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RESEARCH ARTICLE

Electrocardiographic Changes and Arrhythmia Spectrum in Pediatric Patients with Acute Myocarditis

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ABSTRACT

Objective: Myocarditis is a non-ischemic inflammation of the cardiac muscle and causes non-specific and variable electrocardiographic (ECG) changes, indicating the inflammatory process. In this study, we aimed to investigate the prevalence and nature of ECG changes and arrhythmias in patients diagnosed with acute myocarditis (AM).

Materials and Methods: This retrospective study included 75 patients diagnosed with AM between January 1, 2020, and December 31, 2022, at our institution. The diagnosis was based on clinical symptoms, laboratory findings, and imaging procedures.

Results: Of the 75 AM patients evaluated, 67 (89.3%) were male, with a median age of 15 years. The primary symptom was chest pain (89.3%). The peaking times of Troponin I levels varied in presentation times. Elevated creatine kinase-MB and C-reactive protein levels were observed in 62.7% and 78.7% of patients, respectively. Viral serology identified COVID-19, Coxsackievirus, influenza, and parainfluenza as common viruses. ECG and Holter abnormalities were found in 82.7% of patients, with ST-segment elevation, T-wave abnormalities, sinus tachycardia, and interventricular conduction delay being the most common. Extracorporeal membrane oxygenation therapy was required in two critical cases, unfortunately resulting in the deaths of both patients.

Conclusion: Myocarditis presents with varied etiology and manifestations. ECG changes and arrhythmias are commonly observed in patients with this condition. Despite comprehensive care, severe cases can lead to adverse outcomes.

Keywords: Acute myocarditis, arrhythmia, electrocardiography, pediatrics

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INTRODUCTION

Myocarditis represents an inflammation of the myocardium that causes myocytes' degeneration and necrosis with non-ischemic origin.[1] This condition, with a spectrum ranging from asymptomatic to life-threatening scenarios, has been the focus of numerous studies.[1–3] Predominantly, myocarditis results from viral infections or abnormal immune responses and may display symptoms similar to those of various non-inflammatory cardiac diseases, making its diagnosis complex.[1,2] Myocarditis can be caused by several factors, including infectious agents, toxins (ethanol, cocaine, and heavy metals), drugs, systemic diseases, allergic reactions, and autoimmune disorders (systemic lupus erythematosus and Kawasaki disease), with the actual incidence remaining unclear due to the asymptomatic nature of many cases.[1,2,4–6] Indeed, in infants, the disease often presents as a respiratory or gastrointestinal disorder, thus evading easy diagnosis.[1,7,8]

Notably, the clinical presentations of myocarditis can be quite varied. The clinical spectrum is broad, from asymptomatic cases to those with severe symptoms such as chest pain, arrhythmias, heart failure, or even sudden death.[2,4,5,8]

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One of the critical concerns in managing myocarditis is the high prevalence of arrhythmias observed in 29–45% of initial patient consultations and up to 100% in severe cases of acute myocarditis (AM).[3,8,9,10]

The primary aim of this study is to investigate the prevalence and nature of electrocardiographic (ECG) changes and arrhythmias in patients diagnosed with AM.

MATERIALS and METHODS

This retrospective and observational analysis examined the medical files of 75 AM patients diagnosed at our institution between January 1, 2020, and December 31, 2022. All patients who reported myocarditis-related symptoms and whose diagnosis was verified by suitable diagnostic tests were included in the study. Patients with missing medical records and patients admitted after the acute period of 1 month were excluded from the study.

Data Collection

The medical records were searched for demographic information (age and gender), symptoms at presentation, and extensive details about the diagnosis and treatment of myocarditis. Each case underwent a thorough physical examination, a thorough medical history interview, and any relevant laboratory tests.

Diagnostic Standards

AM was defined as the short period between the onset of symptoms and diagnosis of <1 month.[11] The diagnosis was based on the presence of clinical signs (such as chest pain, fatigue, heart failure symptoms, or arrhythmias), laboratory findings (including elevated cardiac enzymes such as Troponin I), ECG features such as ST-segment elevation, T-wave flattening or negative T-waves, and imaging procedures such as echocardiography (ventricular dilatation or dysfunction) or cardiac magnetic resonance imaging (MRI) (late Gadolinium enhancement for myocardial inflammation and damage). This diagnostic approach is consistent with the criteria outlined in the position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases.[1]

The given laboratory reference ranges for the respective markers are as follows: Troponin I: 0–14 ng/L, creatine kinase-MB (CK-MB): 0–6.2 ng/mL, and C-reactive protein (CRP): 0–5 mg/L.

All patients underwent daily ECG evaluations during their hospitalization, and a Holter monitor was applied on the 1st day of admission and when they described symptoms. Twelve-lead ECGs of the patients were recorded at 10 mm/mV and 25 mm/s paper speed. All Holter recordings were examined for arrhythmias, and ECGs were assessed to determine the ECG abnormalities of myocarditis. The ECG pathologies were determined based on the presence of ST-segment elevation or depression, T-wave alterations, low QRS voltage, the emergence or resolution of a first-degree AV block, or QTc prolongation during the course of the patient's presentation. Arrhythmias were also taken into account as ECG pathologies. The PR interval, QRS duration, and QTc interval (Bazett's formula for QTc) were calculated using standard procedures. The Holter recordings revealed the presence of premature atrial contraction (PAC), supraventricular tachycardia (SVT), premature ventricular contraction (PVC), ventricular tachycardia (VT), ventricular fibrillation (VF), and atrioventricular block (AVB).

Statistics and Data Analysis

The results were compiled using descriptive statistics, which included the frequency of arrhythmias, and ECG alterations. All statistical analyses were conducted using the IBM SPSS Statistics software, version 17 (SPSS Inc., Chicago, IL, USA). Categorical variables were presented as frequencies and percentages. The one-sample Kolmogorov–Smirnov test was performed to determine the normality of each continuous variable's distribution. If the data had a normal distribution, they were shown as mean values with standard deviation, while non-normally distributed variables were presented as the median value with range. The Mann–Whitney U test was employed to compare non-parametric independent data sets. P-value <0.05 was considered statistically significant.

Ethics

The study complied with the Helsinki Declaration and our institution's ethical standards (2023.04-41, May 16, 2023). Patient consent was unnecessary for this retrospective investigation since all patient data were anonymized and managed strictly.

RESULTS

Table 1 shows the demographic and clinical features of the patients. A total of 75 patients with AM were evaluated; 67 patients (89.3%) were male, and the median age of presentation was 15 years (5–17 years). In our cohort, recent medical history revealed the cause of AM in some cases. Within the 2 weeks prior, 6 patients (8%) reported having acute gastroenteritis. The medical histories of 12 (16%) patients showed evidence of upper respiratory tract infections. Notably, the COVID-19 virus had previously been acquired by 5 patients

Table 1. The demographic and clinical characteristics of the patients with acute myocarditis

Clinical category	Patients	
	n	%
Number of patients	75	100
Median age (range), years	15 (5–17)	
Median weight (range), kg	65 (17–98)	
Male gender	67	89.3
Referral symptoms		
Chest pain	67	89.3
Chest pain and dyspnea	3	4
Chest pain and fatigue	2	2.7
Fatigue	2	2.6
Syncope	1	1.3
Patient history		
Upper respiratory tract infections within the past 2 weeks	12	16
Acute gastroenteritis within the past 2 weeks	6	8
Previous Covid 19	3	4
Previous myocarditis	1	1.3
Pneumonia	1	1.3

(6.7%). In addition, one patient had a previous diagnosis of myocarditis, while another had a medical history of pneumonia. However, the remaining patients in our study had no apparent medical history that would have shed light on the underlying cause of their myocarditis. The main presentation was chest pain, seen in 67 (89.3%) patients. Three patients presented with chest pain with dyspnea, two with chest pain with fatigue, two with fatigue, and one with syncope.

In our study, all patients exhibited elevated Troponin I levels, with peak levels observed at varying times: At admission (38.7%), on the 1st day of hospitalization (29.3%), on the 2nd day (22.7%), on the 3rd day (for six patients), and on the 6th day (for one patient).

The peak median Troponin I level was observed to be 575.5 ng/L (range 18.55–10000). Elevated laboratory values were detected in our study, with 47 (62.7%) patients having elevated CK-MB levels, and 59 (78.7%) patients having elevated CRP levels. The median CK-MB level manifested as 31.3 ng/mL (range 0.46–300), and CRP levels displayed a median value of 24.7 mg/L (range 1–235). Viral serology was studied in 60 (80%) patients. The viruses that were found to be the most common were COVID-19 (n=2), Coxsackievirus (n=2), influenza, and parainfluenza (n=1). Table 2 presents the diagnostic procedures and management strategies of patients with AM.

ECG features and arrhythmias of the AM patients are demonstrated in Table 3. The ECG and Holter abnormalities were seen in 62 (82.7%) of the 75 patients with AM. ST-segment elevation in the inferior and lateral leads on admission (n=46, 61.3%, Fig. 1), T-wave flattening or negative T-waves (n=38, 50.7%), sinus tachycardia (n=11, 14.7%), and interventricular conduction delay (n=20, 26.7%) were the most common findings. Four patients had low QRS voltage, two had incomplete right bundle branch block, and two had ST depression. Four patients (5.3%) had QTc durations of 450 ms and above. The mean QTc duration was 416 ± 21.8 ms. Nine patients had a 1st-degree AVB. Five patients had PACs on their 24-h Holter ECG, and one had non-sustained SVT. On ECG and Holter monitoring, PVCs were seen in 38 patients (50.7%) (polymorphic, n=17, Fig. 2), non-sustained VT in six (8%), and sustained VT (Fig. 3) with VF in 2 patients (2.6%). Standard ECG and Holter ECG findings were normal in 13 (19.3%) patients at diagnosis. There was a statistically significant difference between the peak Troponin I levels and the presence of the ECG findings, ST-segment elevation, T-wave flattening or negative T-waves, and the occurrence of PVCs (all $p<0.05$). There were no statistically significant correlations between the peak Troponin I level and the presence of the other ECG data or arrhythmias (all $p>0.05$) (Table 4). ST-segment elevation resolved on the 2nd day of admission in 27 (36%) patients, on the 3rd day in 14

Table 2. The laboratory findings and management strategies of the patients with acute myocarditis

Category	Total (n=75)	
	n	%
Laboratory findings		
Viral serology studied	60	80
COVID-19	2	2.6
Coxsackievirus	2	2.6
Influenza/parainfluenza	1	1.3
Troponin I (ng/L), median (range)	575.5 (18.55–10000)	
Creatine kinase-MB (CK-MB, ng/mL), median (range)	20.8 (0.46–300)	
C-reactive protein (CRP, mg/L), median (range)	24.7 (1–235)	
Treatment		
Medications		
Non-steroid anti-inflammatory drug (ibuprofen)	65	86.7
Corticoteroid	2	2.6
Intravenous Immunoglobulin-IVIG	5	6.7
Antiarrhythmic drug	7	9.3
Inotropic support	3	4
ECMO	2	2.6
Death	2	2.6

ECMO: Extracorporeal membrane oxygenation

(18.7%) patients, on the 4th day in two patients, and on the 5th day in one patient. T-wave flattening or negative T-waves were observed on the 1st day of admission in 11 (14.7%) patients, on the 2nd day in 13 (17.3%) patients, on the 3rd day in 11 (14.7%) patients, on the 4th day in one patient (1.3%), and the 5th day in one patient (1.3%). A decreased left ventricular function was observed in three patients at admission. All of them exhibited ST-segment elevation on their ECGs, and two of these also showed a prolongation of the QTc interval.

In the management of inflammation, patients were treated with non-steroidal anti-inflammatory drugs (NSAIDs, e.g., ibuprofen), corticosteroids, or intravenous immunoglobulin (IVIG) therapy. Ibuprofen, a NSAID, was administered at a dosage of 10 mg/kg every 8 h and used in most patients (n=65, 86.7%). Three patients with identified viral etiologies were administered IVIG (4%), while two patients with detected viral agents requiring extracorporeal membrane oxygenation (ECMO) support were treated with corticosteroids and IVIG (2.6%). For 9.3% (n=7) of the patients, antiarrhythmic drugs were required to control ventricular arrhythmias. Four patients (5.3%) needed inotropic support to keep their cardiac output at a healthy level.

In this study, ECMO therapy was required in two critical cases, unfortunately resulting in the deaths of both patients. The first patient was a 14-year-old male patient who was referred to our clinic due to the onset of chest pain 2 days ago. The patient's Troponin I level was 2291 pg/L at admission, and the left ventricle (LV) ejection fraction (EF) was 70%. Coronary arteries were examined with catheter angiography, which revealed the usual. The patient experienced a sudden cardiac arrest on the 3rd day of hospitalization. Despite initiating cardiopulmonary resuscitation (CPR), no response was obtained, necessitating the transition to ECMO support. During the follow-up period, the patient developed VT and VF. Unfortunately, the patient died on the 3rd day of ECMO treatment. The second patient was a 13-year-old boy who was referred to us due to fatigue, LV dilation, and dysfunction. At the time of admission, his LV EF was 30%. Despite receiving inotropic support, the patient developed respiratory distress, hypotension, and eventual bradycardia on the 6th day of hospitalization, necessitating CPR and ECMO support. VT and VF occurred during the patient's follow-up. However, the patient died on the 7th day of ECMO support.

Table 3. Electrocardiographic and Holter monitor findings in patients with acute myocarditis

Parameters	Total (n=75)	
	n	%
Electrocardiographic abnormalities	62	82.7
ST-segment elevation	46	61.3
T-wave flattening or negative T-waves	38	50.7
Sinus tachycardia	11	14.7
ST-segment depression	2	2.6
Low QRS voltage	4	5.3
PR duration (ms), mean±SD (range)	136.3±17.7 (96–194)	
Interventricular conduction delay	20	26.7
QRS duration (ms), mean±SD (range)	85.8±9.8 (68–112)	
Incomplete right bundle branch block	2	2.6
Long QTc interval	4	5.3
QTc interval (ms), mean±SD (range)	416±21.8 (376–490)	
Atrial Arrhythmias		
Premature Atrial Contractions	5	6.7
Atrial Tachycardia	1	1.3
Ventricular Arrhythmias		
Premature Ventricular Contractions (PVCs)	38	50.7
Polymorphic PVCs	17	22.7
Non-sustained Ventricular Tachycardia	6	8
Sustained Ventricular Tachycardia	2	2.6
Heart Block		
First degree atrioventricular block	9	12

SD: Standard deviation

DISCUSSION

Myocarditis is an inflammatory condition that affects the myocardium. It is a complex disease with an array of etiologies and clinical presentations, and it frequently has significant morbidity and mortality.[1,2,6] In this study, 75 patients diagnosed with myocarditis were evaluated. Most of our patients were male (89.3%), with a median age of 15 at presentation. These demographic details support the view expressed in myocarditis research that the condition primarily affects young males.[2,3] The median age of the individuals in our study is consistent with earlier studies that claim myocarditis is not unusual in young patients.[2]

Understanding the interaction between viral stimuli, host immunological responses, and genetic predispositions is necessary to comprehend myocarditis's causes. Infectious etiologies, particularly viral infections, account for many myocarditis patients.[1,5,6,12] Acute viral infections, such as the Coxsackievirus, influenza, parainfluenza, and the SARS-CoV-2 virus, have been related to myocarditis.[1,4,13] A recent history of gastroenteritis and upper respiratory tract infections was recorded in our group, emphasizing the post-infectious, immune-mediated character of myocarditis. The SARS-CoV-2 virus, which caused the COVID-19 pandemic, is a recent addition to the list of infectious triggers. Emerging evidence from our investigation and other recent studies show that the virus can directly infect the myocardium, causing cardiac damage, inflammation, and, ultimately, myocarditis.[12] In five patients, a correlation with the SARS-CoV-2 virus was identified. Two patients underwent acute SARS-CoV-2 virus infection; one patient was diagnosed with MIS-C (Multisystem Inflammatory Syndrome in Children). The other two patients had a history of SARS-CoV-2 virus infection that had occurred 1 month ago.

Myocarditis frequently appears with a wide range of clinical manifestations, making it difficult to diagnose in clinical

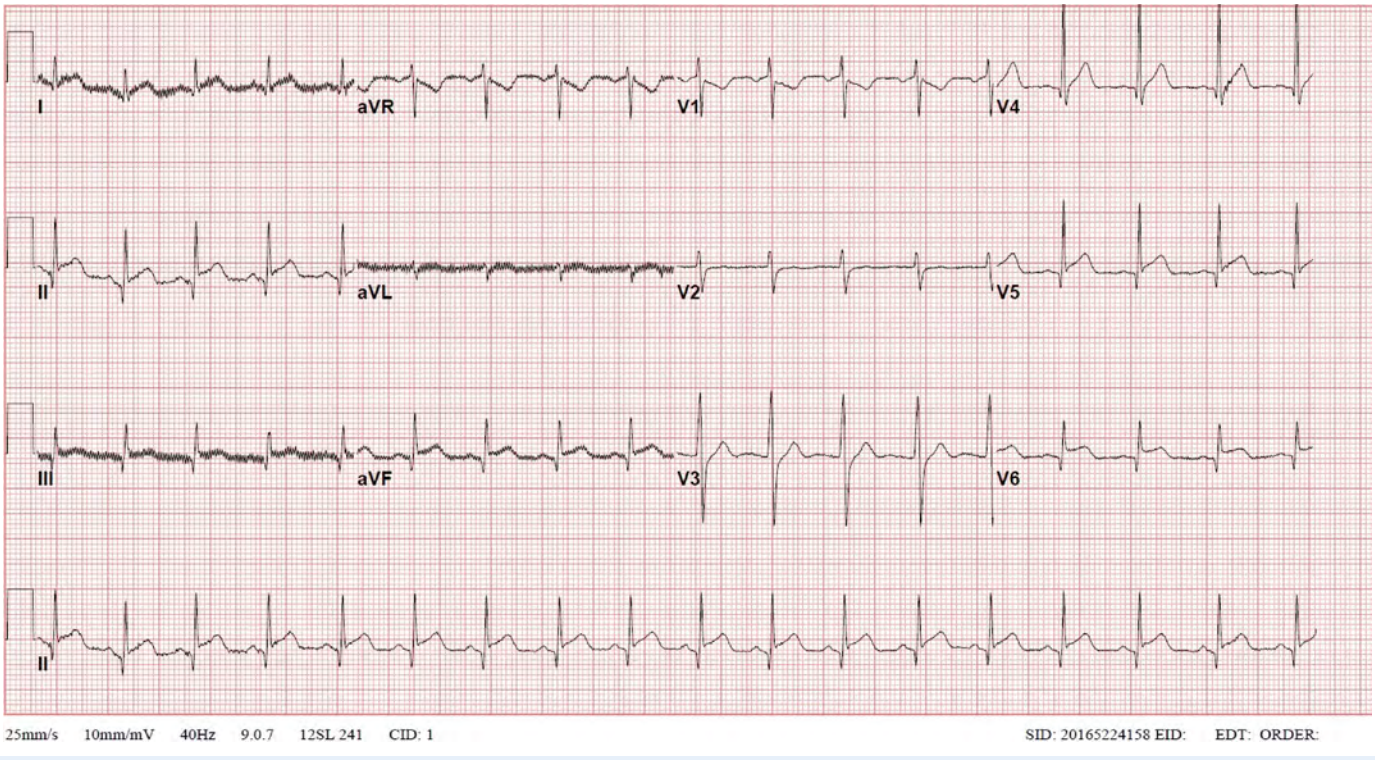

Figure 1. The ST-segment elevation of a patient with acute myocarditis

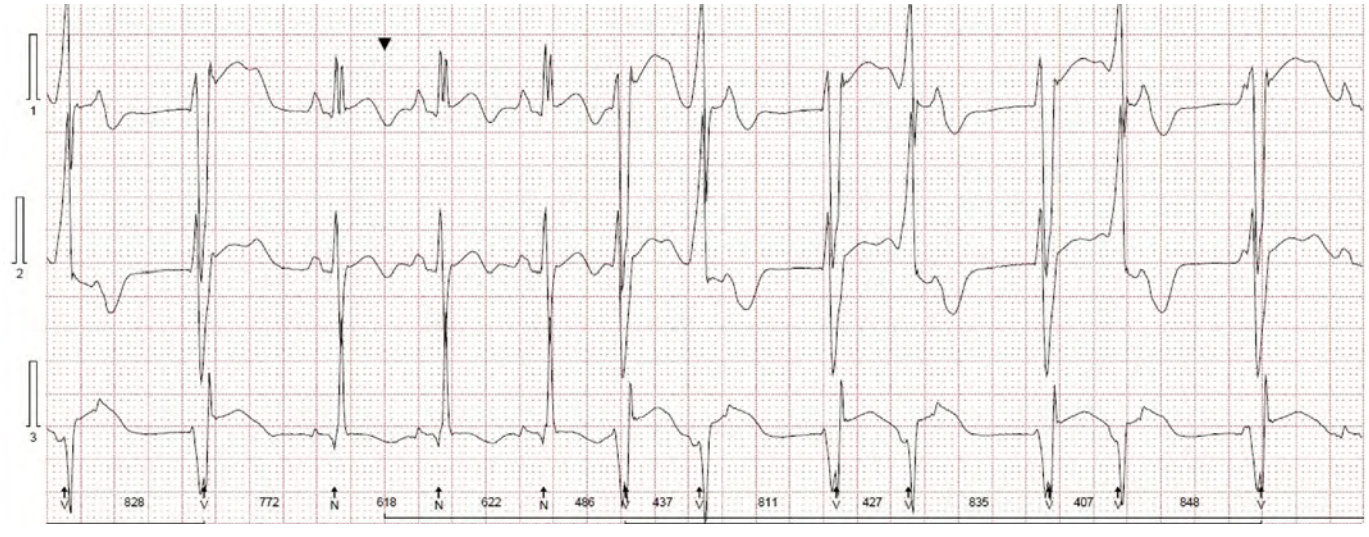

Figure 2. Polymorphic premature ventricular contractions detected by Holter recording of a patient with acute myocarditis

practice. The clinical phenotype can range from asymptomatic cardiac inflammation to fulminant myocarditis, resulting in sudden cardiac death.[1,2,6] Chest pain, which often mimics acute coronary syndrome, is a common initial presentation of myocarditis, as seen in our cohort, with 89.3% of patients presenting with this symptom.[2,4,7,14] It could be due to myocardial inflammation and edema, resulting in ischemia-like sensations even without coronary artery disease.[2,4,6] Troponin I, CK-MB, and CRP levels were raised in our patients, indicating myocardial damage and inflammatory

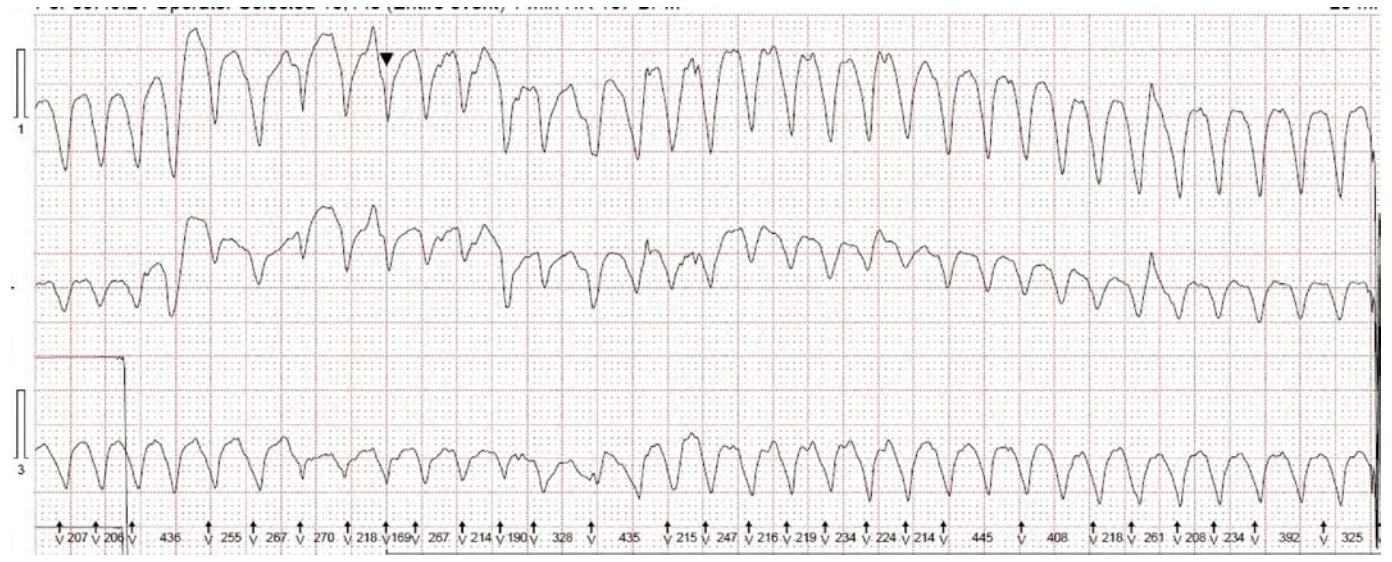

Figure 3. Fast ventricular tachycardia of a patient in ECMO support detected by Holter recording

ECMO: Extracorporeal membrane oxygenation

responses, substantiating their utility in diagnosing myocarditis.[2,5,8] Patients suffering from myocarditis may also exhibit signs of heart failure, such as dyspnea and fatigue, due to impaired cardiac function. Fatigue was observed in a minority of instances, consistent with the literature indicating that it is a less common presenting symptom.[9] In our study, one patient experienced syncope, highlighting the importance of including myocarditis in the differential diagnosis of unexplained syncope, particularly in the pediatric population.[2,4,7]

Myocarditis causes non-specific and varied ECG alterations, suggesting the diffuse and multifocal nature of the inflammatory process affecting the myocardium.[2,3,5] Our study's ECG and Holter findings, which included ST abnormalities, T-wave alterations, conduction disturbances, and arrhythmias in a considerable percentage of our sample, highlight the wide variety of ECG symptoms associated with AM as recorded in the literature.[7,8,10]

ST-segment elevation in inferior and lateral leads was our population's most common ECG change, occurring in 61.9% of patients. This ECG alteration is prevalent in AM, frequently, leading to a misinterpretation of acute myocardial infarction, especially when combined with increased cardiac enzymes.[2] Importantly, ST-segment elevation is often more diffusely distributed with myocarditis, and coronary angiography is usually normal, separating it from genuine myocardial infarction.[1] In our study, 25 (33.3%) patients were diagnosed with perimyocarditis by echocardiography or cardiac MRI. Furthermore, acute coronary syndrome was ruled out by CT angiography in seven patients and catheter angiography in six patients who presented with chest pain, elevated Troponin I levels, and ST-segment elevation.

Another typical ECG finding in myocarditis is T-wave flattening or inversion, which is present in 50.7% of our patients and often indicates myocardial damage or strain.[2,7] Similarly, ST-segment depression (in 2.6% of our patients) can be linked to myocardial inflammation or ischemia. Interventricular conduction delay occurred in 26.7% of our patients, and other conduction abnormalities, such as incomplete right bundle branch block and sinus node dysfunction, can occur due to inflammation and edema interfering with normal electrical conduction pathways.[2,5] Due to the effects of inflammation and necrosis on the cardiac conduction system, both supraventricular and ventricular arrhythmias are commonly observed in myocarditis.[3–5,8] Our findings align with this, as we identified a prolonged QTc interval, first-degree AV block, PACs, SVT, and ventricular arrhythmias in our patient population. Interestingly, the ECG was normal in 19.3% of our patients at diagnosis, consistent with the literature demonstrating that a normal ECG does not rule out myocarditis, emphasizing the importance of a thorough diagnostic assessment when clinical suspicion is high.[7,14]

Therapeutic management of myocarditis primarily hinges on supportive care, which aims to reduce symptoms, avoid or manage complications, and give the myocardium time to heal, which is the mainstay of therapy for myocardi-

Table 4. Correlations between the peak Troponin I levels and presence of electrocardiographic parameters

Parameters	Peak Troponin I level (ng/L) median (range)	p
Electrocardiographic abnormalities		
Yes	680.5 (30–10000)	0.009*
No	114 (18.5–1215)	
ST-segment elevation		
Yes	730 (30–10000)	0.001*
No	221 (18.5–1351)	
T-wave flattening or negative T-wave		
Yes	748 (47–3262)	0.001*
No	302 (18.5–10000)	
Premature Ventricular Contractions		
Yes	711.5 (31–10000)	0.009*
No	302 (18.5–1878)	
Low QRS voltage		
Yes	414.5 (31–10000)	0.847
No	578 (18.5–3262)	
Sinus tachycardia		
Yes	198 (18.5–10000)	0.183
No	540 (30–3262)	
Long QTc interval		
Yes	114 (47–1679)	0.366
No	410 (18.5–10000)	
First-degree atrioventricular block		
Yes	321 (114–1878)	0.819
No	575.5 (18.5–10000)	
Interventricular conduction delay		
Yes	686.5 (54–3262)	0.969
No	518 (18.5–10000)	

P-values are calculated using the Mann–Whitney U test. *: Indicates $p<0.05$

tis. This approach is demonstrated in our dataset, where most patients (86.7%) were treated with anti-inflammatory drugs, a crucial component of this supportive care strategy. Corticosteroids and IVIG were used in a smaller percentage of patients, consistent with earlier research suggesting these therapies for severe or refractory cases.[9,15] Some patients with ventricular arrhythmias required antiarrhythmic drugs (9.3%). This finding is consistent with earlier research showing that this population requires arrhythmia care.[3,4] Three (4%) of our patients received inotropic support for heart failure. Our study also shows that severe myocarditis may necessitate advanced life support procedures. Two patients underwent ECMO, but sadly, they did not survive. This condition emphasizes the potential seriousness of myocarditis and the requirement for quick, forceful treatment in extreme situations.[3,6,16]

Based on our findings, we recommend that patients with AM undergo thorough ECG monitoring, particularly within the 1st week of diagnosis. Early detection of arrhythmias may allow for timely intervention, potentially preventing adverse outcomes such as sudden cardiac death. We also suggest that a high index of suspicion for arrhythmias should be maintained for patients presenting with severe myocardial inflammation, as indicated by markedly elevated cardiac biomarkers.

Study Limitations

Despite the valuable details this study offers, it has limitations. Because the investigation is retrospective and observational, it is difficult to determine the causal relationships between the variables. Of our 75 patients, only five tested positive in viral serology. The timing of tests relative to disease onset can miss transient viremia. The assays' specificity and sensitivity might also impact results. While viruses are a common cause of myocarditis, non-viral etiologies could have been present in some cases. As such, the limited detection of viral agents in our cohort should be viewed with these considerations in mind. Finally, because the study was conducted at a single institution, results may not be applicable to other circumstances necessitating multicenter investigations for more thorough validation.

CONCLUSION

Myocarditis has a variable etiology and presentation. ECG abnormalities and arrhythmias are frequently observed. This condition requires personalized care that is based on the underlying cause, current symptoms, and accompanying consequences. It frequently combines supportive care, targeted medicines, and the control of complications. Even with the best therapy, severe cases can still result in bad results, emphasizing the need for further investigations into improved treatment strategies.

Disclosures

Ethics Committee Approval: The study was approved by the Istanbul Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital Clinical Research Ethics Committee (No: 2023.04-41, Date: 16/05/2023).

Informed Consent: Written informed consent was obtained from all patients.

Peer-review: Externally peer reviewed.

Authorship Contributions: Concept: F.S.Ş., P.Ay.; Design: F.S.Ş., E.D.; Supervision: A.G., Y.E.; Data Collection or Processing: E.D., P.A.; Analysis or Interpretation: A.G., Y.E.; Literature Search: P.A., S.U.A.; Writing: F.S.Ş.; Critical review: A.G., Y.E.

Conflict of Interest: No conflict of interest was declared by the authors.

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