IOSR Journal of Nursing and Health Science (IOSR-JNHS)
e-ISSN: 2320–1959.p- ISSN: 2320–1940 Volume 11, Issue 3 Ser. II (May. – June. 2022), PP 29-37
www.iosrjournals.org

Scientific Approach About Covid-19

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Abstract
Introduction*: COVID-19 is an infectious disease caused by the new coronavirus, responsible for the severe acute respiratory syndrome (SARS), which only in 2020, with the outbreak of SARS, actually emerged the alert of potential health risk, with, currently, the alpha, beta, gamma, delta, omicron, mu and lambda variants, each with their particularities.*
Objective*: To understand and highlight information about COVID-19, as well as its epidemiology, risk factors for transmission, its pathophysiology, diagnosis, and treatments, in addition to understanding its etiological agent, SARS-CoV-2.*
Methods*: A qualitative exploratory study was carried out to identify all the topics already mentioned, using about 37 articles with an approach to this subject.*
Results*: COVID-19 worsened worldwide during the years 2020 and 2021, and in the present year 2022, there was an improvement in the number of cases, however, the disease is not over yet. Conclusions: By having a comprehensive view on the subject, this study can contribute to the expansion of information, mainly due to the current moment in which society is, in addition to a reflection on the main necessary care for the disease.*
Key words: *Pandemic; Covid-19; SARS-CoV-2; Information about Covid-19; Transmission of the SARS-CoV-2 virus.*

Date of Submission: 08-05-2022 Date of Acceptance: 23-05-2022

I. Introduction

COVID-19 is an infectious and complex disease caused by the new coronavirus, responsible for the severe acute respiratory syndrome (SARS), in addition to other systemic symptoms, being variable according to the immune response of each patient [12].

The first record of the virus was carried out in the 1930s in animals and several species were detected, such as the Infectious Bronchiolitis Virus in birds and the Epidemic Diarrhea Virus in swine. In humans, from 1960 onwards, cases were detected and associated with a mild flu, and, only in 2020, with the outbreak of SARS, did the alert of potential health risk emerged [12].

Since the beginning of the SARS-CoV-2 pandemic, decreed by the World Health Organization (WHO), several challenges have developed in the management of infected patients. Underdeveloped countries were the ones that presented greater difficulties [24], both with diagnostic methods, about the lack of material, type and accuracy of exams [20], and with therapeutic methods, concerning the lack of persuasion in the face of the effectiveness of drugs and vaccines [9]. This whole scenario of uncertainties caused a great commotion around the world and apprehension in society.

Given this situation, it is necessary to review the literature on this topic, to characterize the disease in a broad way, which is the objective of this article.

II. Etiological Agent And Etiology

SARS-CoV-2, novel coronavirus, is a single-stranded RNA enveloped virus, measuring from 50 to 200nm [30], it belongs to the order Nidovirales, the family Coronaviridae, the genus Betacoronavirus, and the subgenus Sarbecovirus [25].

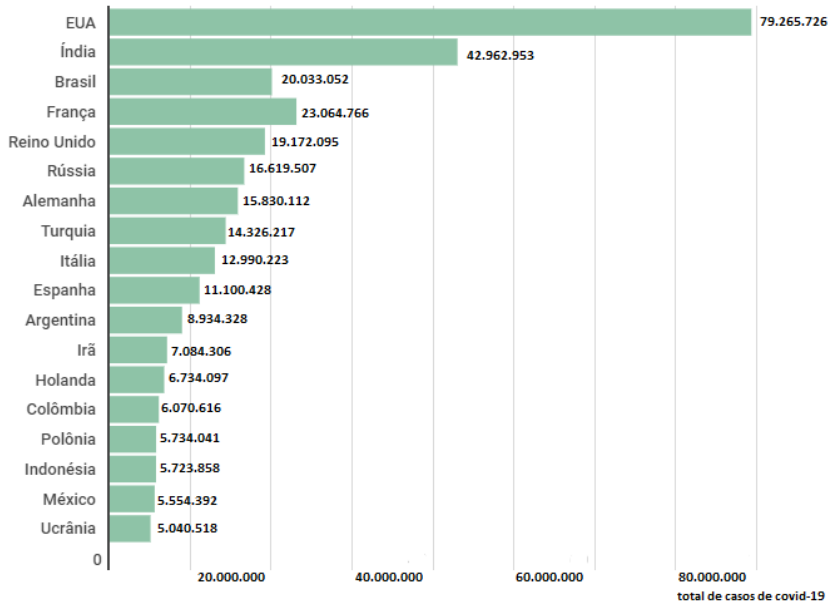
This virus has properties that contribute to its virulence, namely: the S protein – with which SARS-CoV-2 binds to the host cell; N-protein of the nucleocapsid; HE protein – hemagglutinin esterase, responsible for viral binding; and the M protein – responsible for maintaining the pathogen's envelope shape. Furthermore, it can encode certain proteins, such as chymotrypsin-type protease 3, papain-type protease, helicase, and RNA-dependent RNA polymerase [24].

It is not yet known for sure which are the hosts of SARS-CoV-2, being the human being one of them, however, many studies point to the bat as the preponderant natural reservoir [26], being the main reason for the association of the origin of the virus with the animal the phylogenetic similarity with BatCoVRaTG13, bat coronavirus. Pangolins, on the other hand, are still uncertainly placed as possible intermediate hosts for humans, also due to their genomic similarity [17].

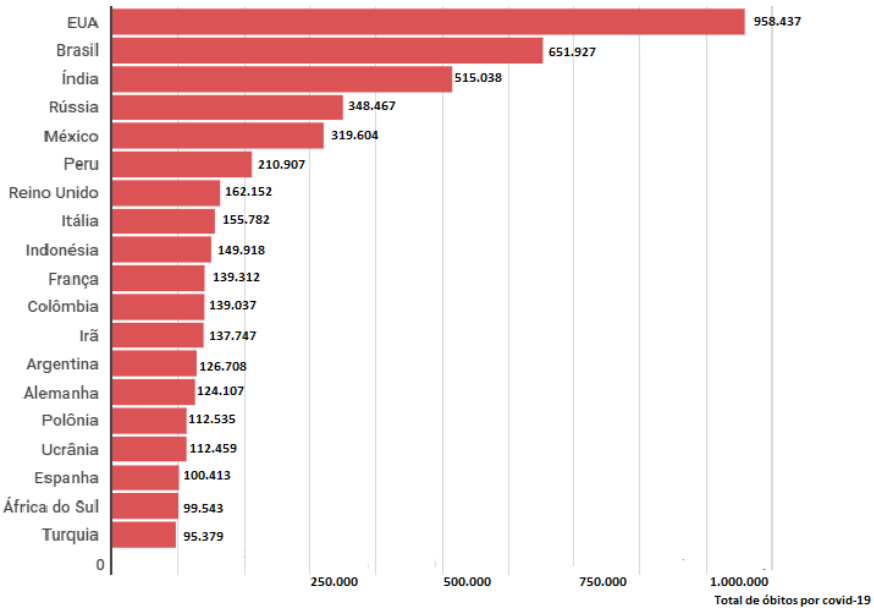
It is noteworthy that, in a study carried out, it was seen that the stability of the SARS-CoV-2 virus in aerosols and surfaces can result in greater transmissibility since the virus was found viable for hours in aerosols (3 hours, with reduction of the infectious titer from 103.5 to 102.7 TCID50 per liter of air) and for days on surfaces made of stainless steel and plastic, where the viable virus was detected up to 72 hours after application to these surfaces, although the titer of the virus was greatly reduced (from 103.7 to 100.6 TCID50 per milliliter of the medium after 72 hours on plastic and from 103.7 to 100.6 TCID50 per milliliter after 48 hours on stainless steel) [35].

III. Epidemiology

According to epidemiological data at the time, on January 5, 2020, the virus would have infected 59 people in the city and 7 were in critical condition [33]. During the month of January, Chinese authorities had reported more than 200 infections and 3 deaths. As of February 20, 2020, a total of 75,995 confirmed cases, including 2,239 deaths in China, and 1,200 confirmed cases, including 8 fatalities outside the country. As of March 29, 2020, more than 700,000 cases have been confirmed worldwide. These reports indicated the rapidity of contamination of the new disease caused by the SAR-COV-2 virus infection, characterizing a pandemic on March 11, 2020 by the World Health Organization (WHO) [23]. According to the epidemiological bulletin [5], the world situation until the end of the epidemiological week (ES) 9 of 2022, on March 5, 2022, 445,129,499 cases of covid-19 were confirmed in the world. The following graphs illustrate how the total distribution of Covid-19 cases and deaths among the 20 countries with the highest number of cases [28].



Graph 1 – Number of Covid-19 cases in countries around the world. Source: Our World in Data - https://ourworldindata.org/coronavírus updated 05/03/2022



Graph 2 – Number of deaths from Covid-19 in countries around the world. Source: Our World in Data - https://ourworldindata.org/coronavírus - updated 05/03/2022

There are a total of five variants of covid-19, the alpha, beta, gamma, delta, omicron, mu and lambda variants. The alpha variant, also known as B. 1 .1 .7, was first reported in September 2020 in the United Kingdom (WHO), is highly transmissible and has already been detected in around 80 countries. The beta variant was first detected in South Africa, it has an E484K mutation, related to an immune escape, and the N501Y mutation, which can influence other variants to be more contagious. The gamma variant, also known as P. 1, was seen for the first time in Brazil, has mutations E484K and N501Y, in addition to more than 30 other mutations (WHO). The Delta variant (B. 1 .617.2), the one with the highest transmission rate among the variants, according to the CDC each infected person infects eight or nine people on average, has existed since the end of 2020, has quickly become dominant in many countries, with the highest prevalence in the United States, according to the Centers for Disease Control and Prevention (CDC). The Omicron variant has been included by the WHO, as a strain of concern, cases have been identified in Botswana, South Africa, Hong Kong, Israel, Belgium, the United Kingdom, Germany, Italy and the Netherlands. The Mu variant identified in Colombia, its global prevalence has decreased considerably with a rate below 0.1 0/0, however, its prevalence in Colombia has increased to 39% and in Ecuador to 13%. Finally, the lambda variant identified in Peru, in December 2020 and in June 2021, was identified as a variant of importance, but of less concern among the other variants [1].

IV. Transmission

Transmission of the SARS-CoV- 2 viruses can be categorized as exposure to respiratory fluids that have infectious viruses. Currently, the modes of transmission are signaled in three ways, namely: inhalation of respiratory droplets and aerosol particles, touching mucous membranes with hands infected by respiratory fluids containing the virus, or indirectly touching surfaces (fomites) that have been contaminated. by SARS-Cov- 2 and by the deposition of droplets and infected respiratory particles on exposed mucous membranes in the mouth, nose or eyes [8]. It is important to emphasize that there is relative importance in identifying and recognizing transmission factors since the implementation of public health strategies aimed at containing the advance of the current pandemic and also of future outbreaks of beta-coronavirus depend on the three situations already mentioned that favor the transmission of the virus [37].

According to one of the studies analyzed, the control of the SARS-CoV- 2 pandemic requires targeted interventions, which in turn, require accurate estimates of quantities that describe transmission, and this factor is influenced by four conditions: (1) the latent period of infection; (2) individual variability in infectivity ; (3) the incubation period; and (4) the serial interval [13]. The exact understanding of these four quantities can help control an outbreak of the virus, however, it is substantial to understand that they can vary depending on disease mitigation interventions, as well as the population structure and inherent properties of the SARS-CoV- 2 variant.

V. Pathophysiology

COVID-19 is a complex and multisystem disease whose symptoms and prognosis depend on the stage in which the patient is. The phases of the disease can be summarized as phase 1 or viral replication, phase 2 or inflammatory, which is divided into 2A, if the patient does not have hypoxia and, 2B, if it evolves to such, and phase 3 or systemic hyperinflammation. [3]. It is important to emphasize that the main clinical characteristic of covid 19 is respiratory symptoms, although there are also neurological, renal, digestive, cardiac and other organ complications, being related to the presence of angiotensin-converting enzyme 2 (ACE2) receptors [29].

The epithelial cells of the host agents have a high expression of ACE2 in the kidneys, intestines, blood vessels and lungs. Considering that viruses of the coronavirus family have a protein, called Spike (S) protein, responsible for communicating with these cells, when there is a connection, the virus replicates polyproteins using ribosomes from the host cells, initiating the infection process. From the moment that SARS-CoV2 enters the body through the respiratory tract through the apical cilia, it binds to the ACE2 receptor present in the lungs and replication occurs in the mucosal epithelium of the respiratory tract and in the alveolar epithelial cells through microvilli, resulting in damage to the bronchial mucosal epithelium and alveolar epithelial cells [6]. Given that ACE2, the entry receptor for the SARS-CoV-2-causing corona virus, is expressed in various extrapulmonary tissues, direct damage to viral tissue is a plausible mechanism of injury. In addition, endothelial damage and thromboinflammation, dysregulation of immune responses, and maladaptation of ACE2-related pathways may contribute to these extrapulmonary manifestations of COVID-19.

Endothelial injury mediated by infection and endothelitis, found in multiple vascular beds, including the lungs, kidney, heart, small intestine, and liver, in COVID-19 patients can trigger excessive thrombin production, inhibit fibrinolysis, and activate pathways. complement, initiating thromboinflammation and ultimately leading to microthrombus deposition and microvascular dysfunction. In this scenario, the cross-communication between platelets and neutrophils and the activation of macrophages can facilitate a variety of pro-inflammatory effects, such as the release of cytokines, the formation of extracellular neutrophil traps (NETs), and the formation of fibrin and/or microthrombi. . In addition, hypoxia-mediated hyperviscosity and upregulation of the hypoxia-inducible factor 1 (HIF-1) signaling pathway subsequent to acute lung injury may also contribute to the prothrombotic state [13].

Regarding cardiovascular tissue, ACE2 expression is high, including cardiac myocytes, fibroblasts, endothelial cells and smooth muscle cells, together with a possible mechanism of direct viral injury. Importantly, patients with pre-existing cardiovascular disease may have higher levels of ACE2, which increases the risk of a more severe condition, as well as isolated right ventricular dysfunction can occur as a result of elevated pulmonary vascular pressures secondary to the syndrome. of acute respiratory distress (ARDS), pulmonary thromboembolism, or potentially virus-mediated injury to vascular endothelial tissue and smooth muscle [13].

The involvement of the virus with the Nervous System was also proven, in which ACE2 receptors were detected in neurons and glial cells. More specifically, SARS-CoV-2 enters the central nervous system through the blood supply, olfactory bulb or lungs to the brain, via the vagus nerve, and infects various cell types through the passage of protein S to receptors. of ECA2 [10]. When this binding occurs, SARS-CoV-2 reduces the action of this enzyme and causes several consequences, such as the local increase in Angiotensin 2 that reacts with AT1 receptors to increase blood pressure, epithelial dysfunction of brain vessels that favors hemorrhage, and reduction in the production of Angiotensin 1-7 which plays a neuroprotective and vasodilator role. Added to this, oxygen deprivation in the tissues also occurs as a possible cause of these bleeding episodes, since hydrostatic and chemical changes can occur in the blood-brain barrier, allowing the leakage of erythrocytes [4]. In addition, the increased rate of D-dimer in some patients with Covid-19 promotes the formation of clots in the body as a whole. For this reason, clotting factors are more consumed and, subsequently, less available to certain areas of the CNS, which makes the blood vessels of this system more vulnerable to bleeding [10].

It is also worth noting the renal complications resulting from SARS-CoV-2 infection in people with COVID-19, such as deposition of immune complexes in renal cells, proteinuria, hematuria, increased blood urea nitrogen, increased serum creatinine and reduced GFR. . These inflammatory complications in kidney tissue can cause severe kidney damage such as AKI due to the cytotropic effect of the virus and the systemic inflammatory response induced by cytokines. In this way, inflammatory responses can trigger a cytokine storm, which induces hypoxia, shock and rhabdomyolysis and thus causes kidney damage [19].

Finally, it is important to remember that host immune responses against SARS-CoV2 may also contribute to the pathogenesis of Covid-19, through recognition of Toll-like receptors on cell membranes and within macrophages, neutrophils and dendritic cells. When the invasion occurs, the cells release type I interferons to recognize and contain the infection through the Complement System, which leads to the appearance of inflammatory signs such as swelling, heat, redness, pain and loss of function in tissues and organs. affected, in addition to fibrin deposition through the activation of several elements. With this, the captured invaders are processed and taken to the main histocompatibility complex (MHC), being made a

connection with the adaptive immune system, and thus, being able to identify, neutralize and destroy the invaders. However, if the patient's immune system is weakened or exposed to a large number of infectious particles, it may be difficult to contain the infection and the damage sustained by invading agents can result in temporary, permanent damage and even death [29].

VI. Prevention

Prevention of COVID-19 consists of imposing public health and infection control measures to prevent or decrease the transmission of the disease, such as the use of face masks, safe physical distancing and isolation. According to the new CONITEC ordinance, isolation must be carried out within 5 days for patients without respiratory symptoms or fever, and can be released with a negative test; 7 days for patients without symptoms without the need for a test to release the isolation, if mild symptoms are necessary a negative test and 10 days for patients without symptoms do not need a test [7].

In addition to these measures, the most crucial step to contain this global pandemic is vaccination to prevent SARS-CoV-2 infection in communities across the world. Currently the vaccines used are: Ad26.COV2.S, ChAdOx1 nCoV-19, NVX-CoV2373, BNT162b2, mRNA-1273. Up to seven other vaccines, including protein-based and inactivated vaccines, have been developed in India (Covaxin), Russia (Sputnik V) and China (CoronaVac) and have been approved or granted emergency use authorization to prevent COVID-19 in many countries. around the world [7].

In addition, in another study, the presence of viable virus was elucidated on the inside of woven and non-woven surgical masks. SARS-CoV-2 RNA was detected in 24/45 (53.3%) on the inside of masks (CTs <38). Viral RNA was detected only on the inside (the part that was in contact with the face) of the masks. None of the masks were positive for RNA on the outside (the part that was in contact with the outside environment). The mean viral load values of the samples on the masks were 2.51x103 (IQR, 0.0–2.51x103), which shows the effectiveness of blocking the virus, interrupting its transmission cycle, and its ability to remain on different surfaces and materials [21].

VII. Clinical Condition

According to the WHO, patients with COVID-19 have a wide range of signs and symptoms, which can appear from 2 to 14 days after exposure [34]. This range ranges from a mild, asymptomatic presentation to critical cases, including septic shock and respiratory failure.

The main symptoms that symptomatic patients present are: fever (>38.2 °C), dry cough, odynophagia, dyspnea, myalgia, fatigue, tiredness, severe headache, coryza, reduced smell and taste, diarrhea, nausea and vomiting [19]. However, not all patients will present such symptoms, they may or may not appear, to a lesser or greater intensity [15].

In general, we can classify the severity of the disease into mild, moderate, severe and critical asymptomatic. Asymptomatic will be those who did not show symptoms. Mild cases will present nonspecific symptoms, such as fever, cough, sore throat or coryza, followed or not by anosmia or ageusia, but without dyspnea, diarrhea, expectoration, normal oxygen saturation, an arterial pressure of oxygen (PaO2) by the fraction inspired oxygen (FiO2), also at normal levels and normal chest X-ray. Moderate cases will be those that will present from progressive signs of disease with symptoms of mild disease associated with signs of lower respiratory tract infection, O2 saturation > 94% in room air. In severe cases, patients will experience symptoms of moderate disease with O2 Saturation < 94%, PaO2/FiO2 < 300mmHg, RR > 30m, or pulmonary infiltrate > 50% and conscious. Finally, in critical cases, they will present acute unit syndrome, organ dysfunction, need for support devices and internal pneumonia in intensive care. Symptoms of severe illness and respiratory failure, septic shock and/or multiple organ dysfunction [3].

In addition, the fact that the patient has a pre-existing risk condition increases their probability of death. Among these conditions are: age over 60 years, Chronic Obstructive Pulmonary Disease (COPD), asthma, structural lung diseases, cerebrovascular disease, heart disease, insulin-dependent DM, renal failure, immunosuppressed, pregnant and obese patients [29]. It is worth remembering that such complications are related to ACE2 with its decrease and increase, being causes: neurological (increased vascular pressure, hemorrhages, oxygen deprivation, in addition to clots), renal (proteinuria, hematuria, increase in urea nitrogen in the blood, can cause hypoxia, shock and rhabdomyolysis), digestive, cardiac (high levels of ACE2 can cause right ventricular dysfunction, ARDS, pulmonary thromboembolism) [29].

Finally, it is worth mentioning that in addition to having symptoms in the face of the disease in its course, patients report some post-Covid sequels. Depending on the length of hospital stay, they demonstrate that coronavirus infections can bring many other problems such as dyspnea, fatigue, insomnia, myalgia, anosmia, arthralgia, cough, within 83 days of hospital discharge. Already when it comes 6-7 months after hospital discharge, one may experience fatigue, post-exercise malaise, cognitive dysfunction, sensory symptoms, headaches, memory problems, insomnia, heart palpitations and muscle pain, dyspnea , dizziness and balance

issues, speech and language issues, joint pain, tachycardia, and other sleep issues [36]. In addition, they may experience fatigue, anxiety, joint pain, ongoing headache, chest pain, dementia, depression, dyspnea, blurred vision, tinnitus, intermittent fever, obsessive-compulsive disorder, pulmonary fibrosis, diabetes mellitus, migraine, stroke , renal failure, myocarditis and arrhythmia, even in cases where there were no hospitalizations [16].

VIII. Diagnosis Emphasizing Differential Diagnosis

Patients with Covid-19 can present from non-specific symptoms to unique symptoms, such as loss of smell and taste. With these symptoms in mind, by mimicking that of a common flu, Covid-19 moved the whole world to improve and advance diagnostic technologies in the containment of infectious diseases, helping to identify cases early, containment and immediate treatment. [34].

To start the diagnosis of Covid, you must first carry out a quality anamnesis (containing the onset of the patient's symptoms, if there was contact with someone infected, their life habits, if they have comorbidities or any risk factor, medications in use) together with serological tests (IgM and IgG), PCR and imaging tests such as radiology, ultrasound and computerized [19; 22].

The nasal and oropharyngeal swab procedure has been approved by the World Health Organization (WHO) and works by detecting COVID-19 RNA. However, this procedure acts more as a screening test rather than a diagnostic test [34]. The current gold standard for detecting active COVID-19 is NAAT in respiratory samples, which may not be readily available for less resourceful areas or limited in supply in many places [18].

Imaging tests help to exclude other diseases that make a differential diagnosis with COVID-19, such as flu syndrome and SARS, caused by Influenza A viruses (such as H5N3 and H1N1) and Influenza B, community-acquired bacterial pneumonia (CAP), tuberculosis, whooping cough and acute exacerbations of COPD [36].

IX. Prognosis

Clinically, COVID-19 can be divided into mild, moderate, severe or critical, taking into account the signs and symptoms and whether the patient has a complete vaccination. The disease predisposes thrombotic and thromboembolic events, due to excessive inflammation, activation and injury of endothelial cells, platelet activation and hypercoagulability [11]. There is an increase in the levels of thrombosis biomarkers, which are associated with the severity and prognosis of the disease. Although some biomarkers of COVID-19-associated coagulopathy, including high levels of fibrinogen and D-dimer, were recognized early in the pandemic, many new biomarkers of thrombotic risk in COVID-19 have emerged.

In addition to the severity of the disease, high levels of coagulation markers are important determinants of the prognosis of the pathology. Early in the pandemic, patients who were hospitalized for the disease manifested disturbances in the levels of clotting biomarkers, including fibrinogen, D-dimer, and activated partial thromboplastin time (PTTA), and routine measurement of these biomarkers was recommended. After a certain time, other clotting markers emerged that helped to refine the understanding of the thrombotic signature of COVID-19 [27].

X. Pharmacological And Non-Pharmacological Treatment

Optimal control of COVID-19 should occur in conjunction with Non-Pharmaceutical Interventions (NPIs). The main NPIs are isolation, hand hygiene, early case detection (PCR testing) and face mask. These NPIs help in mitigating and reducing the size of the disease outbreak Patients with mild and moderate illness may not require emergency interventions or hospitalization, isolation is necessary for all suspected or confirmed cases to contain transmission of the virus. Monitoring a suspected case in a health facility, community or household should be taken on an individual basis. This decision will depend on the clinical presentation, the need for supportive care, potential risk factors for serious illness, and conditions at home, including the presence of vulnerable people in the household [2].

Currently, there is a variety in the drug treatment of SARS-CoV-2 infection, but the most commonly used drug classes include antiviral agents, inflammation inhibitors, low-molecular-weight heparins, plasma, and hyperimmune immunoglobulins. The choice must be based on the pathological characteristics and different clinical stages of COVID-19. Among Antiviral Therapies, the broad-spectrum antiviral chloroquine and its derivative hydroxychloroquine have received much attention. In February 2020, chloroquine was identified as a potential inhibitor of SARS-CoV-2 in vitro, in addition to drugs such as Molnupiravir, remdesivir and Paxlovid that act by curbing infection and viral replication. Although the inclusion of hydroxychloroquine as a pharmacological measure is still controversial, it has not shown efficacy in vivo [31].

Anti-inflammatory therapy: corticosteroids/immunosuppressants such as Dexamethasone and Preidnisone that aim to contain Hyperinflammation. And anti-coagulant therapies, which may be necessary since markers such as D-dimer and fibrinogen are increased leading to hypercoagulability, especially in the

advanced stage of COVID 19, so the goal should be to contain hyperinflammation and its consequences (for with biological drugs) and therapeutic doses of LMWH or unfractionated heparins, known for their anticoagulant properties [32; 7].

Some patients may still have associated respiratory failure and should be closely monitored with continuous pulse oximetry. Supplemental oxygen supplementation via nasal cannula or Venturi mask should be administered to maintain oxygen saturation (SpO2) between 92 to 96%. If clinical and oxygen saturation improve, oxygen supplementation should be continued with periodic reassessment. If there is no clinical improvement or if there is worsening of symptoms and/or oxygen saturation, non-invasive treatments such as High Flow Nasal Cannula (HFNC) or Non-Invasive Positive Pressure Ventilation (NIPPV) are recommended [7].

Along with drug treatment, convalescent plasma (CP) therapy is being advocated as a clinically effective practice. The procedure involves using blood plasma obtained from recovered patients as adaptive immune therapy for critical cases. The underlying principle in the work is that the donor plasma contains a high titer of antibodies that are effective against the pathogen, thus providing a boost of immunity to the recipient. PC has been advocated as an alternative therapy for SARS-CoV-2 infections [32].

XI. Psychosocial Aspects

Faced with the COVID-19 pandemic, concern for the mental health of the population is increasing. Due to the rapid advance of the disease and the excess of available information, sometimes being discordant, the scenario becomes favorable to the occurrence of behavioral changes that can cause psychological illness in healthy people and intensify the symptoms of those with pre-existing mental disorders. For this reason, patients diagnosed with the disease or suspected of having the infection can experience intense emotions and behavioral reactions, in addition to developing disorders such as panic attacks, Post Traumatic Stress Disorder (PTSD), psychotic symptoms, depression and suicide. In the same vein, not being able to provide support and be with family members, regardless of the severity of the individual's health status, can become a trigger for the emergence of feelings of guilt and sadness [19].

It is important to emphasize that before becoming infected, many individuals are developing feelings of insecurity in all aspects of life, from the collective to the individual perspective, from the daily functioning of society to changes in interpersonal relationships. Among confirmed or suspected patients of COVID-19, reports of boredom, loneliness, anger, insomnia, anxiety, decreased concentration, bad mood and loss of energy are common, along with their close family members, who have also been the focus of attention, given the fact that some have shown symptoms related to post-traumatic stress. In addition, the greatest impacts were verified in females, students and psychiatric patients, presenting elevations of emotional and physical burden, which facilitates the triggering, worsening or recurrence of mental disorders or physical diseases [20].

Therefore, correct understanding of information, raising awareness and transparency of health measures, as well as using other non-face-to-face devices to contact family and friends, strengthening social support through phone calls and video calls. , reduces the risk of developing anxiety and depression and encourages the population to participate in the disease prevention and control process. In addition, regular physical activity is important to help control anxiety and regulate sleep [14].

XII. Conclusion

In view of the points observed and listed by this study, it is possible to understand the magnitude of the new coronavirus and the importance of studies on the cause of the pandemic that began in 2020. In view of the complexity of its etiological agent, SARS- CoV -2, and its protein properties that contribute to its virulence, it is not difficult to think about how important it is to review studies on the subject in vogue. This fact becomes concrete when the epidemiology of SARS-CoV- 2 is analyzed, of 59 people infected in January 2020 in the first place to be identified the presence of the virus, the infection advanced to the point of infecting 445,129,499 people worldwide until March 2022. This circumstance is due to the high transmissibility of the etiological agent, which has three ways of spreading: inhalation of contaminated droplets, direct contact of exposed mucous membranes with such respiratory particles and indirect contact with fomites infected by SARS-Cov-2.

In line with what was said above, COVID-19 is a multisystem disease whose symptoms and prognosis depend on the stage of infection that the patient belongs in the moment of diagnosis. With its pathophysiology divided into three phases: I (viral replication), II (inflammatory) and III (systemic hyperinflammation), it is important to highlight the complex development of the virus in the invaded system and also the damage that will appear due to the installation of the infection. As a result, the patient's clinical condition is also classified in a stratified manner that the severity of the infection can be mild, moderate, severe and critical, ranging from asymptomatic to even cases of respiratory failure and multiple organ dysfunction. It is important to emphasize that, in addition to the severity of the disease, high levels of coagulation markers are important determinants of the prognosis of the pathology, as they help in understanding the thrombotic signature of COVID-19.

Therefore, the treatment of the new coronavirus happens through a balanced and ideal control of non-pharmacological and pharmacological interventions. The practical implementation of isolation, hand hygiene, early case detection and face mask in both treatment and prevention stands out , and such pharmacological measures helps to mitigate and reduce the size of the disease outbreak. Among the drugs currently being used in pharmacological treatment, antiviral agents, inflammation inhibitors, low molecular weight heparins, plasma and hyperimmune immunoglobulins are the most cited classes in the articles reviewed by this study, and there are cases in which there will be the need for non- invasive treatments such as Non-Invasive Positive Pressure Ventilation. In conclusion, the present study serves as an updated review of the condition of SARS-CoV- 2 and the pandemic caused by it and affirms the need for more studies that can elucidate unanswered questions about the new corona virus.

Acknowledgment

We thank everyone who participated, directly or indirectly, in the development of this research work, enriching our learning process.
Furthermore, we would like to thank the Centro Universitário do Espírito Santo-UNESC, essential in our professional training process, for its dedication and for everything we have learned over the years of the course.
Likewise, we thank everyone at Fundação de Amparo e Pesquisa do Espírito Santo (FAPAE), for providing data and materials that were fundamental to the development of the research that enabled this work to be done.
And, in particular, to our teachers Fernanda Quintela and Orlando Chiarelli, for having been our advisors and for having performed this role with dedication and friendship.

CONFLICT OF INTEREST

There is no conflict of interest.

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