CARDIAC INDICATION OF CORONAVIRUS (COVID-19): A REVIEW

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Abstract: Intense respiratory disorder Covid 2 (SARS-CoV-2) or COVID-19 contamination is the reason for the continuous worldwide pandemic. Passing’s from COVID-19 contaminations are extremely high in patients with cardiovascular infection. Moreover, COVID-19 patients with previous cardiovascular comorbidities are at more serious danger of death. The super cardiovascular intricacies of COVID-19 are myocardial dead tissue, myocarditis, intense myocardial localized necrosis, arrhythmias, cardiovascular breakdown, stroke, and venous thromboembolism. Clinical intercessions dependent on COVID-19 medications have numerous unfavorable cardiovascular impacts. Here, we audit the adequacy of medication related treatments and symptoms of COVID-19 medications.

Index terms - Covid-19, SARS CoV-2, Cardiovascular complication, myocardial infraction, Arrhythmias

I. Introduction

Covid 2019 (COVID-19) is a flow worldwide, arising, pestilence illness brought about by SARS-CoV-2 disease. SARS-CoV-2 is a sort of RNA infection with a trademark envelope and a direct single strand genome, which is unique in relation to intense respiratory condition (SARS) and Middle Eastern respiratory sickness (MERS) disease and hereditary mutation. Coronaviruses are a huge group of innocuous, covered RNA infections, which can influence numerous types of creatures and people. Human Covid can be arranged by their pathogenicity. Types with high pathogenicity incorporate SARS-CoV, MERS-CoV, and the current adaptation of SARS-CoV2. (1) Chronic respiratory infection Covid 2 (SARS-CoV-2) has prompted the most noticeably awful pandemic of this century with Coronavirus 2019 (COVID-19). Initially known as respiratory framework infection, COVID-19 has been found to intervene and influence the cardiovascular framework prompting myocardial harm and cardiovascular and endothelial brokenness particularly with the Angiotensin-changing over compound 2 (ACE-2) receptor. (2) (3) (4) (5) truth be told, heart harm has been noted even external the clinical highlights of respiratory sickness. Then again, respiratory indications are more terrible in patients impacted by COVID-19 with prior coronary illness; notwithstanding, new cardiovascular breakdown is normal in this subset. Indeed, heart harm has been noted even external the clinical highlights of respiratory sickness. Then again, respiratory side effects are more awful in patients impacted by COVID-19 with prior coronary illness; notwithstanding, new cardiovascular breakdown is normal in this clasp. (6) (7) (8) (9)

II. Etiology

In December 2019, instances of uncommon pneumonia were found in Wuhan, China. The original SARS-CoV2 infection has been distinguished as the reason for the new Corona 2019 illness (COVID-19). (2)
III. Epidemiology

3.1 China

Inclusion of heart conditions was noted toward the start of the pandemic in reports from China. An examination of 187 patients treated at Wuhan Hospital between January 23 and February 23, 2020, found that 35% had existing heart issues, for example, hypertension, coronary illness, and cardiomyopathy, and 28% showed myocardial injury demonstrating significant degrees of troponin T. (9). Other Chinese reports observed that the standard degrees of cardiovascular sickness range from 5% to 16%, hypertension from 15% to 31%, cardiovascular infection 11%, and diabetes by 10%. (10)

![Figure 1: A) Proportion of the Chinese patients and their comorbid conditions that were diagnosed with COVID-19. (B) Proportion of the Chinese patients and their comorbid conditions that died from COVID-19](image1)

3.2 World Scenario

Past China, exceptionally high paces of these illnesses have been accounted for. A progression of review cases from Italy detailed outcomes from 1,591 basically sick patients with COVID-19 conceded to the emergency unit: (49% of patients had hypertension, 21% had coronary illness, and 17% % had diabetes. (11) In a New York study between March 2 and April 1, 2020, 1150 grown-ups with COVID-19 were conceded to two clinics; 257 were truly sick. Of these, 82% had something like one constant ailment, most normally hypertension (63%), diabetes (36%), weight (46%), and coronary illness (19%). (12) In a progression of high-hazard instances of 5700 COVID-19 patients conceded to 12 New York clinics; the predominance of hypertension, diabetes, and coronary conduit illness was 57%, 34%, and 11%, separately. (13)

![Figure 2: C) Proportion of the global patients and their comorbid conditions that were diagnosed with COVID-19. (D) Proportion of the global patients and their comorbid conditions that died from COVID-19. All the four figures indicate that the major complications of COVID-19 are the cardiovascular related diseases.](image2)
4.1 ACE2 Receiver

SARS-CoV-2 uses its S-spark to tie to ACE2 receptors as a cell section point. These ACE2 receptors are communicated in type 1 and type 2 pneumocytes and other cell types, including endothelial cells. ACE2 is the contrary controller of the renin-angiotensin-aldosterone framework. Like other Covids, SARS-CoV-2 uses these ACE2 receptors to target explicit respiratory frameworks. (2)(14)(15)

4.2 SARS-CoV-2 and Immune Response

There are two periods of the invulnerable reaction to COVID-19. Stage 1 happens during the hatching period of the illness, in which the invulnerable framework attempts to destroy the infection; in case there is a particular imperfection in this stage, SARS-CoV-2 will spread and cause harm, with serious harm to organs with significant degrees of ACE2 receptors, including lungs, endothelial cells, heart and kidneys. This significant harm prompts stage 2: extreme irritation of the impacted organs. (16)

Diabetes, atherosclerosis, and corpulence, which add to the danger of cardiovascular sickness, debilitate the invulnerable framework. This is related to a negative expectation on COVID-19. (17)

4.3 Cardiovascular Injury Methods in COVID-19

A few sorts of heart harm have been proposed, in light of past investigations of the SARS and MERS scourge and the continuous COVID-19 plague. Part of the reaction to fundamental irritation in serious COVID-19 is the arrival of significant degrees of cytokines (known as cytokine discharge condition) that can harm many tissues, including the vascular endothelium and heart myocyte. (14)(15)(18)(19)

4.4 Cytokine Release Syndrome

Cytokine discharge condition happens in patients with extreme COVID-19 contamination. Numerous proinflammatory cytokines are exceptionally high in extreme cases, including interleukin (IL) - 2, IL-10, IL-6, IL-8, and cancer corruption factor (TNF) - α. (16)(18)

Cytokines assume a significant part during contamination (stage 1) and during intense persistent aggravation (stage 2), prompting serious respiratory sorrow (ARDS) and opposite end-stage wounds. (16)(20)

4.5 Direct Injury of Myocardial Cells

SARS-CoV-2 collaborations with ACE2 can cause changes in ACE2 pathways, prompting serious harm to lung, heart, and endothelial cells. Few case reports have shown that SARS-CoV2 might be straightforwardly tainted in the myocardium, causing viral myocarditis. Nonetheless, much of the time, myocardial injury has been demonstrated to be expected to an expanded cardiometabolic necessity related to fundamental contamination and moderate hypoxia brought about by pneumonia or ARDS. (18)

4.6 Intense Coronary Syndrome

The crack of the plaque prompting oak seed coronary condition can bring about foundational irritation and expanded catecholamine levels from the infection. (18)(21) Coronary apoplexy has likewise been recognized as a potential reason for intense coronary disorder in patients with COVID-19. (22)

4.7 Other Possible Ways

Certain meds like corticosteroids, antimicrobials, and immunological specialists might have antagonistic cardio poisonous impacts. Electrolyte issues can happen in any genuine foundational sickness and cause arrhythmias, in which patients with lower coronary illness are at more serious danger. There is some worry about hypokalemia in patients with COVID-19, given the connection of SARS-CoV-2 with the renin-angiotensin-aldosterone framework. Hypokalemia is most popular for expanding the danger of different kinds of arrhythmia. (9)(18)
Table 5.1: Adverse Cardiovascular effects of potential drugs to treat Covid-19

<table>
<thead>
<tr>
<th>DRUG</th>
<th>MECHANISM</th>
<th>ADVERSE EFFECTS</th>
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<tbody>
<tr>
<td>Lopinavir/Ritonavir</td>
<td>Protease inhibitor, inhibits 3CLpro</td>
<td>Hypertension</td>
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<td>prolonged P-R and Q-T interval</td>
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<td></td>
<td></td>
<td>Severe conduction disorder</td>
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<tr>
<td>Remdesivir</td>
<td>Protease inhibitor, inhibits SARS-CoV-2RNA dependent RNA polymerase</td>
<td>Hypotension</td>
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<td>Bradycardia</td>
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<td>QTc prolongation</td>
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<tr>
<td>Interferon alpha</td>
<td>Protease inhibitor, viral load reduction through inhibition of replication</td>
<td>Ischemic</td>
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<td>Cardiomyopathy</td>
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<td>Arrhythmias</td>
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<td>Hypertension</td>
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<td>Hypotension</td>
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<td>Ribavirin</td>
<td>Viral load reduction through inhibition of replication</td>
<td>Bradycardia</td>
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<td>Cardiac dysfunction</td>
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<td>Anemia</td>
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<td>Hypomagnesaemia</td>
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<td></td>
<td>Mitochondrial toxicity</td>
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<tr>
<td>Drug</td>
<td>Function Description</td>
<td>Side Effects</td>
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</tr>
<tr>
<td>Chloroquine</td>
<td>Viral load reduction through inhibition of replication</td>
<td>Myocardial suppression, irreversible cardiomyopathy, Atrioventricular Block, Hypokalemia, Hypotension</td>
</tr>
<tr>
<td>Baricitinib</td>
<td>Protease inhibitor, inhibits Janus kinase</td>
<td>Hyperglycemia, Infections, Thromboembolic events</td>
</tr>
<tr>
<td>Arbidol</td>
<td>Protease inhibitor, inhibits glycoprotein</td>
<td>Nausea, Diarrhea, Dizziness, Bradycardia</td>
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<tr>
<td>Dexamethosoe</td>
<td>Combat Cytokine storm by limiting the production of damaging effect of the cytokines</td>
<td>Arrhythmias, Headache, Agitation, Dizziness, Increased appetite</td>
</tr>
<tr>
<td>Tocilizumab</td>
<td>Inhibits the activity of IL-6 receptor, blocks the cytokine strom, caused by IL-6 pathway</td>
<td>Cardiomyopathy, Liver injury</td>
</tr>
<tr>
<td>Fapiravir</td>
<td>Inhibiting RNA synthetase</td>
<td>Hypotension, Arrhythmia</td>
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VI. Case study

Figure 4 – Cardiac MRI in suspected acute covid 19 myocarditis

Figure 5. Column graphs with individual plotted values show distribution of quantitative myocardial MRI parameters in healthy participants and in participants with suspected acute non-COVID-19 and COVID-19 myocarditis. (51)
We identified an example of myocardial edema that spread to members with manifestations of COVID-19 disease and suspected myocarditis. Myocardial edema influences myocardial capacity and might be a sign of broad irritation because of the insoluble framework, direct myocardial injury of SARS-CoV-2, or vascular spillage because of endothelial harm. Strangely, the quantity of LGE injuries, as an indication of myocyte rot, was lower in members with COVID-19 myocarditis contrasted and members with intense non-COVID-19 myocarditis, which is basically brought about via cardio tropic infections. This outcome proposes that the path mechanism of myocardial localized necrosis might be unique in relation to SARS-CoV-2 and is logical depending on the connection of a wide assortment of the cardiovascular framework. Additionally, we noticed examples of stress-incited cardiomyopathy as an option in contrast to myocardial injury in members with COVID-19. Since the event of myocardial injury and the qualities of stress-instigated cardiomyopathy are related to the deadly result of COVID-19, the checked changes in cardiovascular changes are especially intriguing.

VII. Treatment / Management

5.1 Complete Cardiac Care for Patients COVID-19

Cardiac care needs to be developed for COVID-19 patients for the purpose of early detection and management of cardiovascular disease for the purpose of diagnostic and preventive measures to prevent or reduce COVID-19 exposure. As Bonow et al. to put it bluntly, the message to patients should be clear that urgent care should be sought if there are warning signs of a heart attack. Wearing a mask, walking away from the body is still as important as ever. At the same time, doctors and researchers are finding the best procedures for COVID-19-related cardiovascular disease.

5.2 ACE inhibitors (ACEI) / Angiotensin Receptor Blockers (ARB)

In early 2020, controversy arose over the safe use of these drugs for COVID-19 patients. The current consensus is in line with the continued use of these drugs. The BRACE CORONA trial was introduced at ESC Congress 2020, which found no significant difference in the number of days of life and hospitalization out of 30 days between subjects receiving continuous ACEI / ARB and hospitalization due to COVID-19 compared and those who had been on medication for some time. Paused.

5.3 Remdesivir

Remdesivir is an antiretroviral drug (RNA polymerase inhibitor) used to treat COVID-19. A multicenter, double-blind RCT from China studied Remdesivir for adult patients hospitalized due to severe COVID-19. Studies have shown a decrease in clinical improvement in the treatment group compared with controls, but the difference was not significant. The subsequent case, sponsored by the US National Institute of Allergy and Infectious Disease, examined 1063 patients in COVID-19 hospitals in a retrospective study. Results published in the New England Journal of Medicine showed a quick recovery time for patients receiving remdesivir vs. placebo.

The US Food and Drug Administration (FDA) have approved the approval of emergency use of remdesivir to all hospitalized patients with COVID-19. To date, no cardiovascular side effects have been reported with Remdesivir, although this may be reflected in future use during the COVID-19 epidemic.

5.4 Hydroxychloroquine and Chloroquine

Hydroxychloroquine was introduced as a potential treatment for COVID-19 patients based on an open label, a single-group study from France. However, clinical trials of COVID-19 patients in hospitals conducted at a major New York City medical center found no significant benefit from hydroxychloroquine. The use of hydroxychloroquine alone or hydroxychloroquine and azithromycin did not improve the combined result of intubation or death.

Chloroquine has been known to cause atrioventricular block and long QTc blocks, especially when combined with azithromycin. The lack of benefits observed in clinical trials and the potential for adverse cardiac outcomes have led the FDA to withdraw its urgent approval of the use of hydroxychloroquine and chloroquine in patients on COVID-19.

5.5 Azithromycin

Azithromycin was commonly used in combination with hydroxychloroquine as an early treatment for the epidemic. However, a few studies of this combination did not show any clinical benefit. Azithromycin is a macrolide and is known to prolong the QTc interval. Combining azithromycin with chloroquine or hydroxychloroquine increases QTc expansion and potentially increases the risk of torsade de points. Lack of clinical benefit and strength of cardiac arrhythmias have discouraged clinicians from using azithromycin in COVID-19.
5.6 Vaccination

The SARS-CoV-2 genetic sequence was published in January 2020, and since then, teams of researchers around the world have been working hard to develop a vaccine for the SARS-CoV-2 vaccine. More than 90 vaccines are being developed at this time. (43) (44) Vaccination has begun in various countries. MRNA-based vaccines developed by Pfizer and Moderna have been approved for emergency use (EUA) by the US Food and Drug Administration (FDA). Most health workers have already received these vaccines.

Immune thrombotic thrombocytopenia developed after the ChAdOx1nCov-19 vaccine was reported as a rare complication that arose in a few cases. Method of thrombocytopenia similar to heparin-induced thrombocytopenia. (45)

No significant heart problems have been reported with any of the vaccines so far.

VIII. Prognosis

The majority of patients (80%) will develop a mild form of the disease. A serious case of the disease occurs in approximately 15 million patients who need hospitalization and the most serious form occurs in about 5% of patients who need intensive care. The present mortality rate is between 2% to 5% of all patients with COVID-19 but is much higher in patients who require less oxygen intake. The leading cause of death in COVID-19 is severe respiratory depression (ARDS); however, there are other important aspects of organ involvement, including the cardiovascular system and shock. (2) (14) (15) The presence of chronic heart disease or heart involvement leads to a higher mortality rate compared to patients without heart disease. (6) (46)

In the COVID-19 epidemic, very few heart patients have been to clinics or hospitals for fear of contracting the virus. In contrast, heart patients despite the fact that SARS-CoV-2 infection requires immediate diagnosis and management, especially with this disease as resources and staff is often at risk. (47) (48)

The predicted value of cardiovascular disease was fully demonstrated in a group of 191 patients where 50% had high blood pressure and made up 48% of underweight people, while 8% had cardiovascular disease and accounted for 13% of underweight people. (24) In a report on 44672 confirmed COVID-19 cases from the China Center for Disease Control and Prevention, the mortality rate was 2.3% for the whole group but significantly higher in patients with hypertension (6%), diabetes (7%), or heart disease (11%). (49)

IX. Improving the Outcomes of the Health Care Team

SARS-CoV-2 infection has been reported to be complicated by heart failure. Existing heart disease puts the disease at risk and increases the risk of heart disease. Early detection of cardiovascular disease by clinical and laboratory parameters and treatment is very important. Clinical trials are ongoing, and more research will follow to diagnose and treat the disease, which will guide physicians in treatment and policymakers to set guidelines for better management of COVID-19 cardiovascular features.

X. References


