

Covid-19 Inducing Acute Myocardial Infarction with Mitral Regurgitation and Pneumonia; The Risks and Poor Outcome: A Case Report in Cardiology, Infectious Diseases, and Critical Care Medicine

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Abstract

Rationale: A novel COVID-19 with a severe acute respiratory syndrome had arisen in Wuhan, China in December 2019. Thrombosis is a critical clinical entity commonly recognized as a sequel in COVID-19 patients. Interestingly, the presentation of COVID-19 infection with thromboembolism has a risk impact on both morbidity and mortality in COVID-19 patients. There is a correlation between COVID-19 infection and myocardial infarction.

Patient concerns: A 70-year-old married, housewife, Egyptian female patient was admitted to the critical care unit with acute inferior and right ventricular ST-segment elevation myocardial infarction with mitral regurgitation post-COVID-19 pneumonia.

Diagnosis: Acute inferior and right ventricular ST-segment elevation myocardial infarction with mitral regurgitation post-COVID-19 pneumonia.

Interventions: Electrocardiography, oxygenation, streptokinase intravenous infusion, and echocardiography.

Outcomes: Poor outcome and death happening after developing mitral regurgitation and subsequent pulmonary hypertension.

Lessons: Emerging mitral regurgitation and subsequent pulmonary hypertension after acute inferior and right ventricular ST-segment elevation myocardial infarction post-COVID-19 pneumonia in an elderly woman are highly critical. The death after COVID-19 pneumonia, inferior with right ventricular myocardial infarction, and elderly, mitral regurgitation, and pulmonary hypertension may be reasonable.

Key words: COVID-19; Coronavirus; Pneumonia; Thromboembolism; Myocardial infarction; Mitral regurgitation.

ABBREVIATIONS

IMI: Inferior myocardial infarction, COVID-19: Coronavirus disease 2019, ECG: Electrocardiogram, ICU: Intensive care unit, MI: myocardial infarction, O₂: Oxygen, RV: Right ventricle, SGOT: Serum glutamic-oxaloacetic transaminase, SGPT: Serum glutamic-pyruvic transaminase, STEMI: ST-segment elevation myocardial infarction, VR: Ventricular rate

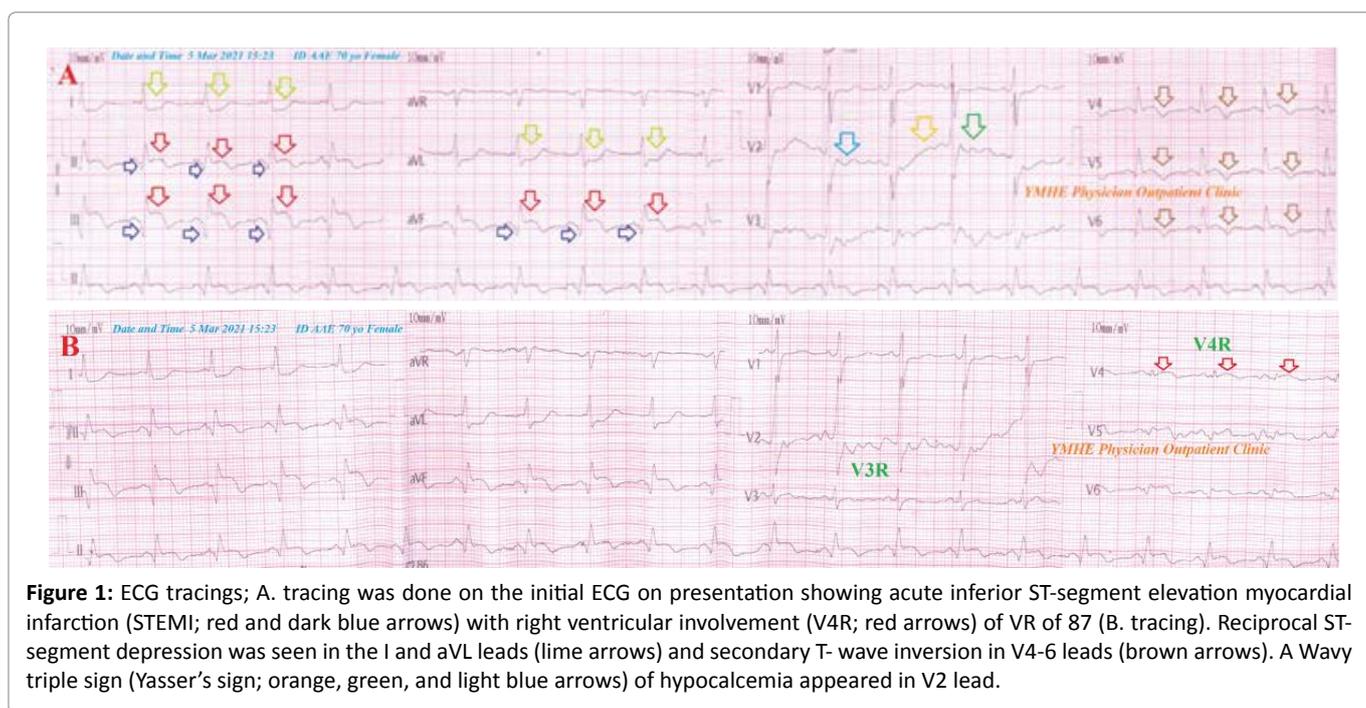
INTRODUCTION

Despite COVID-19 disease being initially reported with respiratory symptoms, but cardiovascular involvement was frequent rather than the presence of higher rates¹. Indeed, cardiac complications may be manifested as a late phenomenon in respiratory COVID-19 patients. Thrombosis is one of the most common cardiovascular complications among COVID-19 patients [1]. There is a higher incidence

of thrombosis in patients with SARS-CoV-2 disease. These complications are seen in multiple organ failures starting from cutaneous thrombosis to pulmonary embolism, cerebrovascular accident, and coronary thrombosis [2]. In some observational studies, thrombotic complications were reported as high as 31% in patients indicating critical care admission [2]. The essential role of thrombo-inflammation and endothelial injury in the pathogenesis of the COVID-19 is detected. Overproduction of pro-inflammatory cytokines such as tumor necrosis factor (TNF), Interleukin (IL)-6, IL-8, and IL-1 β that is known as “cytokine release syndrome” or “cytokine storm” is not unique to this disease. Also, it had described in sepsis and sterile inflammation [2]. This exaggerated cytokine response may lead to multiorgan failure and eventually death in some patients [3]. A high index of clinical suspicion for thrombotic status and their consequences is needed for accurate and quick diagnosis [2]. Multiple mechanisms have been suggested for cardiac damage in the COVID-19 epidemic. The systemic inflammatory response in severe COVID-19 is the producing high levels of cytokines causing cytokine-release syndrome (CRS) that can injure multiple tissues, involving vascular endothelium and cardiac myocytes [4]. Plaque rupture causes acute myocardial infarction due to the systemic inflammation and catecholamine surge in this disease [5,6]. Coronary thrombosis also has been identified as a possible cause of AMI in COVID-19 patients [7].

CASE PRESENTATION

A 70-year-old married housewife Egyptian female patient presented to the physician outpatient clinic (POC) with acute chest pain, tachypnea, and sweating. Fatigue, loss of appetite, loss of smell and taste, and generalized body aches were associated symptoms. The chest pain was crushing, compressive, and ischemic. The patient gave a history of the symptoms for 3 days. Currently, she had a history of contact with her relative who confirmed a COVID-19 patient in the past 10 days. Informed consent was taken. Upon general physical examination; generally, the patient was tachypneic, distressed, with a regular pulse rate of VR; 86 bpm, blood pressure (BP) of 130/70 mm Hg, respiratory rate of 30 bpm, the temperature of 36.5 °C, and pulse oximeter of oxygen (O₂) saturation of 93%. She was referred to the nearest central hospital. The patient was admitted to the COVID ICU unite. She was treated with COVID-19 pneumonia with acute myocardial infarction. Initially, the patient was treated with O₂ inhalation by O₂ cylinder (100%, by nasal cannula, 5L/min), SC enoxaparin 80 mg twice daily), aspirin tablet (75 mg, once daily), clopidogrel tablet (75 mg, once daily, pethidine HCL (100 mg was given on intermittent IV doses), and streptokinase IVI (1.5 million units over 60 minutes) were given. Medications for COVID-19 pneumonia were added according to standard Egyptian protocol were added. The initial ECG on presentation showed acute inferior ST-segment elevation myocardial infarction (STEMI) (Figure 1A) with right ventricular (RV) involvement (V4R) of VR



of 87 (Figure 1B). Reciprocal ST-segment depression was seen in the I and aVL leads and secondary T-wave inversion in V4-6 leads. A Wavy triple sign (Yasser's sign) of hypocalcemia appeared in V2 lead. The initial complete blood count (CBC); Hb was 9.9 g/dl, RBCs; $3.9 \times 10^3/\text{mm}^3$, WBCs; $19.4 \times 10^3/\text{mm}^3$ (Neutrophils; 78 %, Lymphocytes: 13.2%, Monocytes; 8.8%, Eosinophils; 0% and Basophils 0%), Platelets; $182 \times 10^3/\text{mm}^3$. S. Ferritin was high; 544 ng/ml. D-dimer was very high (2209 ng/ml). CRP was high (102.7 g/dl). LDH was high (602 U/L). SGPT was normal (41 U/L), SGOT was normal (37 U/L). Serum

creatinine was normal (1.2 mg/dl) and blood urea was normal (48 mg/dl). RBS was normal (117 mg/dl). Ionized calcium was slightly low (0.9 mmol/L). The troponin test was positive (0.25 U/L). CK-MB was high (250 U/L). The echocardiography was done on the presentation showing regional wall motion abnormalities, grade II, left ventricular diastolic dysfunction, moderate mitral regurgitations, and moderate pulmonary hypertension with an EF of 57%. (Figure 2A). The plain CXR was done on the day of the presentation showing enlargement of both pulmonary arteries and shadow of right ventricular

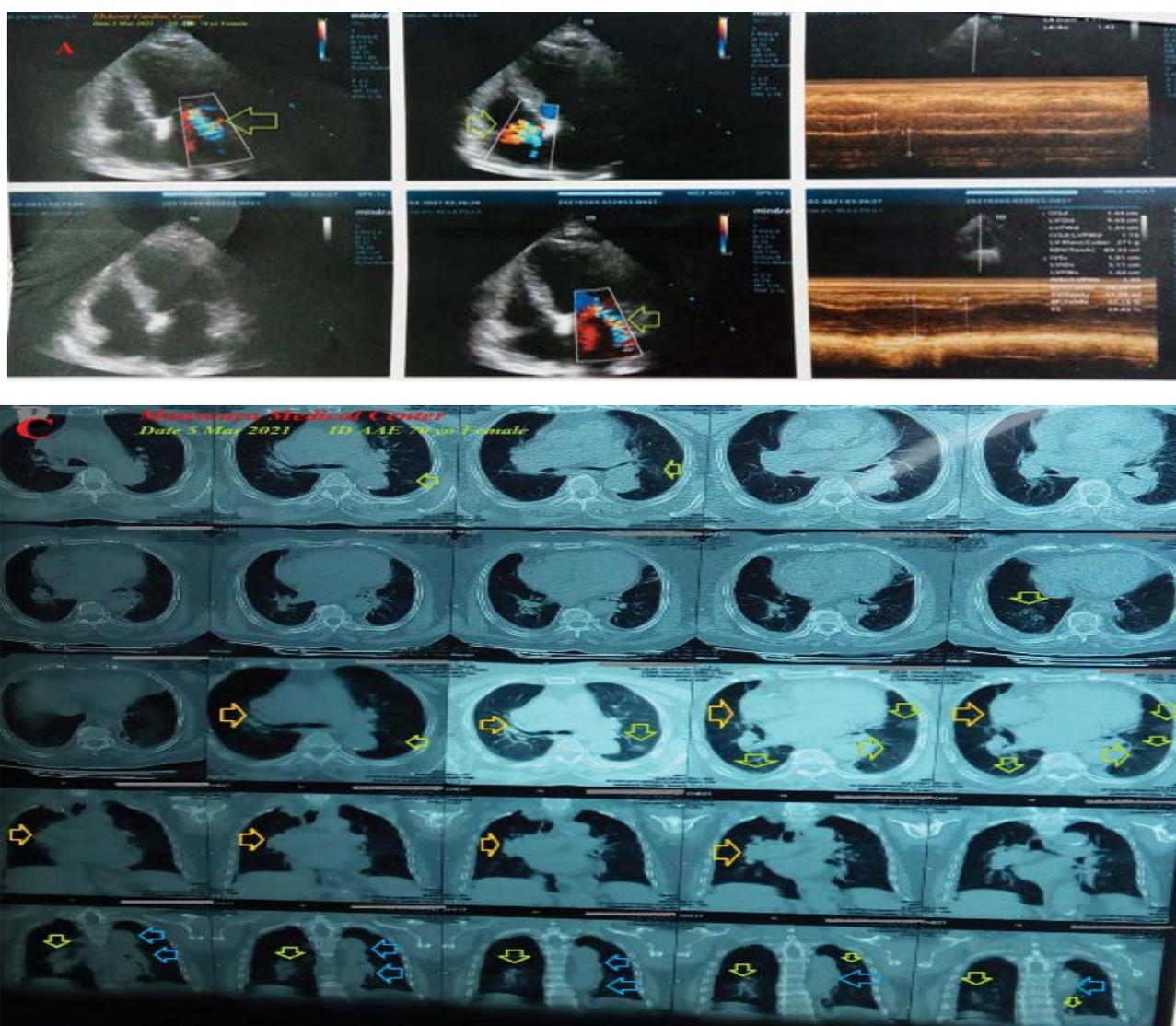


Figure 2: (A). The echocardiography was done on the presentation showing mitral regurgitations with EF of 57%. (B). The plain CXR was done on the day of the presentation showing enlargement of both pulmonary arteries and shadow of right ventricular enlargement (C). The initial chest CT was done on the day of the presentation showing bilateral variable-sized ground-glass opacities (lime arrows), enlargement of both pulmonary arteries (blue arrows), and shadow of right ventricular enlargement (orange arrows).

enlargement (Figure 2B). The initial chest CT was done on the day of the presentation showing bilateral variable-sized ground-glass opacities, enlargement of both pulmonary arteries, and shadow of right ventricular enlargement (Figure 2C). Acute inferior and right ventricular ST-segment elevation myocardial infarction with mitral regurgitation post-COVID-19 pneumonia was the most probable diagnosis. Unfortunately, the patient died within 7 days of ICU admission after the sudden attack of acute ventricular tachycardia.

DISCUSSION

Overview

- A 70-year-old married, housewife, Egyptian female patient was admitted to the ICU with acute inferior and right ventricular ST-segment elevation myocardial infarction (STEMI) with mitral regurgitation post-COVID-19 pneumonia.
- The primary objective for my case study was the presence of a patient who presented with acute inferior and RV STEMI with mitral regurgitation post-COVID-19 pneumonia in the ICU.
- The secondary objective for my case study was the question of; How did you manage the case at home?
- There was a history of direct contact to confirm the COVID-19 case.
- The presence of direct contact to confirm the COVID-19 case, and unilateral ground-glass consolidation on top of acute tachypnea will strengthen the COVID-19 diagnosis.
- There is an existence of acute inferior and right ventricular STEMI. This acute myocardial infarction was indicated for immediate thrombolytic.
- An associated right ventricular infarction in the presence of acute inferior STEMI will be rising the mortality to 50%.
- An elderly, female sex, marked elevated d-dimer, positive troponin in the presence of COVID-19 add other bad prognostic points and is indicating a high-risk condition.

- Emerging mitral regurgitation and subsequent pulmonary hypertension will be another serious risk impact.
- Acute coronary spasm was the possible differential diagnosis for the current case study.
- I can't compare the current case with similar conditions. There are no similar or known cases with the same management for near comparison.
- The only limitation of the current study was the unavailability of cardiac catheterization.

CONCLUSION AND RECOMMENDATIONS

- Emerging mitral regurgitation and subsequent pulmonary hypertension after acute inferior and right ventricular ST-segment elevation myocardial infarction post-COVID-19 pneumonia in an elderly woman is highly critical.
- The death after COVID-19 pneumonia, inferior with right ventricular myocardial infarction, and elderly, mitral regurgitation, and pulmonary hypertension may be reasonable.

CONFLICT OF INTEREST

There are no conflicts of interest.

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