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Pharmaco-invasive Therapy for STEMI in a Patient with COVID-19: A Case Report

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Abstract

Coronavirus disease 2019 (COVID-19) is a pandemic that started in the Wuhan province of China in December 2019. It is associated with increased morbidity and mortality mainly due to severe acute respiratory syndrome 2 (SARS-Cov-2). Cardiac manifestations related to COVID-19 include demand ischemia, fulminant myocarditis, myocardial infarction and arrhythmias. In this report, we present a case of ST-segment elevation myocardial infarction (STEMI) in a 68-year-old man with COVID-19 who initially presented with chest pain and shortness of breath. Patient's STEMI was managed with pharmaco-invasive strategy with tissue plasminogen activator (t-PA). He then developed acute hypoxic respiratory failure that was managed in the intensive care unit (ICU), together with multi-organ failure from which the patient died 2 days after presentation. Although the pathophysiologic mechanisms of STEMI in COVID-19 patients has not been clearly established, we hypothesize that interrelated pathogenetic factors, that we highlight in this report, can play a role in the development of STEMI, including plaque rupture secondary to systemic inflammation, increased pro-coagulants, endothelial dysfunction, impaired fibrinolysis and impaired oxygen utilization leading to demand/supply mismatch and myocardial ischemia.

Keywords

COVID-19; ST-segment elevation myocardial infarction (STEMI); Pharmaco-invasive therapy

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1. Introduction

Coronavirus disease 2019 (COVID-19) is a pandemic encompassing the entire globe at this current time. Similar to previous coronaviruses and influenza epidemics, it has been observed that there has been increased morbidity and mortality in COVID-19 patients with pre-existing cardiovascular disease. [1] There also appears to be an association with myocardial injury. [2,3] We aim to describe the case of a 68-year-old man with coronavirus who presented with ST elevation MI (STEMI).

2. Case Presentation

A 68-year-old male with a past medical history of lung cancer and asthma presented to the emergency department with severe shortness of breath and acute onset chest pain for the last 2 hours. The patient reportedly complained of subjective fevers, cough, dyspnea and diarrhea over the past few days. On arrival, vital signs revealed a blood pressure of 116/80 mmHg, heart rate of 120 beats per minute, oxygen saturation of 89% on room air, and a body temperature of 100.3 F. Physical examination was pertinent for tachypnea and inability to speak in complete sentences. Initial blood work was significant for leukocytosis of 20.36 K/uL, troponin of 75 ng/mL, C-reactive protein of 247 mg/dL, procalcitonin of 7.4 ng/mL and ferritin of 2634.1 ng/m. Complete laboratory test results are shown in (Table 1).

Chest X-ray (CXR) displayed bilateral airspace opacities, more predominant in the right lung (Figure 1). A 12-lead electrocardiogram (ECG) sinus tachycardia, Q waves with ST-elevation in leads III and aVF, ST segment elevation in leads II, V4, V5, V6 and ST segment depression in lead I and aVL (Figure 2). The patient received 325 mg of Aspirin, 600mg of Plavix and 100mg of tissue plasminogen activator (tPA). The patient was subsequently intubated for acute hypoxic respiratory failure. Within 12 hours, the patient suffered a cardiopulmonary arrest, advanced cardiovascular life support was initiated. Return of spontaneous circulation was obtained in 4 minutes. Thereafter, the patient was admitted to the intensive care unit however the patient expired the day after admission from multi-organ failure.

3. Discussion

The emergence of coronavirus disease 2019 (COVID-19) has become a pandemic since its discovery in Wuhan, China in late 2019. It is caused by the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2). It is a single stranded enveloped RNA virus. [1] Infection occurs by the binding of the virus spike protein to the human angiotensin-converting enzyme 2 (ACE2) receptor. [4] ACE 2 is found in the type II pneumocyte in the lung, which is theorized to be the main point of entry of the virus. The ACE2 Receptor is also found in the heart, where it antagonizes the effects of angiotensin 2 in diseases where neurohormonal dysregulation is present. [5] [6]. This explains the observation of various studies done, which show that most of the clinical manifestations from COVID-19 infection are respiratory in nature, although severe cardiovascular events have been reported in the literature. [7]

In a cohort study of 416 patients who tested positive for COVID, Shi et al. [3] described evidence of myocardial injury in 82 patients (19.7%), as evidenced by elevated high sensitivity troponin I (TnI) levels. These patients had a significantly elevated in-hospital mortality rate (42 of 82 [51.2%]) compared to those who did not have biochemical evidence of myocardial injury (15 of 335 [4.5%]). In the group with myocardial injury, greater degrees of troponin elevation were associated with increased mortality.

A study by Guo et al. [2] reported that 27.8% of patients in a cohort of 187 patients. A significant difference within in-hospital mortality for patients with elevated troponin T levels (59.6%) as compared with patients with normal TnT (8.9%). The highest mortality rate was observed in the cohort with pre-existing cardiac disease and elevated troponin (25 of 36 [69.4%]). It was also found that elevated C-reactive protein, procalcitonin and N-terminal-pro-B-type natriuretic peptide (NT-proBNP) were linked with elevated troponin t levels, suggesting a close interaction between inflammation and myocardial stress. [2]

Studies from previous influenza and coronavirus epidemics have shown an association with viral infections and adverse cardiac outcomes, including acute coronary syndromes [8], arrhythmias [9] and exacerbation of heart failure [10]. The mechanism of myocardial injury during COVID-19 infection has been theorized to be multifactorial. Direct toxicity of the virus to cardiac myocytes, systemic inflammatory responses, destabilization of pre-existing coronary plaque and aggravated hypoxia are some of the predominating ideas behind mechanism of injury. [2] It can be extrapolated that diffuse endothelial injury and impaired fibrinolysis are likely additional factors. Impaired myocardial oxygen utilization in the setting of elevated inflammatory cytokine levels may further contribute to cardiac ischemia.

In China, where the outbreak of COVID-19 initially started, there have been reports of COVID associated disseminated intravascular coagulation (DIC). Wang YD, et al [11] describes two cases of COVID-19 infection complicated by DIC. Acute myocardial infarction can be attributed to DIC, due to dysregulation and dysfunction in the coagulation cascade. [12,13] Coagulopathy has also been seen in the dysregulated immune state seen in severe COVID-19 infections. Currently, early stage observation studies have shown some benefit in anticoagulation with heparin. [14,15] (Figure 3).

To date, this case report may be the first documented case of COVID-19 associated ST segment elevation myocardial infarction (STEMI). Due to the novel nature of this virus, we feel that surveillance for STEMI associated with the virus is warranted. As of this point, there is not a clear pathophysiological mechanism to account for STEMI, only theorized mechanisms derived from past coronavirus and influenza epidemics. We hope that further case reports, cohort studies and autopsy data can further our understanding of this disease.

Emergency intravenous thrombolysis (pharmaco-invasive strategy) is indicated as the first line management of STEMI patients who are COVID-19 positive. Our management was consistent with the updated American College of Cardiology guidelines however the patient’s clinical condition deteriorated rapidly. This case reflects the staggering mortality rate associated with COVID-19 infection in patients with myocardial involvement, as

reported in previous cases in China. Given the various mechanisms at play and a limited window for intervention, an ideal approach is yet to be determined; however, for now, thrombolytic therapy remains the recommended approach in COVID-19 confirmed cases.

4. Conclusion

COVID-19 is a disease that can present with severe pulmonary, cardiovascular, hematologic and immunologic manifestations. We believe that all of these interrelated systems can play a role in the development of ST segment elevation myocardial infarction. Further studies and autopsy data will be needed to truly assess the impact of COVID-19 and its relationship to the development of STEMI.

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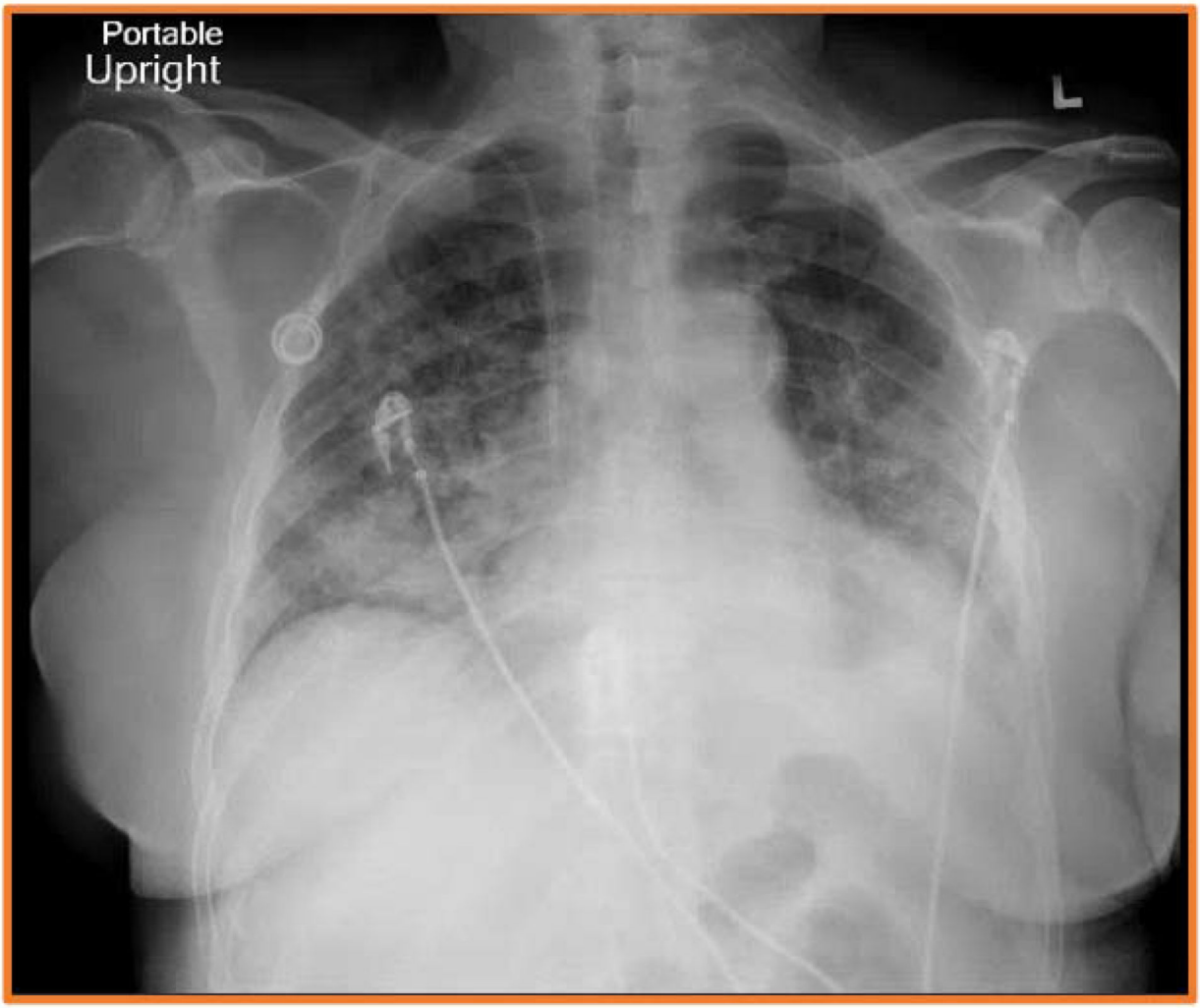

Figure 1.
Chest X-ray showing bilateral opacities

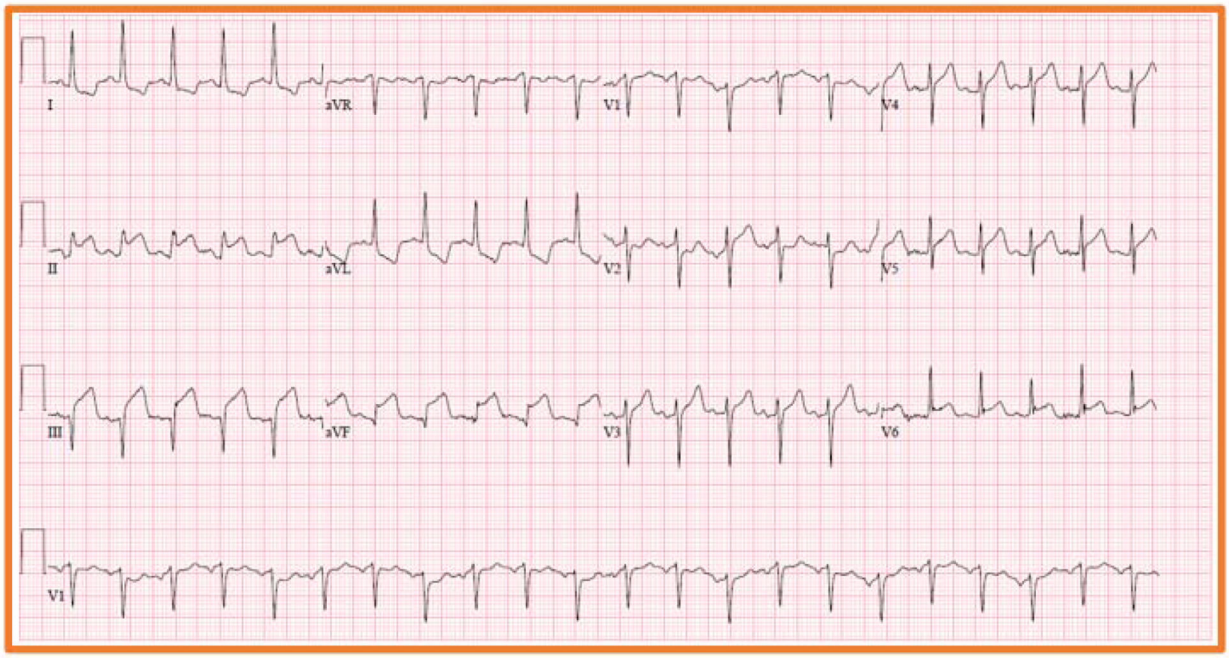

Figure 2.
12-lead ECG showing sinus tachycardia, Q waves with ST-elevation in leads III and aVF, ST segment elevation in leads II, V4, V5, V6 and ST segment depression in lead I and aVL

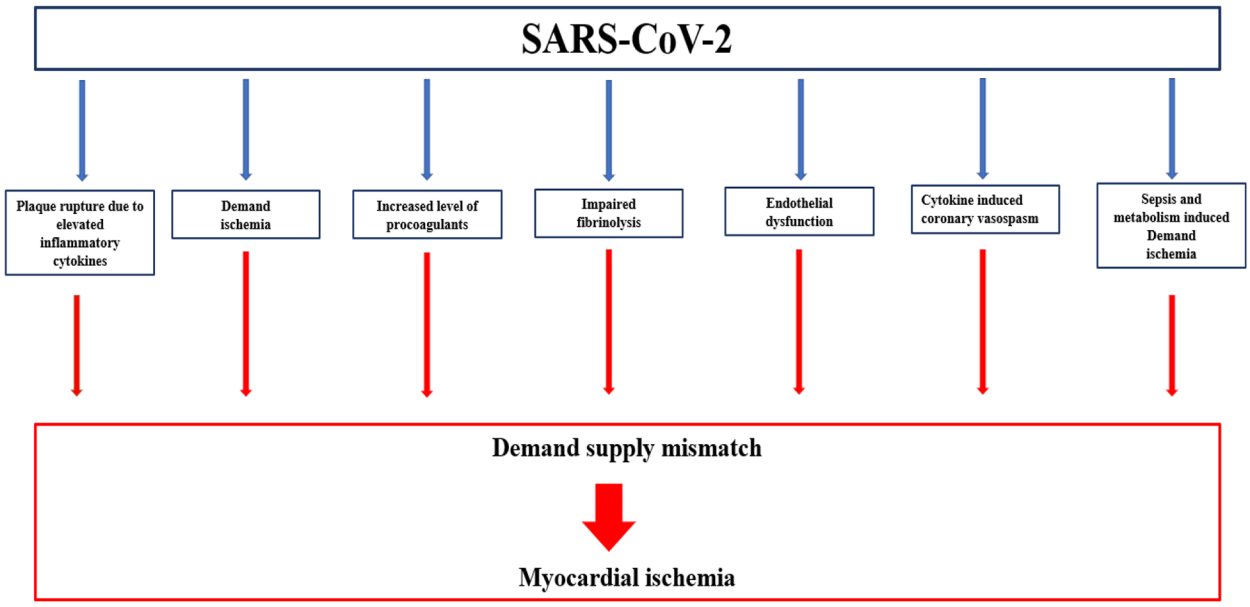

Figure 3.
Summarizing etiopathogenesis of myocardial infarction in SARS-CoV-2

Table 1.

Summary of Laboratory Data

Serum	Day 1 (Admission)	Day 2 (expired)	Reference range
WBC (K/uL)	20.36	16.5	3.5 – 10.8
Hemoglobin (g/dL)	12.3	9.9	14.0 – 18
Hematocrit (%)	41.8	34.3	42.0 – 52.0
Platelets (K/uL)	232	147	130 – 400
Sodium (mmol/L)	142	145	136 – 145
Potassium (mmol/L)	4.8	5.4	3.5 – 5.1
Chloride (mmol/L	106	114	98 – 107
BUN (mg/dL)	59	104	7 – 25
Creatinine (mg/dL)	2.8	5.8	0.7 – 1.3
Calcium (mg/dL)	8.5	8.2	8.2 – 10.0
Total Protein (g/dL)	6.9	5.7	6.0 – 8.3
Albumin (g/dL)	3.62	2.78	3.5 – 5.7
AST (U/L)	67	252	13 – 39
ALT (U/L)	56	61	7 – 52
Alk Phos (U/L)	104	105	34 – 104
Troponin (ng/mL)	75 94.36		[<=0.15
Lactate Dehydrogenase (U/L)	856		140 – 271
CRP (mg/dL)	247		0 – 8
Procalcitonin (ng/mL)	7.4		0.00 – 0.1
Ferritin (ng/mL)	2634.1		16.0 – 294.0
pH (arterial)	7.29	7.35	7.31–7.41
Oxygen partial pressure	68.6	30.7	80 – 105
Carbon dioxide partial pressure	18.1	17.5	23 – 27
Lactic acid level	5.7		0.5 – 1.6