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COVID-19 and Its Impact at the Myocardial Level

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ABSTRACT

T*he disease known as COVID-19 not only affects the respiratory system, but can also involve the cardiovascular system. This work describes the main viral characteristics, the pathophysiology and its repercussion at the cardiac level. SARS-COV2 enters the cells of the respiratory epithelium through the interaction of protein S with the receptor for the angiotensin-converting enzyme 2, which can lead to a severe inflammatory reaction. Among the most important cardiovascular manifestations we have direct myocardial damage, which can cause from acute myocardial infarction to cardiogenic shock, followed by the presence of heart failure and finally the appearance of arrhythmias.*

Keywords: *COVID-19, cardiovascular system, arrhythmias, myocardial infarction.*

INTRODUCTION:

The disease known as COVID-19, not only affects the respiratory system but may involve other organs, including the cardiovascular system, which increases existing health problems in developing countries. The primary objective of this work is to provide general information on the etiology, pathophysiology and its repercussion at the cardiac level, in order to improve diagnosis and therapy.

GENERAL CHARACTERISTICS

In December 2019, a new type of coronavirus was identified as the cause of pneumonia in Wuhan, China. This disease quickly spread initially throughout the country and later throughout the world. Due to its global spread, in February 2020, the World Health Organization (WHO) designated it as a pandemic.1 This beta RNA coronavirus comes from the same subgenus as the 2003 severe acute respiratory syndrome (SARS). The structure of the ligand-receptor is very similar to that of SARS and it has been possible to identify the same receptor for its entry into the cell through the receptor for the angiotensin-converting enzyme 2 (ACE2) 2. Due to discrepancies in the initial nomenclature, an international committee proposed the name of severe acute respiratory syndrome due to coronavirus 2 (SARS-CoV-2). 3 Regarding its morphological characteristics, it has a spherical or elliptical shape with a diameter of 60-140 nm. Like other coronaviruses, it is sensitive to ultraviolet light and heat, on the other hand they are inactivated with lipid solvents such as 75% ether, ethanol, chlorine, peroxyacetic acid and chloroform. Phylogenetically it has been considered that bats are the primary source of these viruses, but their transmission to humans has not yet been precisely elucidated.4 The mechanism of transmission from person to person is through drops, which originate when You sneeze, cough, or speak and can infect another person if you have direct contact with the mucosa. Similarly, infection can occur when the person touches

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a surface contaminated by these drops and subsequently has contact with their mucosa. It should be noted that the drops do not travel more than 2 meters away or persist in the air. 5,6 The incubation period includes up to 14 days after exposure and in most cases occurs between days 4 and 5.7-9

CLINICAL PRESENTATION

Patients may present with fever, cough, dyspnea, and bilateral infiltrates detected by some imaging study when they debut with severe respiratory infection. In a study, 138 patients with COVID-19 were described where the main clinical data were: fever (99%), fatigue (70%), dry cough (59%), anorexia (40%), myalgia (35%), dyspnea (31%), anosmia (34%), dysgeusia (34%), and gastro-intestinal symptoms (18%). 10 We must emphasize that the time interval to say that a patient stops being contagious is still uncertain and may vary depending dela severity of the disease can go from 8 to 37 days 11,12. It is estimated that about 80% of virus carriers can be asymptomatic.

It has been observed that the majority of infections are not severe, specifically in a report, where 44,500 patients were included, it was possible to estimate the severity of the disease in the following spectra: a) Mild-moderate: without pneumonia (81%) , b) Severe: with dyspnea, hypoxia or image-confirmed lung disease in the first 24-48 hours greater than 50% (14%), c) Critical: with data on respiratory failure, shock or multiple organ damage (5%) . 13 According to the WHO, mortality in Wuhan was 5% and 0.7% in the rest of China. However, these figures have changed depending on the geographical region studied, for example mortality in Italy has been 7.2% and in South Korea 0.9% .14

RISK FACTOR'S

Severe disease can occur in any individual, however there are groups with greater susceptibility, especially adults over 60 years of age, the risk increases if the following comorbidities or risk factors are associated: cardiovascular disease, diabetes mellitus, hypertension systemic arterial, chronic obstructive pulmonary disease, obesity, cancer, chronic kidney disease, asthma, immunocompromise. 15-18

Within the evaluation with paraclinical studies, indicators of poor prognosis have been found, such as: lymphopenia, elevation of liver enzymes, lactate dehydrogenase, inflammation markers (C-reactive protein, ferritin, interleukin-6), D-dimer, prothrombin time, troponin, creatinine phosphokinase, and azo. 18.19

People of any age can suffer from severe illness, however older adults are the most susceptible. As an example of this, in one study it was found that the proportion of hospitalizations increased with age, with only 1% being hospitalized in those aged 20-29, 4% between 50-59% and 18% in those over 80 (20)

PATHOPHYSIOLOGY

Initially, the virus enters the cell through the ACE2 receptor, whose main function is the enzymatic conversion of angiotensin II to angiotensin 1-7 and conversion of angiotensin I to angiotensin 1-9 21-24. Protein S together with tyrosine-serine-coupled kinase receptors (TMPRSS2) are responsible for binding to the ACE2 receptor of pulmonary epithelium25. It should be emphasized that the ACE2 receptor is highly expressed at cardiovascular and even gastrointestinal levels in addition to the respiratory epithelium. Furthermore, this virus can enter cells through CD209, which is expressed in macrophages, promoting invasion of immune cells and cardiovascular tissue. 26-28

In severe cases, an uncontrolled inflammatory reaction occurs, which has been called "cytokine storm", whose main effector is interlucin-6 (IL-6). This cytosine is produced by activated leukocytes and promotes the differentiation of B lymphocytes, stimulates the production of acute phase proteins and plays an important role in thermoregulation. Finally, it must be said that it increases in the presence of infections, autoimmune disorders, cardiovascular diseases and even some types of cancer.(21) Cytosine storm also includes the expression of other substances such as IL-2, IL-7, stimulation factor for granulocyte colonies, CXC chymosin 10 (CXCL10), chymosin ligand (CC) and alpha tumor necrosis (TNF-α).

COMPLICATIONS

The main complication is ARDS and can occur immediately after dyspnea. In one study, its presence was reported in up to 20% with a median appearance 8 days after the onset of symptoms, in addition to 12.3% requiring advanced management of the airway. 10 We will now describe the main complications in other organs and at the cardiovascular level.

a) Macrophage activation syndrome and altered thrombotic response

During the evolution of patients, it has been observed that those infected rapidly aggravate presenting a clinical picture compatible with macrophage activation syndrome (SAM). This response has been detected even in young and apparently healthy patients, who have had fatal outcomes. Within the pathophysiology of SAM, we can observe uncontrollable production of T cells, exaggerated activation of macrophages, and hypersecretion of IL-1β, IL-6, interferon, and tumor necrosis factor α (TNF- α). Due to the potential catastrophic events of this syndrome, it is important to make an early diagnosis, paying attention to the levels of ferritin and especially to the D-dimer, because it has been seen that thrombotic phenomena.

b) Direct myocardial damage

This term encompasses all conditions that cause cardiomyocyte cell disruption and is identified with the presence of at least one troponin value above the 99th percentile. 31 Patients with severe COVID-19 disease may experience this type of injury. due to different causes, such as myocarditis, hypoxemic injury, stress cardiomyopathy (Takotsubo), ischemia due to damage to the micro vasculature, coronary artery disease (rupture of the plaque with obstruction of flow, or increased myocardial O2 demand), cor pulmonale and even cytosine storm, however, the contribution of myocardial damage from these events has not yet been determined.32-36 It has been reported that troponin elevation can occur in 7-17% of hospitalized patients and is more frequent in those who enter intensive care (22%) and in those who die (59%). 31,34 -39 Finally, we must mention that direct damage to the myocardium can become so severe that we can observe patients with cardiogenic shock.

c) Heart failure

Very limited information is available on the incidence of heart failure in patients with COVID-19. In a retrospective study of 799 patients in Wuhan it was identified in 49% of deaths and 3% of those who recovered 40. In another study of 191 patients it was identified in 52% of those who died and in 12% of those who died. they recovered. Similarly, elevation of BNP and NT-proBNP has been identified, particularly in those with myocardial damage.(32)

d) Cardiac arrhythmias and conduction disorders

It has been reported that up to 16.7% of hospitalized patients have some type of electrical conduction disorder of the heart. 41 Among the causes for arrhythmias, we can find the following: myocardial damage, ischemia, hypoxia, cardiogenic shock, septic shock, systemic inflammatory response, and pharmacological therapies that help prolong the QT interval, thus favoring the presentation of malignant arrhythmias such as polymorphic ventricular tachycardia that could generate ventricular fibrillation, asystole and death. 42-43

Most patients admitted for COVID-19 may have no symptoms or signs of arrhythmias. Probably only tachycardia are found in addition to other more common symptoms: fever, dyspnea, and pain. Despite this, there is a lot of evidence about the presence of these disorders in COVID-19, for example, in a study of 8,910 patients, 304 patients had tachyarrhythmias (3.4%) and were more common in those who died compared to survivors (6.8% vs. 3.2%) .44 On the other hand, in a Hubei study of 137 patients, 10 patients noted palpitations as the initial symptom, 45 while in another 17% had electrocardiographic confirmation of arrhythmias and 44% of the patients who they were admitted to intensive care they developed them, 10 finally in another report, 260 patients (6.1%) had QTc> 500 at admission after treatment with hydroxychloroquine and azithromycin. 46,47 Similarly, a higher prevalence of atrial tachyarrhythmias has been found in patients with mechanical ventilation (17.7% vs. 1.9%), 48 and a higher frequency of occurrence of ventricular tachycardia49.

It is important to mention that in those patients receiving chloroquine and hydroxychloroquine therapy, it should be known that both prolong the QT interval by blocking the potassium channel IKr and both are metabolized by CYP3A4, so that other drugs that block this enzyme can increase concentrations. of the other two. Hydroxychloroquine has been reported to generate torsades de pointes and the combination with azithromycin has been associated with an increased risk of prolonging QT. Due to this, certain recommendations have been generated to monitor patients who receive any of these medications, such as: taking an electrocardiogram 2-3 hours after ingestion, discontinuing other medications that prolong the QT, correcting the hydroelectrolytic disorders and continuous monitoring with telemetry. 48.49

CONCLUSIONS

Despite the fact that COVID-19 infection has the lung as the main target organ, it is important to recognize the epidemiological and clinical evidence in which cardiovascular alterations have been described, which have a transcendental impact on the short and long-term outcomes. Recognizing these pathologies can prevent complications and improve the evolution of patients affected by COVID-19. These complications originate from the macrophage activation syndrome with altered thrombotic response and its manifestations can be observed by an exaggerated inflammatory response, producing in 20% of cases an acute respiratory distress syndrome, and in 12.3% it is assisted mechanical ventilation is necessary. At the myocardial level, direct damage manifested by myocarditis with varying degrees of heart failure, including cardiogenic shock, was observed. It has been observed that from 7-17% of COVID-19 positive cases there is a significant elevation in serum troponin levels, which in turn has been confirmed to play a role as biomarkers of poor prognosis of the disease. Heart failure has been observed in 3-5%, being more common in up to 49% of patients who died. Arrhythmia and conduction disorders have shown that up to 17% of patients with COVID-19 have some conduction disorder, however the most feared complication is polymorphic ventricular tachycardia (VT) related to QT interval prolongation. (6.1%), In patients under mechanical ventilation, an incidence of TV of up to 17% was observed, and this risk increases when the use of hydroxychloroquine is associated with azithromycin. Finally, the myocardial repercussion by COVID-19 directly affects the mortality of the disease.

REFERENCES

1. World Health Organization [internet]. Director-General's remarks at the media briefing on 2019-nCoV on 11 February 2020 [cited 2020 Feb 11]. Available from: https://www-who-int.pbidi.unam.mx:2443/dg/speeches/detail/who-director-general-s-remarks-at-the-media-briefing-on-2019-ncov -on-11-february-2020

2. Zhou P, Yang XL, Wang XG, Hu B, Zhang L, Zhang W, et al. A pneumonia outbreak associated with a new coronavirus of probable bat origin. Nature 2020; 579: 270-273.

3. Gorbalenya AE, Baker SC, Baric RS, de Groot RJ, Drosten C, Gulyaeva AA, et al. Severe acute respiratory syndrome-related coronavirus: The species and its viruses - a statement of the Coronavirus Study Group. Nat Microbiol 2020; 5: 536–544

4. Tang X, Wu C, Li X, Song Yuhe, Yao X, Wu X, et al. On the origin and continuing evolution of SARS-CoV-2. Natl Sci Rev 2020; 0: 1-12.

5. World Health Organization [internet]. Novel coronavirus situation report -2. January 22, 2020 [cited January 22, 2020]. Available at: https://www-who-int.pbidi.unam.mx:2443/docs/default-source/coronaviruse/situation-reports/20200122-sitrep-2-2019-ncov.pdf

6. van Doremalen N, Bushmaker T, Morris DH, Holbrok MG, Gamble A, Williamson BN, et al. Aerosol and Surface Stability of SARS-CoV-2 as Compared with SARS-CoV-1.N Engl J Med. 2020; 382 (16): 1564-1567.

7. Li Q, Guan X, Wu P, Wang X, Zhou L, Tong Y et al. Early Transmission Dynamics in Wuhan, China, of Novel Coronavirus-Infected Pneumonia.N Engl J Med. 2020; 382 (13): 1199-1207

8. Guan WJ, Ni ZY, Hu Y, Liang WH, Ou CQ, He JX, et al. Clinical Characteristics of Coronavirus Disease 2019 in China.N Engl J Med. 2020; 382 (18): 1708-1720.

9. Chan JF, Yuan S, Kok KH, To KK, Chu H, Yang J, et al. A familial cluster of pneumonia associated with the 2019 novel coronavirus indicating person-to-person transmission: a study of a family cluster.Lancet. 2020; 395 (10223): 514-523.

10. Wang D, Hu B, Hu C, Zhu F, Liu X, Zhang J, et al. Clinical Characteristics of 138 Hospitalized Patients With 2019 Novel Coronavirus-Infected Pneumonia in Wuhan, China.JAMA. 2020; 323 (11): 1061-1069.

11. Zou L, Ruan F, Huang M, Liang L, Huang H, Hong Z, et al. SARS-CoV-2 Viral Load in Upper Respiratory Specimens of Infected Patients.N Engl J Med. 2020; 382 (12): 1177-1179.

12. Wölfel R, Corman VM, Guggemos W, Seilmaier M, Zange S, Müller M, et al. Virological assessment of hospitalized patients with COVID-2019.[published online ahead of print, 2020 Apr 1]. Nature. 2020; 10.1038 / s41586-020-2196-x. doi: 10.1038 / s41586-020-2196-x

13. Wu Z, McGoogan JM. Characteristics of and Important Lessons From the Coronavirus Disease 2019 (COVID-19) Outbreak in China: Summary of a Report of 72 314 Cases From the Chinese Center for Disease Control and Prevention.[published online ahead of print, 2020 Feb 24]. JAMA. 2020; 10.1001 / jama.2020.2648. doi: 10.1001 / jama.2020.2648

14. KCDC [internet]. Updates on COVID-19 in Korea. [Cited 2020 Mar 14]. Available from: https://www.cdc.go.kr/board/board.es?mid=a30402000000&bid=0030

15. Zhou F, Yu T, Du R, Fan G, Liu Y, Liu Z, et al. Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study.Lancet. 2020; 395 (10229): 1054-1062.

16. Liang W, Guan W, Chen R, Wang W, Li J, Xu K, et al. Cancer patients in SARS-CoV-2 infection: a nationwide analysis in China.Lancet Oncol. 2020; 21 (3): 335-337.

17. CDC COVID-19 Response Team. Preliminary Estimates of the Prevalence of Selected Underlying Health Conditions Among Patients with Coronavirus Disease 2019 - United States, February 12-March 28, 2020. MMWR Morb Mortal Wkly Rep. 2020; 69 (13): 382-386. Published 2020 Apr 3. doi: 10.15585 / mmwr.mm6913e2

18. Wu C, Chen X, Cai Y, Xia J, Zhou X, Xu S, et al. Risk Factors Associated With Acute Respiratory Distress Syndrome and Death in Patients With Coronavirus Disease 2019 Pneumonia in Wuhan, China.[published online ahead of print, 2020 Mar 13]. JAMA Intern Med. 2020; e200994. doi: 10.1001 / jamainternmed.2020.0994

19. Shi S, Qin M, Shen B, Cai Y, Liu T, Yang F, et al. Association of Cardiac Injury With Mortality in Hospitalized Patients With COVID-19 in Wuhan, China.[published online ahead of print, 2020 Mar 25]. JAMA Cardiol. 2020; e200950. doi: 10.1001 / jamacardio.2020.0950

20. Waldman FM, Koblin DD, Lampe GH, Wauk LZ, Eger 2nd EI. Hematologic effects of nitrous oxide in surgical patients.Anesth Analg. 1990; 71 (6): 618-624.

21. Cascella M, Rajnik M, Cuomo A, et al. Features, Evaluation and Treatment Coronavirus (COVID-19) [Updated 2020 Apr 6]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK554776/

22. Walls AC, Parky YJ, Tortorici MA, Wall A, McGuire AT, et al. Structure, Function and Antigenecity of the SARS-COV-2 Spike Glycoprotein. Cell. 2020; 181 (2): 281-292.

23. Yan R, Zhang Y, Li Y, Xia L, Xia L, Guo Y, Zhou Q, et al. Structural basis for the recognition of SARS CoV-2 by full-lenght human ACE2. Science. 2020; 367 (6485): 1444-1448

24. Santos R, Sampio W, Alzamora A, Santos D, Alenina N, Bader M, et al. The ACE2 / Angiotensin- (1-7). Physiol Rev. 2018; 98 (1): 505-553.

25. Hoffman M, Kleine-Weber H, Schroeder S, Krüger N, Herrler T, Erichsen S, et al. SARS-CoV-2 Cell Entry Depends on ACE and TMPRSS2 and is Blocked by a Clinically Proven Protease Inhibitor. Cell. 2020; 181 (2): 271-280.

26. Hamming I, Timens W, Bulthius M, Lely AT, Navis GJ, van Goor H. Tissue distribution of ACE2 protein, the functi9onal receptor for SARS coronavirus. A fist step in undertandins SARS pathogenesis. J Pathol. 2004; 203 (2): 631-637.

27. Chen L, Li X, Chen M, Feng Y, Xiong C. The ACE2 Expression in human heart indicates new potential mechanism of heart injury among patient infected with SARS-CoV-2. Cardiovasc Res. 2020; 116 (6): 1097-1100.

28. Li Z, Guo X, Hao W, Wu Y, Ji Y, ZhaoY, et al, The relationship between serum interlukins and T-lymphocytes subset in patient with severe acute respiratory syndrome. Chin Med J (Engl). 2003; 116 (7): 981-984.

29. Huang C, Wang Y, Li X, Ren L, Zhao J, Hu Y, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet. 2020; 395 (10223): 497-506.

30. Mehta P, McAuley DF, Brown M, Sanchez E, Tattesall RS, Manson JJ et al. COVID-19: consider cytokine storm syndromes and immunosuppression. Lancet. 2020; 395 (10229): 1033-1034.

31. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax J, Morrow DA, et al. Fourth Universal Definition of Myocardial Infarction JCirculation. 2018; 138 (20): e618-e651

32. Ruan Q, Yang K, Wang W, Jiang L, Song J. Clinical predictor of mortality due to COVID-19 based on an analysis of data of 150 patients from Wuhan, China, Intensive Care Med. 2020; 46 (5): 846-848

33. Shi S, Qin M, Shen B, Cai Y, Liu T, Yang F, et al. Association of Cardiac Injury With Mortality in Hospitalized Patients With COVID-19 in Wuhan, China.[published online ahead of print, 2020 Mar 25]. JAMA Cardiol. 2020; e200950. doi: 10.1001 / jamacardio.2020.0950

34. Caforio AL, Pankuweit S, Arbustini E, Basco C, Blanes J, Felix SB et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases.Eur Heart J. 2013; 34 (33): 2636-2648

35. Creel-Bulos C, Hockstein M, Amin N, Melhem S, Truong A, Sharifpour M. Acute Cor Pulmonale in Critically Ill Patients with Covid-19. N Engl J Med. 2020; 382 (21): e70

36. Zheng YY, Ma YT, Zhang JY, Xie X. COVID-19 and the cardiovascular system. Nat Rev Cardiol. 2020; 17 (5): 259-260.

37. Tersalvi G, Vicenzi M, Calabretta D, Biasco L, Pedrazzini G, Winterton D. Elevated Troponin in Patients With Coronavirus Disease 2019: Possible Mechanisms.[published online ahead of print, 2020 Apr 18]. J Card Fail. 2020; S1071-9164 (20) 30357-2. doi: 10.1016 / j.cardfail.2020.04.009

38. Lippi G, Lavie CJ, Sanchis-Gomar F. Cardiac troponin I in patients with coronavirus disease 2019 (COVID-19): Evidence from a meta-analysis. [published online ahead of print, 2020 Mar 10]. Prog Cardiovasc Dis. 2020 ;. doi: 10.1016 / j.pcad.2020.03.001

39. Bhatraju PK, Ghassemieh BJ, Nichols M, Kim R, Jerome KR, Nalla AK, et al. Covid-19 in Critically Ill Patients in the Seattle Region - Case Series.N Engl J Med. 2020; 382 (21): 2012-2022

40. Chen T, Wu D, Chen H, Yan W, Yang D, Chen G, et al. Clinical characteristics of 113 deceased patients with coronavirus disease 2019: retrospective study.[published correction appears in BMJ. 2020 Mar 31; 368: m1295]. BMJ. 2020; 368: m1091. Published 2020 Mar 26. doi: 10.1136 / bmj.m1091

41. Chen C, Zhou Y, Wang DW. SARS-CoV-2: A Potential Novel Etiology of Fulminant Myocarditis.Herz. 2020; 45 (3): 230-232.

42. Driggin E, Madhavan MV, Bikdeli B, Chuich T, Laracy J, Zoccai G, et al. Cardiovascular Considerations for Patients, Health Care Workers, and Health Systems During the COVID-19 Pandemic.J Am Coll Cardiol. 2020; 75 (18): 2352-2371.

43. Lakkireddy DR, Chung MK, Gopinathannair R, Patton KK, Gluckmann T, Turagam M et al. Guidance for Cardiac Electrophysiology During the Coronavirus (COVID-19) Pandemic from the Heart Rhythm Society COVID-19 Task Force; Electrophysiology Section of the American College of Cardiology; and the Electrocardiography and Arrhythmias Committee of the Council on Clinical Cardiology, American Heart Association [published online ahead of print, 2020 Apr 1]. Heart Rhythm. 2020 ;. doi: 10.1016 / j.hrthm.2020.03.028

44. Mehra MR, Desai SS, Kuy S, Henry TD, Patel AN. Cardiovascular Disease, Drug Therapy, and Mortality in Covid-19.[published online ahead of print, 2020 May 1]. N Engl J Med. 2020; NEJMoa2007621. doi: 10.1056 / NEJMoa2007621.

45. Liu K, Fang YY, Deng Y, Liu W, Wang M, Ma JP, et al. Clinical characteristics of novel coronavirus cases in tertiary hospitals in Hubei Province. Chin Med J (Engl). 2020; 133 (9): 1025-1031.

46. Richardson S, Hirsch JS, Narasimhan M, Crawford JM, McGinn T, Davidson K, et al. Presenting Characteristics, Comorbidities, and Outcomes Among 5700 Patients Hospitalized With COVID-19 in the New York City Area.JAMA2020; 323 (20): 2052-2059.

47. Chorin E, Dai M, Shulman E, Dai M, Shulman E, Routhier CN, et al. The QT interval in patients with COVID-19 treated with hydroxychloroquine and azithromycin.[published online ahead of print, 2020 May 11]. Heart Rhythm. 2020; S1547-5271 (20) 30435-5. doi: 10.1016 / j.hrthm.2020.05.014

48. Goyal P, Choi JJ, Pinheiro LC, Schenck EJ, Chen R, Jabri A et al. Clinical Characteristics of Covid-19 in New York City.[published online ahead of print, 2020 Apr 17]. N Engl J Med. 2020; NEJMc2010419. doi: 10.1056 / NEJMc2010419

49. Guo T, Fan Y, Chen M, Wu X, Zhang L, He T, et al. Cardiovascular Implications of Fatal Outcomes of Patients With Coronavirus Disease 2019 (COVID-19).[published online ahead of print, 2020 Mar 27]. JAMA Cardiol. 2020; e201017. doi: 10.1001 / jamacardio.2020.1017

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