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REVIEW

Heart failure during the COVID-19 pandemic: clinical, diagnostic, management, and organizational dilemmas

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Abstract

The coronavirus 2019 (COVID-19) infection pandemic has affected the care of patients with heart failure (HF). Several consensus documents describe the appropriate diagnostic algorithm and treatment approach for patients with HF and associated COVID-19 infection. However, few questions about the mechanisms by which COVID can exacerbate HF in patients with high-risk (Stage B) or symptomatic HF (Stage C) remain unanswered. Therefore, the type of HF occurring during infection is poorly investigated. The diagnostic differentiation and management should be focused on the identification of the HF phenotype, underlying causes, and subsequent tailored therapy. In this framework, the relationship existing between COVID and onset of acute decompensated HF, isolated right HF, and cardiogenic shock is questioned, and the specific management is mainly based on local hospital organization rather than a standardized model. Similarly, some specific populations such as advanced HF, heart transplant, patients with left ventricular assist device (LVAD), or valve disease remain under investigated. In this systematic review, we examine recent advances regarding the relationships between HF and COVID-19 pandemic with respect to epidemiology, pathogenetic mechanisms, and differential diagnosis. Also, according to the recent HF guidelines definition, we highlight different clinical profile identification, pointing out the main concerns in understudied HF populations.

Keywords COVID-19; Heart failure; Diagnostic differentiation; Management

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Introduction

The coronavirus disease 2019 (COVID-19) pandemic has affected the care of patients with heart failure (HF) who have contracted COVID-19 as well as those with HF but without COVID-19 who have been impacted by the restructuring of healthcare delivery. The prevalence of new-onset HF during pandemic period and its relationship with COVID-19 is still debated.[1–3] Some reports suggest a decrease of HF hospitalizations in subjects with a previous HF diagnosis,[1] but these

data may have been influenced by the avoidance of hospital services during COVID-19. In addition, previously diagnosed HF increases the risk for a more severe clinical course of COVID-19.[4] HF patients are often elderly with many concomitant non-cardiovascular co-morbidities, conferring a high-risk profile. COVID-19 represents an important trigger for acute decompensation of chronic HF or may be responsible for new-onset HF. Numerous studies reported a high incidence of HF episodes related to the infection, with a high rate of myocardial injury associated with conferring a worse prognosis during COVID-19 hospitalization.[2,5]

New-onset or previously diagnosed HF in the setting of COVID-19 may complicate the diagnostic process. Symptoms from both conditions often overlap and potentiate each other. Furthermore, the management of HF patients concomitantly diagnosed with COVID-19 represents a formidable challenge due to the more severe in-hospital course, complex interactions between COVID-19 and HF medications, and difficulties in performing interventional procedures. Recently, EACVI proposed a diagnostic algorithm aimed at early identification of patients with possible cardiac complications associated with COVID-19.[6] However, the exact HF profile according to the recent HF guidelines[7] classifications occurring during infection has not been sufficiently addressed. The European Society of Cardiology and Chinese Heart Failure Societies recently developed a joint document to provide advice on the management of patients with HF and COVID-19,[8] but several gaps in knowledge remain about the appropriate management in patients hospitalized for acute heart failure (AHF) and in patients with both infection and HF. A specific algorithm focused on the diagnostic differentiation between AHF and COVID-19 with acute respiratory distress syndrome (ARDS), facilitating initial triage and specific therapeutic measures is lacking. When both conditions co-exist, this type of algorithm may help to quantitatively assess the contribution of each component to the clinical presentation. We performed a systematic review selecting studies including HF and COVID complication, guidelines, and recommendation of the ESC and ESC associations, reporting the most important evidences in this topic. The main objective of this review is to prioritize the diagnostic and management challenges of HF patients with concomitant COVID-19 infection.

The main objective of this review is to prioritize the diagnostic and management challenges of HF patients with concomitant COVID-19 infection in the light of the recent HF guidelines and definitions.

Impact of COVID-19 outbreak on HF outcome and health service

Although HF is included among the CV diseases causing clinical deterioration during infection, several manuscripts suggest a decrease of HF hospitalizations during the pandemic. (*Table 1*) A multicentre UK study evaluating the first 6 months of 2020 found a reduction in HF hospitalization with respect to the same period of 2019. This trend is balanced with increased in-hospital mortality.[9] Another study evaluating ED presentations for HF-related causes found a similar reduction regarding ED visits, although it confirms a trend towards an increased in-hospital mortality rate.[10] Data from King's College of London showed a similar reduction of HF hospitalization, but the hospitalized patients experienced a worse clinical condition as compared with the population admitted over a similar time frame from a pre-pandemic period.[11,12] The reduction of HF hospitalizations during pandemic may be attributable to the limited access to medical care or to the sudden increase of fatal events at home due to disease's progression.[13–17] Limited access to medical care resulted in a higher rate of withdrawal of the medical treatment.[14] These findings suggest patients affected by HF during the lockdown went to hospital only in more advanced stages with severe congestion, whereas, when less symptomatic, may have stayed home.[15] However, a study comparing hospital admissions before and during the pandemic did not suggest a significant difference regarding clinical severity at presentation, despite increased mortality in patients with HF and COVID-19 infection.[16]

Rapid surges in COVID-19 admissions can overwhelm hospital services, which are unable to admit patients with worsening HF in a timely fashion or must discharge them quickly. Also, the pandemic reduced the number of nurses and experienced doctors available for HF care,[17] and many hospitals had to decide whether to prioritize care for patients with COVID. Staff may wear personal protective equipment that is cumbersome and time consuming, reducing their contact with patients and efficiency of care. A hospital's reorganization may have been primarily focused on COVID management routes, and this reorganization may lead to a neglect of other diseases including HF. This trend is similar for ambulatory services. Telemedicine programmes were not capable to monitor all HF patients. The reduced rate of ambulatory visits was associated with reduced number of blood test [such as natriuretic peptides (NP), renal function and electrolytes monitoring], increasing the risk of unbalanced fluid homoeostasis and optimization of pharmacotherapies. Therefore, hospital ward reorganization led to postponed device implants, rescheduled CV surgery, delayed ventricular assist device (VAD) implants, and heart transplantations (HT).

Delaying care and undertreatment may explain the much more severe conditions and the consequent mortality risk elevation in hospitalized patients that do seek care.[18,19]

The reduced hospitalization rate should be interpreted in the general context of the pandemic because many of the symptomatic patients remained reluctant to come into the hospital even in the presence of early signs of

Table 1 Epidemiological studies describing the heart failure hospitalization rate and modality during pandemic

First author	Observational period	Patients	Cohort	Main findings
Andersson C. et al.	January 1 to March 11 in 2019 compared with 2020	2197 hospitalization in 2020 vs. 2099 in 2019	Danish nationwide cohort	New-onset HF diagnoses and HF hospitalizations for worsening HF were significantly lower in 2020 vs. 2019. Mortality was similar before and after the national lockdown
Cannata C. et al.	January 7 to June 14 in 2019 compared with 2020	794 vs. 578 admitted	South London hospitals, UK	Significant reduction in hospitalizations during the COVID-19 peak, followed by a return to 2019 levels. Increased in hospital mortality compared with previous period
Frankfurter C et al.	March 1 to April 19 in 2019 compared with 2020	800 ED visits in 2019 vs. 1106 in 2020	Toronto hospital, Canada	Decrease in ADHF-related visits and admissions was observed. A trend towards an increase of in hospital mortality during infection surge compared with 2019
Bromage DI et al.	March 2 to April 19 in 2020 compared with corresponding period in 2017 and 2019	26 admission per week in 2020 vs. 78 in 2019	King's College Hospital, London	A significantly lower admission rate for AHF was observed during the study period compared with all other periods, but hospitalized patients had more severe symptoms at admission
Cox Z et al.	March 22 to April 20 in 2020 compared with corresponding period in 2019	AHF hospitalizations was −11 ± 12% 2019 vs. −46 ± 16% in 2020	Vanderbilt University Medical Center, USA	Decreased number of hospitalizations compared with same period of previous year
Rey JR et al.	March 1 to April 20 followed for 30 days	152 of 3080 infected patients had HF	Madrid hospital, Spain	Infected COVID-19 patients with history of CHF are prone to develop acute decompensation. Patients with CHF showed higher mortality rates (48.7 vs. 19.0%)
Bhatt AS et al.	1 January 2019 to 30 March 2020	6083 patients experienced 7187 hospitalizations for cardiovascular reasons	Retrospective analysis from Mass General Brigham system, USA	Significant decline in hospitalizations in March 2020 associated with reduced length of stay. No differences in terms of in-hospital mortality
Baldi E et al.	21 February to 20 April 2020 with same time frame in 2019	490 out-of-hospital cardiac arrest in 2020 vs. 321 in 2019	Lombardia region, Italy	Out-of-hospital cardiac arrest occurred much more during pandemic period with 52% increase compared with 2019
Marijon E et al.	16 March 16 to 26 April 2020 compared with same period from 2011 to 2019	521 out-of-hospital arrest during observational period vs. 3052 of the same weeks in the non-pandemic period	Observational registry from Paris, France	A transient two times increase in OHCA incidence, coupled with a reduction in survival and delay to intervention
Doolub G et al	7 January to 27 April 2020 dividing in before lockdown (until 2 March) and after the subsequent 8 weeks	164 referred in the 8 weeks before vs. 119 referred after	South-west England, UK	Early period reveals a reduction in hospitalization and mortality respect to late period The 30-day case fatality rate was increased by 10% during late period
Severino P et al	21 February to 31 March 2020, compared with 21 February to 31 March 2019	112 admitted during the case period vs. 201 during intra-year period	Multicentre retrospective Italian study	Significant hospitalization reduction compared with previous year. Admitted patients were in more advanced NYHA class

ADHF, acute decompensated heart failure; CHF, chronic heart failure; COVID-19, coronavirus disease 2019; HF, heart failure; OHCA out-of-hospital cardiac arrest.

decompensation because of fear of potential COVID-19 exposure.[20] It is unclear if a better self-care management when "staying home" did influence the rate of hospitalization in the subsequent surges of COVID pandemic. Current picture shows a trend to a higher incidence of HF hospitalizations as compared with the first wave of pandemic.

For the recent surges of the pandemic, medical community assisted to an increased number of HF hospitalizations. Distinct to the first wave, shortening and more liberal lockdown periods, inadequate management during first wave, attenuation of the fear to go to the hospital in condition of the vaccination, and better hospital organization with more available beds contributed to the increased incidence of HF admissions.

Finally, the pandemic had unfavourable consequences on clinical trial research—patient enrolment and follow-up. Many interventional trials have been stopped because of difficulties in recruiting patients, whereas other studies continued enrolment with a significant decrease of enrolled patients because of patient concerns about in-hospital evaluation and the need to respect distancing rules.[21] Stakeholder resources have been retracted after observing that theoretical number of recruited patients had not been achieved.[22] Conversely, the pandemic stimulated other follow-up modalities, including remote system evaluation with the application of several instruments and devices capable of receiving and recording clinical data collection while maintaining procedural distance and lockdown rules.[1,22] Unfortunately, these facilities cannot replace protocols consisting of blood tests and specific analysis requiring specific bio-profile and metabolomic data. When possible, patient's visits have been replaced by home visits, when study staff to collect required blood samples[20,21] (*Table 2*).

HF and COVID-19: Pathogenetic mechanisms

In an international survey, left or right ventricular dysfunction was reported in more than one third of patients admitted with COVID-19.[23] US data from Mount Sinai Hospital including 6439 hospitalized patients with COVID-19 revealed that 0.6% developed HF, but 25% of patients experienced higher values of both troponin and BNP, irrespective of a history of HF or cardiovascular risk.[24] Currently, these discrepancies depend on how HF or structural cardiac abnormalities were defined. Other discrepancies may arise from enrolling different populations with various baseline CV risk burden and different intensity of care. Patients recruited from intensive care unit (ICU) or cardiology units may have a higher prevalence of CV complications compared with patients admitted in other less intensive departments.[25–27] In patients who recovered from a pneumonia due to severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), RV dysfunction might be more common than left ventricular (LV) dysfunction.[28,29] Time point of enrolment is also important due to the transient nature of the intramyocardial oedema and fibrosis.

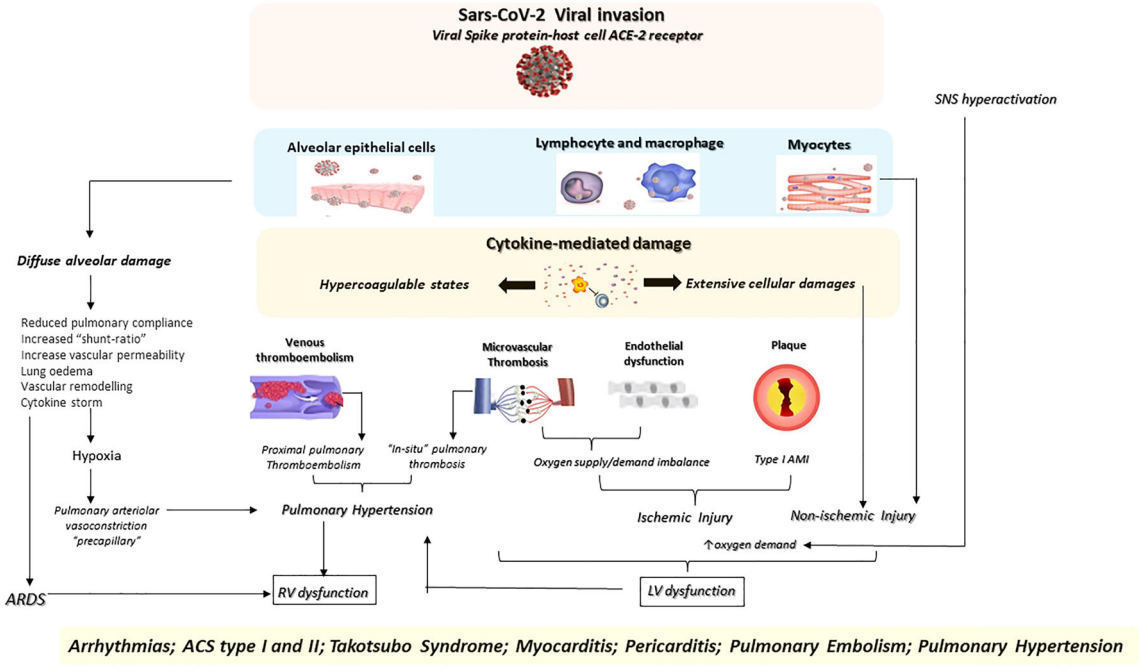
The proposed mechanisms of myocardial injury and dysfunction in patients with COVID-19 include cytokine-mediated damage, oxygen supply–demand imbalance, ischaemic injury from microvascular thrombosis, pulmonary hypertension (PH)-induced right ventricular (RV) dysfunction, and a direct viral infection of the myocardium[30–34] (*Figure 1*). Structural abnormalities including global LV dysfunction, regional wall motion abnormalities, RV dysfunction, Takotsubo cardiomyopathy, and pericardial effusion were detected in the acute phase of COVID-19, and a higher prevalence of echocardiographic abnormalities was found in patients with biomarker evidence of myocardial injury.[28,32,35] In acute phase, in patients with severe LV dysfunction, cardiac magnetic resonance (CMR) revealed abnormalities in T1 and T2 mapping and late gadolinium enhancement images,[31–33] and endomyocardial biopsy revealed active lymphocytic infiltration. However, very few data exist about late effects of infection on cardiac status, and it is still unclear whether the myocardial injury and structural and functional abnormalities observed in the acute phase of infection might be reversible.[32,35,36] Although longitudinal studies have demonstrated gradual declines of cardiac and inflammatory biomarkers, several studies using echocardiography and CMR imaging have reported residual cardiac structural and functional abnormalities in the first 3 months after recovery from COVID-19.[37,38] However, these studies have been limited by their short time interval between COVID-19 diagnosis and follow-up, which may not be long enough for cardiac abnormal-

Table 2 Potential consequences on health system organization during pandemic and future changes

Overall reduction in HF-related hospitalization	Increased self-monitoring and better patient's habits, suppletive role of telemonitoring, positive effects of restriction and isolation, patients' fear to accede into hospital ward despite worsening conditions, limit of studies evaluating only hub hospitals, increased home sudden death
In-hospital ward reorganization	Reduced space and number of site dedicated for HF care, delayed or misunderstood diagnosis, absence of specific diagnostic algorithm, reduced resources for HF units, decreased planned procedures and ICD/CFRT implantation
Reduced ambulatory check-up	Telemonitoring is used only in younger people with increased risk; elderly patients with non-invasive monitorization cannot correctly read data; reduced blood tests; lack of treatment optimization; loss of patients included in VAD or Transplant list
Procedural withdrawal	Planned transcatheter and surgical interventions are delayed; surgical and haemodynamic wards restructured and reallocated only for urgent procedures; prolonged diagnostic and therapeutic time
Research and study investigation	Decreased number of available patients for interventional trial, reduced financial resources, researches breakdown focused on new drugs or new devices benefits, reduced physician's time and availability for investigation

CRT, cardiac resynchronization treatment; ICD, implantable cardiac defibrillator; VAD, ventricular assist device.

Figure 1 Potential contributing factors and mechanisms of cardiac dysfunction in patients with COVID-19. The first step in COVID-19 pathogenesis is viral invasion via its target host cell receptors. SARS-CoV-2 infection induces cellular death and injury in various cellular types and determines overactivation of the host immune and neurohormonal responses. Viral-mediated cell death causes release of damage-associated molecules and cytokines, and maladaptive cytokine release is associated to further cellular destruction and multi-organ dysfunction. The infection of endothelial cells could lead to severe endothelial dysfunction and microvascular thrombosis, factors that contribute to the ischaemic injuries observed in many tissues and organs. The maladaptive immune response can potentially destabilize atherosclerotic plaques and explain the development of Type I acute coronary syndrome. The direct myocyte's viral invasion and infiltration of the myocardium by activated T lymphocytes and macrophages lead to severe cardiac damage and in some instances to fulminant myocarditis. Ischaemic or non-ischaemic myocardial damage, overactivation of the sympathetic nervous system, hypoxaemia, and dyselectrolytemia determine the development of arrhythmias. Pulmonary hypertension could appear as consequence of microvascular thrombosis and systemic coagulopathy that increase the risk of *in situ* pulmonary thrombosis and proximal pulmonary embolism. In addition, hypoxia-induced pulmonary vasoconstriction causes pulmonary hypertension, leading to right ventricular dilatation and dysfunction. ACE-2 receptor, angiotensin-converting enzyme type 2 receptor; ACS, acute coronary syndrome; ARDS, acute respiratory distress syndrome; LV, left ventricle; RV, right ventricle; SNS, sympathetic nervous system.



ities to resolve.[39,40] Furthermore, majority of patients included in these studies had a high cardiovascular burden and a severe in-hospital course, suggesting that the persistent cardiac abnormalities in COVID-19 survivors could be attributed to the pre-existing cardiac conditions and infection severity and to the intensity of care including respiratory support, rather than myocardial injury *per se*. Notably, at longer follow-up time, more than 6 months, most of the studies showed reversibility of the cardiac abnormalities, even if they were present early after diagnosis.[41] A recent study showed that there were no significant differences in echocardiographic parameters, including LV and RV volumes, global longitudinal strain and diastolic function, between COVID-19 survivors and healthy control group, at 327 days after diagnosis regardless of the presence of myocardial injury in the acute phase and disease severity at admission.[42]

Myocarditis is one of the pathogenic contributors, but the prevalence of COVID-19-related myocarditis is unclear and highly dependent on the definition and criteria applied.[43,44] In one study, 7% of COVID-19-related deaths were attributable to myocarditis, but without confirmatory pathological studies. The pathophysiology of COVID-19-related myocarditis is thought to be a combination of direct viral injury and cardiac damage due to the host's immune response.[45] However, a clear demonstration of the presence of the viral genome into myocytes was shown only in sporadic cases, and histology findings showed low-grade inflammation with non-specific myocardial changes and low or absent myocyte necrosis.[34,46] Clinical presentation of COVID-19-related myocarditis is highly variable; some patients may present with relatively mild symptoms, whereas other patients had fulminant myocarditis that progressively deteriorated to cardiogenic shock (CS).[47,48]

The most common cardiovascular complication of COVID-19 is related to arrhythmias, especially atrial fibrillation, and this may be the most important cause of the new onset or worsening of HF due to COVID-19. Pathophysiology of arrhythmias in settings of COVID-19 infection is multifacto-

rial and includes beyond pro-arrhythmogenic effect of the inflammation, electrolyte imbalance, or side effects of additional therapies.[48]

Acute coronary syndrome (ACS) may contribute to the new-onset or worsening HF.[26–28,49,50] Several potential mechanisms have been hypothesized, including systemic inflammatory response with cytokine-mediated injury, microvascular thrombosis, and endothelial dysfunction.[51,52] COVID-19 infection may promote atherosclerotic plaque instability and thrombus formation producing Type 1 MI and may worsen oxygen supply–demand imbalance due to the severe hypoxic state leading to Type 2 MI.[33–35]

Myocardial infarction with non-obstructive coronary arteries (MINOCA) has been also reported in patients with COVID-19, and mechanisms include plaque erosion, endothelial dysfunction microthrombi, or coronary vasospasm.[53,54]

Takotsubo syndrome (TTS), a condition that mimics an ACS at presentation, has been reported during COVID-19 pandemic with an incidence ranging from 2 to 4%.[44,55] TTS may be a direct manifestation of COVID-19 but may be also the consequence of the physical and emotional stress related to the COVID-19 infection leading to sympathetic overdrive.[56,57]

Acute pulmonary embolism (PE) in COVID-19 can be the consequence of the two interrelated processes, a hypercoagulable state responsible for large-vessel thrombosis and direct vascular and endothelial injury responsible for *in situ* microvascular thrombosis.[58] The hypercoagulable state has been characterized early during the pandemic by increased levels of D-dimers, fibrinogen, increased thrombin production, and elevated levels of Factor V and von Willebrand.[59] A cut-off of 2000 ng/mL for D-dimers and/or increase of more than 1.5 times was associated with poor prognosis defined by increased in-hospital risk for critical illness, venous thromboembolism, acute kidney injury, and death.[60] These findings strongly support pathogenic treatment with anticoagulation. In a large observational study, the early initiation within 24 h of admission of prophylactic heparin compared with no anticoagulation was associated with better 30-day survival in hospitalized COVID-19 patients (HR 0.73, 95% CI 0.66–0.81).[61]

The presence of pulmonary thrombosis may explain why hypoxaemia had only a poor correlation to impairment in lung compliance in patients with SARS-CoV-2 pneumonia.[59] Some studies reported a PE incidence of 27% in hospitalized patients with COVID-19[60] and 20% of PEs being diagnosed at admission with signs of severe right HF requiring ICU care.[61] ICU care is very often complicated by multi-organ dysfunction caused by systemic coagulopathy and microvascular thrombotic occlusion.[62,63] Furthermore, hypoxia-induced pulmonary vasoconstriction produces PH and RV dysfunction (*Figure 1*). PE remains a high-risk condition associated with higher rates of CS, ICU transfer, mechanical ventilation, and in-hospital death during COVID-19 infection.[60,62]

Because the viral spike protein-S use the angiotensin-converting enzyme 2 (ACE-2) receptor to enter into the human cells, including pulmonary epithelial cells,[64] it has been hypothesized that the use of RAASi (ACEinh/ARBs/ARNIs) may negatively impact outcomes of patients with COVID-19 by influencing the expression of the ACE-2 receptor.[65,66]

However, this hypothesis has not been confirmed as RAASi have protective CV effects,[67,68] and accumulating evidence confirmed that RAAS inhibitors should generally not be discontinued in patients with HF, because interruption is generally associated with increased mortality risk.[69–71] Discontinuation in patients' low blood pressure or haemodynamic instability should be made on a case-to-case basis.

Different HF phenotypes associated with COVID-19

Hospitalized patients with AHF

Acute decompensated HF Acute decompensated HF (ADHF) remained the most common phenotype and hospitalized patients often presented with severe signs of congestion, altered haemodynamic status, and elevated markers of myocardial injury.[9–12] COVID-19 is a strong trigger for HF decompensation in conditions of extensive pneumonia with hypoxaemia and hyperactivation of the systemic inflammatory response and sympathetic activity. Myocardial oedema, as result of hyperimmune activation and toxic effect of cytokine release, or less often due to direct virus infiltration into cardiac myocytes contributes to progressive contractility dysfunction.[29,50,51,72]

ADHF patients require initiation of decongestive therapies concomitant with antiviral medication. Efficiency of decongestion should be evaluated based on NP reduction, chest B-lines, and jugular venous pressure decrease, because dyspnoea and tachypnoea are not specific and persist after decongestion as result of pulmonary infection. Protective medical equipment hampers repetitive check-up visits for clinical congestion assessment, and pulmonary arterial catheter (PAC) monitoring is available only in a restricted percentage of patients admitted in ICU. In addition to the traditional assessment, urine output and urinary Na are highly indicative of satisfactory decongestion, particularly in the conditions of the limited physical examination.[73]

Isolated right HF Patients with ARDS or PE may develop isolated RV dysfunction secondary to pulmonary pre-capillary hypertension induced by hypoxemic pulmonary vasoconstriction and ventilator-lung injury.[74] A diffuse interstitial pneumonia secondary to the inflammatory status and cytokine release may enhance capillary permeability, leading to lung interstitial oedema and reactive arteriolar vasoconstriction with progressive increase of pulmonary pressure and pro-

gressive RV failure with ventricular–arterial uncoupling.[75,76] Similarly, recurrent pulmonary microembolization or massive embolism will suddenly increase RV pressure with progressive alterations of RV systolic performance and compliance.[77] Therapeutic anticoagulation with unfractioned heparin or low molecular weight heparin (LWMH) is mandatory when PE is diagnosed.[78,79] These patients require very cautious fluid loading to avoid RV overdistension and IV vasopressors in case of haemodynamic instability. Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) may be considered when RV dysfunction progress to CS.[80,81]

CS CS aetiology can originate from different mechanisms and early identification of pathophysiology is crucial for survival.[82] There are several reports of fulminant myocarditis secondary to COVID infection, and the common presentation is sudden haemodynamic deterioration with global hypokinesis and severe biventricular dysfunction leading to severe hypotension and multi-organ dysfunction.[75,76] Mechanical support (MCS) should be early deployed, because the response to IV inotropes is particularly poor in setting of myocarditis.[83,84]

CS may occur in the settings of Type 1 AMI, and registry data suggest AMI patients during the pandemic had longer ischaemic time, more severe Killip class, more mechanical complications, and a higher rate of in-hospital adverse events.[85,86] Early revascularization and effective anti-thrombotic therapies remain critical to improve outcomes in ACS and COVID-19 infection.[87,88]

CS in patients with COVID-19 should be similarly treated to those without COVID-19.[80] Literature does not suggest any benefit for therapeutic anticoagulation in HF patients in sinus rhythm and no other formal indication.[80] Although theoretically hospitalized COVID patients would probably benefit from high-intensity anticoagulation,[81] the pooled data from the several trials showed no survival benefit of more intense anticoagulation regimens.[82] This is probably due to the already increased bleeding risks that have been constantly described in CS patients.[85–88]

Current CS management requires rapid haemodynamic support with VA-ECMO or aortic counter-pulsation to improve systemic perfusion and to prevent multi-organ dysfunction.[78] In the case of refractory hypoxaemia or severe biventricular dysfunction, conversion to veno-arterial-venous (VAV) ECMO cannulation strategy can be considered.[89,90]

Acute pulmonary oedema Acute pulmonary oedema (APO) may occur in different settings such as acute myocarditis, ACS, or hypertensive emergency.[91] The sudden increase in hydrostatic forces leads to increased capillary permeability with rapid extravascular fluid accumulation that overcome clearance capacity of the capillary and lymphatics, resulting in increased water alveolar content.[92] Post-capillary PH occurs in a few hours from initial cardiac damage.[93] Diuretic doses must be carefully adjusted weighing the risks of hypovolemia and COVID-19-related hypotension.[73]

Supplying adequate oxygen to APO patients is a mainstay of treatment. When respiratory distress and/or hypoxaemia cannot be relieved by high-flow oxygen, non-invasive ventilation and prone positioning may be considered. If oxygenation and respiratory distress do not improve or worsen within a short time (1–2 h), endotracheal intubation and invasive mechanical ventilation should be promptly carried out. Mechanical ventilation of patients with COVID-19 requires lung protective ventilation strategy with low tidal volume at 4–8 mL/kg and high levels of positive end-expiratory pressure (PEEP) to prevent further lung injury.[8]

HF degree and severity The prevalence of HFrEF in patients COVID is not clearly established because it was usually included among other CV complications. In single-centre observational studies, it ranges from 4 to 23%[3,35] based on different criteria including NP measurement signs and symptoms of congestion and evidence of pulmonary congestion at chest radiography. Unfortunately, a large registry reporting detailed clinical and imaging data is lacking although EACVI recommendations invited to perform a multi-imaging analysis in symptomatic patients with known CV diseases by an appropriate use of instruments protection and disinfection.[36] For sure, a relevant percentage of ICU patients experienced an elevation of TnT and NP, and these two markers are associated with poor prognosis.[29,46] Whether the biomarkers' increase is synonymous of diffuse cardiac damage and impairment or just a consequence of septic status and restricted myocyte damage without evidence of functional cardiac impairment is not appropriately investigated. In this context, it is hard to identify the severity of HF, perfusional and congestion status, and the exact HF type. The lack of multicentre cross-sectional data comprising detailed clinical evaluation, haemodynamic monitorization, and standardized imaging protocols lead to diverse classifications of the disease's severity. Furthermore, the poor correlation between MRI findings and clinical severity, as well as the transient nature of the myocardial involvement in COVID infection, make difficult to link HF functional severity to cardiac structural damage.[40–42]

In outpatient settings, COVID infection contributes to functional cardiac deterioration with worsening NYHA class, irrespective of LVEF category. Alternatively, history of chronic HF may represent an aggravating factor for COVID infection, as it could further impair respiratory and systemic conditions, and contributing to the increased risk similarly to other CV diseases and risk factors.[48,50]

Ambulatory patients with chronic HF

HF with reduced ejection fraction The prevalence is unclear because it is usually not reported as separated disease

distinct from other CV complications. In observational studies, the prevalence of HFrEF in COVID patients ranges from 4 to 23%, based on different criteria including NP measurement, signs and symptoms of congestion, and evidence of pulmonary congestion at chest radiography.[4,27,28] The large differences in prevalence reflect the variability of the criteria used to diagnose HFrEF, their lower specificity for diagnosis of HF due to the absence of the cardiac imaging data, and the methodological differences between the studies. A relevant percentage of COVID-19 patients experienced an elevation of TnT and NP, and these two markers are associated with poor prognosis.[94,95] Whether the biomarkers' elevation parallels diffuse cardiac damage or is just a consequence of systemic inflammatory and septic status is not appropriately investigated.[96] Cardiac MRI studies demonstrated a higher percentage of oedema in patients with infection even if pauci-symptomatic.[97] The myocardial inflammation is probably transient and proceeds to complete recovery within few weeks. Only few cases have been described as fulminant myocarditis leading to CS in the recovery period.[68,98] HF aggravates clinical course of COVID infection as it could impair respiratory and systemic conditions.[99]

Heart failure with preserved ejection fraction Heart failure with preserved ejection fraction (HFpEF) is a heterogeneous syndrome including a broad population with different pathophysiological triggers, CV risk, and demographic features, resulting in large a spectrum of different phenotypes.[100] Some authors suggested different HFpEF subtypes linked to cardiometabolic alterations, body size conformation, or peripheral maladaptation.[101] These appraisals may be associated to the presence of diabetes, hypertension, chronic kidney diseases, obesity, skeletal muscle metabolism, and vascular rarefaction.[102] A recent study demonstrated a high HFpEF score in COVID-19 patients, associated with significant increase of cardiac biomarkers and more advanced diastolic dysfunction.[102] These findings reinforced the inflammatory hypothesis involving myocytes and vascular districts common in both infection and HFpEF.[102–104] Reduced vascular compliance in the skeletal muscle and the respiratory systems may be the common pathways linking infection with HFpEF onset.[101–104] Additionally, patchy tissue myocardial inflammation and oedema may increase cardiac stiffness and impair relaxation with potential consequences after acute phase resolution.[105,106] Unfortunately, there are no large-scale reports providing morphological data at seriate follow-up in patients with both HFpEF and COVID-19 infection.[107]

Pre-HF stages The recent definition of HF highlights the importance of a combined laboratory and imaging criteria to identify cardiac structural abnormalities.[108,109] The pre-HF phase elapses for long period, but cardiac or extracardiac triggers, supraventricular arrhythmia, hypoxaemia, worsening renal function, and COVID infection may precipitate the natural course and impair the haemodynamic status.[20,24] The long-term consequences of COVID-19 infection in Stages A and B of HF remains unclear,[110] although baseline characteristics may influence HF development.

How to differentiate between AHF and ARDS?

Distinguishing between ARDS COVID-19 and decompensated HF is difficult, and the presence of one does not preclude the other. However, identifying whether one or both is present will appropriately direct management in early phases.[19,20] The common symptoms of both diseases are dyspnoea and fatigue. However, COVID-19-induced dyspnoea is often alleviated assuming prone position, and conversely, in patients with cardiogenic dyspnoea, orthopneic position improves symptoms.[96,111]

Up to 80% of patients admitted with COVID-19 have or have had recent fever, which is often resistant to antipyretic treatment, and may often be associated with a persistent cough and loss of taste and smell and with gastrointestinal symptoms. Isolated pulmonary crackles associated with tachypnoea are much more suggestive of a respiratory infection,[91] but these findings are not entirely specific to COVID-19, and blood cultures should be obtained in all febrile patients. An oxygen saturation below 90% and hypocapnia with respiratory acidosis suggest ARDS or related thromboembolic complication, but the blood gas test has low accuracy to discern the two forms.

A normal chest X-ray does not exclude either a SARS-CoV-19 pneumonia or HF, and a chest-CT is often required.[112]

ECG abnormalities such as sinus tachycardia or atrial arrhythmias are common in both ARDS and AHF because of hypoxaemic status, fever, and electrolyte unbalance.[113]

Elevated levels of inflammatory markers (C-reactive protein or ferritin) associated with relative lymphopenia raise the clinical suspicion of a COVID-19. Troponin is often increased in patients with worsening HF and in those with COVID-19 with or without heart disease. However, higher serum concentrations indicate a greater risk of cardiac and non-cardiac complications.[94,114,115] D-dimer and fibrinogen, reflecting abnormal activation of coagulation and fibrinolytic systems, may be helpful to identify those with a higher risk of thromboembolic events and death, but not to differentiate between the two conditions.[27] A mild elevation of NPs has been described in COVID-19 patients and is related to the direct multi-organ damage related to infection or increased pulmonary pressure occurring in ARDS. Elevated NPs are associated with a poor prognosis.[116,117]A recent meta-analysis found NP elevation within the first 24–48 h after admission significantly associated with higher disease severity, including the need for ICU transfer or mechanical ventilation and increased mortality in COVID-19 patients.[118] Low plasma concentrations of NP may exclude a diagnosis of HF and suggest

a good outcome among those with COVID-19 with or without underlying heart disease.[24,111,116]

A detailed echocardiographic examination is recommended to identify the primary cardiac structural abnormality[6,114,118] and to evaluate LV and RV size and function and PH.[23,29] Notably, a recent analysis based on echocardiographic and MRI suggests that patients with elevated troponin had evidence of reduced myocardial strain and myocardial oedema, even if poorly symptomatic.[119]

Lung ultrasound scan (LUS) may be useful to rule out substantial pulmonary involvement in patients with suspected COVID-19 and HF, but detection of these alterations requires greater skills than the standard LUS approach.[120,121] Distinguishing features for COVID-19 may be an asymmetric or unilateral distribution of B-lines, commonly subpleural, thickening of pleural line, pleural discontinuity, and consolidations. In AHF, B-lines are symmetrically distributed in almost all chest sites with prevalent localization in the inferior lobes.[6] Beside LUS, a point-of-care ultrasound approach including inferior cava vein measurement and jugular vein distention may provide additive value for diagnostic differentiation.[118]

Chest cardiac tomography (CT) can identify in the early phase of infection pulmonary infiltrates that are often limited in extent and usually limited into subpleural district with ground-glass pattern that implies interstitial rather than alveolar oedema and fibrotic consolidation during late ARDS. Micro-embolization or macro-embolization can be visualized in patients with thrombotic complications, whereas in severe cases, main pulmonary artery branch dilatation is observed.[122,123] In patients with AHF, signs of post-capillary hypertension and alveolar oedema are the main diagnostic signs. Additionally, cardiac dilatation and distention of central veins are typical for AHF.

Cardiac magnetic resonance, when available, reveals a detailed segmental and/or global myocardial contractility reduction with signs of diffuse extracellular matrix deposition during T1 mapping scan. Conversely, during isolated COVID-19, cardiac structure and function is usually maintained; no signal defect is visualized except for restricted oedema associated with mild pericardial effusion[124] (*Figure 2*).

Management of patients with AHF and COVID-related ARDS poses significant challenges. Several concerns arise from cardiac side effects of current antiviral and anti-inflammatory drugs and potential interactions between these drugs and cardiovascular agents[125–134] (*Table 3*).

AHF management, including IV vasoactive therapies and MCS, should be tailored according to the clinical profiles (ADHF, APO, RHF, CS) and considering the type and severity of pulmonary involvement.[74,78,80] Intensive care management in patients needing ventilatory support is particularly

Figure 2 Diagnostic process for differentiation between dyspnoea due to ARDS and cardiac dyspnoea: a detailed clinical laboratory and imaging algorithm could facilitate early diagnosis. ACEi, angiotensin-converting enzyme inhibitors; AHF, acute heart failure; ARB, angiotensin receptor blockers; ARDS, acute respiratory distress syndrome; ARNI, angiotensin receptor neprilysin inhibitors; CT, chest tomography; CV, cardiovascular; GI, gastrointestinal; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; IABP, intra-aortic balloon pump; MCS, mechanical circulatory support; MI, myocardial infarction; MV, mechanical ventilation; VA-ECMO, venous arterial extracorporeal membrane oxygenation; VV-ECMO, veno-venous extracorporeal membrane oxygenation.

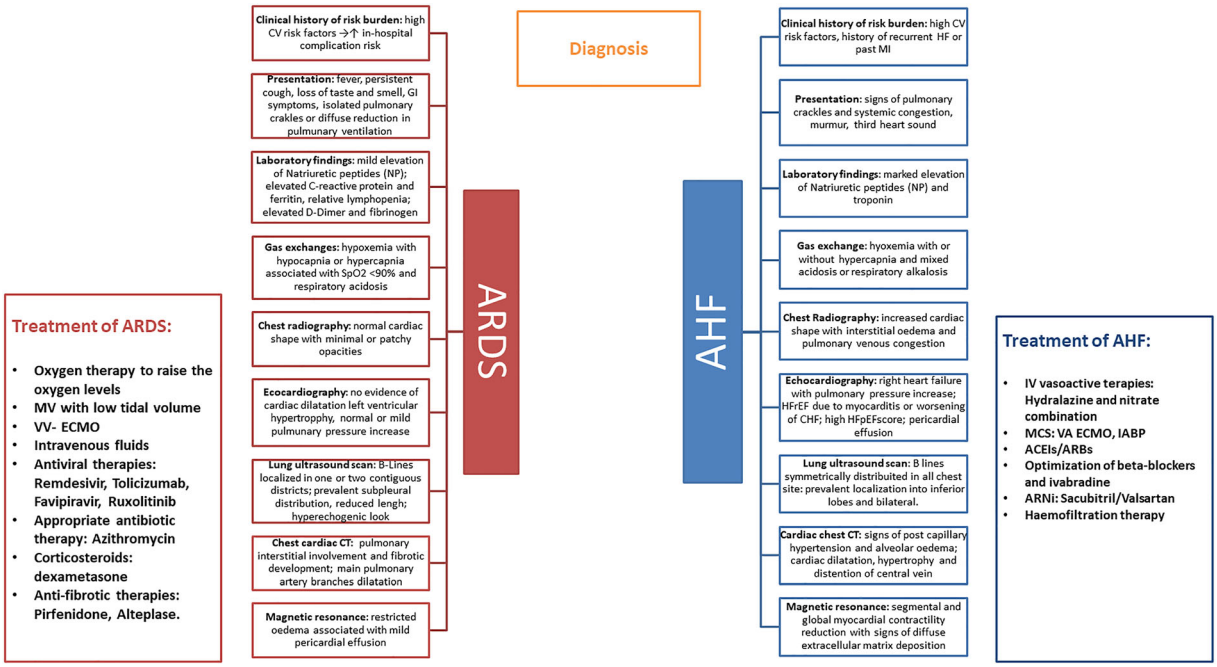


Table 3 Current agents administered during COVID-19 and potential adverse effects/interactions with cardiovascular drugs

COVID-19 treatment	Interaction with cardiac medication	Potential side effects	Monitoring
Dexamethasone Methylprednisolone	Warfarin antihypertensive, metformin, SGLT2i	↑Vascular fragility and haemorrhagic events ↑Vascular resistance ↑Blood pressure and water retention ↓Hypoglycaemic effects	Coagulation state evaluation Blood pressure monitoring Increase antidiabetic therapy
Remdesivir Lopinavir/ritonavir	Statin Anticoagulant Antiarrhythmic ASA Clopidogrel Prasugrel	↓Liver function and drug excretion Q-T interval alteration Prolonged electric potential duration Inhibition of CYP P450 and CYP3A4 Impair vasomotor function and reduce eNOS expression with CV and haemorrhagic risk increase	Close hepatic and coagulation assessment, platelet activation Avoid apixaban and rivaroxaban, recurrent ECG monitoring
Tocilizumab, anakinra, anti-interleukin drugs	Cyclosporine Azathioprine Aspirin, anticoagulant LVAD	Endothelial dysfunction Prothrombotic state, myocarditis Immunosuppressive action in THX	Serial D-dimer and fibrinogen assay, TnT, and NP measurement Reduce immunosuppression
Pirfenidone	Amiodarone Propafenone Statins	CYP1A2 interaction Angiotensin 2 inhibition	Modulate anti arrhythmic treatment, lipid profile, and liver function monitoring Avoid in low blood pressure and CS
Colchicine/ hydroxychloroquine	Antiarrhythmic drug Classes I and III, digoxin	Altered ion channels QT prolongation ↑Risk for torsades des pointes and atrial fibrillation	Look for electrolyte unbalance and avoid use in patients with high arrhythmic risk burden
Molnupiravir Paxlovid	Statins Antiarrhythmic drugs Sildenafil Ranolazine Alfuzosin	CYP3A substrate ↑Antiviral agent plasma concentration, potential vasopressor effects	Avoid contemporary use of Class I and Class III antiarrhythmic drugs, risk for myopathy, avoid in pulmonary hypertension Type 1

ASA, aspirin; CYP, cytochrome; LVAD, left ventricular assistance device; NP, natriuretic peptides; SGLT2i, sodium–glucose co-transporter 2; THX, heart transplantation; TnT, troponin.

challenging and depend on ARDS phase. In early ARDS, non-cardiogenic pulmonary oedema, shunt-related hypoxaemia, and reduced ventilatory area size account for low respiratory compliance.[135] Late ARDS phase is characterized by disproportionate diffuse collagen deposition with amount of fibrotic component and extensive lung consolidation.[136] Another feature is the coagulation cascade activation, with widespread micro-thromboses and macro-thromboses in the lung and in other organs.[136] This aspect requires specific coagulation test monitorization for early prothrombotic identification and anticoagulant treatment. Some antiviral and immunosuppressive agents commonly employed to reduce inflammatory status and cytokine cascade may aggravate this condition and should be immediately interrupted. Specifically, lopinavir/ritonavir decreases the anticoagulant effects of direct Xa inhibitors, such as apixaban, rivaroxaban, and edoxaban by interfering with cytochrome P450; tocilizumab may directly induce coagulation factors' overexpression. Of note, specific attention may be focused on eventual evidence of RV dysfunction and PH subsequent to lung mismatch and embolism.[137]

In case of early ARDS, when hypoxaemia was not corrected by high flow oxygen, ventilatory support includes the use of high levels of positive end-expiratory pressure (PEEP), recruiting manoeuvres, and prone positioning that provides a more homogeneous ventilation in peripheral districts, but should be avoided in patients with severe right HF. FiO_2 (inspiratory oxygen fraction) boosting may avoid inspiratory effort in case of persistent hypoxaemia.[138] In late ARDS, early intubation with a lower PEEP (8–10 cm H_2O) appears more appropriate in order to minimize ventilator-induced lung injury.[125,139]

Special HF populations

Advanced HF

The pandemic has changed both routine hospital admission for HF and planned tests requiring specific skills such as heart catheterization. Interventional therapeutic procedures, such as left ventricular assist device (LVAD) and ICD/CRT implants, have been substantially delayed due to the changes in the priority of healthcare delivery[140,141] and focusing resources in patients with more urgent needing.[142,143] Transitioning to telehealth in order to reduce hospital admission may be advocated in this group of patients.

Heart transplant

Regarding the management of HT waiting list, COVID-19 survivors on the heart transplant (HT) list are required to have

two negative tests after a 14-day interval in order to proceed with transplantation.[144] Only hearts from donors who are negative for COVID-19 infection must be considered for transplantation.

HT patients have an increased likelihood of developing viral infections as result of chronic immunosuppression,[145] and atypical presentation makes the diagnosis difficult. Furthermore, distinguishing rejection from viral myocardial involvement may be difficult. Indeed, microscopic picture of rejection is similar to the infection, with activated T-cell and macrophage infiltration in infected myocardium.

In patients with HT, immunosuppressive treatment should be administered with caution to avoid immunodeficiency status in case of COVID infection. Reducing dose of cyclosporine/tacrolimus and potentially interrupting for few days antimetabolite (mycophenolate and azathioprine) treatment may be considered in acute phase of infection,[146] but these should be immediately resumed when lymphocyte count is increasing and patient recovers from infection. Waiting a patient to become negative at PCR testing may last longer than 7–10 days, and holding mycophenolate for extended periods leads to inacceptable risk of rejection. Particularly, in patients with recent HT, reducing immunosuppression is associated with much higher risk of rejection than late after transplantation.[147] To note, there are no validated strategies to guide immunosuppression in HT recipients exposed to COVID-19 infection. The number of endomyocardial biopsies significantly declined during pandemic, and the evaluation of rejection may be monitored by alternative less invasive methods such as genetic and metabolomic profile or cell-free DNA testing.[148]

HT recipients are at increased risk for morbidity and mortality with COVID-19 based on evidence that respiratory illnesses are associated with greater disease severity and prolonged viral shedding in this population. COVID-19 related mortality in HT recipients has been reported at 20–25%.[145,149] For these reasons, vaccination should be encouraged in all HT patients, and early treatment with recombinant antibodies should be considered in the case of COVID-19 infection.

HF patients with implantable devices

These patients are at high risk for severe COVID infection and cardiopulmonary complications due to the older age and high CV risk profile.[150] Due to increased thrombotic status, a high rate of pulmonary thrombotic events, leading to progressive RV dysfunction, has been reported.[143,151] A strict check-up programme and specific telemonitoring devices should be applied in this setting in order to optimize load conditions, as well as to avoid COVID transmission and potential bacterial superinfections.

HF patients with LVAD

LVAD patients presenting with COVID-19 infection represent a high-risk category, due to abnormal inflammatory profile and possible LVAD complications. Recent case series study showed that 60% of patients require hospitalization with high mortality rate (20%).[143] Although cardiac output provided through the VAD remains theoretically steady even in the setting of a systemic infection, optimizing preload and afterload is very important. If haemodynamics are compromised, various LVAD-related complications can ensue, including RV failure and pump thrombosis, as well as low flow and suction events.[151,152] Close monitoring of anticoagulation is mandatory.

In severe cases with refractory hypoxaemia and ARDS, mechanical ventilation with patients placed in prone position has been recommended by several consensus documents.[139]

HF patients with valvular heart disease

COVID-19 pandemic caused a significant delay in treating valvular heart disease (VHD) in patients with HF. Reorganization of healthcare resources and implementation of algorithm for patients' prioritization based on the severity of their VHD, life expectancy, complexity of the intervention, and resources available have been proposed.[140] Compared with surgery, percutaneous procedures may be associated with a lower risk of COVID-19 infection mainly due to the lower length of stay.

HF and COVID vaccination

Because HF patients are at high risk for complications, vaccination against COVID-19 remains the best approach to control infection, and it is indicated in all patients with HF, including those who are immunocompromised (e.g. HT receiving immunosuppressive therapy) and patients with multiple associated diseases.[153] Although it is preferable to vaccinate HF patients in a stable condition, treatment optimization should not delay vaccination, as recommended by the recent HFA consensus document on vaccination position paper.[154]

One study recently reported permanent cardiac consequences of infection even in vaccinated patients.[155] The late findings depend on previous infection severity, the entity and type of CV complication occurred during infection, the intensity and type of care, and the time elapsing from the infection and subsequent evaluation.[156] The post- COVID syndrome has been classified as a specific condition characterized by fatigue, chest pain, reduced exercise tolerance, and dyspnoea after discharge from hospital. Recent consensus documents focus on some open questions that cardiologists are going to face during the next months in a general cardiology outpatient clinic, in particular how to evaluate a post-consequences.[157] It is not known if post-COVID syndrome is the consequences of CV and lung complications and how to manage it. A recent electronic multisystem health analysis showed that a relevant percentage of previously hospitalized patients had various cardiac lung and metabolic abnormalities, confirming the need of specific healthcare service for monitoring these subjects.[158]

Figure 3 Remaining concerns and dilemmas in HF diagnosis, special population treatment, profile identifications, and research priorities. HF, heart failure; LVAD, left ventricular assist device.

Self-care for HF patients during pandemic

During the pandemic, HF patients should be advised for self-care management. This is important for two main reasons: the isolation and social distance imposed by lockdown and the optimal success of telemedicine that necessarily comprise patient's collaboration, lifestyle education, treatment adherence and careful symptom monitoring. Health professionals delivering care should be focused on recommendation for nutrition, physical activity, medication adherence, psychological status, correct distancing, and symptom monitoring and symptom management[159] (*Figure 3*).

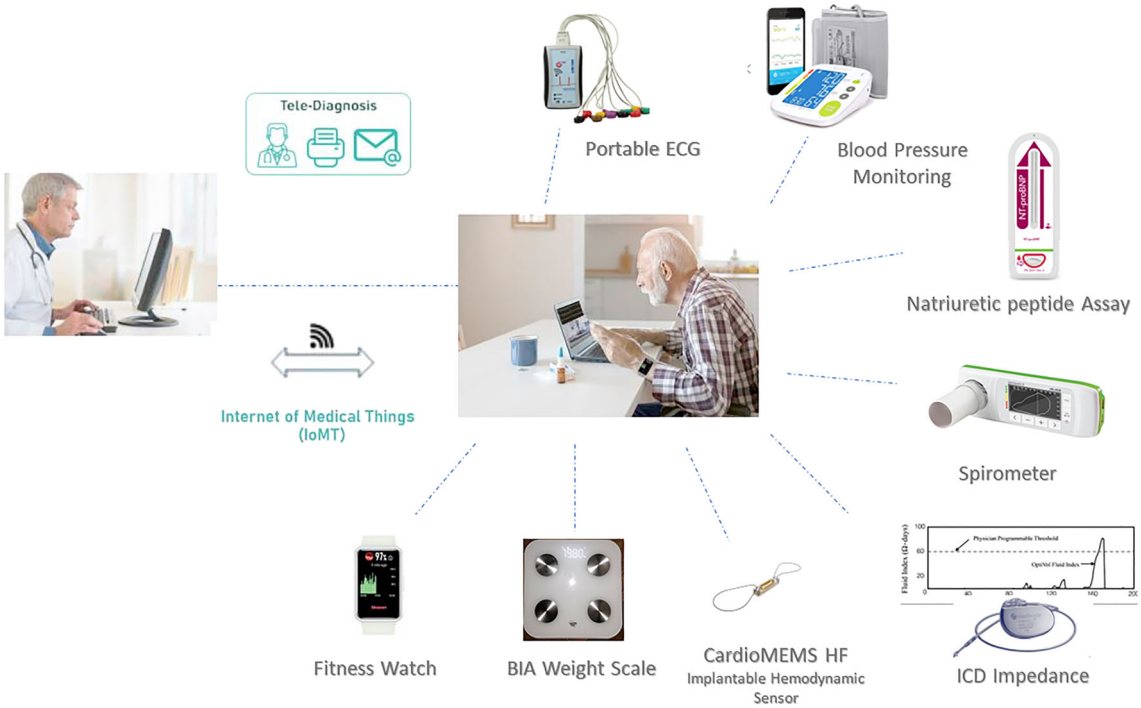
The role of telemedicine

The pandemic provided an input for a larger use of the remote monitoring technology. During the pandemic, remote monitoring of the arrhythmias in ICD recipients[160] coupled with bio-impedance and pulmonary pressure measurements has shown to be effective in preventing and reducing of HF hospitalizations.[161] CardioMEMS' versatility has made it an option for PAP monitoring during the pandemic when clinic visits decreased.[162]

Multi-sensor implanted devices including measurements of heart sounds, temperature, markers of ventilation, and thoracic impedance may provide an early detection of COVID-19 and may distinguish between acute presentations of COVID-19 and cardiac decompensation.[160,163]

Unfortunately, these options are restricted to a small percentage of the whole HF population, and most of patients are constricted to self-monitoring by evaluation of simple clinical parameters such as body weight, daily diuresis, and heart rate.[164] Widespread use of home telemonitoring including simple variables, such as respiratory rate, chest impedance, single lead ECG, and video scans for direct evaluation of jugular vein distention and peripheral oedema would ensure a more regular and continuous delivery of care and will limit risk exposure for both patients and healthcare professionals.[165] A wider adoption of point-of-care testing

Figure 4 Potential applications of telemedicine by a tailored programme by several wireless and interned application programmes addressed to identify common HF symptoms, blood test, ECG, and invasive and non-invasive cardiac pressure. ECG, electrocardiogram; ICD, implantable cardioverter defibrillator.



with NPs in the community would identify those at greater risk of deterioration and death and may determine the priority for a specialist consultation. Further introduction of Internet of medical things (IoMT) may avoid traditional diagnostic tools and complex laboratory-based diagnosis process.[166]

Economic support for implementation of innovative platform and remote monitoring should be advocated and settled according to the geographical area, hospital resources, and level of HF centre.[1,21] Improving access to care should be a top priority of the agenda for governments and medical personnel, to ensure that patients with HF, with or without COVID-19, will receive the adequate healthcare (*Figure 4*).

In a less developed countries with limited health resources and reduced Internet coverage, these techniques are less applicable. Similarly, in these geographic areas, diagnostic and therapeutic assistance is limited, and more recent antiviral agents not available. In these zones, the only real treatment remains prevention by diffuse vaccination booster.[167]

Palliative care

With the rise in patients with AHF and COVID-19 coupled with limited intensive care beds, many HF professionals had to engage in difficult prognostic and ethically challenging conversations. As family members were prohibited from attending the hospital, support could only be facilitated virtually, so patients navigated advanced care planning and contributed to complex decisions.[168–170] Healthcare professionals should aim to integrate palliative care earlier into HF management so that the patient has the opportunity to discuss his/her preferences and wishes with family members to ensure optimal end-of-life care.[171]

Conclusions

Patients with HF and COVID-19 have an increased risk of mortality because they are more likely to develop severe complications from SARS-CoV-2 infection and because of the disruption of access to cardiology services. Some innovative services with remote evaluation and home telemonitoring provide a more regular and continuous delivery of care, reducing risk exposure to both patients and healthcare professionals. From the perspective of the recrudescence of the pandemic, the cardiology community and HF specialists should be better prepared to utilize precise diagnostic algorithms capable of early recognition of different HF subtypes and to address the most appropriate management for specific clinical settings.

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