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Management of STEMI during the COVID-19 pandemic: Lessons learned in 2020 to prepare for 2021 ☆☆☆



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ABSTRACT

As the prevalence of asymptomatic COVID-19 continues to increase, there is an increasing possibility that patients with COVID-19 may present with ST-segment elevation myocardial infarction (STEMI). With social distancing and restricted access to preventive healthcare and emergency services, the management of acute cardiac emergencies such as myocardial infarction has suffered collateral damage. Thus far, global trends suggest a decrease in STEMI activations with possible worse outcomes due to delayed presentation and management. In this review, we discuss the challenges to STEMI management in the COVID-19 era and provide potential solutions for adherence to evidence-based therapies as the pandemic progresses into the year 2021.

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Introduction

Coronavirus disease 2019 (COVID-19) was declared a pandemic by the World Health Organization on March 11, 2020. Since the initial breakout from Wuhan district, China, the disease has spread to over 177 countries, with the United States having the maximum

Abbreviations and acronyms: CAD, coronary artery disease; COVID-19, coronavirus disease 2019; HEPA, high-efficiency particulate air; LUCAS, Lund University cardiopulmonary assist system; PPCI, primary percutaneous coronary interventions; PCI, percutaneous coronary interventions; PPE, primary protective equipment; STEMI, ST-segment elevation myocardial infarction; ECG, electrocardiogram; WMA, wall motion abnormalities.

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number of cases. There is a high prevalence of cardiovascular risk factors such as hypertension and diabetes mellitus among patients with COVID-19 [1,2]. These patients are also at an increased risk of death due to COVID-19 [2].

There are several caveats unique to the management of ST-segment elevation myocardial infarction (STEMI) during the COVID-19 pandemic. In patients with COVID-19, diverse conditions such as myo-pericarditis, coronary artery vasospasm, pulmonary embolism, or stress-induced cardiomyopathy may mimic STEMI [3]. Additionally, there is a delay in patient presentation, referral, and transport to the treatment facility [4,5]. Patients with STEMI and COVID-19 may have significant thrombus burden due to heightened hypercoagulability resulting in suboptimal results after primary percutaneous coronary interventions (PPCI) due to slow flow, or no-reflow warranting novel approaches for treatment [6]. In this review, we highlight the challenges and discuss potential approaches to optimize the treatment of STEMI in patients with COVID-19.

Trends in incidence of STEMI in the COVID-19 era

There has been a decrease in STEMI-related admissions in the COVID-19 period (Fig. 1) [7–10]. STEMI activations in nine high-volume centers (>100 PPCI/year) in the United States decreased

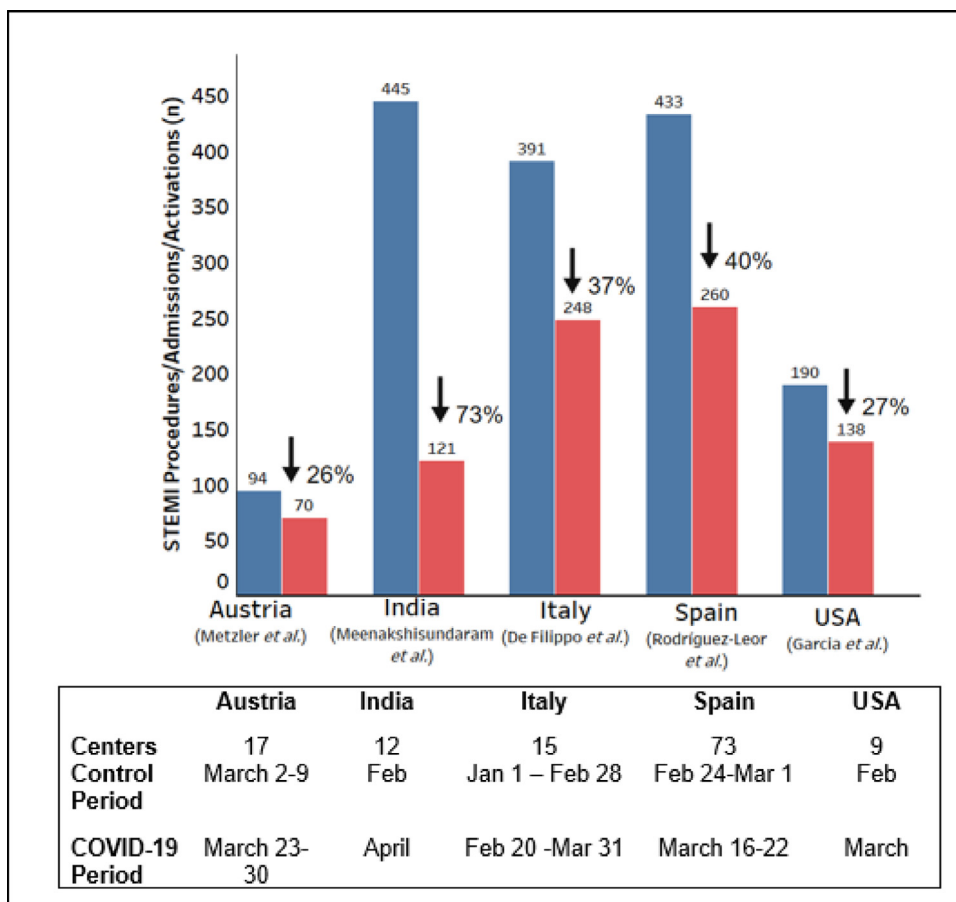


Fig. 1. Trends of STEMI activation across countries with a high burden of COVID-19. Blue and red bars represent the number of activations in the control and study period, respectively. The details of the control and study period are given in the table below the panel. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

from >180/month in the before COVID-19 period to 138/month in the COVID-19 period [7]. Similarly, data from fifteen hospitals in Italy suggests a decrease in admission for STEMI from 8/day to 6.1/day in the COVID-19 period [8].

Along with decreased activations, there is an increase in time to reperfusion. A high-volume center in Ireland reported an increase in ischemic time from 485 to 1,550 min, with an increase in the delay from the point of the first medical contact to the point of activation [5]. A recent study from a hospital offering 24/7 PPCI facilities in Hong Kong suggested an ~300% increase in time from symptom onset to first medical contact, a 30% increase in door-to-device time, and a 60% increase in time from arrival to the cardiac catheterization laboratory to PPCI [4]. This delay in reperfusion is due to both patient-related and systemic factors. Patients may avoid or delay seeking medical care or present at low-volume centers to minimize the risk of exposure to COVID-19 [3,11]. System-related factors may include an increase in emergency department (ED) time for additional COVID-19 testing/confirmation; a decrease in focus on cardiovascular disease and instead, an increased focus on COVID-19 and consequent time spent on performing additional diagnostic tests such as chest X-ray and computed tomography (CT); and possible high threshold to transfer patients with COVID-19 to the cardiac catheterization lab. Although current guidelines advocate avoiding any delays in PPCI in STEMI, it is important to note that a high proportion of patients with COVID-19 may not have significant epicardial coronary artery disease on angiography (Table 1).

The decreased hospitalization for acute care suggests that a large proportion of patients with STEMI may have died without seeking medical care [12]. Emergency Medical System Incident Dispatch data from the New York Emergency Medical Services suggest around a 400% increase in calls for cardiac arrest during March 2020 [13]. A recent analysis of cause-specific mortality in the United States suggested a larger than expected increase in death due to non-respiratory causes, with heart disease and diabetes mellitus as the most notable causes [14].

The personal experience of the authors suggests an increased incidence of STEMI-related mechanical complications such as development of ventricular septal defects and papillary muscle rupture. The treating team needs to be aware of these complications, especially in those presenting late after onset of chest pain.

Pathophysiology of STEMI unique to COVID-19

Infectious illnesses like influenza are associated with systemic inflammation and hypercoagulability, leading to an increased risk of atherosclerotic plaque rupture and thrombosis [15]. However, thromboembolic complications in patients with severe COVID-19 occur at a higher incidence compared to other acute infections [16]. Multiple pathogenic mechanisms contribute to hypercoagulability in COVID-19. The dysregulated systemic immune response to viral replication results in the activation of the complement and coagulation cascade [17] (Fig. 2). Endothelial cell dysfunction mediated by direct viral invasion through angiotensin convert-

Table 1
Studies with COVID-19 patients presenting with ST-segment elevation on electrocardiogram and undergoing invasive coronary angiography.

Author	Country	Sample size	Women (%)	Mean Age (years)	Hypertension (%)	Diabetes Mellitus (%)	Previous MI/PCI/CABG (%)	Culprit lesion identified during invasive angiography n/N (%)
Alaarag et al. [44]	Egypt	26	30.8	57.7	42.3	38.5	15.3	18/26 (69)
Bangalore et al. [24]	United States	18	17	63	65	35	–	6/9 (67)
Choudry et al. [22]	United Kingdom	39	15.4	61.7	71.8	46.2	15.4	38/38 (100)
Hamadeh et al. [45]	Lithuania, Italy, Spain, Iraq	19	53	65	79	11	5	18/19 (95)
NACMI [46]	United States	171	30	–	73	44	48	115/138 (83)
Siudak et al. [47]	Poland	145	28.7	63	46.2	14.5	25.5	123/143 (86)
Secco et al. [48]	Italy	31	22.6	72.3	71	38.7	35.4	8/10 (80)
Stefanini et al. [28]	Italy	28	28.6	68	71.4	32.1	10.7	17/28 (61)

CABG, coronary artery bypass grafting, MI, myocardial infarction, NACMI, North American COVID-19 STEMI Registry, PCI, Percutaneous coronary intervention.

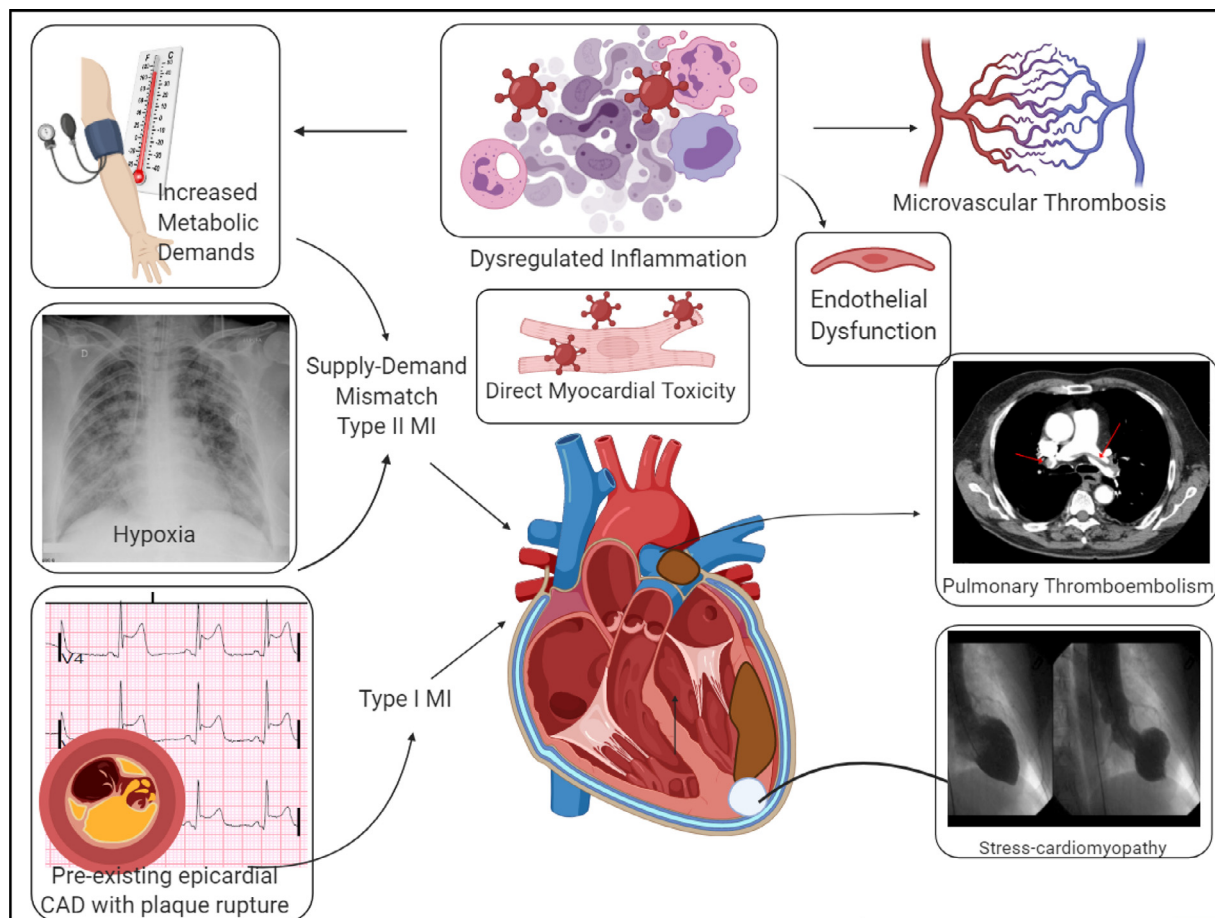


Fig. 2. Potential mechanisms of myocardial injury in COVID-19.

ing enzyme-2 receptors and secondary to systemic inflammation has been found to cause endothelitis and thrombosis in micro-circulation in autopsy studies [18,19]. In particular, the formation of microthrombi in multiple organ systems including the lung and the myocardium may result in troponin elevation even in the absence of ST-elevation, making diagnosis challenging. Systemic inflammation along with profound hypercoagulability predisposes to both arterial and venous thrombosis [20], even without traditional risk factors [21]. Thrombosis increases with worse disease severity and patients with thrombotic events have a significantly elevated risk of all-cause mortality.

Patients with severe COVID-19 and risk factors are at an increased risk of adverse cardiac events such as cardiogenic shock and cardiovascular death [3]. In a study of 3,334 adult patients ad-

mitted to a New York health system with COVID-19, myocardial infarction occurred in 8.9% and accounted for a majority of the arterial thrombotic events [20]. The incidence of myocardial infarction and arterial thrombosis was higher in patients in the intensive care unit vs. others. Another single-center observational study revealed that patients presenting with a STEMI with concurrent COVID-19 had a higher incidence of multivessel thrombosis and stent thrombosis when compared to STEMI patients without COVID-19 [22]. Patients with COVID-19 also had a higher modified thrombus grade resulting in increased utilization of GP IIb/IIIa inhibitors and aspirin thrombectomy [22].

Multiple laboratory markers have been evaluated to identify patients at a higher risk of thrombosis and adverse events with COVID-19. Initial elevation and rising levels of D-dimer during

the hospital stay has been consistently associated with an increased risk of thrombosis, mortality, and adverse clinical outcomes [20,22,23]. In addition to D-dimer, troponin elevation can also help identify patients at higher risk. In COVID-19 positive patients with ST-segment elevation, those with a myocardial infarction had higher mean troponin levels vs. those without [24]. Higher troponin levels are independently associated with an increased risk of mortality after adjusting for baseline characteristics [25].

Differential diagnosis of ST-segment elevation in patients with COVID-19

Besides myocardial infarction, elevated troponin levels may be due to myocarditis, stress-induced cardiomyopathy, myopericarditis, spontaneous coronary artery dissection, systemic microthrombi, and pulmonary embolism [1,3,26,27]. In a case series of 28 patients with STEMI and COVID-19 from the Lombardy region in Italy, ~40% had non-obstructive CAD [28]. In another case series of 18 patients with COVID-19 who presented with ST-segment elevations, eight patients had a myocardial infarction and ten had a non-coronary myocardial injury [24]. Of the nine patients who underwent invasive coronary angiography, only six had obstructive CAD. Troponin-T was elevated in both patients with and without myocardial infarction but peak levels were higher in those with myocardial infarction [24]. Examples of ST-segment elevation patterns in patients with COVID-19 was reported recently by Bangalore and colleagues [24].

Pre-hospital logistics and initial management of patients with COVID-19 and STEMI

Regardless of the COVID-19 status, timely PPCI is the standard of care for patients with STEMI [29]. The ED physicians play an essential role in the timely diagnosis and triage of patients, especially those where the diagnosis of STEMI is unclear due to atypical EKG findings, delayed presentation, or high clinical suspicion for alternate diagnoses. ED evaluation should be focused on rapid risk stratification of COVID-19 status and utilization of point-of-care ultrasound (POCUS) or portable echocardiography to determine the likelihood of coronary occlusion [26]. In COVID-19 positive patients or a person under investigation, assessment of regional wall motion using POCUS may help differentiate STEMI from myocarditis [29]. In a case series of 28 patients with STEMI and COVID-19 from Italy, 22 (82.1%) patients had localized wall-motion abnormalities [28]. The presence of regional wall motion abnormalities suggests STEMI whereas global hypokinesia usually suggests myocarditis. Although there is some evidence to support the possible diagnostic role of computed tomography (CT) coronary angiography in patients with COVID-19 to rule out significant epicardial CAD, the majority of the society guidelines recommend early catheterization laboratory transfer for all patients with suspicion for STEMI [26,30].

The management of patients with STEMI at non-PPCI capable facility may be challenging and needs a multi-system coordinated effort. If initial contact with a PPCI capable facility is >120 min, fibrinolysis should be considered based on physician experience, when appropriate, followed by immediate transfer to a PPCI-capable facility. Timely communication between emergency medical services personnel and physicians on diagnosis, COVID-19 status, fibrinolysis therapy, and early activation of cardiac catheterization laboratory and isolation cardiac intensive care unit beds can help reduce system delays in PPCI (Fig. 3).

Management of cardiac arrest in COVID-19 patients presenting with STEMI

Management of cardiac arrest can be a major challenge in a suspected/confirmed COVID-19 patient. Consensus statements released from various societies support the donning of appropriate primary protective equipment (PPE) including N95 masks, long-sleeve gowns, gloves, and face shields to minimize exposure to healthcare workers [29]. For proper protection, it is critical to understand the correct technique of donning and doffing PPE [31]. Oxygenation and ventilation strategies should be prioritized to minimize aerosolization, and early endotracheal intubation with connection to high-efficiency particulate air filter enabled airway system should be performed in patients with impending respiratory failure [32]. Immediate defibrillation of a shockable rhythm reduces the risk of aerosolization more than chest compressions in patients with an unprotected airway and should be considered where indicated [29]. Wherever feasible, use of the Lund University Cardiopulmonary Assist System (LUCAS) device, a mechanical device that provides automatic chest compressions should be encouraged. Institutions should establish policies to address goals of care discussion with patients to help guide providers for the appropriateness of resuscitation efforts [33]. In patients with known or high probability of COVID-19, placement of venous-venous extracorporeal membrane oxygenation should be considered in the setting of respiratory failure and inability to oxygenate. Whenever appropriate, mechanical circulatory support and extracorporeal membrane oxygenation should be placed at the bedside to avoid the risk of exposure to catheterization laboratory personnel [26].

Reperfusion strategy in patients with COVID-19 and STEMI

PPCI is associated with better outcomes in patients with STEMI relative to thrombolysis and should be the preferred reperfusion strategy even during the ongoing pandemic [34]. PPCI has been shown in multiple trials to have a better success rate in terms of achieving Thrombolysis In Myocardial Infarction-3 flow [35]. Furthermore, most patients treated with thrombolysis at non-PPCI eventually require either rescue or definitive PCI. PPCI, therefore, eventually leads to better outcomes [36]. Moreover, there is also a higher prevalence of coagulation abnormalities in patients with COVID-19 [17]; thus, patients treated with thrombolysis would be at an increased risk of fatal complications such as hemorrhagic cardiac tamponade, intracranial bleeding, or hemorrhagic shock [37]. Taken together, even in the setting of COVID-19, the risk-benefit profile favors PPCI over thrombolysis. The risk of exposure to health care workers in the cardiac catheterization laboratories can be significantly minimized by effective use of PPE [26].

Catheterization laboratory management of STEMI in patients with COVID-19

In compliance with the World Health Organization guidelines for high-exposure procedures, all healthcare workers in the cardiac catheterization laboratory must wear appropriate PPE, including gown, gloves, goggles or face shields, and N95 masks [29]. The number of personnel in the laboratory, the amount of equipment should be minimized, and stratified to avoid over-exposure to COVID-19. Radial artery access helps the early discharge of patients and should be encouraged [38]. If femoral artery access is utilized, closure devices should be favored over manual compression to further minimize patient contact and staff exposure [39]. In the current scenario where critical care beds are extremely valuable, efforts should be made by catheterization laboratory operators to stratify low-risk STEMI patients using scoring systems, such as the CADILLAC and the Zwolle scores, to identify patients who

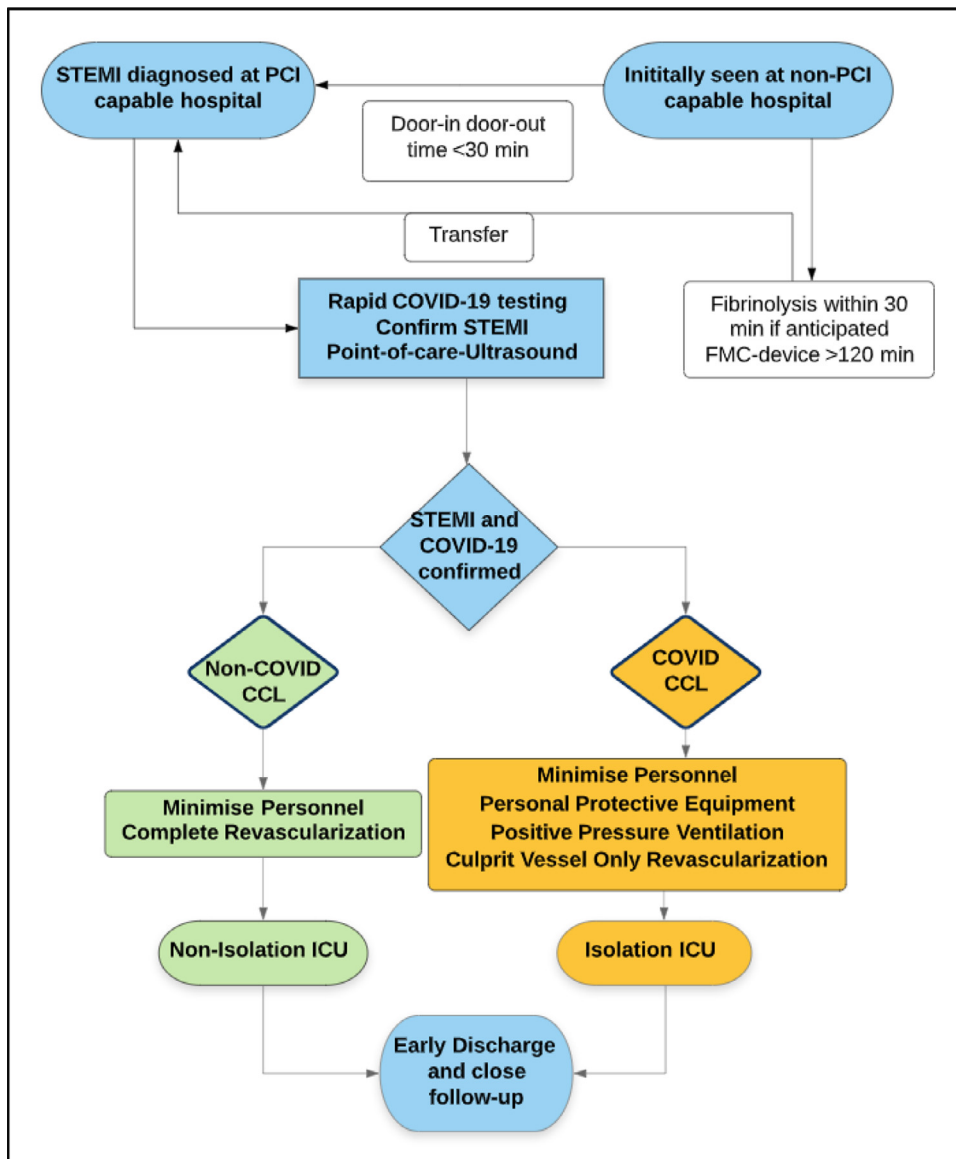


Fig. 3. Multi-disciplinary collaboration of systems for the management of STEMI diagnosed at non-PCI capable locations. CCL, cardiac catheterization laboratory, COVID, Coronavirus disease, FMC, first medical contact, ICU, intensive care unit, PCI, percutaneous coronary intervention, POCUS, point of care ultrasound, STEMI, ST-elevation myocardial infarction.

may be safe for an early discharge without the requirement of cardiac intensive care [40–42].

Post-PPCI management of COVID-19 patients with STEMI

Post-PPCI management should focus on minimizing exposure and early transfer to an intermediate care observation unit for 24–48 h followed by an early discharge for low-risk patients (see above). This would require a careful assessment of procedural success, hemodynamic stability, and the ability of the patient to follow-up in case of adverse events. Interestingly, there may be a role of serial measurements of biomarkers such as cardiac troponins and N-Terminal Pro-B-Type Natriuretic Peptide in the post-PPCI setting to stratify patients who may more likely achieve sustained revascularization and have a lower risk for short term adverse cardiac events, and consequently plan them for early discharge [43]. For high risk or hemodynamically unstable patients, isolation critical care beds are required for a more prolonged stay. Furthermore, as hospital systems open and family members can

visit in a controlled setting, wearing masks, maintaining social distancing and routine handwashing should be strictly enforced. Regardless of the risk, the role of telemedicine and out-patient follow-up, including potentially home-based rehabilitation would be crucial to improve overall outcomes [29].

Conclusion

During the ongoing COVID-19 pandemic, there is a need to further educate patients with symptoms of MI and the need to seek medical attention early. Coordinated care among emergency medical service, ED, and catheterization laboratory personnel is needed to ensure least time to revascularization. All PPCI-capable hospitals should designate a catheterization laboratory with appropriate ventilation for patients with confirmed or suspected COVID-19, and the use of PPE should be enforced when caring for these patients. Along with early discharge, patients should be monitored for electro-mechanical complications. Furthermore, height-

ened knowledge of potential drug-drug interactions when newer COVID-19 specific therapies are used is needed.

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