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Review Article

New Pandemic, New Variant Virus, Heart Complications, Expected?

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Abstract

Coronaviruses belong to a family of single-stranded RNA viruses, already known for a long time, previously 6 strains with the ability to infect humans had been described, 4 of them with generally mild manifestations.

In December 2019, a new strain of this family called "SARS-COV2" was detected in China, whose pathogenic characteristics and virulence have given rise to the pandemic that persists more than a year after its declaration, the knowledge obtained since then has allowed to describe that its pathogenic manifestations range from common cold symptoms to acute respiratory distress syndrome.

The complications or sequelae of this disease called COVID 19 are extensive, and recently it has been described the "Long Covid" syndrome as a manifest of sequelae of the disease at a systemic level; one of the areas of greatest interest are the complications at cardiovascular level, immediately as well as in the long term. The objective of this document is to promote their study as well as to intensify their follow-up in patients recovered from SARS-CoV-2 infection.

Keywords: SARS-CoV-2; Coronavirus; Cardiac complications

Abbreviations

CoV: Coronavirus; RNA: Ribonucleic Acid; CoV(SARS- CoV): Severe Acute Respiratory Syndrome; CoV (MERS- CoV): Middle East respiratory syndrome; SARS-CoV-2: Severe Acute Respiratory Syndrome Coronavirus 2; COVID-19: Coronavirus Disease 19; WHO: World Health Organization; N: Nucleocapsid; S: Spike; HE: Hemagglutinin Esterase; M: Membrane; E: Envelope; ACE2: Angiotensin Converting Enzyme 2; SpO2, (PaO2/FiO2): Partial oxygen saturation; RT-PCR: Reverse Transcriptase Polymerase Chain Reaction; NAAT: Nucleic Acid Amplification; ACC: American College of Cardiology

Introduction

Coronaviruses (CoV) are a large family of single-stranded RNA viruses, with the capacity to infect animals, and 6 strains belonging to this family have the capacity to infect humans, their pathological manifestations are mainly respiratory, but they have also shown gastrointestinal, hepatic and neurological affection, among others [1]. CoV are divided into four genders: alpha-coronavirus, beta-coronavirus, gamma-coronavirus and delta-coronavirus, so far six Human Coronaviruses (HCoV) have been identified, including alpha -CoVs HCoVs-NL63 (Identified in 2004), HCoVs-229E (Identified in 1966), beta-CoVs HCoVs-OC43 (Identified in 1967) and HCoVs-HKU1 (Identified in 2005), these 4 are frequent causes of common cold with mild symptoms in immunocompetent patients. Other two strains are highly pathogenic and transmissible, one called severe acute respiratory syndrome -CoV (SARS-CoV) reported to in 2002 in Guangdong Province, China, and Middle East respiratory syndrome -CoV (MERS-CoV) identified in 2012 in Saudi Arabia. They tend to affect humans, mainly due to their high prevalence and wide distribution, their genetic diversity and the frequent recombination of their genomes, as well as increased human-animal interface activities [2].

New coronavirus

On December 26th of 2019 a 41 years man was admitted to the Central Hospital of Wuhan (China) with severe respiratory syndrome including fever, dizziness and coughing, in the bronchoalveolar lavage fluid a new strain of virus RNA from the family Coronaviridae was identified [3].

Other cases were diagnosed with pneumonia without an isolated agent, finding links between these patients and the seafood wholesaler market of Huanan of exotic species on southern China, this epidemic outbreak made it possible to identify the causative agent, which was designated Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) [4].

The clinical entity was named COVID-19 (Coronavirus Disease) by the World Health Organization (WHO) on January 30 of 2020, it was declared a public health emergency of international concern due to its high risk for countries with vulnerable health systems; to this date its presence has been identified in first world countries and also in countries such as Mexico, Brazil and in all the countries of South America and in most of the subregions of Central America and the Caribbean reaching pandemic figures [5,6].

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Etiology and pathophysiology

Due to its genomic characteristics, it was classified within the *Betacoronavirus* genus, Sarbecovirus subgenus and was called (SARS-CoV-2) [7].

According to the research the genome of this virus is 96% identical to the horseshoe bat (*Rhinolophus affinis*), so it is credited as its natural reservoir and hence has been proposed as intermediate host to Malays pangolins (*Manis javanica*) where it is proposed that there would be a recombination or genomic mutation of the virus, the latter with a genome between 85.5% and 92.4% of similarity with SARS-CoV-2 and these infect humans and probably ferrets and cats continuing the cycle of life [8].

The virus has a genomic size of 29.9 kb, consisting of a nucleocapsid composed of genomic RNA and one Nucleocapsid protein (N) phosphorylated. Its nucleocapsid is located within phospholipid bilayers and is covered by two different types of spike proteins, the spike glycoprotein trimmer (S) that it shares with the rest of the coronavirus and the Hemagglutinin-Esterase (HE) present only in some. The membrane protein (M) and the envelope protein (E) are among the S proteins in the viral envelope, the particle has a diameter of between 60 nm and 140 nm and its shape is round, the virus has quite distinctive peaks, approximately 9 nm to 12 nm long, that give it the appearance of a solar corona.

In its pathogenesis, the role of the Angiotensin Converting Enzyme 2 (ACE2) stands out, being essential in the cardiovascular and immune systems, in addition to being related to the development of hypertension and diabetes mellitus. This enzyme has been identified as a functional receptor for the virus, which fuses the viral membrane with the host cell through the structural rearrangement of the S protein [9,10]. According to what has been seen with other coronaviruses, it is suggested that SARS-CoV-2 can reduce the regulation of ACE2, which leads to a toxic over-accumulation of angiotensin-II that can induce acute respiratory distress syndrome and fulminant myocarditis. [11,12].

Clinical manifestations

Its manifestations appear after an incubation period of approximately 5.2 days, in cases of death a median of 14 days has been reported from the onset of symptoms, this depends on the age of the patient and the immune status. The clinical manifestations with which it occurs have a wide spectrum from mild to moderate symptoms with fever, fatigue, and dry cough, among others such as headache, nasal congestion, pharyngodynia, myalgia, arthralgia, diarrhea, nausea, and severe with dyspnea usually in the second week of illness that could be accompanied by or progress to hypoxemia [13].

Staging

Adults with a diagnosis of SARS-CoV-2 infection can be grouped according to the severity of the disease; however the criteria for each category may vary between guidelines and clinical studies as well as change over time. The National Institutes of Health proposes the following clinical classification of COVID-19:

- Asymptomatic or Presymptomatic Infection: Individuals who test positive for SARS-CoV-2 using a virologic test (i.e., a nucleic acid amplification test or an antigen test) but who have no symptoms that are consistent with COVID-19.
- Mild Illness: Individuals who have any of the various signs and symptoms of COVID-19 (e.g., fever, cough, sore throat, malaise, headache, muscle pain, nausea, vomiting, diarrhea, loss of taste and smell) but who do not have shortness of breath, dyspnea, or abnormal chest imaging.
- Moderate Illness: Individuals who show evidence of lower respiratory disease during clinical assessment or imaging, and who have saturation of oxygen (SpO2) ≥ 94% on room air at sea level.
- Severe Illness: Individuals who have SpO2 <94% on room air at sea level, a ratio of arterial partial pressure of oxygen to fraction of inspired oxygen (PaO2/FiO2) <300 mmHg, respiratory frequency >30 breaths/min , or lung infiltrates >50%.
- Critical Illness: Individuals who have respiratory failure, septic shock, and/or multiple organ dysfunctions.
- Among all the cases, asymptomatic infected people (1.2%), mild to moderate cases (80.9%), severe cases (13.8%) and critical illness (4.7%) are reported. Most cases with mild symptoms can recover in 1 to 2 weeks.

Diagnosis

The diagnosis of suspected cases is attached to the operational definition proposed by WHO and is defined as a confirmed case with or without symptoms plus a positive result of the polymerase chain reaction of reverse transcriptase (RT-PCR), which is considered the gold standard or Nucleic Acid Amplification Test (NAAT) from samples of nasal swab, pharyngeal, sputum, and also serum antigen detection although it is less sensitive than RT-PCR but it has similar specificity [14]. Other tests such as the antibody defection are used as a guideline because of the amount of false negative results that it shows, one of the explanations is that it can take up to 21 days or more after the first symptom for seroconversion occur and therefore be detectable in the blood. It should be noted that there is no serological test for the diagnosis of SARS-CoV-2 approved by the FDA, but some commercially available tests have received authorization for emergency use by the FDA [14,15].

Complications

Patients considered with severe illness often have concomitant factors, which predispose a poor outcome, as advanced age, diabetes, hypertension, previous cardiovascular disease or immunocompromised; in these patients it's common for the acute respiratory distress syndrome to manifest with high morbidity and mortality. As the disease progresses, several complications tend to occur, such as shock, sepsis, acute kidney injury. However the most striking one is the acute cardiac injury, demonstrated by serum levels of cardiac biomarkers (p. Ex., Troponin I) which are above the upper reference limit of the 99th percentile, or new abnormalities in electrocardiography and echocardiography [16-18].

Cardiac complications

It is known that viruses are one of the leading infectious causes of myocarditis, however cardiac affectation as complication of Covid19 is not known thoroughly, so far RNA of coronavirus has not been found in myocardium biopsies, it may be possible that an exaggerated inflammatory response exists and that this response causes myocardial injury, not the virus itself. The American College of Cardiology (ACC) has highlighted the importance of heart implications from this

disease, citing interesting statistics as the development of arrhythmias in 16.7% of patients and severe heart damage in 7.2% [19,20].

Recent studies have postulated hypotheses of COVID19 cardiac injury, including ACE2 direct damage, hypoxia - induced myocardial injury, heart damage and microvascular systemic inflammatory response [21].

Chronic heart damage due to this family of viruses has been described in the past, in a 12-year follow-up survey in patients who recovered from SARS-CoV infection hyperlipidemia was found in 68%, abnormalities of the cardiovascular system in 44% and disorders of the glucose metabolism in 60%. The study also reported an increase in serum free fatty acids, lysophosphatidylcholine, lysophosphatidylethanolamine and phosphatidylglycerol significantly marked compared to people with no history of infection, the mechanisms by which this happens are not clear yet. Since the SARS-CoV-2 is remarkably similar to SARS-CoV, it is possible that the disease develops with chronic damage to the cardiovascular system, so the cardiovascular function should be assessed in light of the current ignorance with practical and valuable methods such as echocardiography [21].

The recent appearance of the disease has led to the fact that not all the long-term side effects of SARS CoV2 are known , in the case of SARS, hypotension, myocarditis, arrhythmias and sudden cardiac death, as well as electrocardiographic changes, subclinical diastolic deterioration of the left ventricle and elevation of biomarkers (troponins) has been reported, with respect to MERS, an association has been found with myocarditis and heart failure; with the above mentioned and the already known effects of other coronaviruses, monitoring must be warranted for patients infected COVID-19 who have now recovered [22-24].

It is also known that other viral groups have a cardiovascular impact, an example of this is the association of influenza infections and an increased risk of myocardial infarction the first days after its onset and even weeks later; some influenza strains have similarity between the amino acid sequences involved in the binding of the cell site to viral hemagglutinin and the amino acids of low-density lipoproteins, which causes alterations in lipids , which causes an increase in the expression of pro-inflammatory cytokines that has also been described in infection by SARS Cov2 [25].

Conclusion

Coronaviruses are a family of viruses known for a long time, they have as natural hosts animals and as an intermediate host the human, in which the clinical manifestations are mostly respiratory. It has been more than a year since the appearance of a new strain that received the name of SARS-CoV-2 that gave rise to a pandemic which has left invaluable losses of millions of humans, which doctors and health personnel have had to deal with, having little knowledge of its management, representing a challenge and a problem that has had repercussions not only on health but also on the global economy.

The follow-up of patients who have managed to recover from this infection is essential, since medium and long-term complications could affect the quality of life and if not detected and treated in time, they could be fatal.

Doctors will aim, in addition to the acute management of the disease, to look for and treat complications, those of the cardiovascular system being very important, it has been found that the myocardium is also affected and the dilemma of the long-term effects that it has are of great importance. For this reason, emphasis is placed on not lowering our guard with all those who are already discharged from the hospital for this infection or who have already passed the acute phase, because perhaps, what we see in the short term is only the tip of an iceberg.

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