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Screening, detection, and management of heart failure in the SARS-CoV2 (COVID-19) pandemic

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Abstract
Observational studies suggest that a heart failure (HF) diagnosis carries a poor prognosis in subjects with severe SARS-CoV2 (COVID-19) infection, but it is unknown whether this association reflects direct myocardial damage due to COVID-19 or the consequence of preexisting cardiac defects and related cardiovascular disease (CVD) risk burden. Although the close relation between CVD and COVID-19 outcomes is well established, contrasting data exists about the occurrence of HF complications during COVID-19 infection. Therefore, a specific algorithm focused on diagnostic differentiation in acute patients distinguishing between acute HF and acute respiratory distress syndrome related to COVID-19 is needed. Further, several concerns exist for the management of patients with an uncertain diagnosis and acute dyspnea, the exact relationship existing between COVID-19 and HF. Therefore, the treatment for subjects with both COVID-19 and HF and which criteria may be defined for domiciliary or hospital management, remain poorly defined. Herein, we describe practices to be adopted in order to address these concerns and avoid further virus spread among patients, l and their familiars involved in such patients' care.

Keywords SARS-CoV-2 · COVID-19 · Acute heart failure · Hospitalization management

Introduction

The coronavirus disease 2019 (COVID-19) pandemic originated in Whuan, China, and is associated with severe acute respiratory syndrome (SARS), resulting in significant mortality risk, despite ventilation support therapy [1]. Many patients admitted to the intensive care unit (ICU) for primitive respiratory deterioration have underlying cardiovascular disease (CVD). Common factors associated with mortality are hypertension, obesity, history of coronary disease, heart failure (HF), atherosclerosis, diabetes, and related metabolic disturbances [2, 3]. Although the close relation between CVD and COVID-19 outcomes is described and well established, no extensive data exists about HF complications occurring during COVID-19 infection. The question regarding whether COVID-19 infection leads to hemodynamic HF deterioration or if chronic HF (CHF) is itself a predisposing factor for ARDS occurrence, remains unexplored [4, 5]. Interestingly, during the period of high COVID-19-related admissions, a reduction in CVD hospitalizations has been noted; this could contribute to an increased rate of cardiovascular death outside of the hospital setting [6, 7]. These topics merit further discussion in order to determine the best management for patients and to safeguard healthcare personnel involved in such patients' care.

Diagnostic tests and screening in patients with acute dyspnea

COVID-19 infection manifestation ranges from modest flu to SARS, which can result in mortality increase [5, 8]. The wide clinical symptoms and the lack of effective management lead to a heterogeneous approach based on personal experience rather than large solid registries or data from clinical trials [4, 9]. A majority of acutely ill patients are not diagnosed

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from community testing, but rather from in-hospital testing after presentation. Because acute HF (AHF) syndromes are characterized by sudden symptom onset, increasing dyspnea, and orthopnea requiring prompt hospitalization, as well as acute respiratory failure due to COVID-19 infection, there is an impelling need to distinguish the two syndromes by an accurate diagnostic protocol. In both cardiac and respiratory complications (AHF and ARDS, respectively), dyspnea represents the most common symptom [10, 11]. Therefore, a customized diagnostic route could be useful in order to reduce contact between AHF and ARDS patients and to avoid in-hospital infection spread. We suggest that the following diagnostic screenings may be appropriately considered during COVID-19 era for patients presenting with the aforementioned symptoms and history: respiratory rate, natriuretic peptides, lymphocyte count, C-reactive protein, D-dimer, fibrinogen, electrocardiogram, and chest X-ray. Analysis of the arterial blood gas could be performed as an additional test to identify the culprit behind the patient's destabilization [12]. Additionally, lung ultrasound could be used as an alternative method to discern a pulmonary lesion associated with interstitial pneumonia or increased comet related to water content [13]. Using these principles, we present an algorithm which assigns the culprit of destabilization as either AHF or COVID-19 with high, uncertain, or low probability. For instance, in the setting of elevated natriuretic peptide levels associated with significant chest X-ray, electrocardiographic alterations, congestion signs, history of previous HF episodes, normal hemocrome and leucocyte formula, D-dimer and fibrinogen levels into the normal range, there is an high probability for AHF diagnosis. Whereas, in the case of increased C-reactive protein associated with lymphocytopenia, increased respiratory rate, elevated D-dimer and fibrinogen, and significant hypoxemia, the diagnosis of respiratory failure related to COVID-19 is high. Conversely, when natriuretic peptide levels (NP) are low, chest X-ray is negative, and congestion is present, the probability of HF is low. Similarly, in the case of coagulative test alterations, normal arterial blood gas, and lack of lymphopenia, even in the presence of increased C-reactive protein associated with radiographic signal suggestive of pneumonia, the probability of COVID-19 lung involvement is low [14]. In order to differentiate the two diseases, the blood gas analysis became mandatory: hypoxemia and hypocapnia associated with low oxygen saturation below 90% respiratory acidosis are specific signs of respiratory distress or related thromboembolic complication; conversely, hypoxemia without CO_2 alteration and relative acidosis or respiratory alkalosis are more typical for hemodynamic derangement [15]. We did not include troponin among laboratory variables because it could be altered in the settings of myocardial cell loss due to increased parietal stress and in reduced oxygen delivery secondary to altered pulmonary gas exchange. Notably, this biomarker is not specific for diagnostic differentiation, although it has demonstrated a remarkable prognostic significance [16, 17].

Among these two pictures, there are several gray situations in which some clinical variables are suggestive for HF, while others are for COVID-19 infection. In those cases, further investigation with more advanced diagnostic tools is required as a second step in the algorithm. Echocardiography may be performed when AHF is more likely, and CT scan may be needed in cases suggestive of interstitial pneumonia and related ARDS [18]. Both diagnostic examinations should be executed in safe conditions with the previous and succeeding rooms disinfected, and mono use material for sound and scan protection [19]. We report below a detailed framework of potential possibilities occurring in clinical practice. In all the other cases, both diagnosis of AHF and COVID-19-related ARDS may be excluded and physicians could shift toward other potential diseases responsible for incremental dyspnea.

Table 1 summarizes the accounted parameters and the related specific variables included in this flowchart.

Table 1 Clinical, laboratory and diagnostic tests useful for initial screening and diagnosis in patients presenting with dyspnoea

High HF/low ARDS-COVID	Uncertain HF ARDS-COVID	Low HF/High ARDS-COVID
• ↑ BNP/NTproBNP • Congestion signs • History of HF • ECG alterations • Positive chest X-ray • Low C-reactive protein • No hemocrome alteration • Normal D-dimer and fibrinogen	LikelyAHF • Mild ↑BNP/proBNP • None or 1 congestion sign • Mild hypoxemia without hypocapnia • Chest X-ray doubtful • Positive chest B lines ultrasound Likely ARDS • ↑C-reactive protein • Mild ↑BNP/proBNP increase • Hypoxemia with mild hypocapnia • ↑D-dimer and fibrinogen increase • Limited chest	• ↓ BNP/NTproBNP • Relevant hypoxemia • Tachipnea • Respiratory acidosis • ↑ C-reactive protein • ↑ Procalcitonin • Localized subpleural and scissural chest X-ray signs • Hemocrome alteration with lymphocitopenia • No history of HF • ↑ D-dimer and fibrinogen

Care management and ward distribution according to clinical presentation

Beyond the initial screening and diagnostic course, many concerns remain unclarified regarding the location and distribution for patients with both AHF and COVID-19 infection, with diagnosis unknown at admission. First, it is imperative to distinguish separate routes for AHF patients with confirmed/suspected COVID-19 and those negative for COVID-19. In the case of a positive test result or those exhibiting symptoms and awaiting a test result, patients should be hospitalized in a well-defined and isolated "red zone" area located in the emergency department: in this site, all patients presenting with dyspnea can be screened by the application of the abovementioned diagnostic algorithm. All the diagnostic tests might be performed in safe condition with face mask worn by patients and health staff (nurses, technicians and doctors). Additional protection of hospital personnel may provide showerproof, single-use coat, gloves, facial protection and FFP3 mask [20]. AHF diagnosis should be performed according to guideline-directed criteria, including traditional blood sample and natriuretic peptide measurement, electrocardiogram and chest X-ray [21]. Lung ultrasound provides additive accuracy in diagnostic differentiation and interstitial pneumonia identification [22]. The second step may require chest CT scan execution in case of high probability for interstitial pneumonia and related ARDS. Conversely, echocardiographic examination could be performed in patients with high HF probability [23].

In AHF COVID-19-positive patients, echocardiography should be performed only in the case of new-onset AHF or unexplored reasons of hemodynamic destabilization such as suspected pericardial tamponade, suspected acute pulmonary embolism, suspected acute coronary syndrome (ACS) and suspected acute valvular heart diseases [24]. In case of contemporary occurrence of infection and AHF, treatment should be customized according to prevalent respiratory or cardiogenic disorder. Because of some interaction between antiarrythmic, anti-inflammatory and antiviral drugs, a consensus treatment decided by cardiologist advisor, infectiologist and pulmonologist may be encouraged. Therefore, because many patients are treated with corticosteroids, which universally influence idro saline and glycemic status, daily diuresis, electrolyte imbalance and metabolic profile may be monitored. Both ARDS and hemodynamic complications should be managed in the "red zone" reassessed as ICU wars. In case of pulmonary embolism or diffuse intravascular coagulopathy suspicion by laboratory measurements (i.e. D dimer and fibrinogen increase associated with persistent hypocapnia and hypoxigenation), an echocardiographic examination to ascertain right ventricle (RV morphology and noninvasive pulmonary pressure estimation) [25]. In case of AHF patients with uncertain COVID-19 diagnosis, it became priority to avoid eventual diffusion among patients and healthcare workers. In all these case, it could be worth to identify a "neutral zone" within the emergency department in which patients can wait for laboratory swab or blood culture response avoiding contagious risk. In this "gray zone," patients should maintain a minimum distance of 2 m and dedicated medical staff should wear showerproof single-use coat, gloves, facial protection, and FFP3 mask [26]. A dedicated cardiologist consultant should support ED clinicians for AHF management during this period following similar procedures and modalities of COVID-dedicated staff. In case of positive coronavirus nasopharyngeal swab, AHF patients should be addressed in "red zone," whereas in case of negative nasopharyngeal swab test, AHF patients could be transferred to the cardiology ward ("green zone") and managed using traditional modalities. In the "green zone," cardiologists and other healthcare workers should keep surgical masks and gloves. All patients may wear surgical masks during hospitalization period. The isolation and disinfection must involve not only healthcare staff but also the environments in which patients remain during transitional period independently from virus test response. Accordingly, ultrasound and radiographic devices must be cleaned with alcoholic solution before and after every examination. All parts nearest to the patient (bed employed for examination, probe, and radiographic scan) must be covered by a plastic mono use protection [19, 27, 28] (Fig. 1).

During early hospitalization, the role of cardiac biomarkers (D-dimer, troponin, and NP) and all the other are key features for both cardiovascular complication recognition and diagnostic differentiation [14, 29]. Therefore, specific laboratory biomarkers should be systematically measured in all patients to recognize treatment efficacy, clinical trend, and cardiovascular and respiratory complications related to COVID-19. Several studies have been reported about potential virus-related complications although a more detailed prevalence is relatively unknown: acute pulmonary embolism, myocarditis, ACS and arrhythmic events, acute kidney injury, sudden cardiac function deterioration, and intravascular disseminated coagulopathy are widely described, but it remains unclear which patients are much more prone to develop current complication and the exact reason of their significant reduction over the last summer period [12, 30] More specifically, although recent magnetic resonance study demonstrated that 60% of COVID patients experienced some signs of cardiac involvement, it remains unknown if current findings lead to significant cardiac dysfunction and which is the primary pathophysiological driver of deterioration (systolic or diastolic dysfunction) potentially causes for HF occurrence during COVID-19 (Fig. 2).

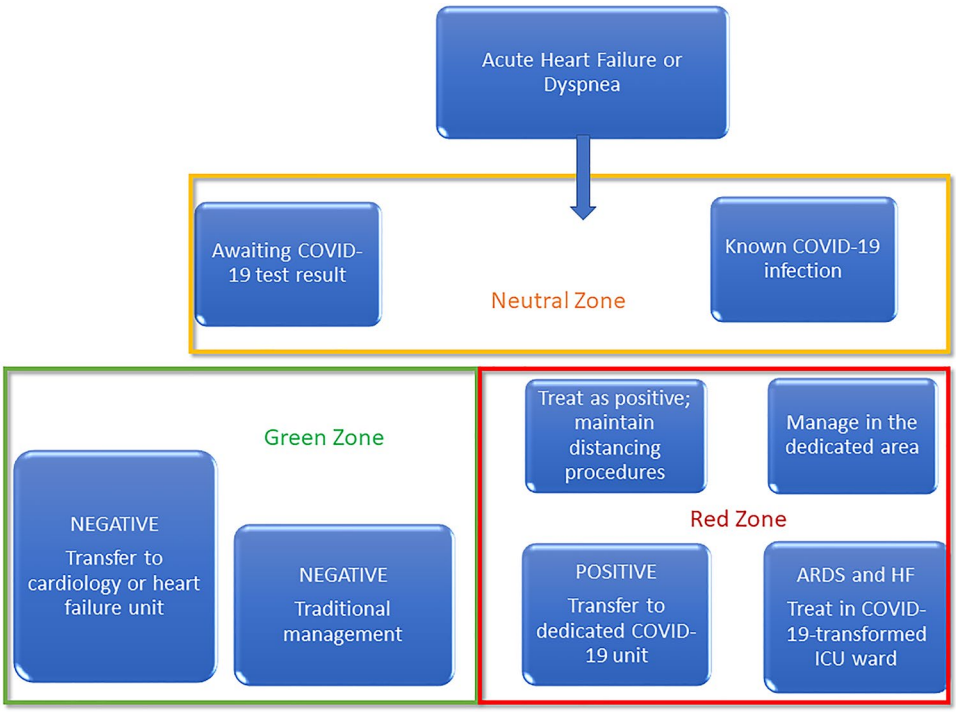

Fig. 1 Diagnostic course and in hospital permanence for patients admitted with acute dyspnea. Patients awaiting serological or swab test might stay in neutral uncontaminated ward, after virus test they can accede in the appropriate area for a customized management

The broad clinical scenario and the lack of a standardized management, lead to an inhomogeneous approach mainly based on personal experience local hospital practice, rather than solid registries and protocols. Before, to address the relationship existing between COVID-19 and HF, it appears priority to recognize the specific diagnostic algorithm for each diseases and the related course that patients may follow during hospitalization. Of note, diagnostic management should be started in each patients presenting with acute dyspnea at ED, with individualized laboratory and imaging tests. Current approach may lead to a better diagnostic differentiation as well as improvement of in hospital avenues with virus spread avoidance.

Alternative management for chronic heart failure patients

Over the last several months, the COVID-19 pandemic has negatively modified patients' attitudes toward receiving routine healthcare and it has modified medical staff conduct [31]. Many patients with CHF followed medical advice (and government indication, if relevant) and carefully isolated, avoiding crowded places and potential sites for virus infection and transmission. While doing so, many did not (or could not continue to) respect HF guidelines regarding diet and exercise: many patients increased their body weight and total body fat, while reducing their

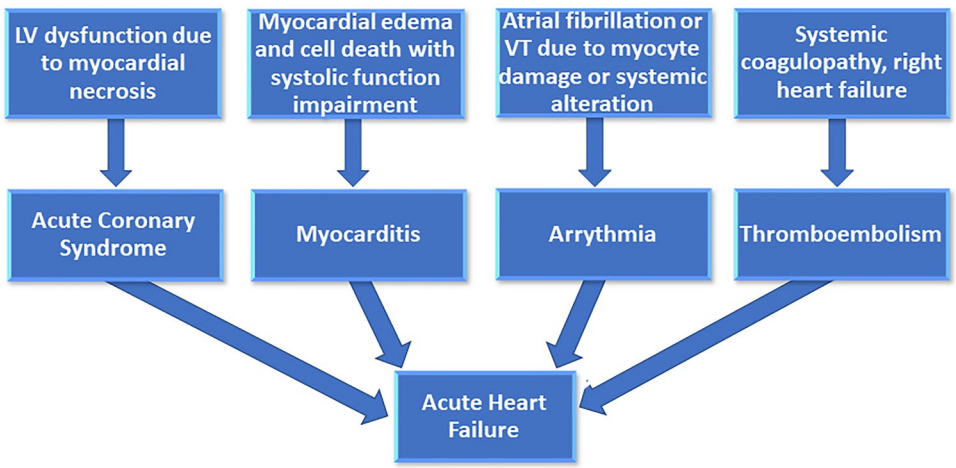

Fig. 2 Potential causes of cardiac complications induced by infection. Different clinical manifestations are related to the main virus location and spread (coronary district, myocites, venous system)

physical activity and aerobic capacity. Such situations increase CVD burden by reducing glycemic control and increasing cholesterol levels, insulin resistance, and blood pressure, with consequent metabolic risk elevation [32]. Such features contribute to HF exacerbation in those with known diagnosis or to HF onset in those with high CVD risk. Otherwise, fear of the virus contributed to delayed diagnosis and preventive/early treatment for congestion. The resultant extremely exacerbated AHF presentation could have caused the healthcare system (hospital ambulatory and peripheral) primarily engaged in virus struggle, to misdiagnose the disease, and those patients requiring hospitalization experienced hemodynamic and congestion deterioration [33, 34]. Current issues raise several concerns about the optimal strategy for patients with CHF and AHF. Indeed, no precise guidelines exist in terms of clinical and laboratory parameters to be evaluated during isolation, and criteria for hospital admission in case of new diagnosis or HF impairment. Although medical devices may be poorly distributed among peripheral environments, telemedicine software should be encouraged [35]. The daily measurement and central registration of some easily measurable variables such as body weight, heart rate, blood pressure, and oxygen saturation could identify the prodromal signs of HF and permit treatment initiation at home in hopes of avoiding hospitalization. Accordingly, asymptomatic patients with sudden increase of CVD risk with known cardiac defects might be closely monitored using telemedicine applications [36, 37]. Different concerns involve AHF patients: diagnostic screening is well established but has been shown to be poorly available with the higher levels of requirements of environmental sanitization and disinfection [38]. A few position papers proposed a multi-modality diagnostic approach with an easier and more rapid protocol compared with traditional examination; however, they do not explain which route or modality should be used to avoid potential virus transmission [4, 25, 39, 40]. In clinical practice, during the COVID-19 era, the possibility to execute a complete examination is often difficult as it requires a substantial amount of time and more personal involvement. Thus, every application should be customized in relation to clinical presentation, patient history, and biomarker profile. Similarly, to avoid virus spread and resource expenditure, each examination needs to be planned in order to achieve useful and additive information [41, 42]. In this setting, a primary role of cardiac biomarkers may be warranted, although no specific studies have been focused on the importance of laboratory examination for early diagnosis and related management. We recognize that the well-supported strategy of early home treatment of SARS-CoV2 infection at the earliest recognition of symptoms is likely to change the natural history of COVID-19 infection including its late cardiac manifestations. Future studies of early treatment with antivirals, immunomodulators, and antithrombotics with attention to short- and longer-term cardiovascular outcomes are warranted.

Conclusions

In light of the COVID-19 pandemic, some diagnostic procedures and in-hospital routes must be adopted in order to guarantee the best clinical management, and avoid infection diffusion following admission. Our protocol suggests to initially screen all patients with worsening dyspnea using a rapid diagnostic clinical and laboratory test, and then offers an additional diagnostic step to identify the most probable cause of admission. Other specific procedures, such as diffusion of telemedicine software, should be encouraged. The systematic application of simple rules will lead to time optimization and prioritize the safety of both patients and healthcare staff.

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