COVID-19 and the Heart

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We have been living with the new coronavirus pandemic since March 11, 2020. Initially, on January 1, 2020, the World Health Organization (WHO) declared the new coronavirus infection a global emergency and proceeded to name the disease COVID-19. The Coronavirus Study Group of the International Committee on Taxonomy of Viruses proposed the adoption of the name SARS-Cov-2.[1] Genomic sequencing and phylogenetic analysis have indicated that it is a betacoronavirus from the same subgenus as the severe acute respiratory syndrome (SARS) that caused an epidemic in China in 2003 and the Middle East respiratory syndrome (MERS) that caused the same condition in the Middle East in 2012. It has 96.2% genetic identity with betaCoV/bat/Yunnan, a virus isolated in bats. The structure of the virus's cell-binding receptor gene is very similar to that of the SARS coronavirus, and the virus appears to use the same angiotensive-converting enzyme 2 (ACE2) receptor to enter the cell.

The clinical picture of COVID-19 is similar to that of other respiratory viruses, namely, fever, generally dry cough, fatigue, and, in severer cases (5%), dyspnea, pulmonary bleeding, severe lymphopenia, and renal failure. Symptoms are mild in 80% of cases. Diagnosis of symptomatic cases should be confirmed by testing for the virus via polymerase chain reaction (PCR) of a nasal swab.

The cardiac complications of this disease have drawn physicians' attention. In a study evaluating 138 patients hospitalized for COVID-19, 16.7% developed arrhythmia, and 7.2% presented acute cardiac injury.[2] On the other hand, cardiologists at the San Raffaele Hospital in Milan, Italy, which is the referral hospital for cardiovascular complications from COVID-19, collected enzymes (BNP, troponin, CK-MB) from all patients to detect the prevalence of cardiac injury. As of March 9, of the 82 patients admitted, 19 of whom were in the intensive care unit, only one 43-year-old female patient had been admitted for chest pain with ST-segment changes and diagnosis of pneumonia. Her coronary angiography was normal.[3]

Keywords

Coronavirus-19/complications; betaCoV/bat/Yunnan/complications; Fever; Severe Acute Respiratory Syndrome; Dyspnea; Respiration Disorders; Risk Factors; Hypertension; Diabetes Mellitus.

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A large study published by the Chinese Center for Disease Control and Prevention, with data from 44,672 confirmed cases of COVID-19, reported a mortality of 2.3%. The most common comorbities in patients who died were arterial hypertension, diabetes mellitus, cardiovascular disease, and age over 70 years.[4]

Another published study[1] based on retrospective analysis of databanks from two hospitals in Wuhan (the Jin Yin-tan Hospital and the Tongji Hospital) evaluated 150 cases of laboratory confirmed infection with SARS-CoV-2, 68 (45%) of which resulted in death. The following discharge criteria were applied: no fever for at least 3 days, significant improvement in respiratory function, and 2 consecutive negative tests for the virus. There was a statistically significant difference for advanced age in patients who died ($p < 0.001$), but there was no difference between sexes ($p = 0.43$). A total of 63% (43/68) of patients who died had underlying diseases, in comparison with 41% (34/82) of patients who were discharged ($p = 0.0069$). Patients with associated cardiovascular disease had a greater risk of death ($p < 0.001$). There was also a higher incidence of secondary infections in patients who died compared to those who were discharged (16% [11/68] vs. 1% [1/82], $p = 0.0018$). Figure 1 shows higher values of inflammatory mediators in patients who died, and Figure 2 summarizes the causes of death.

Another study published in *The Lancet* [5] identified increased interleukin-6, high-sensitivity troponin I, and lactate dehydrogenase values as more frequent findings in hospitalized patients who died ($n = 54$) compared with those who survived ($n = 137$) in 2 hospitals in Wuhan, China. Of all the patients, 91 (48%) had some comorbidity, the following being the most common: arterial hypertension in 58 patients (30%), diabetes mellitus in 36 patients (19%), and chronic coronary disease in 15 patients (8%). However, in multivariate analysis of risk factors for outcome of death, only advanced age, higher Sequential Organ Failure Assessment (SOFA) score, and d-dimer greater than 1 μg/l at admission were statistically significant.

There is still no evidence that the use of angiotensin converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARB) may affect the virus's activity. The Council on Hypertension of the European Society of Cardiology recommends that doctors and patients continue antihypertensive treatment as usual.

Another fundamental recommendation is that everyone is vaccinated against influenza, whose seasonal activity has already begun in Brazil, and which, to date, has higher mortality than COVID-19.

This is a time for vigilance, common sense, and scientific investigation. Medical societies must organize themselves in order to establish protocols for recognizing and treating complications.

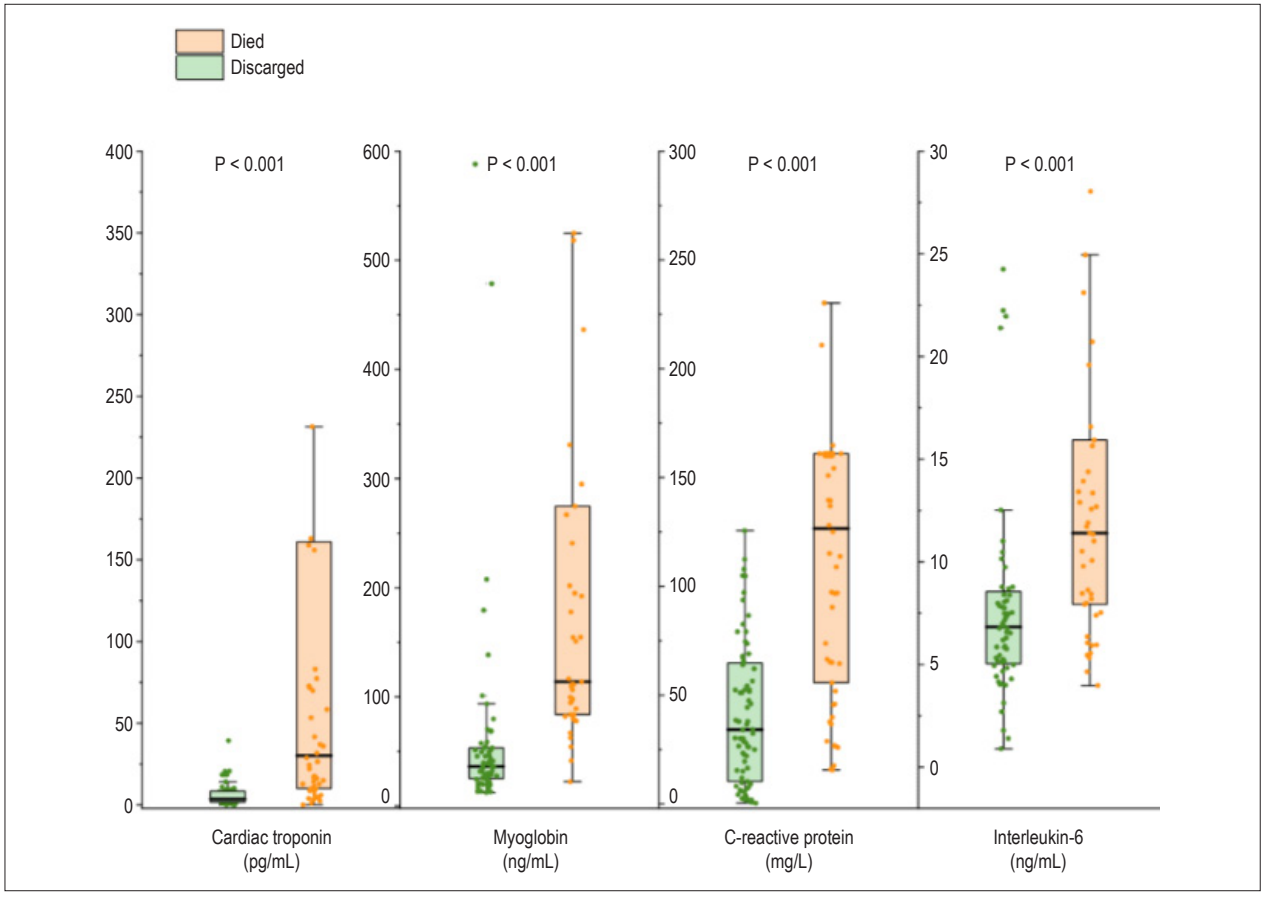

Figure 1 – *Main laboratory parameters in confirmed cases of COVID-19 by outcome.*[1]

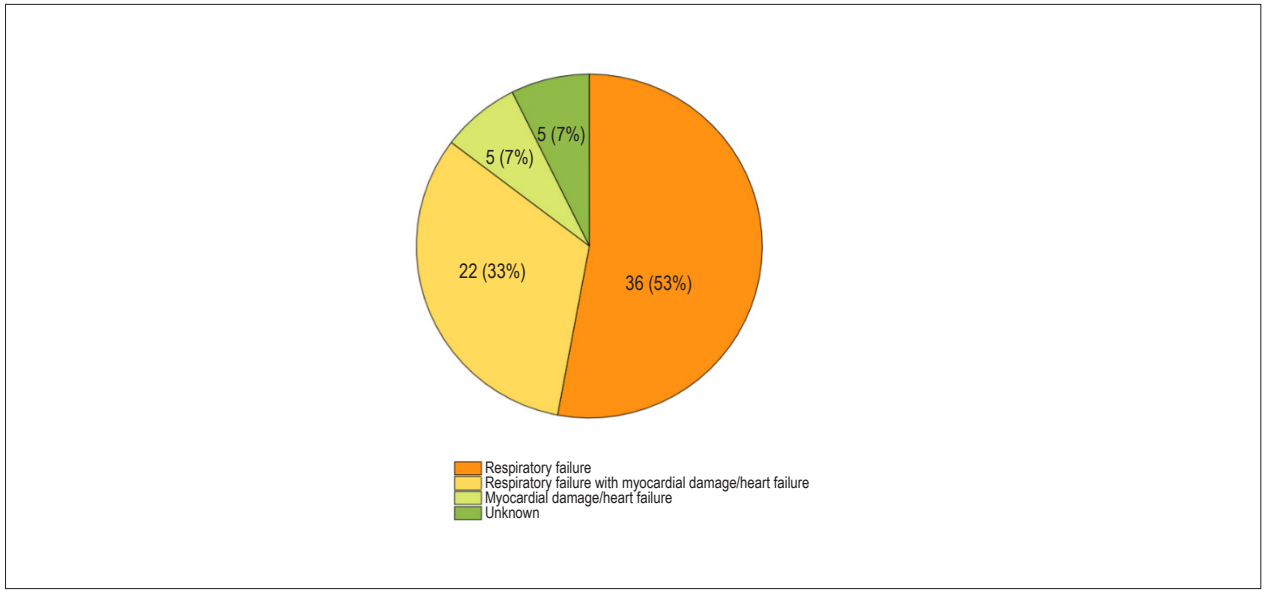

Figure 2 – *Summary of causes of death in 68 patients with confirmed COVID-19.*[1]

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