

World Journal of *Cardiology*

World J Cardiol 2021 August 26; 13(8): 237-380



EDITORIAL

- 237 ISCHEMIA trial: How to apply the results to clinical practice
Vidal-Perez R, Bouzas-Mosquera A, Peteiro J, Vazquez-Rodriguez JM

ACADEMIC ACTIVITY REPORT

- 243 Shortened dual antiplatelet therapy in contemporary percutaneous coronary intervention era
Han J, Attar N

EVIDENCE REVIEW

- 254 Multimodality imaging in the diagnosis and management of prosthetic valve endocarditis: A contemporary narrative review
Lo Presti S, Elajami TK, Zmaili M, Reyalde R, Xu B

REVIEW

- 271 Potential role of an athlete-focused echocardiogram in sports eligibility
Palermi S, Serio A, Vecchiato M, Sirico F, Gambardella F, Ricci F, Iodice F, Radmilovic J, Russo V, D'Andrea A

MINIREVIEWS

- 298 In-depth review of cardiopulmonary support in COVID-19 patients with heart failure
Raffaello WM, Huang I, Budi Siswanto B, Pranata R
- 309 Surgical strategies for severely atherosclerotic (porcelain) aorta during coronary artery bypass grafting
Sirin G
- 325 Angiotensin receptor blocker neprilysin inhibitors
Usuda D, Higashikawa T, Hotchi Y, Usami K, Shimosawa S, Tokunaga S, Osugi I, Katou R, Ito S, Yoshizawa T, Asako S, Mishima K, Kondo A, Mizuno K, Takami H, Komatsu T, Oba J, Nomura T, Sugita M

ORIGINAL ARTICLE**Case Control Study**

- 340 Association of marital status with takotsubo syndrome (broken heart syndrome) among adults in the United States
Appiah D, Farias R, Helo D, Appiah L, Olokede OA, Nwabuo CC, Nair N

Clinical Trials Study

- 348 Nutritional supplement drink reduces inflammation and postoperative depression in patients after off-pump coronary artery bypass surgery
Matsushita S, Shimada A, Yamamoto T, Minematsu K, Inaba H, Kuwaki K, Amano A

Observational Study

- 361 Role of coronary angiogram before transcatheter aortic valve implantation
Beska B, Manoharan D, Mohammed A, Das R, Edwards R, Zaman A, Alkhalil M
- 372 Associations of new-onset atrial fibrillation and severe visual impairment in type 2 diabetes: A multicenter nationwide study
Kaewput W, Thongprayoon C, Rangsri R, Bathini T, Mao MA, Cheungpasitporn W

ABOUT COVER

Editorial Board Member of *World Journal of Cardiology*, Alexander E Berezin, MD, MSc, PhD, Professor, Department of Internal Medicine, Zaporozhye State Medical University, Zaporozhye 69035, Ukraine. aeberezin@gmail.com

AIMS AND SCOPE

The primary aim of *World Journal of Cardiology (WJC, World J Cardiol)* is to provide scholars and readers from various fields of cardiology with a platform to publish high-quality basic and clinical research articles and communicate their research findings online.

WJC mainly publishes articles reporting research results and findings obtained in the field of cardiology and covering a wide range of topics including acute coronary syndromes, aneurysm, angina, arrhythmias, atherosclerosis, atrial fibrillation, cardiomyopathy, congenital heart disease, coronary artery disease, heart failure, hypertension, imaging, infection, myocardial infarction, pathology, peripheral vessels, public health, Raynaud's syndrome, stroke, thrombosis, and valvular disease.

INDEXING/ABSTRACTING

The WJC is now abstracted and indexed in Emerging Sources Citation Index (Web of Science), PubMed, PubMed Central, Scopus, China National Knowledge Infrastructure (CNKI), China Science and Technology Journal Database (CSTJ), and Superstar Journals Database. The 2021 edition of Journal Citation Reports® cites the 2020 Journal Citation Indicator (JCI) for WJC as 0.36. The WJC's CiteScore for 2020 is 0.3, and Scopus CiteScore rank 2020: Cardiology and Cardiovascular Medicine is 289/317.

RESPONSIBLE EDITORS FOR THIS ISSUE

Production Editor: Lin-YuTong Wang; Production Department Director: Xiang Li; Editorial Office Director: Ya-Juan Ma.

NAME OF JOURNAL

World Journal of Cardiology

ISSN

ISSN 1949-8462 (online)

LAUNCH DATE

December 31, 2009

FREQUENCY

Monthly

EDITORS-IN-CHIEF

Ramdas G Pai, Dimitrios Tousoulis, Marco Matteo Ciccone

EDITORIAL BOARD MEMBERS

<https://www.wjnet.com/1949-8462/editorialboard.htm>

PUBLICATION DATE

August 26, 2021

COPYRIGHT

© 2021 Baishideng Publishing Group Inc

INSTRUCTIONS TO AUTHORS

<https://www.wjnet.com/bpg/gerinfo/204>

GUIDELINES FOR ETHICS DOCUMENTS

<https://www.wjnet.com/bpg/GerInfo/287>

GUIDELINES FOR NON-NATIVE SPEAKERS OF ENGLISH

<https://www.wjnet.com/bpg/gerinfo/240>

PUBLICATION ETHICS

<https://www.wjnet.com/bpg/GerInfo/288>

PUBLICATION MISCONDUCT

<https://www.wjnet.com/bpg/gerinfo/208>

ARTICLE PROCESSING CHARGE

<https://www.wjnet.com/bpg/gerinfo/242>

STEPS FOR SUBMITTING MANUSCRIPTS

<https://www.wjnet.com/bpg/GerInfo/239>

ONLINE SUBMISSION

<https://www.f6publishing.com>

In-depth review of cardiopulmonary support in COVID-19 patients with heart failure

Wilson Matthew Raffaello, Ian Huang, Bambang Budi Siswanto, Raymond Pranata

ORCID number: Wilson Matthew Raffaello 0000-0002-4086-1492; Ian Huang 0000-0003-1189-8453; Bambang Budi Siswanto 0000-0003-3998-1590; Raymond Pranata 0000-0003-3998-6551.

Author contributions: Raffaello WM contributed in literature reviewing, manuscript writing and editing; Huang I, Siswanto BB, and Pranata R contributed in reviewing and performing extensive editing of the manuscript.

Conflict-of-interest statement: There is no conflict of interest associated with the senior author or other coauthors contributed their efforts in this manuscript.

Open-Access: This article is an open-access article that was selected by an in-house editor and fully peer-reviewed by external reviewers. It is distributed in accordance with the Creative Commons Attribution NonCommercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: <http://creativecommons.org/licenses/by-nc/4.0/>

Manuscript source: Invited

Wilson Matthew Raffaello, Raymond Pranata, Faculty of Medicine, Universitas Pelita Harapan, Tangerang 15810, Indonesia

Ian Huang, Department of Internal Medicine, Faculty of Medicine, Universitas Padjadjaran, Bandung 40161, Indonesia

Bambang Budi Siswanto, Department of Cardiology and Vascular Medicine, National Cardiovascular Center Harapan Kita, Universitas Indonesia, Jakarta 11420, Indonesia

Corresponding author: Raymond Pranata, MD, Doctor, Faculty of Medicine, Universitas Pelita Harapan, Jl. Jend. Sudirman No.20, Bencong, Kec. Klp. Dua, Tangerang 15810, Indonesia. raymond_pranata@hotmail.com

Abstract

Coronavirus disease 2019 infection has spread worldwide and causing massive burden to our healthcare system. Recent studies show multiorgan involvement during infection, with direct insult to the heart. Worsening of the heart function serves as a predictor of an adverse outcome. This finding raises a particular concern in high risk population, such as those with history of preexisting heart failure with or without implantable device. Lower baseline and different clinical characteristic might raise some challenge in managing either exacerbation or new onset heart failure that might occur as a consequence of the infection. A close look of the inflammatory markers gives an invaluable clue in managing this condition. Rapid deterioration might occur anytime in this setting and the need of cardiopulmonary support seems inevitable. However, the use of cardiopulmonary support in this patient is not without risk. Severe inflammatory response triggered by the infection in combination with the preexisting condition of the worsening heart and implantable device might cause a hypercoagulability state that should not be overlooked. Moreover, careful selection and consideration have to be met before selecting cardiopulmonary support as a last resort due to limited resource and personnel. By knowing the nature of the disease, the interaction between the inflammatory response and different baseline profile in heart failure patient might help clinician to salvage and preserve the remaining function of the heart.

Key Words: COVID-19; Heart failure; Cardiopulmonary support; Extracorporeal membranous oxygenation; Ventricular assist device; Coagulopathy

©The Author(s) 2021. Published by Baishideng Publishing Group Inc. All rights reserved.

manuscript

Specialty type: Cardiac and cardiovascular systems**Country/Territory of origin:**
Indonesia**Peer-review report's scientific quality classification**

Grade A (Excellent): 0

Grade B (Very good): B

Grade C (Good): C

Grade D (Fair): 0

Grade E (Poor): 0

Received: March 2, 2021**Peer-review started:** March 3, 2021**First decision:** March 31, 2021**Revised:** April 13, 2021**Accepted:** August 5, 2021**Article in press:** August 5, 2021**Published online:** August 26, 2021**P-Reviewer:** Wang MK**S-Editor:** Ma YJ**L-Editor:** A**P-Editor:** Liu JH

Core Tip: Coronavirus disease 2019 (COVID-19) infection might cause severe respiratory distress and demonstrates an extrapulmonary involvement. Recent evidence shows direct involvement of COVID-19 and deterioration of the heart function. Severe infection is commonly found in high risk population, indicates a complex interaction between host inflammatory response and the infection itself, signifies the use of cardiopulmonary support and associated with high mortality. There are relatively scarce information regarding the use of ventricular assist device and extracorporeal membrane oxygenation and here we will be discussing the possible mechanism of how cardiopulmonary support may improve COVID-19 infection outcome.

Citation: Raffaello WM, Huang I, Budi Siswanto B, Pranata R. In-depth review of cardiopulmonary support in COVID-19 patients with heart failure. *World J Cardiol* 2021; 13(8): 298-308

URL: <https://www.wjgnet.com/1949-8462/full/v13/i8/298.htm>

DOI: <https://dx.doi.org/10.4330/wjc.v13.i8.298>

INTRODUCTION

Coronavirus disease 2019 (COVID-19) is an emerging viral infection which has caused global pandemic with resulting both high global economic burden and mortality rate [1,2]. It also caused an alteration and restructuration in our healthcare system, especially in treating patients with infection and chronic disease[3]. Patients with both condition of COVID-19 and prior cardiovascular disease have an increased risk of cardiovascular complications which severely affect the mortality rate.

COVID-19 is also associated with higher incidence of cardiovascular complication in compare to previous coronavirus outbreaks[4,5]. Deterioration of the cardiac function is prominent in COVID-19 and those with lower baseline function are prone to further decline in cardiac function. A study demonstrate the preexistence of chronic heart failure (CHF) and high cardiac biomarkers is associated with worse outcome[6]. Recent study also shows that patient with heart failure (HF) is associated with an increased risk of mechanical ventilation and overall mortality regardless of left ventricular ejection fraction (LVEF)[7].

The use of cardiopulmonary support in COVID-19 shares the same prominent role in managing severe conditions such as severe respiratory distress and heart failure[3, 8]. However, there are several distinct clinical characteristics of HF patients which may differ from non-pre-existing HF patients. These factors have to be considered before choosing cardiopulmonary support as a treatment of choice. Another challenging issue is the highly selective inclusion and exclusion criteria before a patient is eligible for the use of extracorporeal membranous oxygenation (ECMO). Therefore, the management of both HF and COVID-19 has to be tailored since the concept of one treatment fits all might not suitable in these patients.

HEART FAILURE AND COVID-19

The mechanism of cardiac function disturbance in COVID-19 is poorly understood and it is thought to be an interaction between several mechanisms including direct myocardial injury, cytokine release, prothrombotic state causing microvascular thrombosis and exacerbation of underlying cardiovascular disease[3,9-13]. In the context of COVID-19, CHF patients are vulnerable to acute exacerbation. These patients are at risk because of their lower baseline status which unable to cope with the increasing metabolic demand in systemic inflammation triggered by the infection[14]. In the settings of advanced HF with left ventricular assisted device (LVAD) support, functional capacity of the lung has already impaired and contributes even more to the decrease of the cardiopulmonary reserve[15,16].

Several studies have demonstrated more severe infections and higher mortality rate in patients with preexisting cardiovascular disease[6,7,14,17]. Older age along with other comorbid such as obesity, hypertension, diabetes mellitus, chronic obstructive pulmonary disease, atrial fibrillation, chronic kidney disease and frailty are seems to

be more prevalent in the HF group and might contribute to an increased rate of mortality and morbidity[7,18]. Moreover, these patients generally have a reduced immunity, general frailty, and tend to be in an inflammatory state[14,19].

A lower pulmonary function is commonly found in HF patients and is contributed mainly by chronic obstructive pulmonary disease (COPD)[20]. Combination of underlying parenchymal disease and elevated left ventricular filling pressure leads to the development of pulmonary hypertension[21]. In COVID-19 infection, respiratory failure and acute respiratory distress syndrome (ARDS) further exacerbates pulmonary vasoconstriction and interstitial edema. This condition is further worsened by pre-existing biventricular failure and ARDS that eventually lead to right ventricular function impairment.

Recent studies have shown that COVID-19 is associated with acute myocardial involvement which is described as an acute cardiac injury[3,12,13]. A suspicion of cardiac involvement in COVID-19 is best described by an elevation in cardiac biomarker [high-sensitivity troponin (hs-TnI)] above 99th percentile upper reference limit[13,22]. Early reports had been analyzed, with notable findings suggest an increased level of cardiac troponin was associated with admission to intensive care and higher in hospital mortality[12,23-26]. A careful observation of endomyocardial biopsy in COVID-19 patients has revealed an evidence of acute myocardial inflammation through the presence of viral particles and a diffuse myocardial edema on cardiac magnetic resonance, therefore raising the suspicion of direct viral myocardial invasion[27]. Severe acute respiratory distress syndrome coronavirus 2 (SARS-CoV2) binds with angiotensin-converting enzyme 2 (ACE2) receptor and with the help of transmembrane protease serine 2 (TMPRSS2), facilitates viral entry through the cell[28, 29]. ACE2 and TMPRSS2 are widely expressed in various tissue, including the heart, and might explain the involvement of heart during the course of infection[29]. Viral inclusion bodies were found from the biopsy of myocardial tissue along with identification of SARS-CoV2 genomic RNA in patients with suspected COVID-19 myocarditis, therefore raising the possibility of direct viral invasion to the myocardium[30-33]. Although there was a high viral load which is associated with higher proinflammatory cytokine expression in the cardiac tissue, these findings were not accompanied with an elevated inflammatory cell infiltrates[34]. The exact proportion of myocarditis is still hard to be determined, mainly because of the lack of definitive diagnostic procedure done in the patients[13,27]. However, myocarditis is important to be considered as it may cause abnormal electrical conductance in the myocardium[10].

Myocardial infarction plays a significant role in the development of acute HF in COVID-19 infection. Both Type I and II myocardial infarction might occur in patient with COVID-19, worsening the function in an already impaired baseline function[3]. This may lead to worse outcome in patient with a history of HF[3].

Cardiac arrhythmia is a major concerning issue in the context of HF and COVID-19 infection. Careful observation of the heart rate and rhythm are vitals in the clinical settings. Atrioventricular block, atrial fibrillation, polymorphic ventricular tachycardia and pulseless electrical activity have been closely associated with COVID-19 despite of the unknown mechanism of how these rhythm abnormalities may develop[22,35]. QT prolongation is constantly a threat, considering that QT prolongation is an independent risk factor of adverse outcome in advanced heart failure[36]. Even though the use of hydroxychloroquine and azithromycin is no longer recommended due to the lack of evidence to reduce mortality and severity, patients with HF are often already have underlying structural abnormalities. These abnormalities are related to the delay of ventricular repolarization which is manifested on electrocardiogram (ECG) as prolonged QT interval[37-39]. The use of another agents such as loop diuretic, may promote electrolyte imbalance and increase the risk of developing malignant arrhythmia. Sepsis is a known risk factor for QT prolongation that has to be considered in these patients[40]. The proposed mechanism of cardiac function deterioration is depicted in [Figure 1](#).

ROLE OF INFLAMMATORY RESPONSE IN HEART FAILURE PATIENTS WITH COVID-19

Profound cytokine release in the setting of severe COVID-19 infection is commonly found. The release of proinflammatory cytokines, increased metabolic demand and coagulation disorder in sepsis may contribute to the development of new onset HF and decompensation episodes in CHF[41]. Cytokine storm is observed in viral induced infection such as influenzae and COVID-19 and in the setting of graft-versus-host

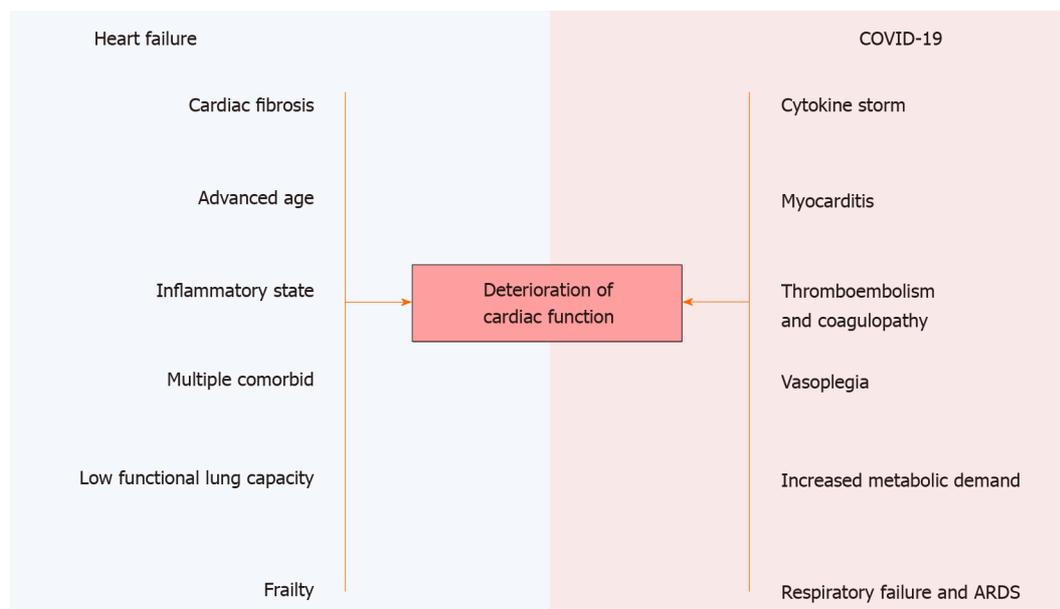


Figure 1 Proposed mechanism of deterioration of cardiac function in preexisting chronic Heart failure patient with coronavirus disease 2019 infection. CHF: Chronic Heart failure; COVID-19: Coronavirus disease 2019; ARDS: Acute respiratory distress syndrome.

disease. Lymphopenia, C-reactive protein, lactate dehydrogenase, ferritin, D-dimer and troponin are among the biomarkers that reflect the severity of the hyperinflammatory state[42,43]. In HF, the inflammation happened as a response to the myocardial stressors. Multiple preexisting comorbidities that might further contribute to the profound inflammatory response are coronary artery disease, hypertension, arrhythmia, diabetes, and obesity. Increased level of inflammatory cytokine directly linked to the deterioration of the heart function[44,45]. The increased level of several cytokines such as tumor necrosis-alpha (TNF- α), interleukin-1 beta (IL-1 β), interleukin-6 (IL-6) and galectin-3 may predict worse outcome in HF patients[46,47]. Interestingly, an elevation of IL-6 Level is also seems to be correlated with mortality in COVID-19 infection[48]. Moreover, the systemic hyperinflammation state that might occur in the setting of COVID-19 has extrapulmonary involvement and causing additional burden to the heart. The effect of hyperinflammation is well reflected by an elevation of cardiac biomarkers such as troponin and N-terminal pro B-type natriuretic peptide [43]. In this stage, vasoplegia and myocarditis might also occur[43].

Pro-inflammatory response which is induced by the infection might worsen hypoxia which in turn causing more stress to the damaged heart[42]. Hypoxia serves as a risk factor of survival in COVID-19 patients and should not be overlooked since it has deleterious effect in patients with HF and COVID-19[49]. Acute respiratory distress syndrome and exaggerated inflammatory response contributes to the development of hypoxia which may cause cardiac lesion and further exacerbates HF[50-53].

COAGULOPATHY IN THE WORSENING HEART DURING COVID-19

Coagulopathy is a common disorder found in COVID-19 infection. It is thought that the interaction between host defense mechanism and coagulation system during COVID-19 infection may lead to hypercoagulability and a high prevalence of thrombotic events[54-56]. This finding is reinforced by an elevated D-dimer level which is often present in the setting of COVID-19 infection[5,55]. The combination of endothelial dysfunction, inflammatory state, oxidative stress and platelet activation are thought to be responsible for a hypercoagulable state[1,5,55]. The true nature of the course is remain unknown to date, however the role of endothelial activation cannot be overlooked. It is thought that ACE2 receptor that serves as an entry point for the SARS-CoV-2 into the cell, plays an important role[57,58]. The presence of ACE2 receptors on the endothelial cells as well as antithrombin (AT), heparin and other anticoagulation might play an important role in regulating the inflammatory response [57]. AT interacts with heparin-like glycosaminoglycan (GAG) on endothelial surface and therefore involved in the release of prostacyclin which will inhibit leukocyte

activation by decreasing IL-6 Level[57,59]. The hypercoagulability state is also involved in the development of both micro- and macrovascular thrombus and may also plug the extracorporeal circuits[5,55]. Grave consequence of thrombosis may occur in COVID-19 patients as it may present as pulmonary venous thrombosis leading to right heart failure and or microvascular thrombosis may leads to myocardial dysfunction, worsening the heart function[5,55,60-62].

Patients with a preexisting history of heart failure are already in an increased risk for developing thromboembolism due to venous stasis, endothelial injury, ischemic cardiomyopathy and atrial fibrosis[63,64]. This condition might be worsened by the presence of COVID-19 infection which may trigger coagulopathy and the presence of implantable device which also may trigger thrombosis[65,66]. The pre-existing cardiopulmonary support such as left ventricular assisted device (LVAD) alone might increase the risk of developing pump thrombosis, although the risk of stroke with co-existing COVID-19 infection has not been assessed yet[3,67].

CARDIOPULMONARY SUPPORT IN COVID-19 PATIENTS WITH HEART FAILURE

Patient with COVID-19 might require mechanical circulatory support as a consequence of COVID-19 induced cytokine release syndrome or cytokine storm[3]. Hypoxemic respiratory failure may cause circulatory collapse in a small subset of patients and the need of lung-protective ventilation (LPV) in these patients is evident [68,69]. The use of extracorporeal life supports (ECLS) is reserved for patients with refractory hypoxemia or hypercarbia, right ventricular failure as a consequence of hypercarbia and acidemia, and hypoxic pulmonary vasoconstriction. These patients might have benefit from the use of venovenous (V-V) ECMO while those who suffer from refractory cardiogenic shock might consider the use of venoarterial (V-A) ECMO which might improve cardiac condition by pronounced LV unloading[70-72].

However, the use of these devices is remain in question since significant resources such as specialized equipment and trained personnel are needed to plant and maintain the device[4,42]. The decision of implanting the device might be considered for patients with ARDS and or cardiogenic shock refractory to traditional management with favorable outcome with the use of the device[68]. Close monitoring is also essential and health care workers exposure is also needed to be looked closely[42]. Still, despite the use of cardiopulmonary support, management of patients with HF and COVID-19 infection remain difficult due to complex interaction between the volume status and the biventricular dynamics[42]. There are also strict criterias have to be met before implanting ECMO in COVID-19 patient. While there are no difference for indication of ECMO between COVID-19 and non-COVID-19 patient, there are several things to be remembered[68]. First, careful selection is needed as patients with advanced age and significant comorbidities might not have much benefit from the use of ECMO. Also, patient with underlying CHF tend to be older, have multiple existing comorbidities and often fall into profound clinical status in the natural course of COVID-19 infection[6,7,14]. Therefore, the use of ECMO should be restricted in these patients unless there are a reasonable chance of recovery[5]. The use of ECMO should be carefully taken, considering that the hospital capacities and resources are limited in most settings and the possible outcome that the patient might achieve[5,42,68].

There are some small subset of patient who may choose another options in regard to the treatment of advanced HF. Patients who are not eligible for the heart transplant might use the left ventricular assist device (LVAD). However, the use of this device is not without risk, especially in the context of COVID-19 infection. These patients are known to have different types of HF that may produce different inflammatory profile in response to the implanted device[47]. To the best of our knowledge, until now there are no specific indication of when to implant ventricular assist device (VAD) in the context of severe COVID-19. First reported case of VAD implantation in patient with COVID-19 infection demonstrate the possibility of VAD as an alternative in a setting of prolonged cardiogenic shock and hemodynamical instability with modest chance of VA ECMO weaning[65]. Careful consideration and assessment of patient's clinical status has to be put in top priority in determining when to implant the device. The indications and contraindications for ECMO in COVID-19 is described in [Table 1](#).

The presence of hardware in the body and prolonged support such as LVAD may cause immune dysregulation, increase the risk of infection and cellular immunity impairment as prior studies had already demonstrated[47,66,73,74]. In COVID-19, severe inflammatory response might induce profound patient's clinical status and

Table 1 Indications and contraindications for Extracorporeal membranous oxygenation in coronavirus disease 2019

Indications for ECMO	Contraindications for ECMO
V-V ECMO; PaO ₂ /FiO ₂ < 60 mmHg for > 6 h < 50 mmHg for > 3 h; pH < 7.2 with PaCO ₂ > 80 mmHg for > 6 h	Relative contraindications; Age ≥ 65 yr old; Body mass index ≥ 40; Immunocompromised status; No legal medical decision maker available; Advanced chronic underlying systolic heart failure; High dose vasopressor requirement (and not under consideration for VA or V-VA ECMO); Absolute contraindications; Advanced age; Clinical frailty scale category ≥ 3; Mechanical ventilation > 10 d; Significant comorbidities: Chronic kidney disease ≥ III; Cirrhosis; Dementia; Baseline neurological disease which might prohibit rehabilitation potential; Disseminated malignancy; Advanced lung disease; Uncontrolled diabetes with chronic end-organ dysfunction; Severe deconditioning; Protein-energy malnutrition; Severe peripheral vascular disease; Other preexisting life-limiting medical condition; Nonambulatory or unable to perform activities; Severe multiple organ failure; Severe acute neurologic injury (example: anoxic, stroke); Uncontrolled bleeding; Contraindications to anticoagulation; Inability to accept blood products; Ongoing cardiopulmonary resuscitation
V-A ECMO; Refractory cardiogenic shock; Persistent tissue hypoperfusion; Systolic blood pressure < 90 mmHg; Cardiac index < 2.2 L/min/m ² while receiving noradrenaline > 0.5 mcg/kg/min; Dobutamine > 20 mcg/kg/min or equivalent	

ECMO: Extracorporeal membranous oxygenation; V-V: Venovenous; V-VA: Venovenous-arterial; V-A: Venous-arterial.

worse outcome. The pre-existing LVAD in severe COVID-19 infection may raise some concerns in the context of management. The risk of pump thrombosis has to be kept in mind, as hypercoagulability state in COVID-19 infection and the pump itself may induce thrombosis[65]. Despite of severe hypoxemia might improve by prone position, there is a specific concern of the outflow graft compression, driveline damage and elevated pressure in the right ventricle with subsequent right ventricular failure[66, 67]. Moreover, additional load to the right ventricle may predispose to right heart failure which is well known as a potential etiology of hypotension in the setting of LVAD use and inflammatory surge[66,67]. However, prone position in patients with LVADs is not contraindicated in the management of hypoxemic respiratory failure although more data are needed[3].

Anticoagulant use in LVAD patient has to be closely monitored due to a high risk of thrombosis in this population[3,14]. More interestingly, patient with COVID-19 infection may often shows a hypercoagulability state despite therapeutic dose of anticoagulation and to overcome this state, requires an increase dose which will carry the risk of life-threatening bleeding[75]. Still, thromboembolism carries a significant risk of adverse outcome and the use of closely monitored anticoagulation might have a beneficial role[55].

Several biomarkers that reflect the severity of hyperinflammation in COVID-19 might be obtained before the infection and serves as a baseline markers in patient with LVAD. Baseline lactate dehydrogenase (LDH), absolute lymphocyte count, troponin and natriuretic peptide that are taken prior the infection might bring an important information that should not be overlooked[42]. These indicators are valuable in following LVAD patients with COVID-19 infection[42]. The role of cardiopulmonary support in COVID-19 infection is illustrated in Figure 2.

CONCLUSION

As discussed above, COVID-19 infection has deleterious effect on the heart function. In addition, exaggerated inflammatory response in severe COVID-19 infection in combination with preexisting impairment of the heart function and multiple comorbid as seen in the HF patients may severely affect the outcome. Cardiac function in patient with HF should not be overlooked as deterioration of the heart function may occur rapidly as a consequence of the infection. Therefore prompt diagnosis and early monitoring of the heart function are critical in the management. Careful monitoring of inflammatory marker during the course of the disease might also play an important role, as patient with advanced HF often have their baseline checked regularly. Any elevation of the inflammatory marker might serve as a clue of worsening inflammation and the heart function.

Another thing needs to be considered is the use of anticoagulation in severe COVID-19 patients with heart failure might have beneficial effect. Hypercoagulability state is often found in the patient, it is possibly because of the inflammatory response and the implanted device that may induce coagulation. However the risk of bleeding has to be kept in mind since fluctuant international normalized ratio and overt bleeding is not uncommon[55,76-78].

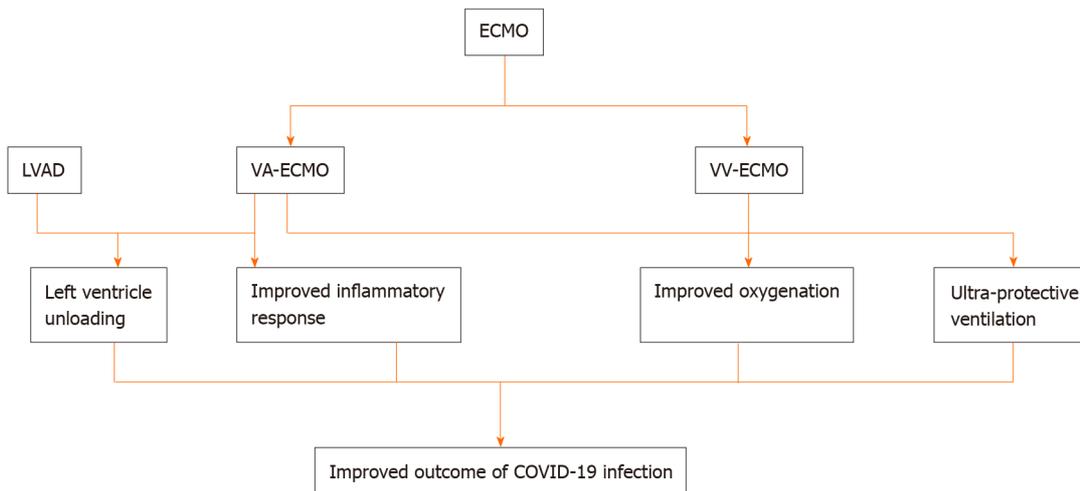


Figure 2 Proposed role of cardiopulmonary support in coronavirus disease 2019 infection. ECMO: Extracorporeal membranous oxygenation; LVAD: Left ventricular assist device; VV: Venovenous; VA: Venoarterial; COVID-19: Coronavirus disease 2019.

The use of cardiopulmonary support in this patient remains an issue. A small subset of patients with implanted LVAD also need to be concerned as unfamiliarity of the healthcare personnel to the device and the possible manipulation of the patient's position such as prone position might increase the right ventricular pressure and might lead to hypotension[66]. The use of ECMO and COVID-19 is very challenging due to its highly selective criteria and contraindicated in most patient with COVID-19 due to multiorgan dysfunction, significant comorbidities and the risk of bleeding[5,8].

Currently, supportive treatment remain the mainstay of treatment for COVID-19 infection. Focus is now directed on primary prevention and vaccination program. High burden and mortality rate was found in patient with preexisting cardiovascular disease, therefore American college of cardiology recommends to prioritize vaccination program in this high risk group[79].

REFERENCES

- Bikdeli B**, Madhavan MV, Jimenez D, Chuich T, Dreyfus I, Driggin E, Nigoghossian C, Agho W, Madjid M, Guo Y, Tang LV, Hu Y, Giri J, Cushman M, Quéré I, Dimakakos EP, Gibson CM, Lippi G, Favaloro EJ, Fareed J, Caprini JA, Tafur AJ, Burton JR, Francese DP, Wang EY, Falanga A, McLintock C, Hunt BJ, Spyropoulos AC, Barnes GD, Eikelboom JW, Weinberg I, Schulman S, Carrier M, Piazza G, Beckman JA, Steg PG, Stone GW, Rosenkranz S, Goldhaber SZ, Parikh SA, Monreal M, Krumholz HM, Konstantinides SV, Weitz JI, Lip GYH; Global COVID-19 Thrombosis Collaborative Group, Endorsed by the ISTH, NATF, ESVM, and the IUA; Supported by the ESC Working Group on Pulmonary Circulation and Right Ventricular Function. COVID-19 and Thrombotic or Thromboembolic Disease: Implications for Prevention, Antithrombotic Therapy, and Follow-Up: JACC State-of-the-Art Review. *J Am Coll Cardiol* 2020; **75**: 2950-2973 [PMID: 32311448 DOI: 10.1016/j.jacc.2020.04.031]
- Kabir M**, Saqib MAN, Zaid M, Ahmed H, Afzal MS. COVID-19, economic impact and child mortality: A global concern. *Clin Nutr* 2020; **39**: 2322-2323 [PMID: 32499056 DOI: 10.1016/j.clnu.2020.05.027]
- DeFilippis EM**, Reza N, Donald E, Givertz MM, Lindenfeld J, Jessup M. Considerations for Heart Failure Care During the COVID-19 Pandemic. *JACC Heart Fail* 2020; **8**: 681-691 [PMID: 32493638 DOI: 10.1016/j.jchf.2020.05.006]
- Chow J**, Alhussaini A, Calvillo-Argüelles O, Billia F, Luk A. Cardiovascular Collapse in COVID-19 Infection: The Role of Venoarterial Extracorporeal Membrane Oxygenation (VA-ECMO). *CJC Open* 2020; **2**: 273-277 [PMID: 32363334 DOI: 10.1016/j.cjco.2020.04.003]
- McLean DJ**, Henry M. Con: Venoarterial ECMO Should Not Be Considered in Patients With COVID-19. *J Cardiothorac Vasc Anesth* 2021; **35**: 707-710 [PMID: 33288431 DOI: 10.1053/j.jvca.2020.11.029]
- Dalia T**, Lahan S, Ranka S, Acharya P, Gautam A, Goyal A, Mastoris I, Sauer A, Shah Z. Impact of congestive heart failure and role of cardiac biomarkers in COVID-19 patients: A systematic review and meta-analysis. *Indian Heart J* 2021; **73**: 91-98 [PMID: 33714416 DOI: 10.1016/j.ihj.2020.12.002]
- Alvarez-Garcia J**, Lee S, Gupta A, Cagliostro M, Joshi AA, Rivas-Lasarte M, Contreras J, Mitter SS, LaRocca G, Tlachi P, Brunjes D, Glucksberg BS, Levin MA, Nadkarni G, Fayad Z, Fuster V, Mancini

- D, Lala A. Prognostic Impact of Prior Heart Failure in Patients Hospitalized With COVID-19. *J Am Coll Cardiol* 2020; **76**: 2334-2348 [PMID: 33129663 DOI: 10.1016/j.jacc.2020.09.549]
- 8 **Bartlett RH**, Ogino MT, Brodie D, McMullan DM, Lorusso R, MacLaren G, Stead CM, Rycus P, Fraser JF, Belohlavek J, Salazar L, Mehta Y, Raman L, Paden ML. Initial ELSO Guidance Document: ECMO for COVID-19 Patients with Severe Cardiopulmonary Failure. *ASAIO J* 2020; **66**: 472-474 [PMID: 32243267 DOI: 10.1097/MAT.0000000000001173]
 - 9 **Xiong TY**, Redwood S, Prendergast B, Chen M. Coronaviruses and the cardiovascular system: acute and long-term implications. *Eur Heart J* 2020; **41**: 1798-1800 [PMID: 32186331 DOI: 10.1093/eurheartj/ehaa231]
 - 10 **Driggin E**, Madhavan MV, Bikdeli B, Chuich T, Laracy J, Biondi-Zoccai G, Brown TS, Der Nigoghossian C, Zidar DA, Haythe J, Brodie D, Beckman JA, Kirtane AJ, Stone GW, Krumholz HM, Parikh SA. Cardiovascular Considerations for Patients, Health Care Workers, and Health Systems During the COVID-19 Pandemic. *J Am Coll Cardiol* 2020; **75**: 2352-2371 [PMID: 32201335 DOI: 10.1016/j.jacc.2020.03.031]
 - 11 **Madjid M**, Safavi-Naeini P, Solomon SD, Vardeny O. Potential Effects of Coronaviruses on the Cardiovascular System: A Review. *JAMA Cardiol* 2020; **5**: 831-840 [PMID: 32219363 DOI: 10.1001/jamacardio.2020.1286]
 - 12 **Tersalvi G**, Vicenzi M, Calabretta D, Biasco L, Pedrazzini G, Winterton D. Elevated Troponin in Patients With Coronavirus Disease 2019: Possible Mechanisms. *J Card Fail* 2020; **26**: 470-475 [PMID: 32315733 DOI: 10.1016/j.cardfail.2020.04.009]
 - 13 **Tavazzi G**, Pellegrini C, Maurelli M, Belliato M, Sciutti F, Bottazzi A, Sepe PA, Resasco T, Camporotondo R, Bruno R, Baldanti F, Paolucci S, Pelenghi S, Iotti GA, Mojoli F, Arbustini E. Myocardial localization of coronavirus in COVID-19 cardiogenic shock. *Eur J Heart Fail* 2020; **22**: 911-915 [PMID: 32275347 DOI: 10.1002/ejhf.1828]
 - 14 **Bader F**, Manla Y, Atallah B, Starling RC. Heart failure and COVID-19. *Heart Fail Rev* 2021; **26**: 1-10 [PMID: 32720082 DOI: 10.1007/s10741-020-10008-2]
 - 15 **Moss N**, Rakita V, Lala A, Parikh A, Roldan J, Mitter SS, Anyanwu A, Campoli M, Burkhoff D, Mancini DM. Hemodynamic Response to Exercise in Patients Supported by Continuous Flow Left Ventricular Assist Devices. *JACC Heart Fail* 2020; **8**: 291-301 [PMID: 32035893 DOI: 10.1016/j.jchf.2019.10.013]
 - 16 **Lang CC**, Agostoni P, Mancini DM. Prognostic significance and measurement of exercise-derived hemodynamic variables in patients with heart failure. *J Card Fail* 2007; **13**: 672-679 [PMID: 17923361 DOI: 10.1016/j.cardfail.2007.05.004]
 - 17 **Epidemiology Working Group for NCIP Epidemic Response**, Chinese Center for Disease Control and Prevention. [The epidemiological characteristics of an outbreak of 2019 novel coronavirus diseases (COVID-19) in China]. *Zhonghua Liu Xing Bing Xue Za Zhi* 2020; **41**: 145-151 [PMID: 32064853 DOI: 10.3760/cma.j.issn.0254-6450.2020.02.003]
 - 18 **Yonas E**, Alwi I, Pranata R, Huang I, Lim MA, Gutierrez EJ, Yamin M, Siswanto BB, Virani SS. Effect of heart failure on the outcome of COVID-19 - A meta analysis and systematic review. *Am J Emerg Med* 2020 [PMID: 33071085 DOI: 10.1016/j.ajem.2020.07.009]
 - 19 **Riehle C**, Bauersachs J. Key inflammatory mechanisms underlying heart failure. *Herz* 2019; **44**: 96-106 [PMID: 30715565 DOI: 10.1007/s00059-019-4785-8]
 - 20 **Canepa M**, Franssen FME, Olschewski H, Lainscak M, Böhm M, Tavazzi L, Rosenkranz S. Diagnostic and Therapeutic Gaps in Patients With Heart Failure and Chronic Obstructive Pulmonary Disease. *JACC Heart Fail* 2019; **7**: 823-833 [PMID: 31521680 DOI: 10.1016/j.jchf.2019.05.009]
 - 21 **Vachiéry JL**, Adir Y, Barberà JA, Champion H, Coghlan JG, Cottin V, De Marco T, Galie`N, Ghio S, Gibbs JS, Martinez F, Semigran M, Simonneau G, Wells A, Seeger W. Pulmonary hypertension due to left heart diseases. *J Am Coll Cardiol* 2013; **62**: D100-D108 [PMID: 24355634 DOI: 10.1016/j.jacc.2013.10.033]
 - 22 **Yasmin Kusumawardhani N**, Huang I, Martanto E, Sihite TA, Nugraha ES, Prodjosoejojo S, Hamijoyo L, Hartantri Y. Lethal Arrhythmia (*Torsade de Pointes*) in COVID-19: An Event Synergistically Induced by Viral Associated Cardiac Injury, Hyperinflammatory Response, and Treatment Drug? *Clin Med Insights Case Rep* 2020; **13**: 1179547620972397 [PMID: 33402858 DOI: 10.1177/1179547620972397]
 - 23 **Huang C**, Wang Y, Li X, Ren L, Zhao J, Hu Y, Zhang L, Fan G, Xu J, Gu X, Cheng Z, Yu T, Xia J, Wei Y, Wu W, Xie X, Yin W, Li H, Liu M, Xiao Y, Gao H, Guo L, Xie J, Wang G, Jiang R, Gao Z, Jin Q, Wang J, Cao B. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. *Lancet* 2020; **395**: 497-506 [PMID: 31986264 DOI: 10.1016/S0140-6736(20)30183-5]
 - 24 **Guo T**, Fan Y, Chen M, Wu X, Zhang L, He T, Wang H, Wan J, Wang X, Lu Z. Cardiovascular Implications of Fatal Outcomes of Patients With Coronavirus Disease 2019 (COVID-19). *JAMA Cardiol* 2020; **5**: 811-818 [PMID: 32219356 DOI: 10.1001/jamacardio.2020.1017]
 - 25 **Shi S**, Qin M, Shen B, Cai Y, Liu T, Yang F, Gong W, Liu X, Liang J, Zhao Q, Huang H, Yang B, Huang C. Association of Cardiac Injury With Mortality in Hospitalized Patients With COVID-19 in Wuhan, China. *JAMA Cardiol* 2020; **5**: 802-810 [PMID: 32211816 DOI: 10.1001/jamacardio.2020.0950]
 - 26 **Zhou F**, Yu T, Du R, Fan G, Liu Y, Liu Z, Xiang J, Wang Y, Song B, Gu X, Guan L, Wei Y, Li H, Wu X, Xu J, Tu S, Zhang Y, Chen H, Cao B. Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study. *Lancet* 2020; **395**: 1054-

- 1062 [PMID: [32171076](#) DOI: [10.1016/S0140-6736\(20\)30566-3](#)]
- 27 **Sala S**, Peretto G, Gramegna M, Palmisano A, Villatore A, Vignale D, De Cobelli F, Tresoldi M, Cappelletti AM, Basso C, Godino C, Esposito A. Acute myocarditis presenting as a reverse Tako-Tsubo syndrome in a patient with SARS-CoV-2 respiratory infection. *Eur Heart J* 2020; **41**: 1861-1862 [PMID: [32267502](#) DOI: [10.1093/eurheartj/ehaa286](#)]
 - 28 **Gheblawi M**, Wang K, Viveiros A, Nguyen Q, Zhong JC, Turner AJ, Raizada MK, Grant MB, Oudit GY. Angiotensin-Converting Enzyme 2: SARS-CoV-2 Receptor and Regulator of the Renin-Angiotensin System: Celebrating the 20th Anniversary of the Discovery of ACE2. *Circ Res* 2020; **126**: 1456-1474 [PMID: [32264791](#) DOI: [10.1161/CIRCRESAHA.120.317015](#)]
 - 29 **Liu PP**, Blet A, Smyth D, Li H. The Science Underlying COVID-19: Implications for the Cardiovascular System. *Circulation* 2020; **142**: 68-78 [PMID: [32293910](#) DOI: [10.1161/CIRCULATIONAHA.120.047549](#)]
 - 30 **Kim IC**, Kim JY, Kim HA, Han S. COVID-19-related myocarditis in a 21-year-old female patient. *Eur Heart J* 2020; **41**: 1859 [PMID: [32282027](#) DOI: [10.1093/eurheartj/ehaa288](#)]
 - 31 **Inciardi RM**, Lupi L, Zaccone G, Italia L, Raffo M, Tomasoni D, Cani DS, Cerini M, Farina D, Gavazzi E, Maroldi R, Adamo M, Ammirati E, Sinagra G, Lombardi CM, Metra M. Cardiac Involvement in a Patient With Coronavirus Disease 2019 (COVID-19). *JAMA Cardiol* 2020; **5**: 819-824 [PMID: [32219357](#) DOI: [10.1001/jamacardio.2020.1096](#)]
 - 32 **Wenzel P**, Kopp S, Göbel S, Jansen T, Geyer M, Hahn F, Kreitner KF, Escher F, Schultheiss HP, Münzel T. Evidence of SARS-CoV-2 mRNA in endomyocardial biopsies of patients with clinically suspected myocarditis tested negative for COVID-19 in nasopharyngeal swab. *Cardiovasc Res* 2020; **116**: 1661-1663 [PMID: [32562489](#) DOI: [10.1093/cvr/cvaa160](#)]
 - 33 **Escher F**, Pietsch H, Aleshcheva G, Bock T, Baumeier C, Elsaesser A, Wenzel P, Hamm C, Westenfeld R, Schultheiss M, Gross U, Morawietz L, Schultheiss HP. Detection of viral SARS-CoV-2 genomes and histopathological changes in endomyocardial biopsies. *ESC Heart Fail* 2020; **7**: 2440-2447 [PMID: [32529795](#) DOI: [10.1002/ehf2.12805](#)]
 - 34 **Giustino G**, Pinney SP, Lala A, Reddy VY, Johnston-Cox HA, Mechanick JI, Halperin JL, Fuster V. Coronavirus and Cardiovascular Disease, Myocardial Injury, and Arrhythmia: JACC Focus Seminar. *J Am Coll Cardiol* 2020; **76**: 2011-2023 [PMID: [33092737](#) DOI: [10.1016/j.jacc.2020.08.059](#)]
 - 35 **Kochav SM**, Coromilas E, Nalbandian A, Ranard LS, Gupta A, Chung MK, Gopinathannair R, Biviano AB, Garan H, Wan EY. Cardiac Arrhythmias in COVID-19 Infection. *Circ Arrhythm Electrophysiol* 2020; **13**: e008719 [PMID: [32434385](#) DOI: [10.1161/CIRCEP.120.008719](#)]
 - 36 **Vrtovec B**, Delgado R, Zewail A, Thomas CD, Richartz BM, Radovancevic B. Prolonged QTc interval and high B-type natriuretic peptide levels together predict mortality in patients with advanced heart failure. *Circulation* 2003; **107**: 1764-1769 [PMID: [12665499](#) DOI: [10.1161/01.CIR.0000057980.84624.95](#)]
 - 37 **Geleris J**, Sun Y, Platt J, Zucker J, Baldwin M, Hripesak G, Labella A, Manson DK, Kubin C, Barr RG, Sobieszczyk ME, Schluger NW. Observational Study of Hydroxychloroquine in Hospitalized Patients with Covid-19. *N Engl J Med* 2020; **382**: 2411-2418 [PMID: [32379955](#) DOI: [10.1056/NEJMoa2012410](#)]
 - 38 **Rosenberg ES**, Dufort EM, Udo T, Wilberschied LA, Kumar J, Tesoriero J, Weinberg P, Kirkwood J, Muse A, DeHovitz J, Blog DS, Hutton B, Holtgrave DR, Zucker HA. Association of Treatment With Hydroxychloroquine or Azithromycin With In-Hospital Mortality in Patients With COVID-19 in New York State. *JAMA* 2020; **323**: 2493-2502 [PMID: [32392282](#) DOI: [10.1001/jama.2020.8630](#)]
 - 39 **Link MG**, Yan GX, Kowey PR. Evaluation of toxicity for heart failure therapeutics: studying effects on the QT interval. *Circ Heart Fail* 2010; **3**: 547-555 [PMID: [20647490](#) DOI: [10.1161/CIRCHEARTFAILURE.109.917781](#)]
 - 40 **Tisdale JE**, Jaynes HA, Kingery JR, Mourad NA, Trujillo TN, Overholser BR, Kovacs RJ. Development and validation of a risk score to predict QT interval prolongation in hospitalized patients. *Circ Cardiovasc Qual Outcomes* 2013; **6**: 479-487 [PMID: [23716032](#) DOI: [10.1161/CIRCOUTCOMES.113.000152](#)]
 - 41 **Tufan A**, Avanoğlu Güler A, Matucci-Cerinic M. COVID-19, immune system response, hyperinflammation and repurposing antirheumatic drugs *Turk J Med Sci* 2020; **50**: 620-632 [PMID: [32299202](#) DOI: [10.3906/sag-2004-168](#)]
 - 42 **Chau VQ**, Oliveros E, Mahmood K, Singhvi A, Lala A, Moss N, Gidwani U, Mancini DM, Pinney SP, Parikh A. The Imperfect Cytokine Storm: Severe COVID-19 With ARDS in a Patient on Durable LVAD Support. *JACC Case Rep* 2020; **2**: 1315-1320 [PMID: [32292915](#) DOI: [10.1016/j.jaccas.2020.04.001](#)]
 - 43 **Siddiqi HK**, Mehra MR. COVID-19 illness in native and immunosuppressed states: A clinical-therapeutic staging proposal. *J Heart Lung Transplant* 2020; **39**: 405-407 [PMID: [32362390](#) DOI: [10.1016/j.healun.2020.03.012](#)]
 - 44 **Aukrust P**, Ueland T, Lien E, Bendtzen K, Müller F, Andreassen AK, Nordøy I, Aass H, Espevik T, Simonsen S, Frøland SS, Gullestad L. Cytokine network in congestive heart failure secondary to ischemic or idiopathic dilated cardiomyopathy. *Am J Cardiol* 1999; **83**: 376-382 [PMID: [10072227](#) DOI: [10.1016/S0002-9149\(98\)00872-8](#)]
 - 45 **Torre-Amione G**, Kapadia S, Benedict C, Oral H, Young JB, Mann DL. Proinflammatory cytokine levels in patients with depressed left ventricular ejection fraction: a report from the Studies of Left Ventricular Dysfunction (SOLVD). *J Am Coll Cardiol* 1996; **27**: 1201-1206 [PMID: [8609343](#) DOI: [10.1016/0735-1097\(95\)00589-7](#)]

- 46 **Deswal A**, Petersen NJ, Feldman AM, Young JB, White BG, Mann DL. Cytokines and cytokine receptors in advanced heart failure: an analysis of the cytokine database from the Vesnarinone trial (VEST). *Circulation* 2001; **103**: 2055-2059 [PMID: [11319194](#) DOI: [10.1161/01.cir.103.16.2055](#)]
- 47 **Radley G**, Pieper IL, Ali S, Bhatti F, Thornton CA. The Inflammatory Response to Ventricular Assist Devices. *Front Immunol* 2018; **9**: 2651 [PMID: [30498496](#) DOI: [10.3389/fimmu.2018.02651](#)]
- 48 **Silberstein M**. Correlation between premorbid IL-6 Levels and COVID-19 mortality: Potential role for Vitamin D. *Int Immunopharmacol* 2020; **88**: 106995 [PMID: [33182059](#) DOI: [10.1016/j.intimp.2020.106995](#)]
- 49 **Mikami T**, Miyashita H, Yamada T, Harrington M, Steinberg D, Dunn A, Siau E. Risk Factors for Mortality in Patients with COVID-19 in New York City. *J Gen Intern Med* 2021; **36**: 17-26 [PMID: [32607928](#) DOI: [10.1007/s11606-020-05983-z](#)]
- 50 **Babapoor-Farrokhran S**, Gill D, Walker J, Rasekhi RT, Bozorgnia B, Amanullah A. Myocardial injury and COVID-19: Possible mechanisms. *Life Sci* 2020; **253**: 117723 [PMID: [32360126](#) DOI: [10.1016/j.lfs.2020.117723](#)]
- 51 **Nan J**, Jin YB, Myo Y, Zhang G. Hypoxia in acute cardiac injury of coronavirus disease 2019: lesson learned from pathological studies. *J Geriatr Cardiol* 2020; **17**: 221-223 [PMID: [32362921](#) DOI: [10.11909/j.issn.1671-5411.2020.04.010](#)]
- 52 **Rali AS**, Ranka S, Shah Z, Sauer AJ. Mechanisms of Myocardial Injury in Coronavirus Disease 2019. *Card Fail Rev* 2020; **6**: e15 [PMID: [32537248](#) DOI: [10.15420/cfr.2020.10](#)]
- 53 **Adeghate EA**, Eid N, Singh J. Mechanisms of COVID-19-induced heart failure: a short review. *Heart Fail Rev* 2021; **26**: 363-369 [PMID: [33191474](#) DOI: [10.1007/s10741-020-10037-x](#)]
- 54 **Danzi GB**, Loffi M, Galeazzi G, Gherbesi E. Acute pulmonary embolism and COVID-19 pneumonia: a random association? *Eur Heart J* 2020; **41**: 1858 [PMID: [32227120](#) DOI: [10.1093/eurheartj/ehaa254](#)]
- 55 **Iba T**, Levy JH, Levi M, Connors JM, Thachil J. Coagulopathy of Coronavirus Disease 2019. *Crit Care Med* 2020; **48**: 1358-1364 [PMID: [32467443](#) DOI: [10.1097/CCM.0000000000004458](#)]
- 56 **Guan WJ**, Ni ZY, Hu Y, Liang WH, Ou CQ, He JX, Liu L, Shan H, Lei CL, Hui DSC, Du B, Li LJ, Zeng G, Yuen KY, Chen RC, Tang CL, Wang T, Chen PY, Xiang J, Li SY, Wang JL, Liang ZJ, Peng YX, Wei L, Liu Y, Hu YH, Peng P, Wang JM, Liu JY, Chen Z, Li G, Zheng ZJ, Qiu SQ, Luo J, Ye CJ, Zhu SY, Zhong NS; China Medical Treatment Expert Group for Covid-19. Clinical Characteristics of Coronavirus Disease 2019 in China. *N Engl J Med* 2020; **382**: 1708-1720 [PMID: [32109013](#) DOI: [10.1056/NEJMoa2002032](#)]
- 57 **Kowalewski M**, Fina D, Słomka A, Raffa GM, Martucci G, Lo Coco V, De Piero ME, Ranucci M, Suwalski P, Lorusso R. COVID-19 and ECMO: the interplay between coagulation and inflammation—a narrative review. *Crit Care* 2020; **24**: 205 [PMID: [32384917](#) DOI: [10.1186/s13054-020-02925-3](#)]
- 58 **Permana H**, Huang I, Purwiga A, Kusumawardhani NY, Sihite TA, Martanto E, Wisaksana R, Soetedjo NNM. In-hospital use of statins is associated with a reduced risk of mortality in coronavirus-2019 (COVID-19): systematic review and meta-analysis. *Pharmacol Rep* 2021; **73**: 769-780 [PMID: [33608850](#) DOI: [10.1007/s43440-021-00233-3](#)]
- 59 **Levy JH**, Sniecinski RM, Welsby IJ, Levi M. Antithrombin: anti-inflammatory properties and clinical applications. *Thromb Haemost* 2016; **115**: 712-728 [PMID: [26676884](#) DOI: [10.1160/TH15-08-0687](#)]
- 60 **Hendren NS**, Drazner MH, Bozkurt B, Cooper LT Jr. Description and Proposed Management of the Acute COVID-19 Cardiovascular Syndrome. *Circulation* 2020; **141**: 1903-1914 [PMID: [32297796](#) DOI: [10.1161/CIRCULATIONAHA.120.047349](#)]
- 61 **Park JF**, Banerjee S, Umar S. In the eye of the storm: the right ventricle in COVID-19. *Pulm Circ* 2020; **10**: 2045894020936660 [PMID: [32655856](#) DOI: [10.1177/2045894020936660](#)]
- 62 **Clerkin KJ**, Fried JA, Raikhelkar J, Sayer G, Griffin JM, Masoumi A, Jain SS, Burkhoff D, Kumaraiah D, Rabbani L, Schwartz A, Uriel N. COVID-19 and Cardiovascular Disease. *Circulation* 2020; **141**: 1648-1655 [PMID: [32200663](#) DOI: [10.1161/CIRCULATIONAHA.120.046941](#)]
- 63 **Goldhaber SZ**. Venous Thromboembolism in Heart Failure Patients: Pathophysiology, Predictability, Prevention. *J Am Coll Cardiol* 2020; **75**: 159-162 [PMID: [31948644](#) DOI: [10.1016/j.jacc.2019.11.028](#)]
- 64 **Fanola CL**, Norby FL, Shah AM, Chang PP, Lutsey PL, Rosamond WD, Cushman M, Folsom AR. Incident Heart Failure and Long-Term Risk for Venous Thromboembolism. *J Am Coll Cardiol* 2020; **75**: 148-158 [PMID: [31948643](#) DOI: [10.1016/j.jacc.2019.10.058](#)]
- 65 **Rassaf T**, Totzeck M, Mahabadi AA, Hendgen-Cotta U, Korste S, Settelmeier S, Luedike P, Dittmer U, Herbstreit F, Brenner T, Klingel K, Hasenberg M, Walkenfort B, Gunzer M, Schlosser T, Weymann A, Kamler M, Schmack B, Ruhparwar A. Ventricular assist device for a coronavirus disease 2019-affected heart. *ESC Heart Fail* 2021; **8**: 162-166 [PMID: [33219613](#) DOI: [10.1002/ehf2.13120](#)]
- 66 **Singh R**, Domenico C, Rao SD, Urگو K, Prenner SB, Wald JW, Atluri P, Birati EY. Novel Coronavirus Disease 2019 in a Patient on Durable Left Ventricular Assist Device Support. *J Card Fail* 2020; **26**: 438-439 [PMID: [32305569](#) DOI: [10.1016/j.cardfail.2020.04.007](#)]
- 67 **Sobol I**, Yuzefpolskaya M, Roth Z, Colombo PC, Horn E, Takeda K, Sayer G, Uriel N, Naka Y. Characteristics and Outcomes of Patients With a Left Ventricular Assist Device With Coronavirus Disease-19. *J Card Fail* 2020; **26**: 895-897 [PMID: [32956813](#) DOI: [10.1016/j.cardfail.2020.09.011](#)]
- 68 **Papazian L**, Aubron C, Brochard L, Chiche JD, Combes A, Dreyfuss D, Forel JM, Guérin C, Jaber S, Mekontso-Dessap A, Mercat A, Richard JC, Roux D, Vieillard-Baron A, Faure H. Formal guidelines: management of acute respiratory distress syndrome. *Ann Intensive Care* 2019; **9**: 69 [PMID: [32305569](#)]

- 31197492 DOI: [10.1186/s13613-019-0540-9](https://doi.org/10.1186/s13613-019-0540-9)]
- 69 **Thompson BT**, Chambers RC, Liu KD. Acute Respiratory Distress Syndrome. *N Engl J Med* 2017; **377**: 1904-1905 [PMID: [29117492](https://pubmed.ncbi.nlm.nih.gov/29117492/) DOI: [10.1056/NEJMc1711824](https://doi.org/10.1056/NEJMc1711824)]
- 70 **Hardin CC**, Hibbert K. ECMO for Severe Acute Respiratory Distress Syndrome. *N Engl J Med* 2018; **379**: 1092-1093 [PMID: [30230806](https://pubmed.ncbi.nlm.nih.gov/30230806/) DOI: [10.1056/NEJMc1808731](https://doi.org/10.1056/NEJMc1808731)]
- 71 **Goligher EC**, Tomlinson G, Hajage D, Wijeyesundera DN, Fan E, Jüni P, Brodie D, Slutsky AS, Combes A. Extracorporeal Membrane Oxygenation for Severe Acute Respiratory Distress Syndrome and Posterior Probability of Mortality Benefit in a Post Hoc Bayesian Analysis of a Randomized Clinical Trial. *JAMA* 2018; **320**: 2251-2259 [PMID: [30347031](https://pubmed.ncbi.nlm.nih.gov/30347031/) DOI: [10.1001/jama.2018.14276](https://doi.org/10.1001/jama.2018.14276)]
- 72 **Bunge JJH**, Caliskan K, Gommers D, Reis Miranda D. Right ventricular dysfunction during acute respiratory distress syndrome and veno-venous extracorporeal membrane oxygenation. *J Thorac Dis* 2018; **10**: S674-S682 [PMID: [29732186](https://pubmed.ncbi.nlm.nih.gov/29732186/) DOI: [10.21037/jtd.2017.10.75](https://doi.org/10.21037/jtd.2017.10.75)]
- 73 **Ankersmit HJ**, Tugulea S, Spanier T, Weinberg AD, Artrip JH, Burke EM, Flannery M, Mancini D, Rose EA, Edwards NM, Oz MC, Itescu S. Activation-induced T-cell death and immune dysfunction after implantation of left-ventricular assist device. *Lancet* 1999; **354**: 550-555 [PMID: [10470699](https://pubmed.ncbi.nlm.nih.gov/10470699/) DOI: [10.1016/s0140-6736\(98\)10359-8](https://doi.org/10.1016/s0140-6736(98)10359-8)]
- 74 **Kimball PM**, Flattery M, McDougan F, Kasirajan V. Cellular immunity impaired among patients on left ventricular assist device for 6 mo. *Ann Thorac Surg* 2008; **85**: 1656-1661 [PMID: [18442560](https://pubmed.ncbi.nlm.nih.gov/18442560/) DOI: [10.1016/j.athoracsur.2008.01.050](https://doi.org/10.1016/j.athoracsur.2008.01.050)]
- 75 **Bilaloglu S**, Aphinyanaphongs Y, Jones S, Iturrate E, Hochman J, Berger JS. Thrombosis in Hospitalized Patients With COVID-19 in a New York City Health System. *JAMA* 2020; **324**: 799-801 [PMID: [32702090](https://pubmed.ncbi.nlm.nih.gov/32702090/) DOI: [10.1001/jama.2020.13372](https://doi.org/10.1001/jama.2020.13372)]
- 76 **Thachil J**, Tang N, Gando S, Falanga A, Cattaneo M, Levi M, Clark C, Iba T. ISTH interim guidance on recognition and management of coagulopathy in COVID-19. *J Thromb Haemost* 2020; **18**: 1023-1026 [PMID: [32338827](https://pubmed.ncbi.nlm.nih.gov/32338827/) DOI: [10.1111/jth.14810](https://doi.org/10.1111/jth.14810)]
- 77 **Ortega-Paz L**, Capodanno D, Montalescot G, Angiolillo DJ. Coronavirus Disease 2019-Associated Thrombosis and Coagulopathy: Review of the Pathophysiological Characteristics and Implications for Antithrombotic Management. *J Am Heart Assoc* 2021; **10**: e019650 [PMID: [33228447](https://pubmed.ncbi.nlm.nih.gov/33228447/) DOI: [10.1161/JAHA.120.019650](https://doi.org/10.1161/JAHA.120.019650)]
- 78 **Tang N**, Bai H, Chen X, Gong J, Li D, Sun Z. Anticoagulant treatment is associated with decreased mortality in severe coronavirus disease 2019 patients with coagulopathy. *J Thromb Haemost* 2020; **18**: 1094-1099 [PMID: [32220112](https://pubmed.ncbi.nlm.nih.gov/32220112/) DOI: [10.1111/jth.14817](https://doi.org/10.1111/jth.14817)]
- 79 **Driggin E**, Maddox TM, Ferdinand KC, Kirkpatrick JN, Ky B, Morris AA, Mullen JB, Parikh SA, Philbin DM Jr, Vaduganathan M. ACC Health Policy Statement on Cardiovascular Disease Considerations for COVID-19 Vaccine Prioritization: A Report of the American College of Cardiology Solution Set Oversight Committee. *J Am Coll Cardiol* 2021; **77**: 1938-1948 [PMID: [33587998](https://pubmed.ncbi.nlm.nih.gov/33587998/) DOI: [10.1016/j.jacc.2021.02.017](https://doi.org/10.1016/j.jacc.2021.02.017)]



Published by **Baishideng Publishing Group Inc**
7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA
Telephone: +1-925-3991568
E-mail: bpgoffice@wjgnet.com
Help Desk: <https://www.f6publishing.com/helpdesk>
<https://www.wjgnet.com>

