Journal of Cardiovascular Disease Research

ISSN:0975 -3583,0976-2833 VOL 15, ISSUE 04, 2024

Original research article

Myocarditis masquerading cardiovascular disorders: A case series

[1]Dr. Ashwani Kumar, [2]Dr. Saurabh Biswas, [3]Dr. Jahanvi Grover, [4]Dr. Kuldip Singh Laller

[1]Associate Professor, Department of Cardiology, PT. B.D. Sharma Post Graduate Institute of Medical Sciences, PGIMS, Rohtak, Haryana, India

[2]Senior Resident, Department of Cardiology, PT. B.D. Sharma Post Graduate Institute of Medical Sciences, PGIMS, Rohtak, Haryana, India

[3]MBBS Intern, PT. B.D. Sharma Post Graduate Institute of Medical Sciences, PGIMS, Rohtak, Haryana, India

[4]Senior Professor and Head, Department of Cardiology, PT. B.D. Sharma Post Graduate Institute of Medical Sciences, PGIMS, Rohtak, Haryana, India

Corresponding Author:
Dr. Saurabh Biswas

Abstract

Myocarditis is defined as an inflammatory infiltration of the myocardium with associated necrosis or degeneration, or both. The disease is also known as inflammatory cardiomyopathy, with various aetiologies contributing to its onset. Among these, viral infections have garnered substantial attention due to their potential to trigger myocardial inflammation and lead to severe cardiac complications. Post-viral myocarditis, a subset of myocarditis, occurs as a consequence of viral infections and represents a critical intersection between infectious diseases and cardiology. Viruses such as enteroviruses, adenoviruses, and more recently, SARS-CoV-2, have been implicated in post-viral myocarditis, underlining the broad spectrum of viral triggers that clinicians must consider. Myocarditis presents in various forms, including Fulminant myocarditis (17%), which can lead to complete resolution or severe cardiac compromise resulting in rapid deterioration and death; Acute myocarditis (65%) typically causes moderate cardiovascular compromise and incomplete recovery with the potential for cardiac dysfunction or subsequent death; Chronic active myocarditis (11%) resembles acute myocarditis but tends to progress to mild or moderate cardiac dysfunction, often with restrictive physiology, and exhibits ongoing fibrosis on histologic examination; and Chronic persistent myocarditis (7%) characterized by non-resolving active or borderline inflammatory infiltrates on histologic examination, typically without cardiovascular compromise.

Keywords: Myocarditis, ACS/STEMI, SVT with aberrancy, pleomorphic VT, heart block, heart failure with reduced ejection fraction (HFrEF), cardiovascular disorders, a case series

Introduction

Myocarditis is defined as an inflammatory infiltration of the myocardium with associated necrosis or degeneration, or both. The disease is also known as inflammatory cardiomyopathy, with various aetiologies contributing to its onset. Among these, viral infections have garnered substantial attention due to their potential to trigger myocardial inflammation and lead to severe cardiac complications. Post-viral myocarditis, a subset of myocarditis, occurs as a consequence of viral infections and represents a critical intersection between infectious diseases and cardiology. Viruses such as enteroviruses, adenoviruses, and more recently, SARS-CoV-2, have been implicated in post-viral myocarditis, underlining the broad spectrum of viral triggers that clinicians must consider [1]. As viral infections continue to affect millions globally, the incidence of post-viral myocarditis remains a pressing concern in clinical practice. The incidence and prevalence of myocarditis are unclear but it is usually seen that myocarditis affects younger individual wherein the median age of patients is 42 years.

Despite advancements in diagnostic modalities and therapeutic strategies, the diagnosis of myocarditis remains a clinical challenge. Differentiating it from other cardiac conditions with similar presentations requires a multifaceted approach, including serological markers, cardiac imaging, and endomyocardial biopsy. Recent studies have proposed novel biomarkers and imaging techniques that enhance the accuracy of diagnosis and monitoring. However, challenges persist in developing a standardized diagnostic algorithm that accounts for the diversity of viral triggers and their variable clinical manifestations.

Three stages of the disease process are listed below.

- **Acute stage (1-7 days):** Defined by direct viral cytotoxicity and focal or diffuse necrosis of the

ISSN:0975 -3583,0976-2833 VOL 15, ISSUE 04, 2024

myocardium with subsequent exposure to host proteins and activation of the innate immune response. Adenovirus and enterovirus cause direct cell toxicity, whereas parvovirus infests endothelium and releases pro-inflammatory cytokines. Influenza causes T-cell-regulated inflammatory response.

- **Subacute stage (1-4 weeks):** An increase in autoimmune-mediated injury with activated T cells and B cells and subsequent antibody production, creating cardiac autoantibodies and inflammatory proteins. There are higher concentrations of anti-b-myosin antibodies in patients with myocarditis with dilated cardiomyopathy than in control groups.
- **Recovery or chronic:** Depends on the presence or elimination of the viral genome in the myocyte. Chronic T-cell-mediated inflammation results in remodeling and diffuse myocardial fibrosis, affecting cardiac dysfunction and leading to dilated cardiomyopathy and its sequelae, such as CHF, ventricular dysrhythmias, and abnormal ECG findings. Th17 cells are believed to play a significant role in this chronic phase of myocarditis.

Myocarditis presents in various forms, including Fulminant myocarditis (17%), which can lead to complete resolution or severe cardiac compromise resulting in rapid deterioration and death; Acute myocarditis (65%) typically causes moderate cardiovascular compromise and incomplete recovery with the potential for cardiac dysfunction or subsequent death; Chronic active myocarditis (11%) resembles acute myocarditis but tends to progress to mild or moderate cardiac dysfunction, often with restrictive physiology, and exhibits ongoing fibrosis on histologic examination; and Chronic persistent myocarditis (7%) characterized by non-resolving active or borderline inflammatory infiltrates on histologic examination, typically without cardiovascular compromise. These distinct presentations highlight the diverse clinicopathological classification of this cardiac condition.

Myocarditis presents with a wide spectrum of clinical manifestations, ranging from total asymptomatic cases to various chest pain syndromes, which can range from mild persistent chest pain (found in 35% of cases) associated with acute myopericarditis to severe symptoms mimicking acute myocardial infarction [2]. Rarely, coronary artery vasospasm may lead to chest pain in myocarditis patients, or the presentation may resemble pericarditis-associated chest pain. Preceding the cardiac symptoms, approximately 60% of patients may experience arthralgias, malaise, fever, sweats, or chills, often consistent with recent viral infections such as pharyngitis, tonsillitis, or upper respiratory tract infections, occurring 1 to 2 weeks prior to symptom onset. The hallmark symptoms of myocarditis are those of heart failure, including dyspnea, fatigue, and edema. Fatigue and reduced exercise capacity are common initial manifestations in patients who develop heart failure. However, severe, diffuse myocarditis can progress rapidly, leading to acute heart failure and cardiogenic shock. Additionally, some patients may present with arrhythmias, such as syncope, palpitations due to heart block, ventricular tachyarrhythmias, or even sudden cardiac death. Understanding the diverse clinical presentations of myocarditis is crucial for timely diagnosis and management in clinical practice.

Myocarditis masquerades many cardiovascular diseases so following different cases are all about how myocarditis can presented as ACS/STEMI, SVT with Aberrancy, Pleomorphic VT, Heart Block, Heart Failure with reduced Ejection Fraction (HFrEF). Covering all the spectrum of cardiac sign and symptoms.

Post Viral Myocarditis

1. Myocarditis Presenting as Acute ST-Elevation Myocardial Infarction (STEMI)

Case-1: A 33-year-old female presented with sudden onset retrosternal chest pain radiating to the left arm. Her Pulse was 90/m, BP-94/60mmHg. ECG-ST-segment elevation in lead V2-V5, I, and aVL with reciprocal depression in lead II, III, and aVF.2D-Echo revealed Global Hypokinesia of left ventricular with ejection fraction (LVEF) of 40% but no RWMA. Troponin I was 5.77 ng/ml (reference range 0.00-0.30 ng/ ml) with persistent rising trend. Her coronary angiography revealed normal epicardial coronaries. Serial ECGs showed persistent ST-segment elevation, intermit-tent junctional rhythm, and frequent ventricular premature complexes (VPC). No history of prior drug intake or any chronic illness. Serological for Epstein-barr virus, HIV, hepatitis B and C were negative. RT-PCR for COVID-19 was negative. Cardiac magnetic resonance imaging (CMR) declined due to financial constraints. We (cardiologist) diagnosed her with idiopathic inflammatory myocarditis. Along with the supportive therapy, she was treated with intravenous methylprednisolone I/V/O hypotension and rising Trop-I, to which she showed dramatic improvement. The ECG changes settled by day seven of steroid therapy, and LVEF improved to 55%. At follow-up of one year, she remained asymptomatic, and her review 2D-Echo revealed normal left ventricular (LV) functions with LVEF of 65%. Our patient presented with typical clinical symptoms of ACS. ECG changes and raised cardiac enzymes directed towards a diagnosis of AWMI. However, the absence of coronary artery atherosclerotic risk factors and absence of RWMA on echo made us think of the possibility of myocarditis masquerading as acute MI. Prior cases of myocarditis presenting as acute MI have been reported.

ISSN:0975 -3583,0976-2833 VOL 15, ISSUE 04, 2024

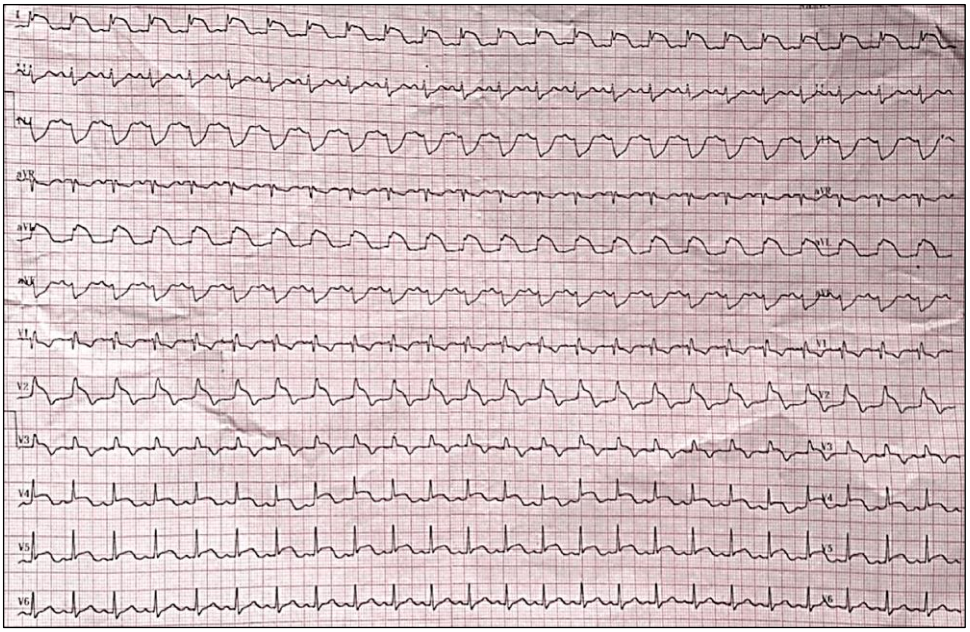


Fig 1: ECG showing ST elevation in V2-V6, I, aVL with reciprocal ST Depression in II, III, aVF

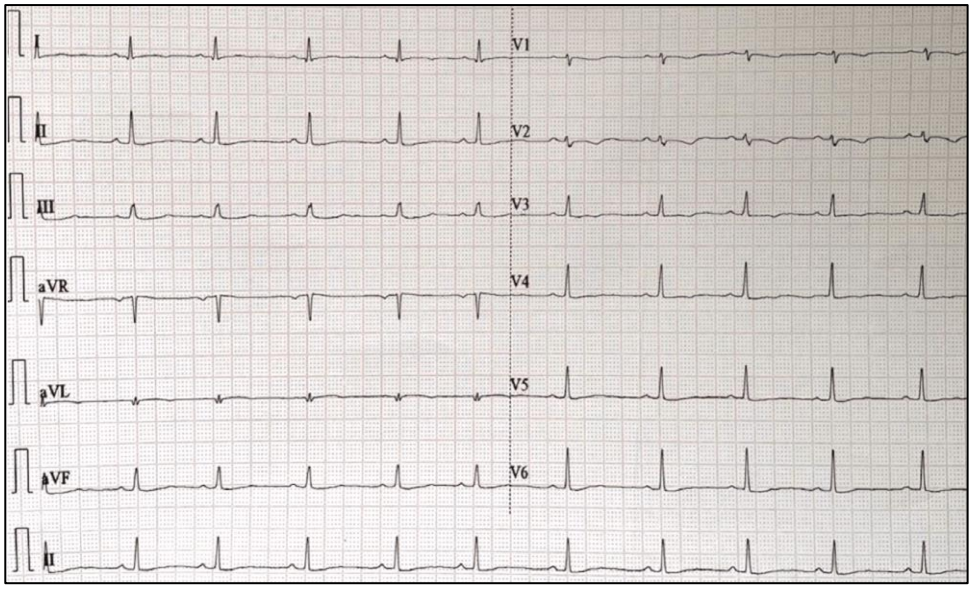
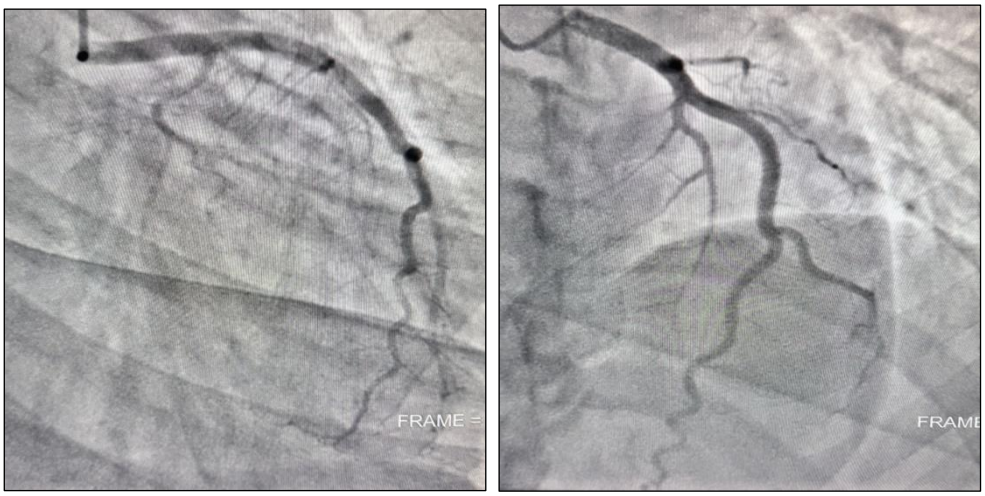


Fig 2: ECG showing normalisation of ST segment in all leads, T-inversion in V2



ISSN:0975 -3583,0976-2833 VOL 15, ISSUE 04, 2024

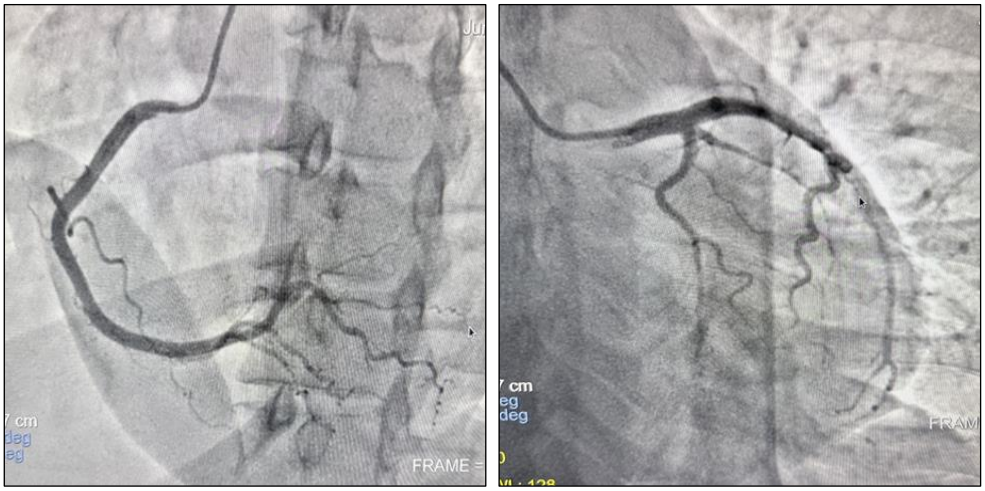


Fig 3: CAG of same patient showing normal epicardial coronaries, with multiples viewing angles

2. Myocarditis Presented as Electrical Abnormality

Case-2: A 19 year old male presented to the emergency department, PGIMS Rohtak with complaint of palpitations and localised chest pain which was atypical. ECG was done and s/o wide complex tachycardia with HR-212/m on presentation. Patient was hemodynamically stable, conscious and oriented. Then cardiology consultation was taken by on duty emergency medicine resident. He was admitted in cardiology department, ICCU-30 where initially he was managed with supportive care as the patient was conscious and oriented, afebrile to touch, blood pressure was 112/76mmhg, a heart rate of 174/min and oxygen saturation of 98% at room air. A 12 lead electrocardiogram was performed and confirmed the presence of a supraventricular tachycardia with aberrancy with 174/m. Patient was put on INJ. AMIODARONE I.V. (loading dose followed by maintenance dose). Finally, echocardiography was done and s/o Global hypokinesia of LV with EF-40%, rest normal study. Laboratory investigations revealed elevated Trop-I levels of 484.50 ng/L (Normal: <19ng/L), suggestive of cardiac involvement. The blood counts, pulmonary findings, liver and kidney function tests were all withing normal limits. Viral markers with hepatitis and HIV came out negative. There was history of fever, nausea, vomiting which lasted for 2 days, 1 week before only for which he consulted nearby clinic and treated conservatively without any investigation. On 3rd day he presented with above to us. In view of fever, Acute Idiopathic Myocarditis (? post viral) considered as diagnosis.

Steroid was given for 3 days as dose of 1mg/Kg and by the next morning ECG reverted to normal sinus rhythm.

Patient was discharged after 3 days with hemodynamic stable condition and Echo on follow up after 1 month was NORMAL study with EF-60-65%.

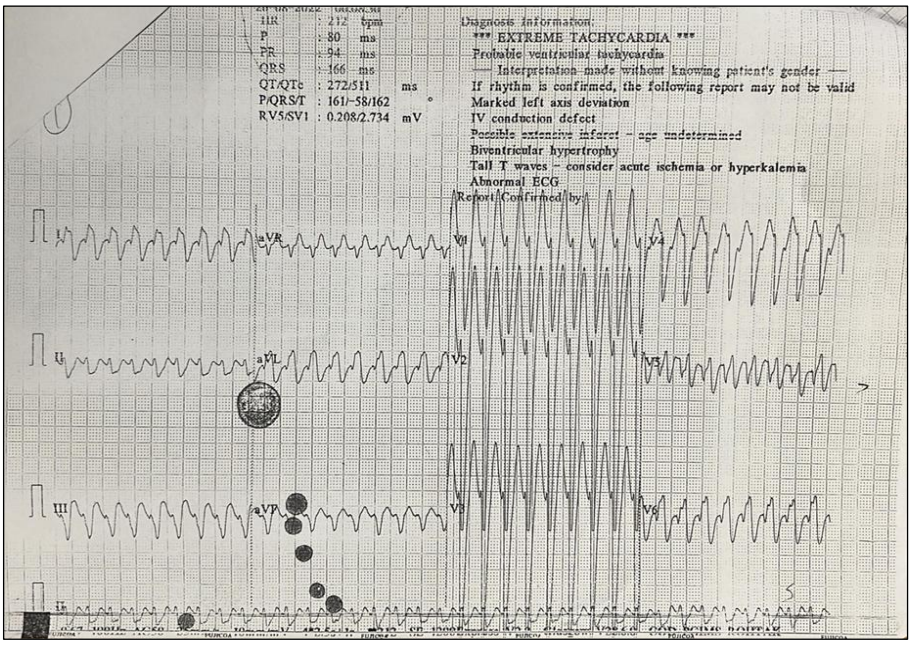


Fig 4: ECG on presentation-wide complex tachycardia-SVT with aberrancy

ISSN:0975 -3583,0976-2833 VOL 15, ISSUE 04, 2024

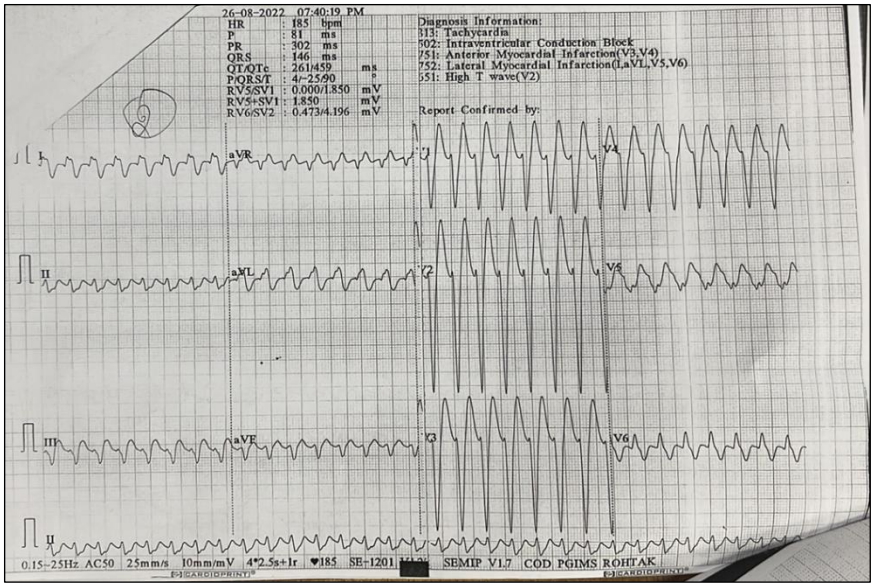


Fig 5: ECG in ICCU– wide complex tachycardia- SVT with aberrancy

After Steroid Therapy

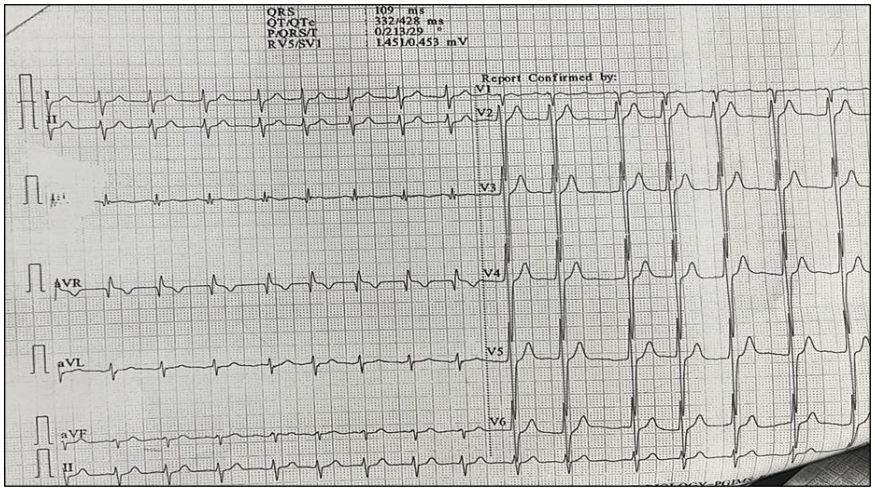


Fig 6: ECG after steroid therapy-Atrial Fibrillation with controlled ventricular rate

Next Morning

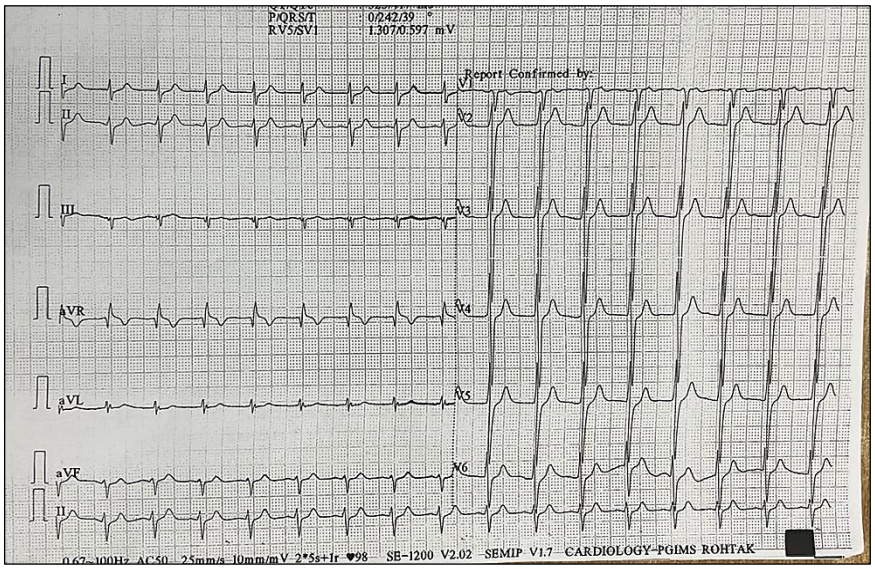


Fig 7: After 3 days of steroid-ECG-normal sinus rhythm

ISSN:0975 -3583,0976-2833 VOL 15, ISSUE 04, 2024

Case-3: A 26 years old female came to the emergency room of our hospital for the management of acute resting localised chest pain which was non-radiating. The interrogation found a patient with no cardiovascular risk factor, but she reported a flu syndrome associated with a fever in the week preceding the chest pain, treated with antipyretics outside. Moreover, there was no similar case in the patient's family. On examination, the patient was conscious, of normal weight and height with a BMI of 22 kg/m^2. Her pulse was feeble and BP-84/56mmHg. Immediately bed sided ECG was done, it was suggestive of Ventricular Tachycardia (VT). DC Cardioversion done immediately and reverted to normal sinus rhythm. On next day she was fully investigated and routine blood investigation were normal. Echo was done in view of VT, it was suggestive of global hypokinesia if LV, moderate LV Dysfunction (EF-30-35%). On next day she developed breathlessness, tachypnea, tachycardia then COVID RTPCR was done in view of fever and flu like symptoms and it was positive. Chest X-ray was normal lung field with no cardiomegaly. She was put on SARS-CoV-2 infection treatment: vitamin c, zinc, azythromicin, and preventive anticoagulation enoxaparin and paracetamol. Her breathlessness was not improving so again ECHO was done suggestive of global hypokinesia if LV, severe LV Dysfunction (EF-20-25%). Troponin-I was high. COVID-19 was positive, new myocardial injury (defined as marked troponin elevation), cardiac dysfunction, myocarditis leading to heart failure (HF) was the initial impression. Oral prednisolone started with other HFrEF treatment.

After 5 days she improved, no complaints. She was discharged with prednisolone and standard HFrEF treatment with LV systolic dysfunction (EF-30-35%). After a month on follow up her echo was done, it was normal LV systolic function (EF-60%).

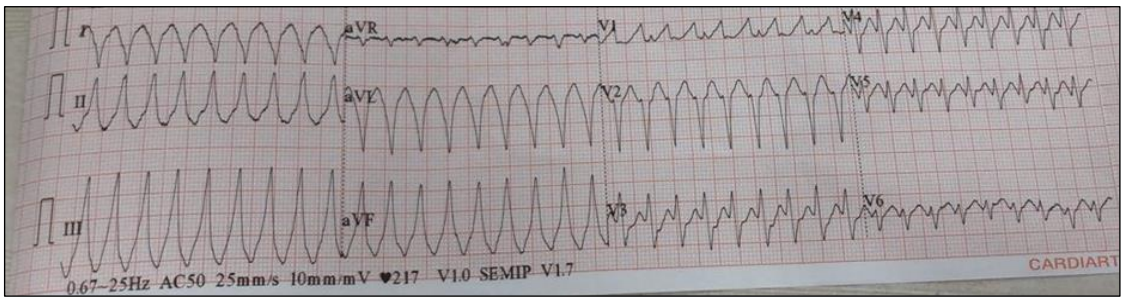


Fig 8: ECG suggestive of monomorphic ventricular tachycardia

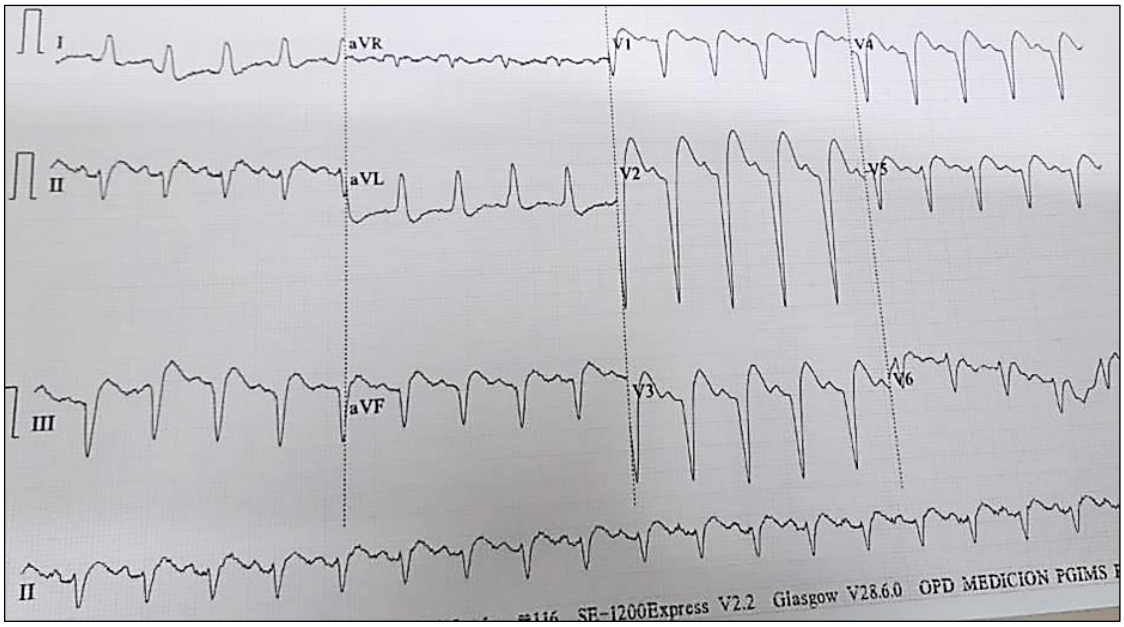


Fig 9: ECG of same patient after DC Cardioversion

ISSN:0975 -3583,0976-2833 VOL 15, ISSUE 04, 2024

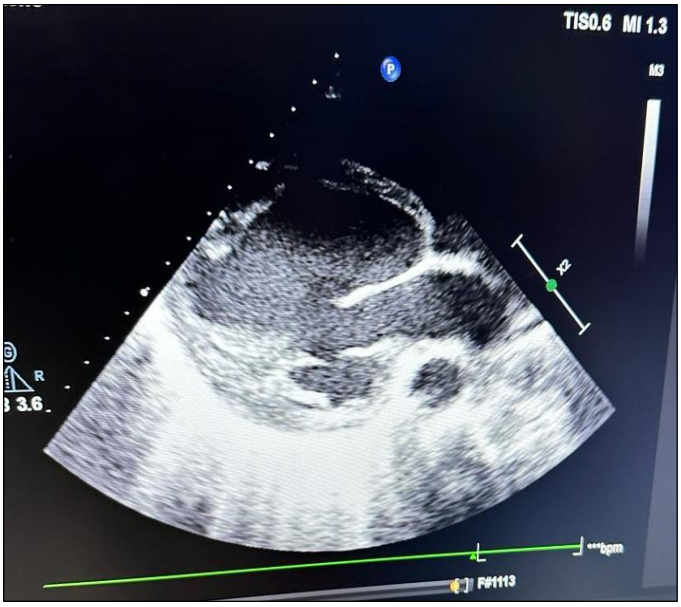


Fig 10: Echocardiography of same patient showing dilated LV

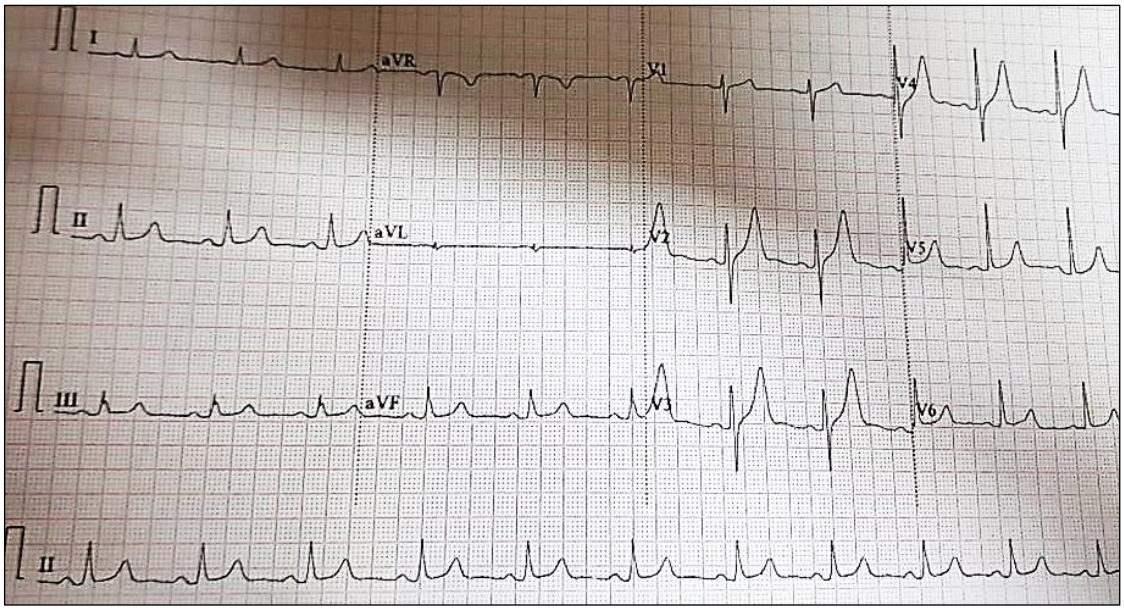


Fig 11: ECG of same patient after 1 month during follow up

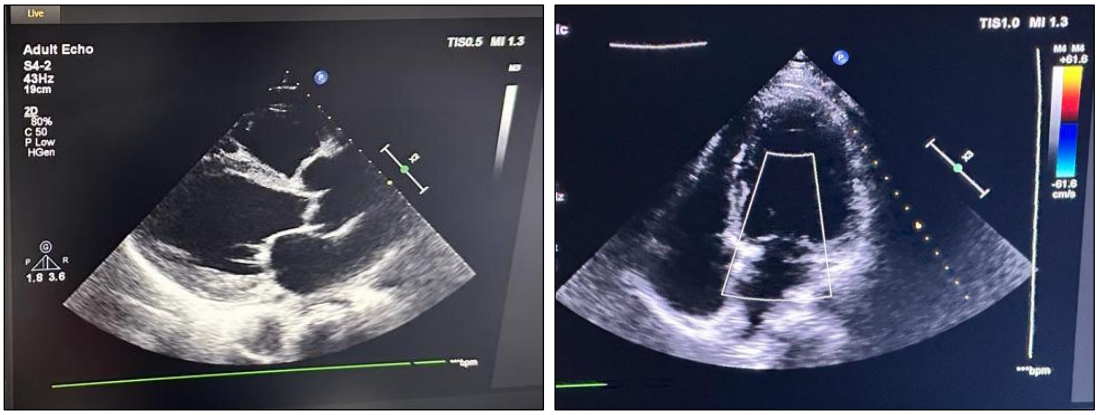


Fig 12: Echo after 3 months during follow up showing normal LV dimension and function

ISSN:0975 -3583,0976-2833 VOL 15, ISSUE 04, 2024

Case-4: A 28-year-old man presented to our hospital with breathlessness on exertion and easy fatiguability from last 3 days which exaggerated from last six hours before, with no fluctuations. He was a healthy, non-smoking adult with no history of cardiovascular disease or obesity (BMI 23.8 kg/m^2). There was no known family history of cardiovascular or autoimmune disease. ECG on the day of presentation showed sinus tachycardia, LAFB, LVH with LV volume overload changes, RS in v3-v4.

Fever was reported in the last 7-10 days and his temperature was around 100-101° F for which he never consulted expert physician. After his fever subsides he developed breathlessness on exertion and easy fatiguability making him to consult us. His blood pressure was 166/100 mmHg, his heart rate was 112 beats per minute, and the oxygen saturation was up to 98%. The physical exam did not reveal any pathological findings, and no pericardial rub sound was auscultated. The chest X-ray showed a normal cardiothoracic ratio and physiological findings from the imaging of both lung fields. The patient was admitted to the Intensive Cardiac Care Unit (ICCU) for monitoring. COVID-19 nasal swab testing using polymerase chain reaction was negative. The transthoracic echocardiogram (TTE) showed severe global hypokinetic segments in the left ventricular myocardium, with the left ventricular ejection fraction (LVEF) estimated up to 30%. Laboratory blood tests confirmed the suspicion of myocardial injury. Troponin-I high sensitive (TnI-hs) was 0.092 ng/ml (optimal values <0.014 ng/ml), and creatine phosphokinase myocardial band (CPK-MB) was 31 IU/L (normal range: <25 IU/L). C-reactive protein (CRP) was also elevated at 1.12 mg/dl (normal: <0.5 mg/dl) indicating Inflammation, whereas white blood cells (WBC) were counted normal at 8.59 (normal range: 4-11 × 10^9/L). The peak of the values was reached in the next 24 hours, with TnI-hs 0.365 ng/ml, CPK-MB 65 IU/L, and CRP 2.3 mg/dl, respectively. The patient was not taken for an emergent coronary angiogram since he was at a low clinical probability of having coronary heart disease, due to his young age and lack of family history, but also because of the absence of risk factors for atherosclerotic disease. TTE also ruled out the possibility of Takotsubo or any other cardiomyopathy. Patient was put on HFrEF treatment with Diuretic, ACE-Inhibitor, MRA, SGLT-2 inhibitor. The patient was discharged after a total of five days of hospitalization, with instructions for restriction of physical activity, as recommended after myocarditis and with treatment as above along with Beta-Blockers. After four months of follow-up, the patient is completely asymptomatic with a satisfactory left ventricular performance.

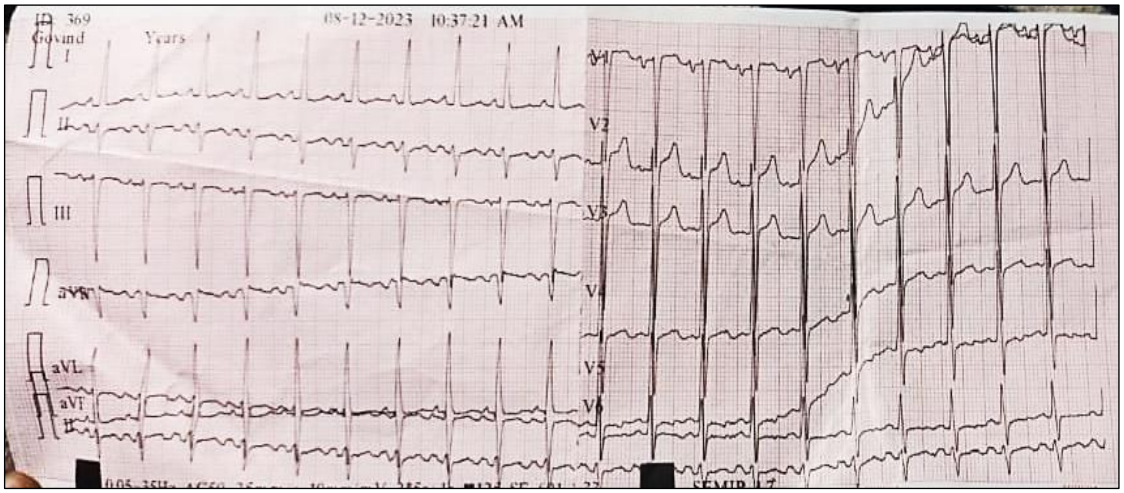
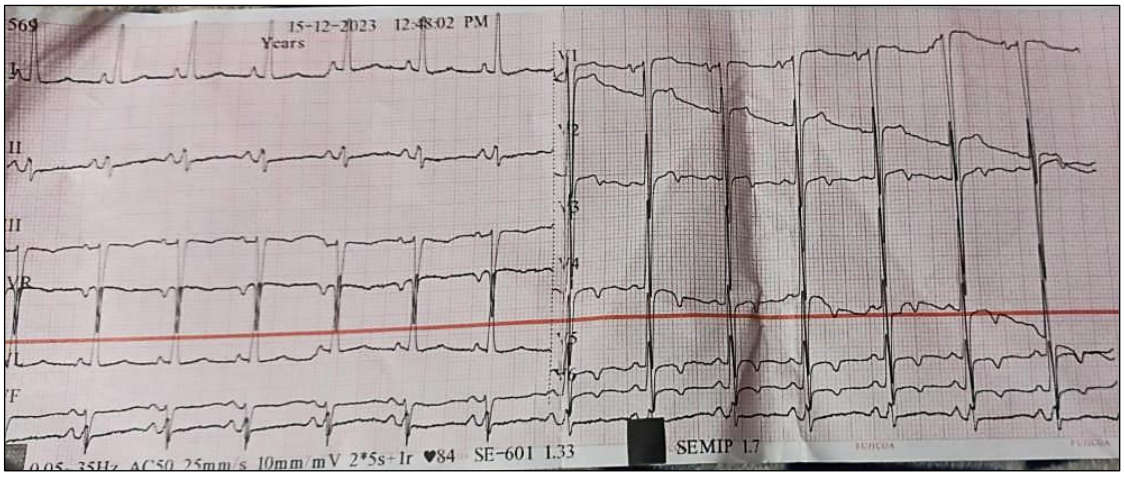


Fig 13: ECG on day-1 shows-showed sinus tachycardia, LAFB, LVH with LV volume overload changes, RS in v3-v4



ISSN:0975 -3583,0976-2833 VOL 15, ISSUE 04, 2024

Fig 14: ECG (day-5) shows NSR, LAFB, T-inversion in V3-V6

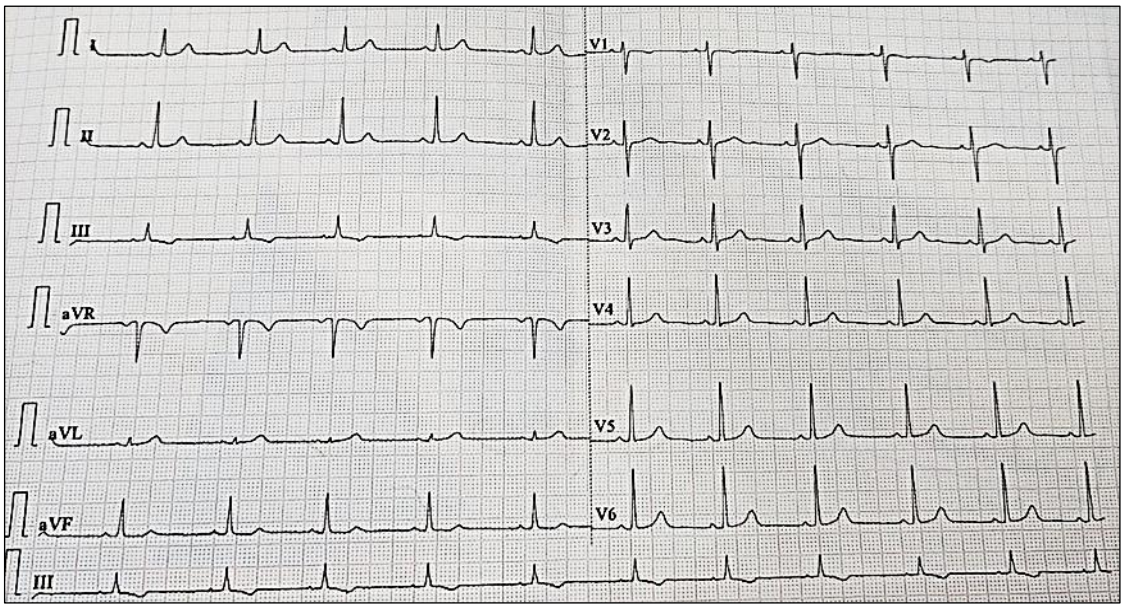


Fig 15: ECG-Normal Sinus Rhythm with No ST-T Changes

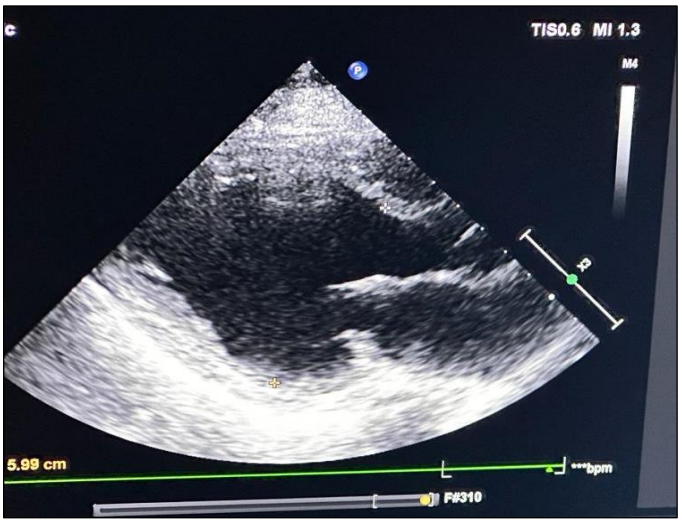


Fig 16: Echocardiography of same patient during hospitalisation showing Dilated LV with poor systolic function (severe LV dysfunction)

3. Post Viral Fulminant Myocarditis Presented as Heart Failure

Case-5: A 60yrs old, female presented to emergency with dizziness, chest pain from 1 day. Initially patient was taken to private hospital, Rohtak. There, she was in Shock and ECG s/o Ventricular Tachycardia (VT). Immediately DC Shock was given there and patient rushed to Cath lab. CAG was done s/o Insignificant CAD (RCA Prox. 20% disease). Then patient referred to PGIMS for further management. Patient then presented on same night with Chest Pain (Atypical) and Worsening of Dyspnea NYHA-IV. Patient was immediately admitted in Cardiology ICCU. Initially managed conservatively as ADHF with Cardiogenic Shock with dual ionotropic support. In ICCU, pt. again developed seizure like activity and monitor ECG s/o pleomorphic VT. Reverted to DC SHOCK as patient was hemodynamically unstable.

There was H/o Fever 7-8days back which lasted for 3 days and treated nearby clinic conservatively without any investigations.

ECG-as below.

Echo-Global Hypokinesia of LV with EF- 15-20%.

CAG-RCA-Prox. 20% disease.

RFT-Normal, Hb-9.3 TLC-15,000.

Trop-I >40ng/ml.

We were managing conservatively then on 2nd day patient become drowsy, monitor ECG s/o-Complete Heart Block (CHB) so Temporary Pacemaker Implantation (TPI) was done urgently.

ISSN:0975 -3583,0976-2833 VOL 15, ISSUE 04, 2024

In view of Fever, several episodes of VT, Cardiogenic Shock then CHB-Post Viral Fulminant Myocarditis was the impression and consideration.

And therefore, in view of fulminant course and progressive deterioration, we decided to give Steroids and Inj. Methylprednisolone (MPS) I.V. according to weight was given. On 3rd day of I.V. MPS, Patient reverted to SR and inotropes was tapered to minimal dose. TPI was removed. After 5 days, Ionotropic support was tapered and stopped. After 7 days, patient was discharged with hemodynamic stable condition. Repeat ECHO was done s/o Global hypokinesia of LV with EF-35%. Patient discharged with HFrEF Rx and with oral prednisolone.

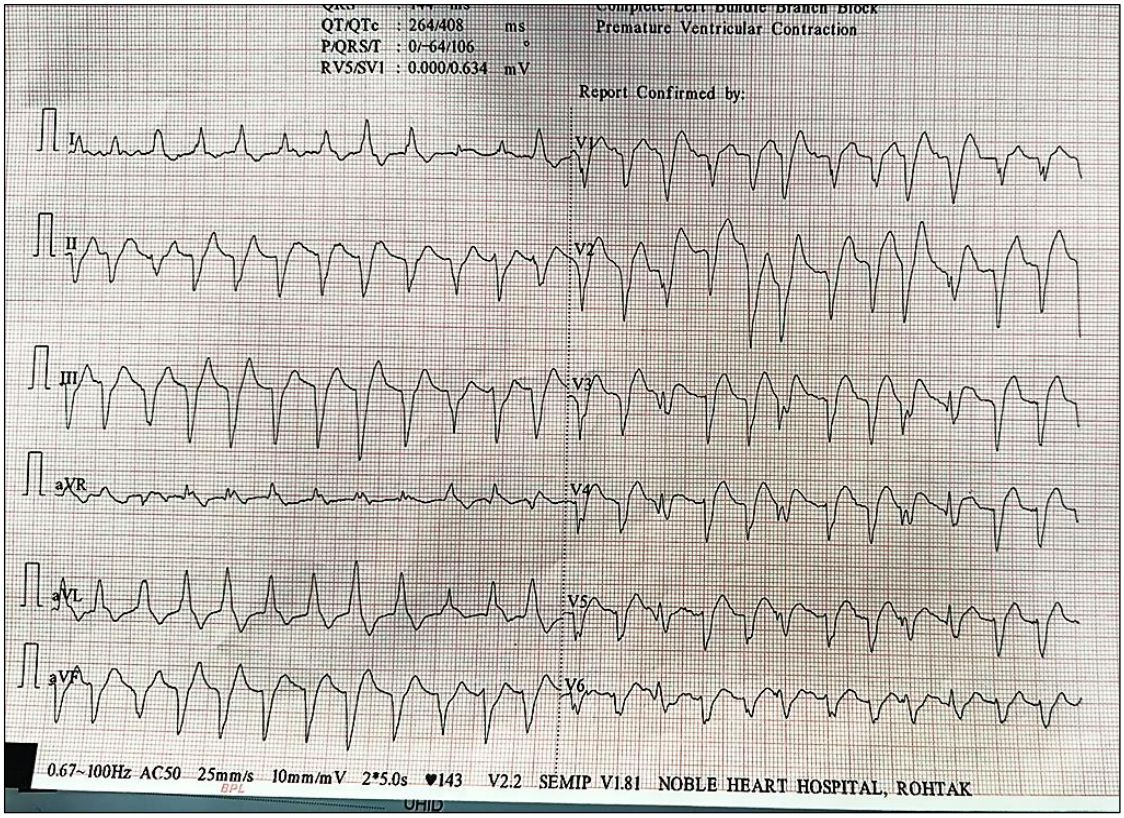


Fig 17: ECG (Outside private hospital)-Pleomorphic VT

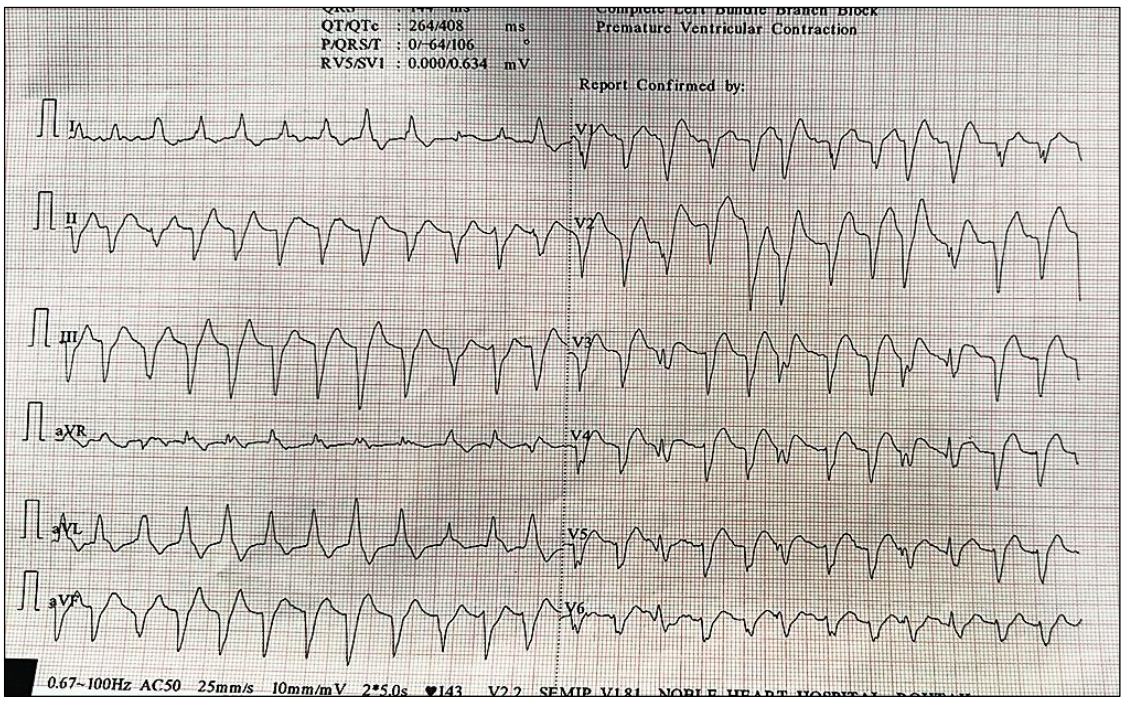


Fig 18: ECG (on presentation to PGIMS Rohtak)-Pleomorphic VT

ISSN:0975 -3583,0976-2833 VOL 15, ISSUE 04, 2024

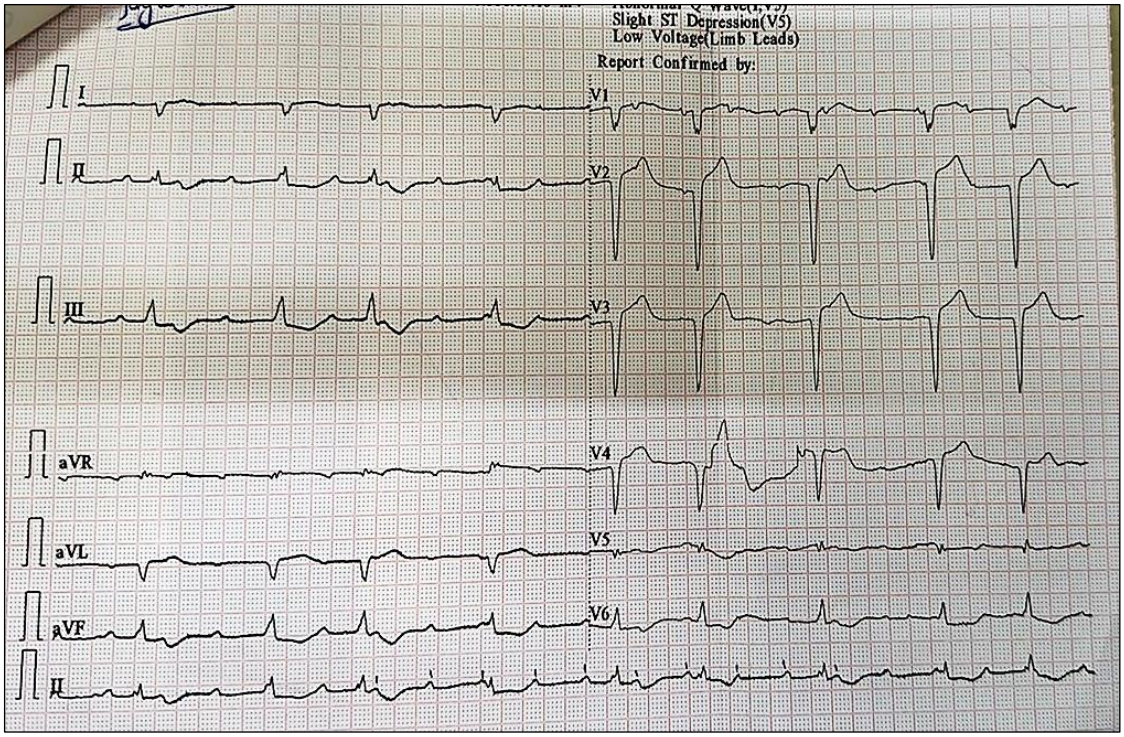


Fig 19: On 2nd day ECG-Complete Heart Block (CHB)

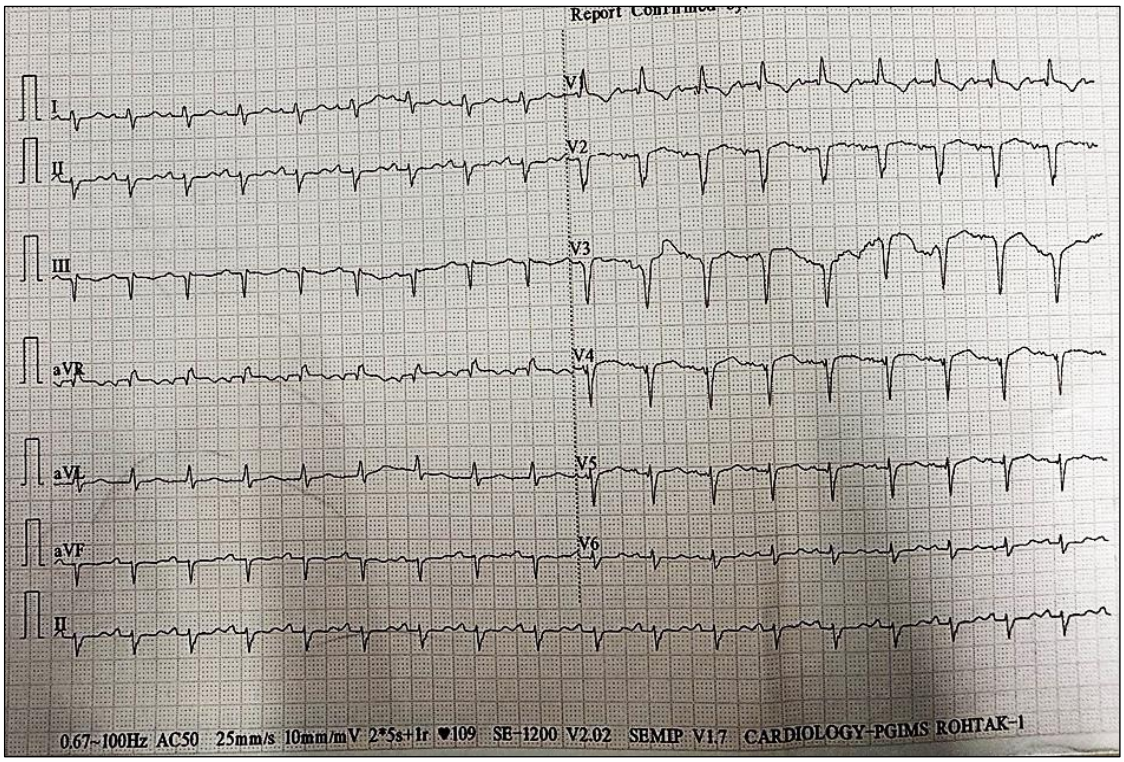


Fig 20: ECG-Normal Sinus Rhythm (after 3 days of IV Steroids)

ISSN:0975 -3583,0976-2833 VOL 15, ISSUE 04, 2024

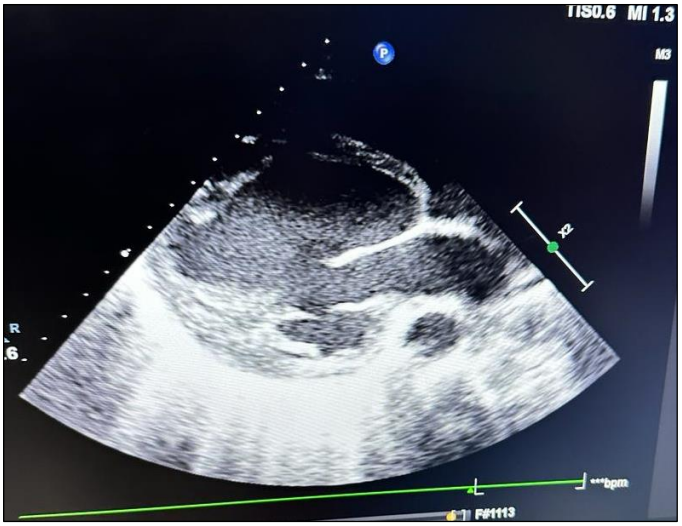


Fig 21: Echocardiography of same patient showing dilated LV with severe LV Dysfunction

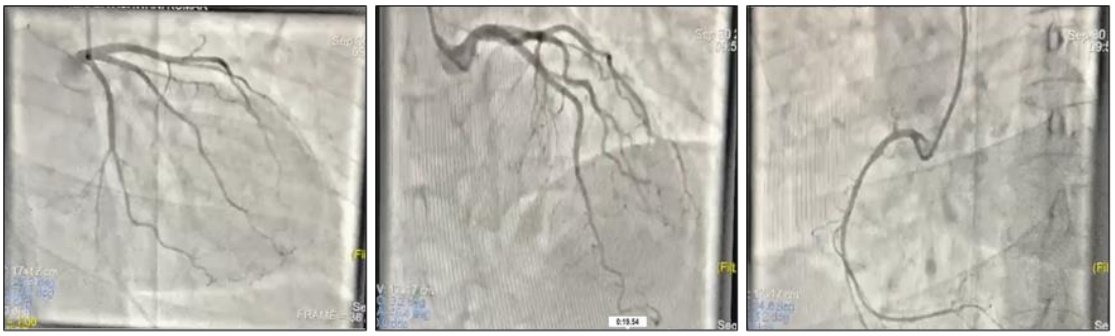


Fig 22: CAG of the same patient s/o-Plaque in Mid RCA, rest normal coronaries

Discussion

Myocarditis is generally mild and self-limiting pathology but may have a grave prognosis leading to inflammatory cardiomyopathy. The prognosis of inflammatory cardiomyopathy is poor in patients with LV dysfunction and heart failure [3]. Myocarditis may present with a fulminant course characterised by sudden onset severe LV dysfunction leading to cardiogenic shock or arrythmias. Fulminant myocarditis should be considered among the top differential diagnoses in young patients with cardiogenic shock [2, 4]. When treated promptly by early diagnosis and specific treatment strategies, myocarditis can have a good prognosis. The clinical presentation of a myocarditis patient may vary from mild symptoms to frank heart failure or sudden cardiac death (SCD). The patient may present with chest pain, fatigue, dyspnea, palpitations or syncope [2]. Myocarditis is the cause of SCD in 10% of cases of SCD in young patients less than 35 years old [5]. Careful history taking can elicit a history of prodromal events like fever, flu-like symptoms, and gastrointestinal upset in up to 80% of patient's weeks before presentation with myocarditis [3].

The proposed three-tiered classification for acute myocarditis, which is primarily distinguished by increasing diagnostic certainty (table-1). An asymptomatic patient can be classified as having possible subclinical acute myocarditis if other causes of acute cardiac disease are excluded, and if they have a recent trigger for myocarditis, such as a recent viral illness, and one of the following findings:

1. An otherwise unexplained rise in troponin concentrations.
2. Electrocardiographic changes suggestive of acute myocardial injury.
3. Abnormal cardiac function on echocardiogram or cardiac MRI.

If a patient meets the criteria for possible subclinical myocarditis but also has one of four clinical syndromes consistent with acute myocarditis (acute heart failure, chest pain, presyncope or syncope, or myopericarditis), then they can be categorised as having probable acute myocarditis. If myocarditis is confirmed by histological studies, then the diagnosis is definite myocarditis, irrespective of the clinical syndrome.

ISSN:0975 -3583,0976-2833 VOL 15, ISSUE 04, 2024

Table 1: A three-tiered clinical classification for the diagnosis of myocarditis on the basis of level of diagnostic certainty

DIAGNOSTIC CATEGORY	CRITERIA	HISTOLOGIC CONFIRMATION	BIOMARKER, ECG, OR IMAGING ABNORMALITIES CONSISTENT WITH MYOCARDITIS	TREATMENT NEEDED
Possible or subclinical myocarditis	In the clinical context of possible myocardial injury **without** cardiovascular symptoms but with at least one of the following: 1. Biomarkers of cardiac injury raised 2. ECG findings suggestive of cardiac injury 3. Abnormal cardiac function on echocardiogram or CMR	Absent	Required	Not known
Probable or Clinical myocarditis	In the clinical context of possible myocardial injury **with** cardiovascular symptoms and at least one of the following: 1. Biomarkers of cardiac injury raised 2. ECG findings suggestive of cardiac injury 3. Abnormal cardiac function on echocardiogram or CMR	Absent	Required	As per clinical syndrome
Definite myocarditis	Histologic or immunohistologic evidence of myocarditis	Present	Not Required	Specific to cause/ biopsy finding

According to the ESC position statement [6], clinically suspected myocarditis is defined in presence of ≥1 clinical presentation and ≥1 diagnostic criteria from different categories in the absence of angiographically detectable coronary artery disease (coronary stenosis, ≥50%) and known pre-existing cardiovascular disease or extracardiac causes which could explain the syndrome (e.g., valve disease, congenital heart disease, hyperthyroidism) [7]. Suspicion is higher with higher number of fulfilled criteria; if the patient is asymptomatic, ≥2 diagnostic criteria should be met.

Clinical presentation could include:

- Acute chest pain.
- New-onset dyspnea (days up to 3 months).
- Subacute/chronic dyspnea [> 3 months].
- Palpitations and/or unexplained arrhythmia symptoms.
- Unexplained cardiogenic shock.

Diagnostic criteria are defined as follow:

- ECG features of cardiac damage.
- Elevated markers of myocardial necrosis.
- Functional and/or structural abnormalities on cardiac imaging (echocardiogram or angiogram or CMR).
- Tissue characterization by CMRI (edema and/or LGE of classical myocarditis pattern).

It should be noted that the treatment of acute viral myocarditis differs according to the severity and initial presentation of the patient, for example, in fulminant forms, the initial management is that of cardiogenic shock and is based on hospitalization in an intensive care unit, continuous monitoring of cardiac rhythm and invasive blood pressure, careful correction of possible hypovolemia, and the use of inotropic and vasopressor therapies, mainly dobutamine and noradrenaline. In acute and chronic forms, treatment is by analogy identical to the treatment of chronic heart failure. In the absence of signs of low output, treatment combines diuretics in case of pulmonary edema or signs of overload, converting enzyme inhibitors, beta blockers, and anti-aldosterone. However, no studies have specifically looked at myocarditis [8].

In recent years and with the appearance of COVID-19 and other viral etiology, the diagnosis of acute coronary syndrome based only on ECG has become difficult especially in cases where COVID-19 is complicated by acute myocarditis which leads to ST-segment elevation with sometimes a typical chest pain, in this context we decided to write this paper to know better how to manage this association which remains until now little described in the literature in terms of additional examinations, and therapeutic management.

Hou *et al*. reported case of middle-aged male patient presenting with chest pain and raised cardiac enzymes after a flu like illness. Viral serology revealed high titres of rubella immunoglobulin and late gadolinium enhancement (LGE) on CMR. The patient showed significant improvement with anti-viral and supportive therapy [9]. A study of 45 patients suspected of acute MI with normal coronary angiogram was undertaken to assess a myocarditis diagnosis. 35 out of 45 patients showed either diffuse or focal

ISSN:0975 -3583,0976-2833 VOL 15, ISSUE 04, 2024

myocarditis on myocardial indium-111 antimyosin antibody or Thallium-201 imaging [10].
Our patients were negative for viral serology. Careful history taking did not reveal any flu-like illness. No history of any toxin or drug intake were found. We diagnosed them with a case of myocarditis of idiopathic origin. Along with supportive therapy she was treated with intravenous methylprednisolone 1mg/kg body weight for a week followed by oral prednisolone 1mg/kg therapy to which she showed dramatic improvement. Her symptoms resolved, and ECG changes reverted to normal at the seven of starting steroids.
CMR play an important role in the risk stratification of patients with myocarditis and preserved LVEF. The pattern of LGE on CMR provides important prognostic information. In the ITAMY study, it was found that LGE in mid wall layer of anteroseptal segment is associated with a worse prognosis and was the best and independent predictor of SCD, appropriate ICD firing, resuscitated cardiac arrest and hospitalisation for heart failure [11]. A prospective study on 672 patients with suspected myocarditis showed that tissue characterisation provides effective risk stratification in patients with suspected myocarditis. The presence of LGE was associated with more than doubling the risk of MACE, i.e., 4.8% versus 2.1% annual MACE rate when LGE was present and absent, respectively. The same study showed that septal and mid-wall LGE had the strongest association with MACE [12]. Acute myocarditis may present clinically similarly to ACS; hence, a high suspicion level is required to differentiate the two entities. Because of the potential to develop inflammatory cardiomyopathy and its serious consequences of SCD, arrhythmia, and heart failure, acute myocarditis should be properly treated. Patients should be investigated to look for causative factors and treated accordingly. CMR is the gold standard non-invasive modality of choice for diagnosing myocarditis. It is a class one recommendation for the characterisation of myocardial tissue for the diagnosis of myocarditis [13]. The ability to recognise myocarditis in patients presumed to be acute MI is invaluable because when treated timely, the prognosis in such patients can be good. Finally, this report has been made following SCARE guidelines [14].

Conclusion

- Acute myocarditis may present clinically similarly to ACS, HF, Tachyarrhythmia, AV Block or even CHB; hence a high level of suspicion is required to differentiate. The causes of myocarditis are diverse, but a viral etiology is the most common.
- The absence of significant cardiovascular risk factors for atherosclerotic coronary artery disease, with presence of global hypokinesia with absence of RWMA, elevation of cardiac troponins and normal coronary angiogram are further suggestive of myocarditis. CMR plays an important role in the diagnosis and prognostication of myocarditis [15]. The definite diagnosis by endomyocardial biopsy is rarely performed [5]. Some patients can present as fulminant course and may need Mechanical Circulatory Support (MCS).
- In **Cochrane systematic review [16]**, corticosteroid therapy had no effect on mortality. These drugs may improve (or at least stabilize) LV systolic function, but this needs to be interpreted with caution since the evidence is based on small-scale trials of poor methodological quality with significant risk of bias. At 1-3 months follow-up significant differences were seen in LV systolic function as assessed by LVEF about 7% to 13%. If the clinical condition deteriorate faster and patient is unstable, clinician can use corticosteroids for initial stabilisation of vitals and LVEF, further with supportive management.

Myocarditis, defined as inflammatory disease of the myocardium, has a first phase of direct or indirect tissue damage due to infection, followed by an autoimmune phase [17]. Corticosteroids would thus be expected to affect the pathophysiological process. However, in this Cochrane systematic review, corticosteroid therapy had no effect on mortality. 1 Improvement or at least stabilization of LV function was only seen in small trials of poor methodological quality; none of the RCTs included were blinded. The Position Statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases recommends immunosuppressive therapy (including corticosteroids) for autoimmune, eosinophilic or toxic myocarditis and cardiac sarcoidosis with ventricular arrhythmias or dysfunction or heart failure refractory to standard therapy only after ruling out active infection by polymerase chain reaction analysis of endomyocardial biopsy [18].

References

1. Tscho¨pe C, Ammirati E, Bozkurt B, *et al*., Myocarditis and inflammatory cardiomyopathy: current evidence and future directions, Nat. Rev. Cardiol. 2021;18:169-193.
2. Kociol RD, *et al*., Recognition and initial management of fulminant myocarditis: a scientific statement from the American Heart Association, Circulation. 2020;141:e69-e92.
3. Ammirati E, *et al*., Clinical presentation and outcome in a contemporary cohort of patients with acute myocarditis, Circulation. 2018;138:1088-1099.
4. Ammirati E, *et al*., Fulminant versus acute non-fulminant myocarditis in patients with left ventricular systolic dysfunction, J Am. Coll. Cardiol. 2019;74:299-311.

ISSN:0975 -3583,0976-2833 VOL 15, ISSUE 04, 2024

5. Corrado D, Basso C, Thiene G. Sudden cardiac death in young people with apparently normal heart, Cardiovasc. Res. 2001;50:399-408.
6. Caforio AL, Pankuweit S, Arbustini E, Basso C, Gimeno-Blanes J, Felix SB, *et al*. Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. European heart journal. 2013 Sep;34(33):2636-48.
7. Schmitt P, Demoulin R, Poyet R, *et al*. Acute Myocarditis after COVID-19 vaccination: a case report. Rev Med Intern. 2021;42(11):797-800.
8. Hékimian G, Franchineau G, Bréchot N, M S, A N, S B, *et al*. Diagnosis and management of myocarditis. Méd Intensive Réa. 2017;26:196-206.
9. Ya-Min Hou, Peng-Xi Han, Xia Wu, *et al*., Myocarditis presenting as typical acute myocardial infarction: a case report and review of the literature, World J Clin. Cases. 2020 Jan;8(2):415-424.
10. Sarda L, Colin P, Boccara F, *et al*., Myocarditis in patients with clinical presentation of myocardial infarction and normal coronary angiograms, J Am. Coll. Cardiol. 2001 March;37(3):786-792.
11. Aquaro GD, *et al*., Cardiac MR with late gadolinium enhancement in acute myocarditis with preserved systolic function: ITAMY study, J Am. Coll. Cardiol. 2017;70:1977-1987.
12. Grani C, *et al*., Prognostic value of cardiac magnetic resonance tissue characterization in risk stratifying patients with suspected myocarditis, J Am. Coll. Cardiol. 2017;70:1964-1976.
13. McDonagh TA, Metra M, Adamo M, *et al*., ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure [published correction appears in Eur Heart J. 2021 Oct 14;:], Eur. Heart J. 2021;42(36):3599-3726. https://doi.org/ 10.1093/eurheartj/ehab368, 2021.
14. Agha RA, Franchi T, Sohrabi C, Mathew G, Kerwan A, Thoma A, *et al*. The SCARE 2020 guideline: updating consensus surgical case report (SCARE) guidelines, Int. J Surg. 2020 Dec;84:226-230.
15. Kindermann I, Barth C, Mahfoud F, *et al*. Update on myocarditis. J Am Coll Cardiol. 2012;59:779-92.
16. Caldeira D, Lopes LR, Vaz-Carneiro A, Costa J. Cochrane Corner-administração de corticosteroides para miocardite de etiologia viral. Revista Portuguesa de Cardiologia. 2015 Jan;34(1):65-7.
17. Liu PP, Mason JW. Advances in the understanding of myocarditis. Circulation. 2001;104:1076-82.
18. Caforio AL, Pankuweit S, Arbustini E, *et al*. Current state of knowledge on aetiology, diagnosis, management and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. Eur. Heart J. 2013;34:2636-48, 2648a-2648d.