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General Medicine

COVID-19 PRESENTING WITH FAILED HEART, CONGESTED LIVER AND PATCHY LUNG IN A YOUNG PATIENT: A CASE REPORT

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ABSTRACT Coronavirus disease 2019 (COVID-19) caused by Severe acute respiratory syndrome coronavirus 2 (SARS CoV-2) was declared as a pandemic by world health organisation (W.H.O) on 11th March 2020. SARS CoV-2 enters the host cells by binding with ACE-2 receptors. Studies have shown that apart from lung, ACE- 2 receptors are expressed in many other organs which may explain the non-respiratory symptoms. Apart from thrombotic complications like acute coronary syndrome and venous thromboembolism, a wide range of arrhythmias and myocarditis with acute heart failure were reported in association with COVID-19. We report a young male admitted with complaints of exertional dyspnea for 4 days. He was subsequently diagnosed to have COVID-19 pneumonia and bi-ventricular dysfunction. Any patient presenting with acute heart failure with no history of any comorbidities and cardiovascular disease should be evaluated for COVID-19 in current scenario. Patient should be started on guideline directed medical therapy and expectant management in patients with stable hemodynamics. All the patients should receive anticoagulants to prevent thrombotic complications. ACE inhibitors and ARBs can be continued. Serial inflammatory bio markers monitoring, QTc monitoring and electrolyte monitoring is recommended.

KEYWORDS : COVID-19, SARS CoV-2, viral myocarditis, acute heart failure.

INTRODUCTION:

Coronavirus disease 2019 (COVID-19) caused by Severe acute respiratory syndrome coronavirus 2 (SARS CoV-2) was declared as a pandemic by world health organisation (W.H.O) on 11th March 2020. As on 29-06-2020 10 million cases and nearly 500000 deaths of COVID-19 have been reported globally. Older age groups and people with co-morbidities are associated with poor outcome. Hypertension is the most common co-morbidity seen in patients admitted in intensive care units. Others include cardiovascular diseases, diabetes mellitus, chronic lung diseases, chronic liver disease, malignancy, and obesity (1, 2, 3).

Though it is primarily a respiratory pathogen, extra pulmonary involvement is well reported. SARS CoV-2 enters the host cells by binding with ACE-2 receptors. Studies have shown that apart from lung, ACE- 2 receptors are expressed in many other organs like gastro intestinal tract, endothelial cells, nervous system, skeletal muscles, kidney, and cardiomyocytes which may explain the non-respiratory symptoms.

Significant minority of patients with COVID-19 were found to have cardiovascular implications. Previous coronavirus outbreaks like SARS and MERS were also associated with cardiovascular complications like hypotension, myocarditis, heart failure, arrhythmias and sudden cardiac death (4, 5). Apart from thrombotic complications like acute coronary syndrome and venous thromboembolism, a wide range of arrhythmias and myocarditis with acute heart failure were reported in association with COVID-19, especially in those with history of established cardiovascular disease. We present a young male with history of recently diagnosed hypertension (on life style modification), admitted with complaints of exertional dyspnea for 4 days. He was subsequently diagnosed to have COVID-19 pneumonia and bi-ventricular dysfunction. In this report we describe approach in evaluating and treating COVID-19 positive patients presenting with acute heart failure.

Case discussion:

A 44-year-old gentleman with history of recently diagnosed systemic hypertension (not on any medications) presented to us with shortness of breath, swelling of both lower limbs and mild abdominal distension for 5 days. He denied fever, cough, chest pain and diarrhea. No history of sick contacts, recent travel. On admission, he was afebrile (98.4^0F); his heart rate was 128/min, blood pressure 160/90 mm of hg, and oxygen saturation of 96% with 2L of oxygen. The initial electrocardiogram (ECG) showed sinus tachycardia and T wave inversion in Lead I, II, AVL, and V5 (Figure: 1). Plasma high sensitive Troponin-I was 69.3 pg/ml.

COVID-19 by RT-PCR turned out to be positive. Laboratory analysis showed white cell count of 5,700 cell/mm3, serum creatinine 1.0 mg/dl, aspartate aminotransferase 20 U/L, and alanine aminotransferase 16 U/L. N-terminal pro B-type natriuretic peptide was 6568 pg/ml, prothrombin time 13(11) and activated partial thromboplastin time 34(26) . Inflammatory biomarkers were mildly elevated (C-reactive protein 10.3, interleukin-6 38.6, D-dimer 5.44). Ultrasound abdomen revealed normal sized liver with coarse echotexture and moderate ascites. HBsAg and Anti HCV were negative. Diagnosis was considered to be COVID-19 pneumonia with myocarditis and biventricular dysfunction.

He was started on broad spectrum antibiotics, short course steroids and prophylactic anticoagulation. Drugs like Azithromycin and Hydroxychloroquine were avoided in view of risk of QTc prolongation. He was treated with diuretics, Aldosterone antagonist and angiotensin receptor blockers (ARBs) for his heart failure and hypertension. There was significant clinical improvement, inflammatory bio markers were in decreasing trend with stable haemodynamics throughout hospital stay except for the minimal oxygen support. He was discharged with oral diuretics, β- blockers, ARBs, statins and antiplatelet agents. He was advised home isolation for 10 days and to review in 1 month for elective coronary angiogram to rule out coronary artery disease.

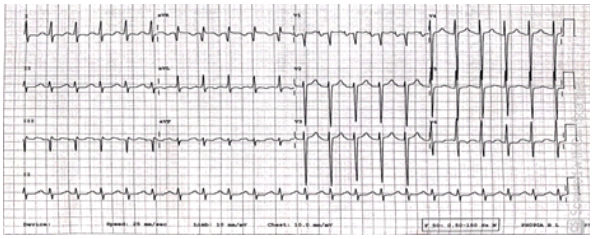
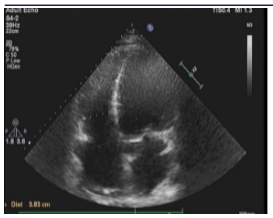
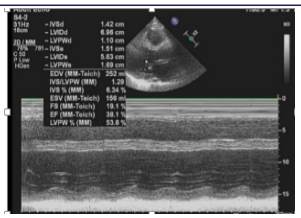


Figure- 1: ECG showing sinus tachycardia and T wave inversion in Lead I, II, AVL, and V5.

Chest X-RAY PA view was notable for cardiomegaly and bilateral lower zone infiltrates. Echocardiogram was notable for global hypokinesia of left ventricle, dilated left ventricle with severe dysfunction (ejection fraction – 35%), dilated right atrium and right ventricle with dysfunction (Figures: 2, 3).

Figures: 2, 3: Echocardiogram images showing dilated chambers with reduced ejection fraction.

Computed tomography (CT) scan revealed patchy areas of consolidation and ground glass opacities suggestive of viral etiology (Figures: 4, 5, 6).

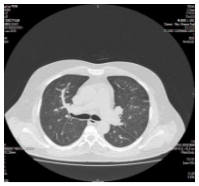
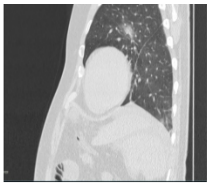
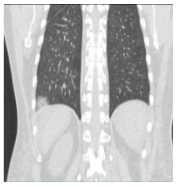




Figure-4: CT chest showing **Figure-5: CT chest showing** **Figure-6: CT chest showing**

Ground glass opacities in consolidation in left upper lobe consolidation in left lower Upper lobe

DISCUSSION:

Most common presenting symptoms in COVID-19 are fever, cough with dyspnea, anorexia, myalgia, sputum production, rhinorrhea, and headache. Altered smell or taste can be the first manifestation, reported in about 64% of patients (6). But patient presenting with acute heart failure without usual symptoms, as in our patient is rare. Cardiac injury is seen nearly in one quarter of hospitalised patients. COVID-19 associated cardiac injury is multifactorial. It may be due to direct viral invasion, cytokine storm resulting in vascular and myocardial inflammation, plaque instability. It may be due to sepsis or disseminated intravascular coagulation (DIC).

We hypothesize that acute heart failure in our case is probably secondary to viral myocarditis. This is based on acute onset, previous normal cardiac reserve, elevated HS-Troponin I and sinus tachycardia with other ECG changes. An experimental model in rabbit has shown that coronavirus infection can progress to myocarditis and congestive cardiac failure (7). It is recommended to asses these inflammatory markers (Troponin I, NT pro BNP, D-Dimer, IL-6 etc.,) as soon as admission and follow up the trend, for risk stratifying the patients. In our case, inflammatory biomarkers were elevated mildly and remained stable throughout the hospital stay which would explain the favourable outcome. QTc interval on ECG should be monitored and should be cautious with drugs causing QTc prolongation like azithromycin, hydroxychloroquine, especially if it is more than 500ms. Invasive hemodynamic monitoring and mechanical circulatory support should be limited to most critically ill patients.

Traditionally myocarditis is established by edomyocaridal biopsy. But such an approach may not be feasible in setting of covid-19, especially when there is no impact on clinical outcome. As per European society of cardiology, in COVID-19 infected patients, echocardiography and CT-coronary angiogram should be performed only if there is potential impact on the clinical outcome (7). Since he recovered significantly, cardiac imaging and angiogram were planned after a month. In a white paper published in American heart journal, Hendren et al. proposed that a majority of patients with an abnormal troponin in the setting of COVID-19 infection can be followed up with expectant management until recovery from the acute viral syndrome (8). These are to reduce the risk of transmission to health care personnel and to other patients which is very important especially in a resource limited country like ours.

COVID-19 is highly prothrombotic state and studies have shown that prophylactic heparin therapy had mortality benefit in patients with 6 fold increase in D-Dimer levels or sepsis induced coagulopathy (SIC) score more than 4 (9). We have started our patient on low molecular weight heparin on Day 1 till day of discharge and prescribed antiplatelet and statins to continue.

Initial reports suggested that ACE inhibitors and ARBs are associated with increased susceptibility for infection. But there is also contradicting evidence that these may have protective effect on lung (10). Since there is no strong evidence to suggest that these drugs will increase risk of infection or complications, we started our patient on ARBs as per standard guidelines for management of hypertension (7). COVID-19 patients are at increased risk of electrolyte disturbances and arrhythmias. In our patient since he was on diuretics for heart failure we closely monitored his serum potassium levels to prevent risk of hypokalemia and arrhythmias. In case of severe hemodynamic instability and cardiogenic shock in association of covid-19 intra-aortic balloon pump (IABP), or veno- arterial ECMO should be considered

Conclusion: In conclusion key learning points are, all the clinicians should be aware of the atypical presentations of COVID-19. Any patient presenting with acute heart failure with no history of any comorbidities and cardiovascular disease should be evaluated for COVID-19 in current scenario. Patient should be started on guideline directed medical therapy and expectant management in patients with stable hemodynamics. All the patients should receive anticoagulants to prevent thrombotic complications. ACE inhibitors and ARBs can be continued. Serial inflammatory bio markers monitoring, QTc monitoring and electrolyte monitoring is recommended.

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