

Covid-19 Learning in Cardiovascular Diseases

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Heart failure is a growing public health issue. As 1 out of 5 persons is expected to develop heart failure during their lifetime. In the world, there are 63 million people that suffer heart failure. This event would cost 31 trillion dollars in 2012 and probably 70 trillion in 2030 [1].

The viral infection that started at the city of Wuhan-China, COVID 19 beta Coronavirus, is similar to COVID 1 and Mers of 2002. The virus produced a flu-like state with a particular connotation with loss of smell and taste [2].

COVID 19 cases were divided into asymptomatic, symptomatic patients with moderate and symptomatic patients with severe respiratory failure. The patient with severe respiratory failure was hypoxemic even with mechanical ventilation prompted the physicians to think that something else was happening. The Italian doctors reported high mortality in vulnerable people, elderly, diabetics, hypertensive, children, pregnant women [3]. When autopsies were performed, and they were surprised that the examined patients had venous thrombosis in the lower limbs and pulmonary embolism [4]. It was suggested then that interstitial pneumonia reported by the Chinese had to be combined with another event. Parameters such as dimer D, ferritin, BNP, troponin, PRO BNP, inflammatory cytokines, especially interleukin 2 and 6 were studied [5].

What did we learn?

That the beta coronavirus generated a high macrophage uncontrolled inflammatory response producing a massive amount of proinflammatory cytokines (cytokine storm) that cause injuries to the lung, heart, kidneys and brain and induce the high hypercoagulability described in these patients [6].

It has been suggested that ACE 2 inhibitor antihypertensive drugs and ARA 2 may increase infectivity, which is why several physicians proposed that the treatment protocol should be changed in order to stop inflammation and block intravascular coagulation [7].

Italian observational studies carried out in Lombardy (Italy) suggested that the antihypertensive drug treatment maintained in general; however, each patient should be carefully screened for individualized therapy. For example, patients over 60 years of age with severe respiratory failure, with HT, coronary heart disease and diabetes with positive COVID, must go to ICU and continue with the treatment of ACEI and anti-ARA 2. This approach agrees with what was previously described by cardiovascular societies and observational studies, despite the theoretical basis that it could increase infectivity [8].

What happens when the heart fails?

The heart fails because cardiomyocyte injury is caused by a massive, uncontrolled, inflammatory cytokine secretion (cytokine storm) generated upon COVID 19 infection. Cardiomyocyte injury is detected by the increase in serum levels of troponin, BNP and PRO BNP, markers of fibrosis and heart failure. There are several potential outcomes: hypotension, viral myocarditis, microvascular ischemia, hypoxia, type 2 myocardial infarction, and coagulopathy.

When does this happen?

Depending on the incubation time, it can be a week or two weeks; it is not known exactly. We do know is that the new treatments for heart failure Sacubitril (neprisinil) Vericiguat, and the new “inotropes” (calcitropes, mitotropes, mymiotropes) could change the spectrum of myocardial damage [9].

In the case of generalized thromboembolism, low molecular weight heparin and the new non-vitamin k-dependent anticoagulants, like Rivaroxaban, are recommended. For pulmonary hypertension, we have drugs such as Macitentan, Ambrisentan, Riociguat, selexipag [10].

In the treatment of this pandemic, it is necessary to reason what happened with COVID 1, Ebola, HIV and Mers, and classify each one of the symptomatic, asymptomatic patients with positive COVID with respiratory failure, heart failure, etc. The initial treatments proposed were chloroquine and hydroxychloroquine with azithromycin despite the reported side effects. In treated hospitalized patients, Q-T interval, which is the ventricular repolarization, can be lengthened and it may cause fatal arrhythmias [11].

Lopinavir and Ritonavir, protease inhibitors, have emerged from different groups as possible treatments, but the responses have not optimal. Remdesivir, which is an inhibitor of RNA-polymerase, has just been approved for treatment. Other immunomodulatory drugs have been used, steroids and interleukin blockers (tocilizumab) with success [12]. Convalescent plasma with COVID 19 has also proven to be useful in some patients [12].

With the advent of portable ultrasound, the “new stethoscopes” currently called “sonoscopes”, we can decipher how the heart works, know the ejection fraction, the pressure of the pulmonary artery, the area of fibrosis (strain) and it also works as a tissue Doppler. That is, we can perform noninvasive cardiac hemodynamic studies that allow us to make fast decisions in these complicated patients. The lung can also be evaluated with the same portable instrument and decipher the dry and mixed wet patterns, and consolidation of the lung [13]. All these advances have incorporated portable low-radiation tomographs and latest-generation fans.

To conclude, there is a risk of vulnerability in the patient with previous heart disease, heart failure, hypertension and risk factors.

Each patient should be informed and individualized according to the recommended evidence-based guidelines. Insist on maintaining cardiovascular health, reducing risk factors, maintaining medications and their doses, not self-medicating and according to each case. You can consult by telemedicine for orientation treatment at home or if you are very symptomatic, go to the emergency service.

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