Phase I, II, and III clinical trials have demonstrated encouraging efficacy and safety; nevertheless, additional research is required to assess its potential carcinogenic hazards and genotoxicity.[62] Insufficient data exists to substantiate the application of corticosteroids in the management of COVID-19. If glucocorticoids are administered, antiviral medications must be used in addition to them to prevent compromise of the host defense mechanisms required to fend against SARSCoV-2 infection. Anakinra and tocilizumab, two cytokine receptor antagonists, have been demonstrated to lessen the inflammatory response, whereas therapeutic plasma exchange (TPE) quickly and non-selectively eliminates excess cytokines from plasma, changing lymphocyte function and proliferation status while boosting immunity against SARS-CoV-2. The International Society of Thrombosis and Haemostasis (ISTH) has recommended that all hospitalized COVID-19 patients who do not have any contraindications take anticoagulant medication, namely low molecular weight heparin (LMWH).[63] Greater therapeutic dosages of LMWH would be necessary since certain patients receiving preventive LMWH still have a chance of developing thrombotic problems because of possible heparin resistance brought on by elevated NET levels.[64]

5. Global Impact of the SARS-CoV-2 (B.1.1.529) Variant

More than 650 million confirmed cases and more than 6.6 million deaths worldwide are the results of the highly contagious infectious disease known as COVID-19, which is caused by the severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2). The disease has had a severe negative impact on humanity's health and socioeconomic well-being. Owing to this worry, on March 11, 2020, the World Health Organization (WHO) declared COVID-19 to be a global pandemic, which is still in effect today.[65]

Based on advice from the Technical Advisory Group on the Virus Evolution of the WHO, the WHO introduced a variation of concern B.1.1.529 on November 26, 2020. This novel strain was linked to multiple mutations, including 26–32 in the S protein, and was thought to be substantially distinct from previous strains. A possible transmission rate was associated with some alterations that raised concerns about their ability to evade the defense system. The number 66 According to the most recent data, which was released on December 16, 2021, the B.1.1.529 strain has been found in over 85 countries throughout all WHO regions.The 67th The four main components that directly contribute to the Omicron danger are the strain's ability to spread, the vaccine's ability to neutralize the strain, the strain's spectrum of infectivity, and public health and safety protocols about the strain.[68] In addition to these, robust and consistent evidence is also observed for Omicron's wide growth rate above delta strain. With a doubling period of 36–72 hours, Omicron's transmission rate is faster than delta strain's, increasing the likelihood that Omicron will surpass delta strain in community transmission.[69]
As of right now, not much is known about the clinical severity of Omicron, and a wide range of data is typically needed to comprehend the severity profile as well as the impact of vaccination and host immunity on severity.[70] A significant spike in instances causes hospital admissions in South Africa and the UK to grow daily. Most healthcare systems had the potential to soon become overburdened. It was observed that patients with COVID-19 infection or those who had previously received vaccinations had less ability to neutralize Omicron when compared to the initial data, indicating humoral defense system evasion..[71]

Proof of the vaccine's effectiveness against Omicron, as evaluated by peer review, was lacking. South Africa and the UK provided historical vaccination efficacy statistics. Because the blueprint, subject selection bias, and results were all based on tiny numbers, this information needed to be carefully evaluated. AstraZeneca-Vaxzevria or Pfizer BioNTech-Comirnaty vaccines were given in two doses, but the vaccination response from England demonstrated a discernible decline in vaccine effectiveness against Omicron when compared with the fourth VoC. When a booster dosage of Pfizer BioNTech-Comirnaty was administered after two weeks, better efficacy was seen.[72]

Omicron risk assessment needs to draw attention to relevant issues with persuasive justifications. The first is due to the significant danger of SARS-CoV-2 globally, and the second is because of data that shows how quickly the Omicron is spreading across the community, increasing the number of patients who may need to be admitted to the hospital. WHO is continuously refining its understanding of risk assessment and will do so as new data become available.[73]

In a recent update, the World Health Organization requested that all linked nations conduct daily reviews and revisions to their national policies for managing Omicron to reduce its spread. To reduce the transmission of COVID-19 among people, the World Health Organization has released preventive measures globally and mandated the use of proper masks, social distancing, avoiding crowds, and practicing good hand cleanliness.[74] Along with a prompt diagnosis, public protection measures will be the key to preventing transmission. The most important thing to do to lower the hospitalization and death rates should be to vaccinate the entire population. Furthermore, in certain countries, a booster dosage of the corresponding vaccination has also been initiated, which has a fantastic effect on patient safety, a specific population at high risk of death, important infection, and reinfection. The International Health Regulation (IHR) system encouraged all countries to

report the first cases associated with the fifth VoC to the WHO. Every official body should link data and evidence on Omicron and other mild strains with suitable public guidelines regularly and in the cleanest possible way, considering all known and unknown elements as well as the work being done by specialized authorities [75].

6. Conclusion

This review concludes with insights into the SARS-CoV-2 B.1.1.529 variant, also known as the Omicron variant, highlighting its distinct characteristics compared to previous variants. Omicron has a higher mutation rate, particularly in the spike protein, leading to increased transmissibility and partial immune escape. This has resulted in widespread infections, even among vaccinated individuals. Despite generally causing less severe disease, the sheer volume of cases has significantly strained healthcare systems worldwide. The global response to Omicron underscores the importance of genomic surveillance, rapid public health measures, and ongoing vaccination efforts to mitigate its spread and impact. The variant's ability to evade immunity highlights the need for continuous adaptation of vaccines and treatments to maintain their effectiveness. Overall, the Omicron variant has reinforced the critical need for global cooperation and preparedness in managing and responding to emerging infectious diseases.

List of abbreviations

ACE2 angiotensin-converting enzyme 2

COVID-19 coronavirus disease 2019

GISAID Global Initiative on Sharing All Influenza Data

ECDC European Centre for Disease Prevention and Control

CRS cytokine release syndrome

IgG Immunoglobulin G

IgM Immunoglobulin M

LAMP Loop-mediated isothermal amplification

Ag-RDTs antigen-detecting rapid diagnostic tests

NAAT nucleic acid amplification testing

NGS next-generation sequencing

RBD receptor binding domain

RDT rapid diagnostic test

RNA ribonucleic acid

RT-PCR reverse transcriptase-polymerase chain reaction

SARS-CoV-2 severe acute respiratory syndrome coronavirus 2

VOC variants of concern

VOI variants of interest

TMPRSS Transmembrane protease, serine

NSP Non-structural protein

WHO World Health Organization

SGTF S gene target failure

TriSb92 Triplex assay S gene target detection

MSA Multiple sequence alignment

RdRp RNA-dependent RNA polymerase

LMWH Low molecular weight heparin

NETs Neutrophil extracellular traps

Conflict of Interest

The author declares no conflict of interest.

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