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RESEARCH ARTICLE

PERSISTENT COMPLETE ATRIOVENTRICULAR BLOCK IN A COVID PATIENT

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Abstract

Myocardial injury is one of the important pathogenic features of COVID-19. As a surrogate for myocardial injury, multiple studies have shown increased cardiac biomarkers mainly cardiac troponins I and T in the infected patients especially those with severe disease. Myocarditis is depicted as another cause of morbidity amongst COVID-19 patients. The exact mechanisms of how SARS-CoV-2 can cause myocardial injury are not clearly understood. The proposed mechanisms of myocardial injury are direct damage to the cardiomyocytes, systemic inflammation, myocardial interstitial fibrosis, interferon mediated immune response, exaggerated cytokine response by Type 1 and 2 helper T cells, in addition to coronary plaque destabilization, and hypoxia. This is a 62-year-old M.K with a poorly monitored type 2 diabetes and hypertension as ATCDS who was admitted to the emergency department for syncope, a complete AVB responding to the bolus of atropine and the sternal punch. After electro systolic training probe we were able to find a regular sinus rhythm with gradual recovery of consciousness Evidence of myocardial injury is common among adults hospitalized with COVID-19. Possible causes of myocardial injury in patients with COVID-19 include myocarditis, hypoxic injury, stress (Takotsubo) cardiomyopathy, ischemic injury caused by cardiac microvascular damage or coronary artery disease, right heart strain (acute cor pulmonale) and systemic inflammatory response syndrome. COVID-19 infection has been associated with myocardial injury, which has been implicated with more severe disease courses and even death. Remarkable efforts are being done to elaborate underlying mechanisms of myocardial injury. Due to the acuteness of this pandemic, the scientific world currently lacks randomized controlled trials in order to fully elucidate the pathophysiological mechanisms and therapeutic measures.

Introduction:-

Coronavirus Disease 2019 (COVID-19) has quickly progressed to a global health emergency. Respiratory illness is the major cause of morbidity and mortality in these patients with the disease spectrum ranging from asymptomatic subclinical infection, to severe pneumonia progressing to acute respiratory distress syndrome. There is growing

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evidence describing pathophysiological resemblance of SARS-CoV-2 infection with other coronavirus infections such as Severe Acute Respiratory Syndrome coronavirus and Middle East Respiratory Syndrome coronavirus (MERS-CoV). Angiotensin Converting Enzyme-2 receptors play a pivotal role in the pathogenesis of the virus. Disruption of this receptor leads to cardiomyopathy, cardiac dysfunction, and heart failure. Patients with cardiovascular disease are more likely to be infected with SARS-CoV-2 and they are more likely to develop severe symptoms. Hypertension, arrhythmia, cardiomyopathy and coronary heart disease are amongst major cardiovascular disease comorbidities seen in severe cases of COVID-19. There is growing literature exploring cardiac involvement in SARS-CoV-2. Myocardial injury is one of the important pathogenic features of COVID-19. As a surrogate for myocardial injury, multiple studies have shown increased cardiac biomarkers mainly cardiac troponins I and T in the infected patients especially those with severe disease. Myocarditis is depicted as another cause of morbidity amongst COVID-19 patients. The exact mechanisms of how SARS-CoV-2 can cause myocardial injury are not clearly understood. The proposed mechanisms of myocardial injury are direct damage to the cardiomyocytes, systemic inflammation, myocardial interstitial fibrosis, interferon mediated immune response, exaggerated cytokine response by Type 1 and 2 helper T cells, in addition to coronary plaque destabilization, and hypoxia.

Observation:-

This is a 62-year-old M.K with a poorly monitored type 2 diabetes and hypertension as ATCDS who was admitted to the emergency department for syncope.

On admission the patient was unconscious with a heart rate of 15bpm and BP = 70/60. After rapid initial conditioning, the patient underwent an emergency ECG**(Fig.1)**demonstrating a complete AVB responding to the bolus of atropine and the sternal punch.

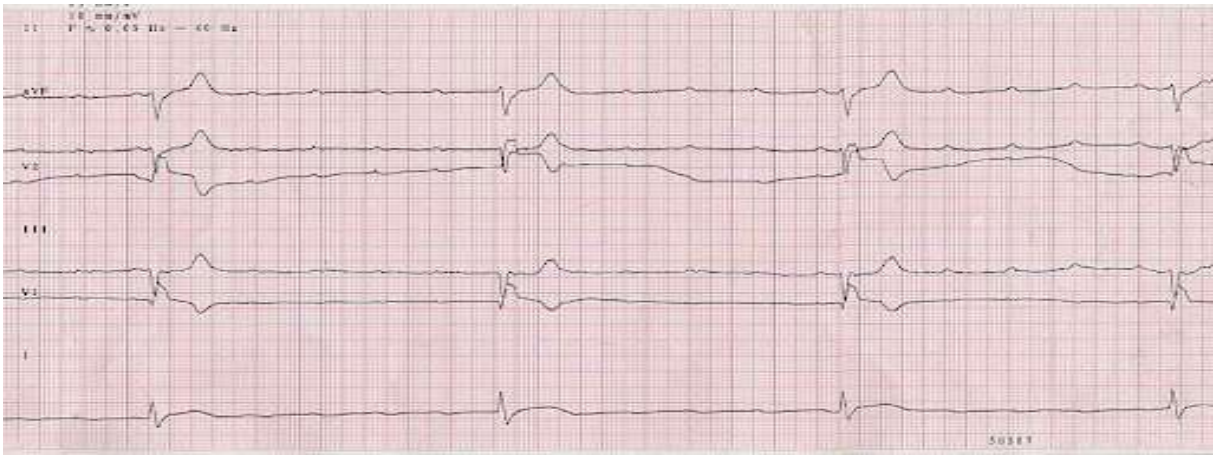

Figure 1:- ECG taken on admission showing complete atrioventricular block.

We carried out a rise of the electro systolic training probe allowing to find a regular sinus rhythm with gradual recovery of consciousness**(Fig.2).**

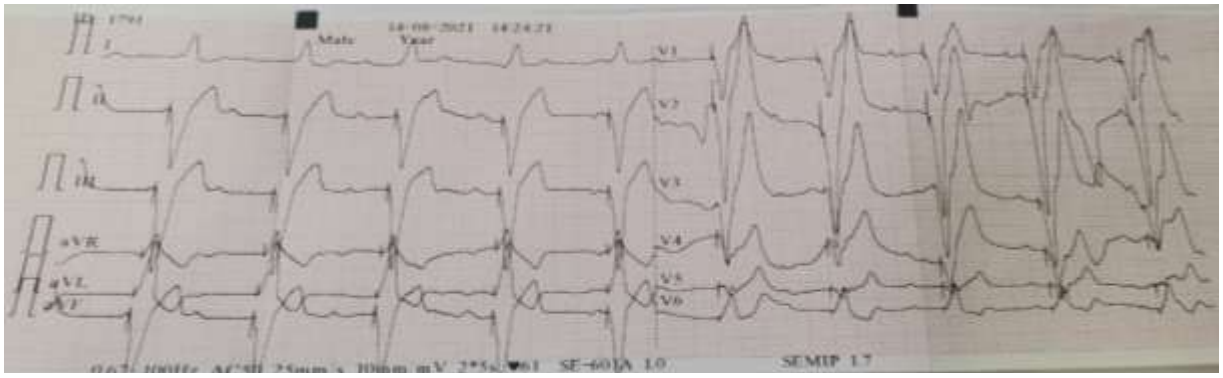

Figure 2:- ECG after installation of the electrosystolic training probe.

After stabilization, the patient underwent a cerebral CT showing no abnormalities**(Fig.3)**and a thoracic CT showing an impairment of frosted glass in favor of the covid involving 50% of the pulmonary parenchyma**(Fig.4)**.

The patient was transferred to a covid intensive care unit where the course was marked by clinical and biological improvement and was transferred to the cardiology department.

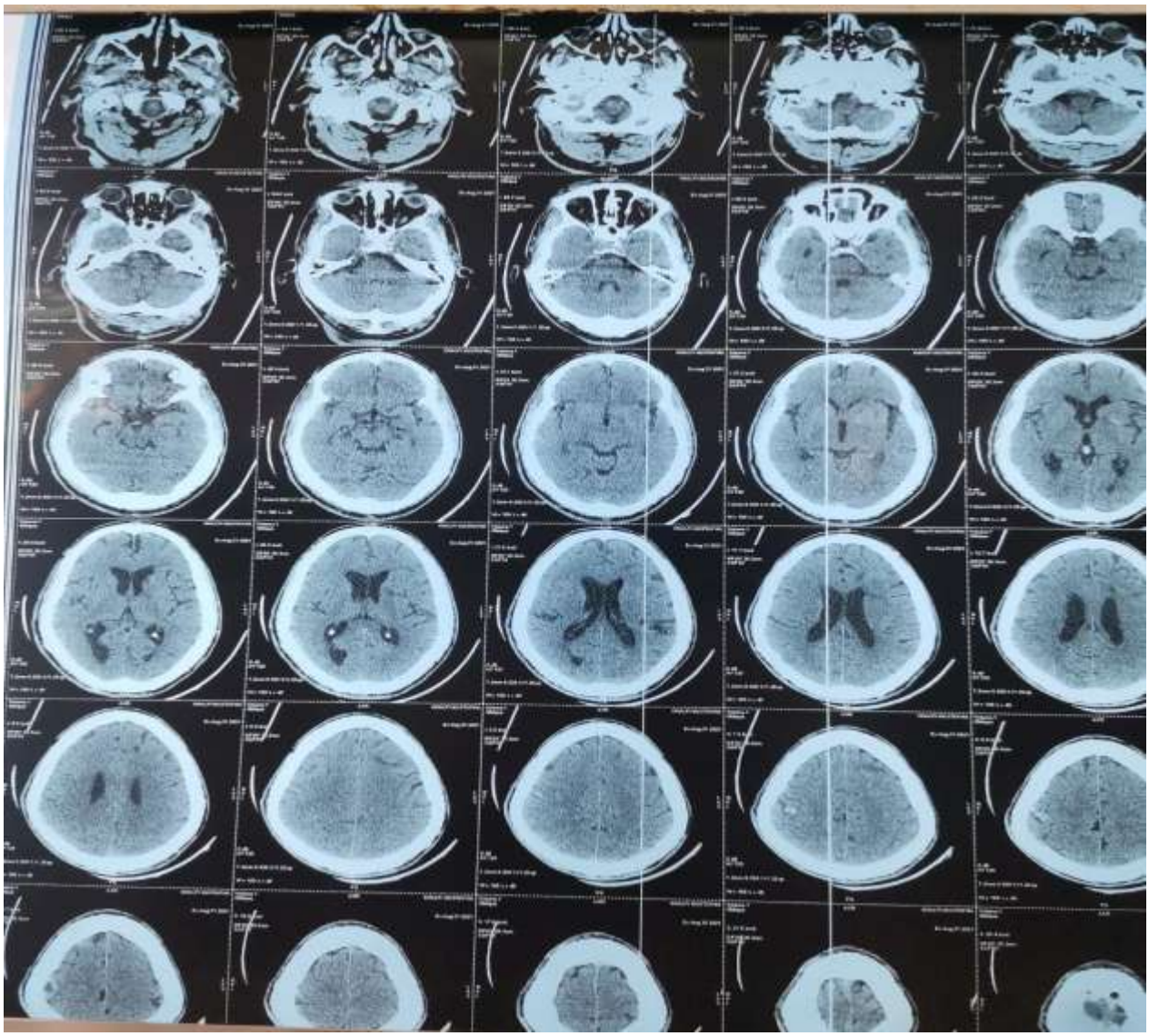

Figure 3:- Cerebral CT scan performed after stabilization of the patient.

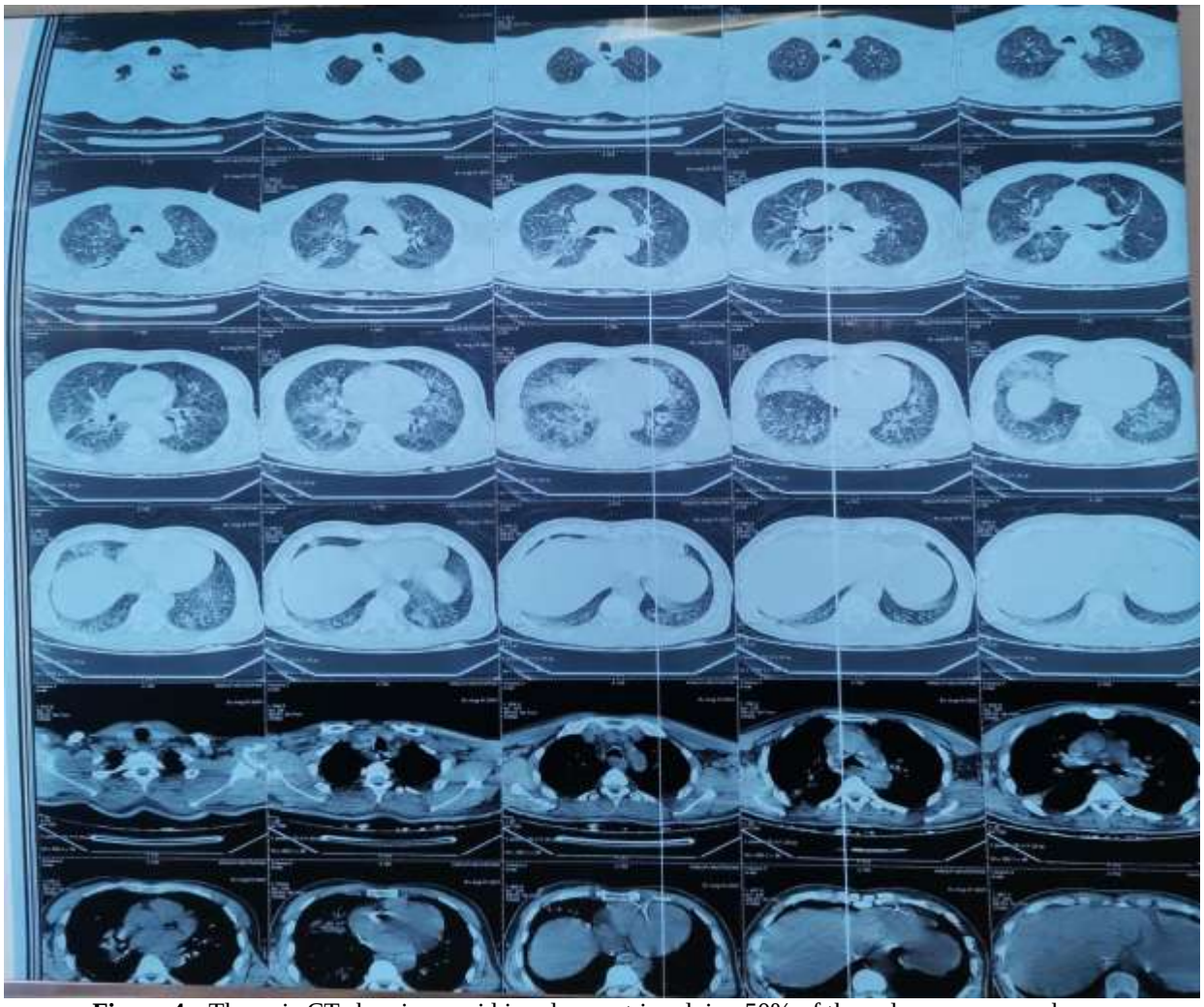

Figure 4:- Thoracic CT showing covid involvement involving 50% of the pulmonary parenchyma.

Discussion:-

Since we have limited knowledge regarding pathophysiology of cardiovascular complications related to COVID-19, especiallyconduction defects. There are different hypotheses suggested to explain the mechanism of AVB in this setting.

Evidence of myocardial injury is common among adults hospitalized with COVID-19. Possible causes of myocardial injury in patients with COVID-19 include myocarditis, hypoxic injury, stress (Takotsubo) cardiomyopathy, ischemic injury caused by cardiac microvascular damage or coronary artery disease, right heart strain (acute cor pulmonale) and systemic inflammatory response syndrome.(1-5)Clinical presentation ranges from asymptomatic troponin elevation to fulminant myocarditis requiring extracorporeal membrane oxygenation support. Atrial and ventricular arrhythmias have been reported in 3-17% of adults hospitalized with COVID-19.(6-8) Bradyarrhythmias have not been typically seen, but there was one reported case of transient complete heart block in a critically ill 54-year-old woman with COVID-19 pneumonia, who required cardiopulmonary resuscitation for ~ 10 minutes until resumption of normal sinus rhythm.(9)Increased expression of the angiotensin-converting enzyme 2 receptor (ACE-2) due to SARS-CoV-2 may also compromise normal conduction. This mainly contributes to sinus node dysfunction rather than AVB. (10)Likewise, a remarkable activation of immune response including cytokine/chemokine storm as well as amplification of the inflammatory cascade occurs.(11)Exacerbation of the preexisting conduction disorders is likely to cause advanced blocks. Moreover, infrequent reports have raised caution regarding aggravation of AVB following initial doses of azithromycin and hydroxychloroquine (12)as well as their long-term use. (13)

The etiology of the AVB in our patient remains unclear, but may result from inflammation of the conduction tissue as part of a more diffuse process of myocardial injury. Although we thought that the systemic inflammation in conjunction with hypoxemia and a transient hypercoagulable state might explain the incident AVB. Myocardial involvement was not confirmed in our patient. Maybe a subclinical injury has occurred in this case. After correction of hypoxia and regression of inflammation, the block was persistent. Infranodal damage seems unlikely since the QRScomplexes are narrow, and the rate of the escape rhythm exceeds 40 bpm. There is only one report with similar presentation of advanced AVB in non-severe stable COVID-19 patients. (14)

The hypothesis that may explain BAV in our case, is direct viral injury or prolonged antibody-mediated aggression of the AV node or adjacent tissues by virus or inflammatory factors in order to cause damage or electrical remodeling. But this hypothesis must be validated by biological, immunological and above all histological examinations.

Strong data is lacking regarding persistent AVB as well as its management in the patient with COVID. Large, multi-center studies are required to better understand the pathophysiology, clinical presentation, and impact of treatment on atrioventricular conduction disease in this category of patients.

Conclusion:-

COVID-19 infection has been associatedwithmyocardialinjury, which has been implicatedwith more severedisease courses and evendeath. Remarkable efforts are beingdone to elaborateunderlyingmechanisms of myocardialinjury. Due to the acuteness of thispandemic, the scientific world currentlylacksrandomizedcontrolled trials in order to fullyelucidate the pathophysiologicalmechanisms and therapeuticmeasures. However, there are a handful of clinical trials on the way to assess possible therapeutictargets for the treatment and prevention of thisdisease. Even whilelackingsubstantialevidence, certain conclusions can bedrawnfromthisreview. Namely, itappearsextrapulmonary manifestations are more likelywiththis SARS outbreak, and cliniciansshouldmaintain a high index of suspicion for COVID-19 infection even in patients withoutrespiratorysymptoms, as delayedtestingwillresult in increasedcommunity and healthcareworker spread.

Conflict of interest :

There arent anyconfilcts of interests to be mentioned

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