

Acute ST Elevation Myocardial Infarction Complicated by Out of Hospital Ventricular Fibrillation Cardiac Arrest as the First Clinical Presentation of COVID-19

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Abstract

Coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), has posed many challenges to emergency physicians with various unexpected clinical presentations. Although the majority of the admissions to the emergency department are related to respiratory symptoms and signs, cardiovascular presentations are not uncommon. We report a 50-year-old male who was brought to the emergency department following successful out of hospital cardiopulmonary resuscitation. The post-cardiac arrest electrocardiogram demonstrated ST segment elevation in the lateral chest leads and a chest radiograph performed in the emergency department showed extensive bilateral consolidation. A subsequent COVID-19 PCR (polymerase chain reaction) test was positive. To our knowledge and as per the literature reviewed, this is an uncommon presentation of COVID-19 with an acute ST-elevation myocardial infarction complicated by out of hospital ventricular fibrillation cardiac arrest. COVID-19 has been associated with various cardiac complications like myocarditis, cardiac failure, STEMI, cardiomyopathy. Presentation of COVID-19 with such cardiac complications carries a guarded prognosis.

Keywords: COVID-19; SARS-CoV-2; ARDS; STEMI; Cardiac arrest; Ventricular fibrillation

1. Introduction

Coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) was declared by the World Health Organization (WHO) as a public health emergency on January 30th, 2020 and as a pandemic on March 11th, 2020.

As of October 1st, 2020, COVID-19 cases have soared to 34 million in 213 countries around the world, with over 1 million deaths. Although it was initially recognized as a disease of the respiratory system, it soon became apparent that COVID-19 affects multiple organ systems with potentially devastating consequences.

Patients at higher risk of cardiovascular complications of COVID-19 are those with advanced age, male sex, diabetes, hypertension, obesity, and pre-existing cardiovascular disease. Various mechanisms have been postulated for cardiovascular injury in COVID-19 patients [1,2].

- Direct cardiac injury by binding to angiotensin-converting enzymes II. Binding of SARS-CoV-2 to CE2 can results in alteration, leading to acute myocardial injury.
- Systemic inflammation and cytokines storm
- Altered myocardial demand-supply ratio
- Systemic inflammation induced coronary thrombosis
- Plaque rupture secondary to increased stress

Aside from presenting as Acute Coronary Syndrome secondary to thrombosis, COVID-19 can present as myocarditis or in the form of an arrhythmia.

Although there is an increased risk of cardiovascular complications [3] in patients with COVID-19, the association between COVID-19 and out of hospital cardiac arrest is not clear. Lombardia Cardiac Arrest Registry (Lombardia CARe) studied the incidence of out of hospital cardiac arrest during the first 40 days of the COVID-19 outbreak in the Lombardy region in Italy. This registry showed a 58% increase in out of hospital cardiac arrest as compared to the same time frame last year [4].

We report a 50-year-old male who was brought into the Emergency Department with ventricular fibrillation cardiac arrest, resuscitated successfully with ECG showing an acute lateral wall STEMI and CXR showed extensive bilateral consolidation with a PCR Positive COVID-19.

2. Setting

This case was reported at the Emergency Department (ED) of Mediclinic City Hospital (MCH), Dubai Healthcare City (DHCC) Dubai, United Arab Emirates. MCH is a 230-bed multidisciplinary tertiary care hospital and a Level 1 cardiac center in the region. We treat 45,000 patients per year in our ED, of which 2400 patients present with cardiac symptoms. On average, we perform approximately 300 coronary angioplasty procedures per year.

3. Case Report

A 50-year-old Indian male patient contacted emergency services complaining of chest pain. On arrival at the scene, paramedics witnessed the collapse of the patient with sudden loss of consciousness. Cardiopulmonary Resuscitation (CPR) was commenced, and ventricular fibrillation was diagnosed. Return of spontaneous circulation (ROSC) was achieved after 1 direct current (DC) shock with 200 Joules.

On arrival at the emergency department, the patient was alert and awake and was complaining of chest pain. His blood pressure was 98/68 mm Hg and heart rate was 100 beats per minute. Oxygen saturation was 90% on room air. The patient was afebrile and had a random blood sugar of 9.1 mmol/l.

Clinical history revealed that the patient complained of fever, cough, and shortness of breath for 3 days. He did not have any history of diabetes, hypertension, hyperlipidemia, chronic pulmonary disease, or smoking. There was no significant family history of any illnesses and the patient was not taking any regular medications.

On examination, auscultation of the chest revealed bilateral crackles but no wheeze. The jugular venous pressure was not elevated and there was no lower limb edema. The Glasgow Coma Score was 15 and there were no focal neurological signs. There were no signs of head or limb trauma.

12-lead electrocardiogram (ECG) demonstrated ST segment elevation in leads I, aVL, aVR, and V2 and ST segment depression with T wave inversion in leads II, III, aVF and V6 (FIG. 1). The findings suggested an acute lateral wall ST-elevation myocardial infarction (STEMI) with inferior wall ischemia and therefore the primary percutaneous coronary intervention pathway was activated.

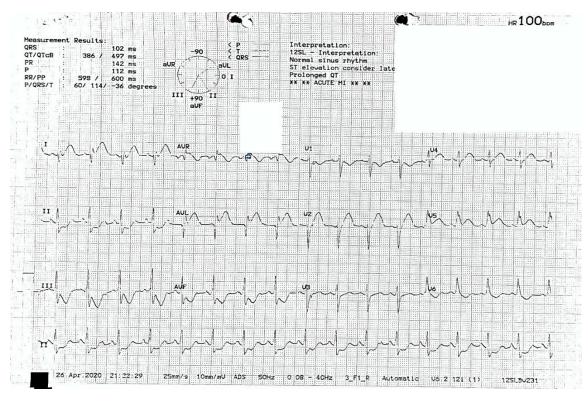


FIG. 1. ECG at Presentation.

The patient was given aspirin (acetylsalicylic acid) 300 mg and Brilinta (Ticagrelor) 180 mg per oral. Opiate analgesia was administered intravenously (morphine 5 mg) along with intravenous metoclopramide 10 mg.

A chest radiograph demonstrated extensive bilateral peripheral pulmonary consolidation with no overt signs of cardiac failure. Costophrenic angles were clear. These findings were consistent with COVID-19 pneumonitis. Laboratory investigations were done for acute coronary syndrome as well as for COVID-19 presentation (TABLE 1).

of 7.48, pCo2 30, p02 51 14.7 K/Ul 86.7% 273.72 mg/L 2.787 ng/ml 2.87 ug/ml FEU	4.00-11.00 K/Ul 45%-65% 0.00-5.00 mg/L <0.034 ng/ml 0.0-0.5 ug/ml FEU
86.7% 273.72 mg/L 2.787 ng/ml 2.87 ug/ml FEU	45%-65% 0.00-5.00 mg/L <0.034 ng/ml
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2.787 ng/ml 2.87 ug/ml FEU	<0.034 ng/ml
2.87 ug/ml FEU	-
	0.0-0.5 ug/ml FEU
15.4	
15.4 seconds	11-15 seconds
1.2	
664 U/L	125-243 U/L
25 g/L	35-50 g/L
87 IU/L	0-55 IU/L
104 IU/L	5-34 IU/L
2128 ng/ml	21.8-274.7 ng/ml
0.47 ng/ml	<0.05 ng/ml
Positive	
	tic Peptide (BNP), Thyroid Stimulatir
	664 U/L 25 g/L 87 IU/L 104 IU/L 2128 ng/ml 0.47 ng/ml Positive

TABLE 1. Laboratory	Investigations.
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4. Coronary Angiography Report

Posterior-anterior cranial projection of the right coronary artery demonstrating a dominant vessel that is free of atheroma. The right coronary artery gives rise to the posterior descending artery and posterior left ventricular branch which are also free of atheroma (FIG. 2).





The posterior-anterior caudal projection of the left coronary arterial system demonstrated that the left main stem and nondominant left circumflex artery are normal. The left anterior descending artery was occluded at the ostium with intracoronary thrombus (FIG. 3).



FIG. 3.

A balanced middleweight coronary wire was positioned in the distal left anterior descending artery and an Export Advance Aspiration catheter (Medtronic, USA) was delivered on the wire. After the aspiration of intracoronary thrombus, TIMI 2-3 flow was restored in the vessel (FIG. 4). Intravenous bolus and infusion of Aggrastat (Tirofiban) was administered during the procedure.





Repeat imaging did not reveal any obvious coronary artery stenosis. Intravascular ultrasound or optical coherence tomography was not performed as the patent's respiratory function deteriorated during the procedure and he was not able to lie flat. The patient was intubated and invasively ventilated shortly after the coronary intervention due to hypoxia and respiratory failure secondary to COVID-19 bilateral pneumonitis.

At the end of the procedure, there was TIMI 2-3 flow in the left anterior descending artery. Coronary stenting was not performed as there did not appear to be any coronary artery stenosis. The patient's chest pain and the ECG changes of ischemia resolved with intracoronary thrombus aspiration. The post-procedure ECG can be seen in (FIG. 5).

We postulate that the presentation with anterior ST-elevation myocardial infarction might be due to COVID-19 related coronary artery embolization of thrombus.

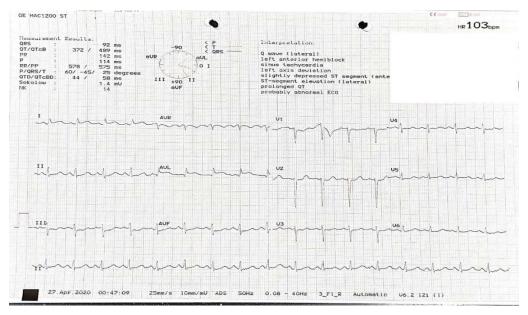


FIG. 5. ECG Post-Coronary Intervention.

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5. Clinical Course

Following the coronary intervention, the patient was transferred to the intensive care unit for invasive ventilation and supportive care. He developed cardiogenic and septic shock.

Transthoracic echocardiography demonstrated severe global hyperkinesia with overall severe left ventricular systolic dysfunction and an estimated left ventricular ejection fraction of 20%. There was no evidence of valvular heart disease, atrial or ventricular septal defects or pericardial effusion.

Unfortunately, despite maximal intravenous inotropic support and aggressive invasive ventilation, the patient's condition deteriorated with intractable hypoxia and organ hypo perfusion. Extra-corporeal membrane oxygenation (ECMO) was considered but not initiated given the very poor prognosis with COVID-19 infection and multi-organ failure. The patient suffered a cardiac arrest on the 18th day of admission and could not be resuscitated despite prolonged CPR.

6. Discussion

Cardiac arrest outcomes have steadily improved due to the emphasis on rapid recognition, high-quality chest compressions, rapid defibrillation, and early access to reperfusion strategies [1,2]. There are standardized, measurable time goals which assist in achieving these outcomes, such as door-to-ECG and door-to-needle time. However, in the rush to deliver the patient with a STEMI to the cardiac catheterization suite, emergency physicians may overlook the mimics masquerading as a STEMI, often to the detriment of both the patient and the emergency staff [5].

In the new era of COVID-19, additional clinical and logistical challenges have surfaced regarding the rapid workup and treatment of a patient with an ST elevation myocardial infarction [5].

Firstly, there is the risk of exposure to healthcare workers:

Administration of CPR involves performing numerous aerosol-generating procedures, including chest compressions, positive pressure ventilation, and establishment of an advanced airway; viral particles can remain suspended in the air with a half-life of approximately 1 hour and be inhaled by those nearby; the extent of exposure to multiple providers and the close proximity to the patient during the resuscitation efforts increases their risk and it is possible that under high-stress emergent events such as the resuscitation of a cardiac patient, that lapses in infection control may occur [6,7].

The second major challenge given the urgency of reperfusion strategies is to have a rapid and accurate diagnosis for the STEMI seen on ECG bearing in mind the differential diagnoses in the COVID-19 pandemic that mimic STEMI. These STEMI mimics include myocarditis, microvascular thrombosis, cytokine-mediated injury, and stress-induced cardiomyopathy [8,9]. One particular question remains as to whether the normal reperfusion pathways need to be adjusted bearing in mind the increased risk for acute kidney injury with a contrast induced nephropathy [10].

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7. Conclusion

COVID-19 has been associated with various cardiac complications like myocarditis, cardiac failure, STEMI, cardiomyopathy. Presentation of COVID-19 with such cardiac complications carries a guarded prognosis. We postulate that the presentation with anterior ST-elevation myocardial infarction might be due to COVID-19 related coronary artery embolization of thrombus.

8. Ethics Approval & Patient Consent

Ethics approval was obtained from the hospital research and ethics committee. No written consent has been obtained from the patient as there is no patient identifiable data included in this case report. Patient died and we lost follow up with the next of kin of the patient, therefore a consent waiver has been obtained from Mediclinic research and ethics committee for publication of this case report and accompanying images. A copy of the same is available for review by the Editor-in-Chief of this journal on request.

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