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Review of different clinical scenarios in patients with cardiovascular disease in the era of the coronavirus pandemic

Short title: Clinical review of Coronavirus Disease 2019 and cardiovascular disease

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Introduction

Cardiovascular comorbidities are common in patients with Coronavirus Disease 2019 (COVID-19) and are associated with a greater risk for morbidity and mortality [1]. This summary reviews clinical data on the impact of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection in patients with cardiovascular disease (CVD) [1] (Figure 1). The spread of COVID-19 requires attention to the immediate and long-term cardiovascular implications of viral infection. This is especially important for patients with pre-existing CVD.

Hypertension

Arterial hypertension is one of the most frequent comorbidities in patients with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). A recent meta-analysis of six studies including 1,527 infected patients showed that the prevalence of hypertension, cardio- and cerebrovascular disease and diabetes was 17.1%, 16.4% and 9.7%, respectively [1]. What is more, the prevalence of comorbidities was higher among patients who were admitted to intensive care units (ICUs), required mechanical ventilation, or had fatal outcomes [2-5]. However, according to current knowledge, hypertension is not an independent risk factor for worse outcomes in patients with COVID-19. Notably, the aforementioned comorbidities are often treated with renin-angiotensin system (RAS) inhibitors, including angiotensin-converting enzyme inhibitors (ACEi) and angiotensin receptor blockers (ARB). Their beneficial effect on cardiovascular outcomes has been demonstrated in many studies. Recently, however, the potential for unfavorable effects from ACEi and ARB, specifically an increased risk of SARS-CoV-2 infection and a more severe course of COVID-19, has emerged [6,7]. SARS-CoV-2 binds to its target cells through angiotensin-converting enzyme 2 (ACE2). ACE2 is expressed in the lungs, intestines,
kidneys, blood vessels and in the heart [6,7]. In animal models, up-regulation of ACE2 expression in various organs resulted from ACEi and ARB treatment [8-10]. ACE2 converts angiotensin II to angiotensin 1–7, which has a protective role in the cardiovascular system and potentially has a vasodilatory effect. Moreover, ACE2 prevents acute lung injury [11,12]. Possible up-regulation of ACE2 expression as a receptor for viral cell entry (with ACE inhibitors or ARBs) may theoretically increase susceptibility to COVID-19.

Interestingly, an opposite hypothesis was also raised. ARBs have been suggested to act beneficially in COVID-19 patients by ACE2 up-regulation, thus increasing the concentration of vasodilating angiotensin 1-7 at the expense of vasoconstricting angiotensin II, which contributed to lung protection [13,14]. Therefore, in the absence of clear evidence regarding beneficial or adverse outcomes of ACEi and ARB treatment in patients with COVID-19, the European Society of Cardiology (ESC) and American College of Cardiology and American Heart Association (ACC/AHA) do not recommend discontinuation or any modification of clinically indicated therapy. The risk of discontinuing the above-mentioned drugs is well known, this may destabilize blood pressure control, and consequently may increase cardiovascular mortality in COVID-19 patients [15, 16].

**Chronic Coronary Syndromes**

Reports of outpatient and inpatient cohorts with COVID-19 showed that 24% of patients had at least one comorbidity and 2.5-25% had chronic coronary syndromes (CCS) [2-5, 17]. CCS were more frequently observed in critically ill patients with respiratory failure (9%) and in non-survivors (24%). CCS were also a risk factor for in-hospital death in univariable analysis (OR 21.40; 95% CI 4.64–98.76) [3]. It is warranted to presume that patients with coronary
artery disease are susceptible to cardiac injury and complication from COVID-19 leading to sudden deterioration [18].

Acute inflammation can exacerbate the course of pre-existing CCS and may contribute to atherosclerotic plaque rupture leading to cardiovascular events. Therefore, administering drugs like aspirin, statins, beta-blockers, and ACEi which have the potential to stabilize plaque is strongly recommended according to current CCS guidelines [19].

Due to the increased likelihood of stent thrombosis due to pro-coagulant effects of systemic inflammation, some experts advise assessing platelet function and considering the intensification of anti-platelet therapy, especially in patients with a history of previous coronary intervention [20].

In the era of COVID-19 pandemic, many hospitals are limiting or cancelling elective diagnostics and treatment procedures. High-risk patients on waiting lists should be prioritized. What is more, single-day percutaneous coronary interventions (PCI), with special attention paid by dedicated nurse and the doctor during an about 6-hour-long hospital stay after the procedure, should be considered to reduce the risk of infection transmission [21]. As for treatment options, medical management or PCI may need to be preferentially considered over coronary artery bypass graft surgeries (CABG), if feasible, according to current guidelines. One reason for this is the limited number of ICUs, beds and respirators. Second, there is currently a limited number of packed red blood cells due to a lack of blood donors [22].

**Acute coronary syndromes**

The clinical presentation of acute coronary syndrome (ACS) may have signs and symptoms overlapping with those of COVID-19, including nausea, isolated dyspnea, chest discomfort, fatigue and tachycardia, making it challenging to differentiate between these two entities [23].
While the optimal management strategy in ACS patients during the COVID-19 epidemic is being widely discussed, no ultimate consensus has been reached, so far. Decisions should be individualized, considering the risk of SARS-CoV-2 exposure versus the risk of delay in diagnosis or therapy. However, in cases of ST-segment elevation myocardial infarction (STEMI), it is reasonable to follow the guideline recommended treatment, which implies primary PCI within 90 minutes from first medical contact [24].

In STEMI patients with active or suspected COVID-19 in whom PCI is to be performed, appropriate personal protective equipment (PPE) should be worn, such as gowns, gloves, goggles, shields and FFP 2/3 masks. However, in our opinion, it is reasonable to assume that each patient is a possible SARS-CoV-2 case. This assumption is based on the fact that 15-20% of infected patients remain asymptomatic but contagious [25]. Such a strategy, although it requires utilization of additional resources, ultimately minimizes the risk of personnel exposure and contamination of the catheterization laboratory (CathLab). Adoption of this precautions should reduce the risk of CathLab quarantine/shut-downs allowing full availability of primary PCI service in a continuous and uninterrupted fashion.

There are yet no specific recommendations to alter routine PCI technique, stent selection, and periprocedural pharmacotherapy. However, in patients with acute cardiovascular and respiratory decompensation, intubation should be considered prior to CathLab admission, as emergency intubation, suction, and active cardiopulmonary resuscitation (CPR) in the CathLab setting is likely to result in aerosolization of respiratory secretions increasing the likelihood of personnel exposure.

Of note, pandemics may pose a threat to the STEMI patient population due to the risk of a time delay to primary PCI, resulting from multiple factors. Firstly, there seems to be a significant patient factor, which most probably results from patient reluctance to hospitalization. Specifically, as was found in a preliminary report from China, researchers
noted a nearly fourfold increase in the time from symptom onset to first medical contact [25]. Secondly, the availability of emergency medical transport may be limited due to system overload and unavailability of some emergency medical response teams due to post-exposure quarantine. Last but not least, one can expect some hospital delay related to the need of additional above-mentioned safety procedures as identified in recent publication [25]. Taking into consideration the increased risk of delays from onset of symptoms to PCI, a fibrinolysis with an early pharmaco-invasive strategy should be considered in STEMI patients in whom the expected delay is more than 90 minutes. Patients with non-ST-segment elevation myocardial infarction (NSTEMI) should undergo biomarker evaluation (i.e. high sensitivity troponin), symptom and electrocardiogram (ECG) monitoring, followed by risk stratification. Current ESC guidelines recommend an immediate invasive strategy only in very high-risk patients, which means that for the majority of the NSTEMI population timing should allow for diagnostic testing for COVID-19 prior to cardiac catheterization and more informed decision-making regarding infection status [26]. Of note, a troponin rise is observed in up to 17% of COVID-19 patients, which is likely related to acute cardiac injury and/or type 2 myocardial infarction [3]. That is why, in light of a lack of any other signs of ischemia, a conservative treatment should be considered as the default strategy.

**Myocarditis**

The actual incidence of myocarditis in patients with a SARS-CoV-2 infection is impossible to be determined, as endomyocardial biopsy (EMB) is used infrequently. Out of more than 21 millions known COVID-19 cases worldwide the scenario of clinically suspected myocarditis is uncommon. Using the search terms (“coronavirus” OR “SARS-CoV-2” OR “COVID-19” AND “myocarditis”), so far there are only several dozen cases reports and a low-number of retrospective studies reporting clinically suspected myocarditis, few with positive cardiac
magnetic resonance (CMR) imaging, but none were biopsy-proven [3, 27-31]. CMR should be performed in all cases of suspected myocarditis, in order to increase the likelihood of myocarditis diagnosis, but it increases the risk of facilities contamination. What is more, sputum or nasal/throat swab virology testing is not sufficient to prove the association of clinically suspected myocarditis with SARS-CoV-2 infection, since only biomolecular testing (polymerase chain reaction or in-situ hybridization) of the myocardial tissue provides certain diagnosis. In addition, SARS-CoV-2 is not among cardiotropic viruses that are known to be associated with myocarditis (like parvovirus B19, enterovirus, adenovirus). In one case, autopsy showed only few interstitial mononuclear inflammatory infiltrates but no signs of myocardial damage [32]. However, it is postulated that there is a potential way of direct cardiac involvement through ACE2 – the receptor for SARS-CoV-2 [33]. This theory requires further investigation.

Myocarditis is more common in children and young adults, may occur in middle age and is rare in the elderly, while the median age of COVID-19 patients was approximately 55 years and comorbidities were present in at least half of patients. In patients with COVID-19 the predominant clinical presentation was pneumonia with associated signs and symptoms of systemic infection, while chest pain (2% of patients) and/or palpitations/arrhythmia were observed at low frequency [30,34]. In overall populations, elevated natriuretic peptides and troponins levels were observed in a low proportion of patients (approximately 30% and 10-20%, respectively), with higher prevalence amongst patients requiring intensive care [4,35, 36]. Deceased patients had significantly more complications such as acute respiratory distress syndrome, acute kidney injury, shock, and disseminated intravascular coagulation. In deceased groups acute cardiac injury with elevated troponins and natriuretic peptides was also significantly more frequent than in survivors, which is more likely due to multiorgan damage (including liver and renal failure), cytokine storm or undefined etiology [37]. There is
evidence that acute infections are associated with an increased risk of myocardial damage [38]. An abnormal troponin level does not equate to heart failure, myocarditis or myocardial infarction in COVID-19. Other potential mechanisms of troponin elevation include high myocardial oxygen demand accompanied by hypoxia caused by acute respiratory failure, plaque rupture and coronary thrombosis enabled by systemic inflammation, dyselectrolytemia, especially hypokalemia concerning interaction between SARS-CoV-2 and RAS [1, 20, 39]. Additionally, the drugs used in COVID-19 such as antivirals, corticosteroids, chloroquine and azathioprine may pose a risk of cardiac toxicity, interact with cardiac drugs and prolong QT interval, thus leading to arrhythmias [40]. Therefore, current epidemiological data do not support the hypothesis that myocarditis is associated with COVID-19, or that it is common.

In the above-mentioned studies, patients with clinically suspected myocarditis were treated with corticosteroids, intravenous immunoglobulin (IVIG) or antiviral treatment (alone or in combination), although these agents have no proven beneficial role in clinically suspected myocarditis [41]. According to the current ESC recommendations, immunosuppressive or immunomodulative treatment should be prescribed after ruling out viral presence in the myocardium on EMB [27]. What is more, COVID-19 patients who received corticosteroids and/or IVIG did not benefit from this treatment or even had increased risk of death [3]. Use of corticosteroids in viral infection (in COVID-19 there was also reported a high risk of coinfection of other respiratory pathogens) is controversial and may lead to reduced viral clearance and an increased risk of sepsis [42]. Administration of IVIG for a hypothetical COVID-19 myocarditis might be useless, since human IVIG do not contain IgG to COVID-19 (except for cured COVID-19 patients). It is also postulated that up to 50% of acute myocarditis patients undergo spontaneous healing [27].
**Intensive cardiac care**

Current research shows that patients requiring ICUs treatment were more likely to suffer from hypertension, diabetes or ischemic heart disease [43, 44]. The infection may indirectly affect the circulatory system, exacerbating the symptoms of already existing diseases, but also may directly contribute to the occurrence of specific diseases and cardiological complications (e.g., myocarditis, ACS, myocardial injury, acute heart failure leading to cardiogenic shock, malignant ventricular arrhythmias or venous thromboembolism). Most likely, this is due to uncontrolled immune response to the presence of the virus resulting in acute neurohormonal stress, release of a large number of pro-inflammatory cytokines and leading to significant disorders of the coagulation system [3]. In addition, the infection can lead to complications specific to COVID-19 such as pneumonia, acute respiratory distress syndrome (ARDS), septic shock or multiple organ failure. All this means that individual disease syndromes can superimpose each other, which can greatly impede the diagnostic process and implementation of appropriate treatment. Priority patients with severe COVID-19 should be referred to the ICU due to the specific equipment available and the medical staff experienced in intensive care. However, it seems very likely that a patient with COVID-19 will be transferred to an intensive cardiac care unit (ICCU) due to a primary cardiac disease or lack of ICUs. This fact sets new challenges for cardiologists and certainly breaks from the current routine.

Available reports clearly show that the presence of CVD in patients infected with SARS-CoV-2 is associated with a dramatic increase in in-hospital deaths when the existing cardiovascular pathologies overlap with respiratory failure, infectious myocardial damage, or new, resistant to treatment cardiac arrhythmias [43].

**Heart failure**
It has been shown that heart failure occurred in 52% of non-survivors and in 12% of survivors [3]. Surprisingly, although respiratory illness is the major clinical manifestation of COVID-19, the case-fatality rate for patients with pre-existing CVD is greater (10.5%) than in patients with pre-existing chronic respiratory disease (6.3%) [43].

Heart failure in patients with suspected/confirmed COVID-19 should be managed according to the current guidelines [45]. To limit the risk of viral exposure, it is recommended to avoid blood testing (to measure troponins and natriuretic peptides) or echocardiography in COVID-19 patients unless it is necessary and affects treatment [46]. Treatment of patients with acute left ventricular failure complicated by pulmonary edema in COVID-19 patients should follow the same rules as for non-infected patients. The difficulty physicians face is to establish the cause of acute left ventricular decompensation and differentiate with non-cardiogenic pulmonary edema caused by massive pneumonia or ARDS. It is known that in the acute phase of edema, radiological symptoms are unreliable and more advanced diagnostics are difficult to perform due to the patient's clinical condition. Probably in this case, the initial inference should be based on assessing the dynamics of withdrawal of pulmonary edema symptoms in response to the use of typical pharmacological treatment. It should be assumed in persons who, despite treatment, develop respiratory failure symptoms leading to the use of mechanical ventilation, may have primary pulmonary or mixed pathology. Properly performed endotracheal intubation by qualified medical staff in PPE and patient sedation enables imaging, including chest computed tomography (CT) and lung ultrasound, which in combination with serial arterial blood gas tests will allow staff to establish a diagnosis and implement appropriate treatment [47]. The management of COVID-19 patients should follow the accepted principles, but with special emphasis on the prognostic aspect of patients [48].

**Pulmonary embolism**
Another cardiological condition which requires treatment in ICCU is pulmonary embolism, which may occur as a direct consequence of COVID-19. Patients with COVID-19 are at increased risk of developing venous and arterial thromboembolism. In the group of hemodynamically unstable patients, rescue fibrinolysis should be used or, in case of its ineffectiveness, qualification for surgical or percutaneous thrombectomy should be considered. In many cases, this may unfortunately involve the need to transfer the patient to a center with the possibility of highly specialized treatment with simultaneous access to methods of hemodynamic support. It seems that especially in the era of COVID-19, such decisions should be made by interdisciplinary teams in the treatment of severe pulmonary embolism. According to the current guidelines low risk patients may be hospitalized shortly and, after exclusion of the virus infection, should be discharged on oral treatment [3].

**Acute aortic syndrome**

Patients with acute aortic syndrome should be referred to cardiac surgery centers after establishing serological status, as patient condition allows. Possibly, if serological assessment is not available, the abnormalities seen in chest CT may support the decision to take appropriate precautions to prevent virus transmission.

**Dissimilarity of ventilation support principles**

Interestingly, the recommended first-line method of ventilator support in selected patients with acute heart failure is non-invasive ventilation (NIV) (continuous positive airway pressure [CPAP] and bilevel positive airway pressure [BiPAP]) [49]. Currently, NIV is not recommended due to high risk of virus transfer to medical staff while the preferred method is endotracheal intubation and mechanical ventilation. As experts suggest, a strategy should be adopted for the active identification of patients who will require intubation in the near future.
to avoid performing a procedure with a high risk of virus transfer without adequate protection of the personnel with PPE. The passive methods include using only high-flow nasal catheters and face masks, without positive inspiratory pressures. The National Early Warning Score (NEWS 2) scale takes into account the following criteria: respiratory rate, hypercapnic respiratory failure, room air or supplemental O₂, temperature, systolic blood pressure, pulse and consciousness. The score is now validated for COVID-19 patients.

In severe ARDS it is recommended to adjust ventilation therapy to serial arterial oxygen pressure measurements. Often, there is a need to use high peak end-expiratory pressure (PEEP) (in normal conditions it is 4-6 mm H2O) values with relatively small inspiratory volumes (4-6 ml / kg body weight). However, the increased value of PEEP may not be sufficient to improve oxygenation [47]. Simultaneously, increased PEEP may itself worsen hemodynamic instability and hypotension. In this case, the effectiveness of ARDS treatment will be significantly limited by poor hemodynamic parameters and a consequent need for extracorporeal membrane oxygenation (ECMO) therapy [50].

**Cardiopulmonary resuscitation**

Some distinctions regarding CPR procedures in COVID-19 patients should be emphasized. The key recommendation is to identify as early as possible patients who may require CPR, to ensure required equipment and avoid unprotected CPR. Use of the NEWS 2 scale can help to select patient at increased risk.

Importantly, it is recommended to start compression-only CPR with quick assessment of patient cardiac arrest rhythm until endotracheal intubation with full PPE protection is provided. Do not use mouth-to-mouth ventilation to limit aerosol spread. In the case of defibrillating rhymes (hemodynamically unstable ventricular tachycardia or ventricular fibrillation), it is recommended to perform defibrillation as soon as possible [17].
**Potential serious adverse events of currently proposed treatment protocols of COVID-19**

Based on encouraging results of preliminary studies, chloroquine/hydroxychloroquine and azathioprine were widely prescribed to patients with COVID-19. However, these drugs favour QT interval prolongation and therefore may provoke subsequent ventricular arrhythmias, including torsade de pointes, and are not currently recommended. Clinicians caring for these patients should be aware of the potential cardiovascular side-effects of proposed therapeutic options. Moreover, adequate monitoring of treatment safety is warranted, especially in highly vulnerable patients with underlying cardiac, renal or hepatic dysfunction and in those receiving medications with a known effect on QT interval prolongation [40].

**Arrhythmias, ablations and implantable devices**

In patients with COVID-19 the risk of arrhythmia is increased due to possible heart failure, suspected myocarditis, proinflammatory effects, cardiac injury and increased sympathetic stimulation. Although arrhythmia is listed as a potential cardiovascular complication of a SARS-CoV-2 infection, the precise distribution of different heart rhythm disturbances is not available.

Nonspecific heart palpitations were present in 7.3% of 137 patients admitted due to COVID-19 [34]. In 138 COVID-19 patients hospitalized in Wuhan, cardiac arrhythmia was noted in 16.7% of patients and was more common in ICUs patients (44.4%) compared to non-ICUs patients (6.9%) [4]. Unfortunately, there were no precise specifics about the types of arrhythmia. In another study evaluating the association of underlying CVD and myocardial injury with fatal outcomes, patients with high Troponin T level presented a higher incidence of malignant arrhythmias compared to those with normal levels (11.5% vs 5.2%) [49].
It is recommended as a “best practice” to postpone all elective procedures: ablation in clinically stable patients, device upgrades, most primary prevention implantable cardioverter defibrillator (ICD) implants, left atrial appendage closure device implants, and implantable loop recorders. This is due to the impact of COVID-19 cases on healthcare resources and the increased risk to patients and medical personnel from contact with individuals who are infectious. EP procedures in patients with confirmed COVID-19 should be performed only in cases of life-threatening arrhythmias in dedicated hospitals. Cardiac implantable electronic device (CIED) implantations due to urgent indications in non-infected patients should also be performed. In cases of urgent electrophysiology (EP) procedure in known SARS-CoV-2 infection, it is recommended to supply appropriate PPE for all members of care teams including powered air-purifying respirators or N-95 masks due to the potential risk of urgent bag-mask ventilation or intubation (Heart Rhythm Section of Polish Cardiac Society COVID-19 Task Force Message published online).

In patients with CIED who are already followed by remote monitoring and do not require in-person evaluation, it is recommended to consider replacing routine visits with remote visits (e.g., video, telephone, remote monitoring of CIED). For other patients, it is recommended to enroll in remote monitoring. For patients without CIED who require rhythm monitoring, ambulatory monitors can also be mailed to each patient. ECG acquisition by smartphone or smartwatch may be considered. Routine in-hospital in-person device interrogation in stable patients in long-term care facilities is not recommended (Heart Rhythm Section of Polish Cardiac Society COVID-19 Task Force Message published online).

**Telemedicine**

A global pandemic was officially announced on March 11th by World Health Organization (WHO) [51]. The spread of SARS-CoV-2 across the world has changed the organization of
many industries, including the health care system, which had to adjust to prevailing conditions and clinical demands. Telemedicine accelerated its growth together with a limitation of social contact, which includes limiting regular doctor’s appointments. Telemedicine had been recommended for use by international societies before the COVID-19 pandemic [52,53]. However, there have always been some barriers, which stopped the expansion of telemedicine [54]. Reorganization of health systems, clinician willingness, poor computer skills and financial reimbursement are listed among the reasons for its slow development so far [55]. Nonetheless, nowadays physicians across the world are forced to provide consultations with mobile phones and computers in order to stop the virus spread and enable the treatment of patients, especially isolated patients or during quarantine. Telemedicine consulting has been legalized by several governments. This approach also has a basis in evidence-based medicine and has been justified by several randomized trial publications. Video-consultations proved to be a useful tool, with high satisfaction rates among both patients and physicians, without differences in disease progression and is financially profitable [56-58]. However, we should be careful in selecting patients for consultations for reasons that differ from those that are COVID-19 related, who can and will benefit from this solution. Patients whose general condition is unstable or require acute care still need to have a traditional visit. Telecare can abolish barriers to receiving cardiac care in this SARS-CoV-2 pandemic. First of all, telecare diminishes patient exposure, thus reducing the risk of infection. Secondly, telecare resolves patient problems with transportation during a pandemic, when public transport is not recommended. What is more, this will allow for a better allocation of hospital beds to the seriously ill or COVID-19 patients. For the sake of safety, it is necessary to review upcoming appointments in advance with the goal of identifying three groups of patients:
- Those who must be seen face-to-face (traditional visit)
- Those who might be seen virtually (telecare visit)
- Those who can be safely rescheduled a few months later during a more appropriate time with lower risk of infection (postponed visit)

It is necessary to mention some practical issues of telecare. There are different types of virtual visits such as video consultation, consultation by phone or special dedicated system for e-consults. During virtual visits physicians can review results from previous examinations with patients and discuss vital sign trends from remote monitoring. Televisits can be done with the goal of educating patients about disease process and treatment targets. Education and counselling about diet or physical activity can be also provided through these visits.

Another form of telecare apart from the televisits is the remote monitoring of implanted cardiac devices by special dedicated systems. It is recommended to call every patient whose scheduled visit for routine monitoring and reprogramming of cardiac devices cannot be conducted, with the goal of assessing their status (new symptoms such as dizziness, lack of consciousness, history of shock delivery) [59-61].

Key points:

- Patients with cardiovascular (CV) risk factors and cardiovascular diseases (CVD) are associated with a greater risk for morbidity and mortality when suffering from Coronavirus Disease 2019 (COVID-19).
- Regarding acute cardiac conditions such as acute coronary syndromes, heart failure, cardiogenic shock decisions of management strategy should be individualized, considering the risk of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) exposure versus the risk of delay in diagnosis or therapy.
- Troponin release is a common and multifactorial cardiac manifestation of COVID-19 and is associated with increased risk of morbidity and mortality.

- Myocarditis is highly overdiagnosed as one of the common cardiac complications of COVID-19, but there is no proof that SARS-CoV-2 is a novel cardiotropic virus causing direct cardiomyocyte damage. The term „Myocarditis” should be used only for endomyocardial biopsy/ autopsy- proven diagnosis.

- Different not evidence-based treatment strategies in patients with COVID-19 disease (i.e. antivirals, corticosteroids, chloroquine, azathioprine) may pose a risk of cardiac toxicity and should not be used at all or with extreme caution.

- A COVID-19 global pandemic poses challenges to health care system. Implementation of telemedicine solutions for daily care is beneficial, particularly for the patients with stable, chronic conditions. However, it should not delay the management of patients who require look out.
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