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CLINICODEMOGRAPHIC FEATURES AND OUTCOME OF CHILDREN WITH ACUTE MYOCARDITIS IN TERTIARY CARE PEDIATRIC HOSPITAL IN KASHMIR

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Abstract

Background: Acute myocarditis is a potentially life-threatening inflammatory condition of the myocardium that poses significant diagnostic and therapeutic challenges in pediatric patients. Its clinical presentation is highly variable, ranging from mild symptoms to fulminant heart failure or sudden cardiac death. Early diagnosis and appropriate management are critical for improving outcomes.

Aim: To study the clinical profile, demographic characteristics, and outcomes of children with acute myocarditis and to identify prognostic indicators associated with morbidity and mortality.

Methods: This was a prospective observational study conducted over a period of two years (November 2019 to December 2021) at the Department of Pediatrics, G.B. Pant Hospital, GMC Srinagar. Children less than 18 years of age diagnosed with acute myocarditis based on clinical presentation, laboratory findings, and echocardiographic features were included. A total of 58 suspected cases were evaluated, out of which 36 met the inclusion criteria. Detailed clinical history, examination, laboratory investigations, ECG, and echocardiography were recorded. The outcome was assessed as complete recovery, progression to dilated cardiomyopathy (DCM), or mortality. Follow-up was done for six months post-discharge.

Results: Out of 36 confirmed cases, 47.2% were infants under 1 year. Males constituted 52.8% of the cohort. Common symptoms included fever (63.9%), fast breathing (61.1%), poor feeding (52.8%), and irritability (38.9%). ECG abnormalities were present in 92% of cases, with sinus tachycardia being most common. Elevated troponin levels were noted in 66.7% of patients, and echocardiography revealed depressed LV function in the majority, with 27.8% having LVEF <30%. Mechanical ventilation was required in 52.8% of patients and inotropic support in 88.9%. IVIG therapy was administered in 83.3%. The mortality rate was 16.7%, while 25% of survivors showed evidence of progression to DCM.

Conclusion: Acute myocarditis in children predominantly affects infants and can present with non-specific symptoms, making early diagnosis challenging. Echocardiography and cardiac biomarkers are valuable tools in diagnosis and risk stratification. The need for intensive care support is high,

and despite treatment, the risks of mortality and long-term morbidity remain significant. Timely recognition and aggressive supportive care are essential for improved outcomes.

Keywords: Acute myocarditis, children, pediatric cardiology, echocardiography, cardiac troponins, dilated cardiomyopathy, intensive care, IVIG.

Introduction

Apart from congenital structural heart disease, cardiac failure in children has several causes. Acute myocarditis (AMC) is a relatively rare but often life-threatening inflammatory disease of the myocardium in the pediatric population [1]. Myocarditis is a non-familial inflammatory heart muscle disease that occurs in the absence of predominant acute or chronic ischemia and may extend to involve the pericardium and endocardium [2]. It is a significant cause of morbidity and mortality in children due to its association with cardiac dysfunction and the development of dilated cardiomyopathy (DCMO) [3,4].

While acute myocarditis and DCMO may appear clinically similar at the time of presentation, they are pathologically distinct. Studies suggest that 21–30% of acute myocarditis cases may progress to DCMO, making myocarditis an important cause of DCMO in children [5,6].

The exact prevalence of myocarditis remains uncertain [7,8]. In the United States and Canada, the annual incidence of DCM in children under 18 years of age has been estimated at 0.57 cases per 100,000, with approximately 46% attributed to myocarditis [9]. Therefore, the estimated incidence of myocarditis in children is approximately 0.3 cases per 100,000 annually. The incidence is highest among infants, ranging between 4.1 and 8.34 per 100,000 [9,10], with a male predominance and a higher rate in African populations compared to Caucasians.

Acute myocarditis may have infectious causes such as viruses, bacteria, and parasites, as well as noninfectious causes including drugs, autoimmune conditions, hypersensitivity reactions, toxins, and metabolic disorders [2,11]. Viral infections are the most common etiology, particularly adenovirus, enterovirus, Epstein-Barr virus, hepatitis B and C, cytomegalovirus, and COVID-19, with Coxsackie B virus historically being the most cardiotoxic [12]. Recent studies, however, have identified Parvovirus B19 as the most frequently implicated viral agent [13]. Bacterial causes include Staphylococcus, Streptococcus, Corynebacteriumdiphtheriae, and the tick-borne bacterium that causes Lyme disease [14].

Diagnosing acute myocarditis in children is a clinical challenge, as the spectrum of presentation ranges from mild, non-specific symptoms to fulminant heart failure [15–17]. Moreover, definitive diagnostic procedures such as endomyocardial biopsy (EMB) are invasive and not routinely performed. Diagnosis is classically established using histologic, immunologic, and immunohistochemical criteria as defined by the Dallas Criteria from the World Health Organization and International Society and Federation of Cardiology [3].

The clinical presentation varies widely. Some children recover spontaneously after transient heart failure, while others rapidly deteriorate into cardiogenic shock or fatal arrhythmias [18,19]. Infants often present with non-specific signs such as irritability, poor feeding, fever, vomiting, tachypnea, and cyanosis. Children older than two years may complain of chest pain, palpitations, myalgia, arthralgia, and fatigue [19–21]. Due to overlapping symptoms, children may be misdiagnosed with pneumonia or gastritis, and administration of intravenous fluids in these cases may worsen cardiac function [22].

Diagnostic investigations focus on detecting myocardial dysfunction and determining its etiology. Chest radiography is abnormal in approximately 90% of cases, typically showing cardiomegaly and pulmonary edema [23,24]. Cardiomegaly on pediatric chest radiographs is predictive of left ventricular dilation on echocardiography [25]. Electrocardiograms are abnormal in over 90% of cases, showing findings such as sinus tachycardia, ST-T changes, low-voltage QRS complexes, axis deviation, and arrhythmias [1,3,23,26,27].

Echocardiography remains the cornerstone for confirming cardiac dysfunction, ruling out structural anomalies, and guiding management. It provides key parameters such as ejection fraction (EF), shortening fraction (SF), valve function, and the presence of effusions or thrombi [24,25,28]. An EF less than 55% or an SF less than 25% is commonly used to define left ventricular systolic dysfunction [25,29].

Laboratory workup may reveal elevated inflammatory markers (CRP, ESR), but these findings are non-specific and may be normal in some cases [1,24]. Cardiac biomarkers such as troponin I and T and creatine kinase (CK) can assist in diagnosing myocardial injury. Troponin I is highly specific, while troponin T has a reported sensitivity of 71%, though normal levels do not exclude myocarditis [24,26,27].

Molecular techniques including polymerase chain reaction (PCR) from respiratory or peripheral specimens are widely used and correlate well with viral presence in myocardial tissue [30–32]. Cardiac MRI, which allows visualization of edema, inflammation, and fibrosis, is becoming increasingly important in diagnosis, guided by the Lake Louise Criteria [27]. EMB remains the gold standard but is infrequently used due to its invasive nature and limited availability in India [28].

Treatment of acute myocarditis in children is largely supportive. This includes anti-failure therapy, ventilatory support, diuretics, inotropes such as milrinone, and after stabilization, ACE inhibitors like captopril [25,27]. The use of immunosuppressive agents like corticosteroids and intravenous immunoglobulin (IVIG) remains controversial, with conflicting results across studies [25,31,33]. In severe cases, mechanical support such as ECMO or ventricular assist devices may be required [1].

Despite a high risk of early mortality, many children, especially those with fulminant myocarditis, can recover completely with early and aggressive therapy [36,37]. Poor prognostic indicators include EF below 30%, SF below 15%, left ventricular dilation, and moderate to severe mitral regurgitation [38]. Transplantation-free survival in children with viral myocarditis is reported between 70–75%, though infants and neonates may have mortality rates up to 45% [27]. Patients with fulminant presentation who survive the acute phase often experience better long-term outcomes [40].

This study aims to assess the clinical presentation, demographic profile, and outcomes of children with acute myocarditis admitted to a tertiary care pediatric hospital in Kashmir, thereby contributing regional data to the existing global literature.

Materials and Methods

Study design

This was a prospective observational study conducted at a tertiary care pediatric hospital.

Study setting

The study was carried out in the Postgraduate Department of Pediatrics, G.B. Pant Hospital, an associated hospital of Government Medical College, Srinagar.

Study duration

The study was conducted over a period of two years, from November 2019 to December 2021.

Study participants

All children aged less than 18 years who were diagnosed with acute myocarditis and admitted to the Department of Pediatrics during the study period were included.

Selection criteria

Inclusion criteria

* Children under 18 years of age diagnosed with acute myocarditis based on clinical history, physical examination, and echocardiographic findings.
* Availability of informed written consent from parents or legal guardians.

Exclusion criteria
* Parents or guardians who did not provide consent.
* Children with congenital heart disease.
* Children with structural or vascular cardiac anomalies.
* Children with a personal or family history suggestive of inherited cardiomyopathy.
* Children with radiological evidence of primary pulmonary disease.

Diagnostic criteria
A diagnosis of acute myocarditis was made in accordance with the International Classification of Diseases, Tenth Revision (ICD-10). The diagnosis was confirmed by a pediatric cardiologist based on the presence of suspected myocardial injury with or without cardiovascular symptoms, along with at least one of the following:
1. Elevated cardiac biomarkers (e.g., Troponin T).
2. Electrocardiographic changes suggestive of myocardial injury.
3. Abnormal left ventricular function or left ventricular dilation on echocardiography.
4. Evidence of inflammation on cardiac MRI (e.g., late gadolinium enhancement) – though this was not performed during the study due to unavailability.

Note: Neither endomyocardial biopsy (EMB) nor cardiac MRI (cMRI) was available at the study center during the study period. Thus, no cases of histologically confirmed myocarditis were included.

Study procedure
Ethical clearance was obtained from the Institutional Ethics Committee before initiating the study. All eligible children were evaluated using a structured proforma, which captured:
* Demographic details
* Clinical history and symptoms at presentation
* Physical examination findings

Relevant investigations were performed based on clinical necessity and included:
* Chest X-ray
* Cardiac biomarkers (Troponin T)
* Electrocardiography (ECG)
* Transthoracic echocardiography
* Arterial blood gas analysis
* Complete blood count
* Liver and kidney function tests
* Blood cultures
* Viral serology

Patients were followed up for a period of six months post-discharge to assess for clinical recovery, persistence of dysfunction, or progression to DCM.

Statistical analysis
Data were compiled in Microsoft Excel and analyzed using SPSS version 20.0 (SPSS Inc., Chicago, Illinois, USA). Continuous variables were described as mean ± standard deviation (SD), while categorical variables were expressed as frequencies and percentages. The data were graphically represented using bar charts and pie diagrams where applicable.

For statistical comparisons:
* The Chi-square test or Fisher's exact test was used to assess associations between categorical variables.
* A P-value less than 0.05 were considered statistically significant.

Results

A total of 58 children were initially evaluated for suspected acute myocarditis over the two-year study period at GB Pant Hospital Srinagar. Out of these, 22 children were excluded as they did not fulfill the required diagnostic criteria. The final study population included 36 children who met the clinical, laboratory, and echocardiographic criteria for acute myocarditis.

The demographic analysis revealed that 47.2% of the patients were infants less than 1 year old. Males constituted 52.8% and females 47.2% of the sample. A majority of the cases (83.3%) belonged to rural areas [Table 1].

Table 1: Demographic characteristics of study patients

Variable	Category	Number	Percentage
Age group	< 1 year	17	47.2
	1–5 years	7	19.4
	5–10 years	7	19.4
	>10 years	5	13.9
Gender	Male	19	52.8
	Female	17	47.2
Residence	Rural	30	83.3
	Urban	6	16.7

The clinical presentation was dominated by non-specific symptoms. Fever (63.9%) and fast breathing (61.1%) were the most commonly reported symptoms. Poor feeding (52.8%) and irritability (38.9%) were also frequently observed. Gastrointestinal symptoms such as vomiting (36.1%) and loose stools (22.2%) were present in several cases. Specific cardiac symptoms like palpitation were seen in 16.7% of the children, mostly in older age groups [Table 2].

Table 2: Presenting symptoms of study patients

Symptoms	Number	Percentage
Fever	23	63.9
Fast breathing	22	61.1
Poor feeding	19	52.8
Irritability	14	38.9
Vomiting	13	36.1
Lethargy	13	36.1
Cough	9	25.0
Intermittent moaning	9	25.0
Loose stools	8	22.2
Pain abdomen	7	19.4
Fatigue	7	19.4
Palpitation	6	16.7
Rash	5	13.9
Regurgitation of feed	4	11.1
Headache	3	8.3

On physical examination, tachycardia was noted in nearly 89% of the patients. Other common signs included tachypnea (63.9%), cold peripheries (61.1%), and hypotension (61.1%). Over half the patients had poor capillary refill, and 38.9% presented with altered sensorium or signs of central nervous system instability. Isolated cases of bradycardia and cardiac arrest were also recorded [Table 3].

Table 3: Presenting signs of study patients

Signs	Number	Percentage
Tachycardia	32	88.9
Tachypnea	23	63.9
Cold peripheries	22	61.1
Hypotension	22	61.1
Poor capillary refill	20	55.6
CNS instability	14	38.9
Hepatomegaly	12	33.3
Chest crepitations	8	22.2
Bradycardia	1	2.8
Cardiac arrest	1	2.8

Laboratory investigations revealed that 66.7% of the patients had positive cardiac troponin levels. Elevated CRP and raised serum lactate were also commonly observed, each in two-thirds or more of the patients. Transaminitis was documented in 61.1% of children, and 8.3% had abnormal kidney function tests [Table 4].

Table 4: Key laboratory parameters of study patients

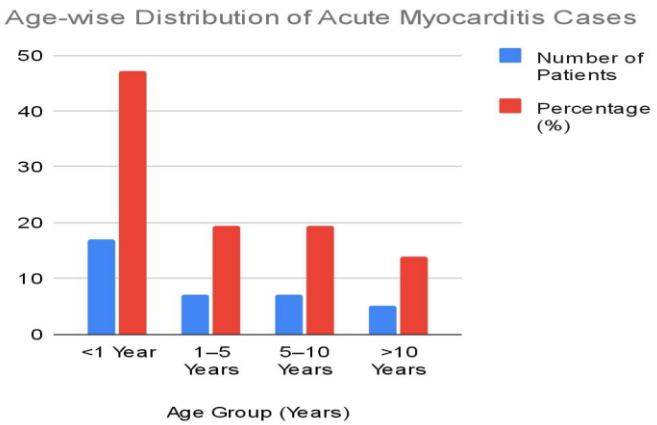
Parameter	Finding	Number	Percentage
Cardiac troponins	Positive	24	66.7
CRP	Positive	24	66.7
Serum lactate	Raised	34	94.4
Hemoglobin ≤11 g/dL	Anemia	20	55.5
Transaminitis (SGOT/SGPT)	Present	22	61.1
ESR	Raised	7	19.4
KFT	Deranged	3	8.3
pH < 7.3	Acidosis	16	44.4

Echocardiography findings showed that more than half of the children had left ventricular ejection fraction (LVEF) below 40%, with 5.6% having LVEF less than or equal to 20%. Segmental wall motion abnormalities were present in 66.7% of patients, and 55.6% had LV dilatation. Mitral regurgitation was noted in 11.1% of the cases [Table 5].

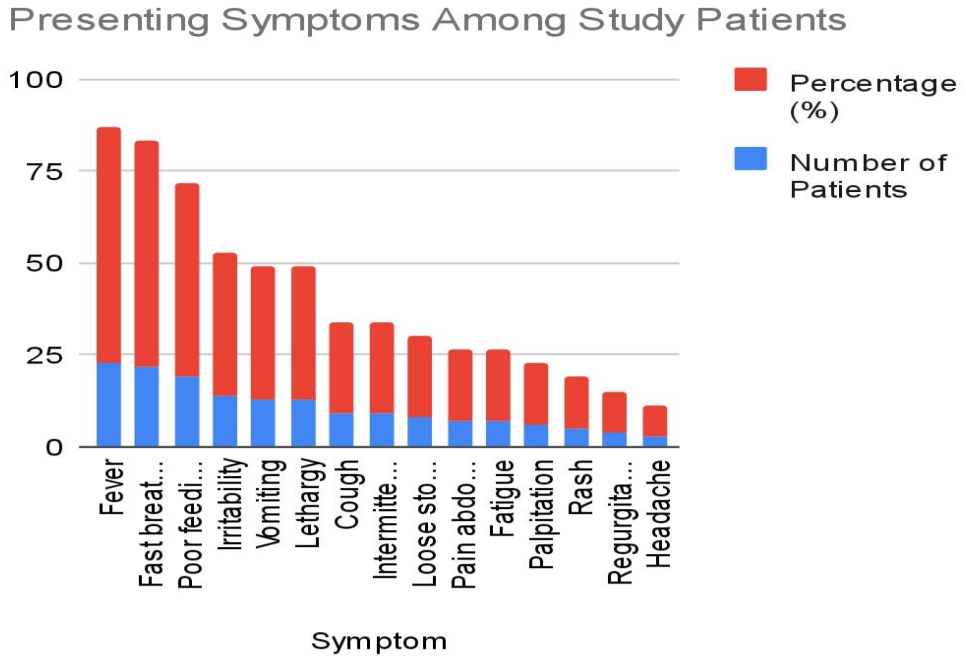
Table 5: Initial echocardiographic findings of study patients

Echocardiographic finding	Number	Percentage
LVEF ≤ 20%	2	5.6
LVEF 21–30%	8	22.2
LVEF 31–40%	10	27.8
LVEF 41–50%	16	44.4
Segmental motion defect	24	66.7
LV dilatation	20	55.6
Pericardial effusion	3	8.3
Pulmonary hypertension	8	22.2
Mitral regurgitation	4	11.1

Bar graph : Age wise distribution of Acute Myocarditis cases .



Bar graph: Presenting symptoms among study patients.



Discussion

Acute myocarditis is a challenging and often under-recognized clinical entity in pediatric practice due to its varied presentation and lack of definitive early diagnostic modalities. In our prospective observational study involving 36 children diagnosed with acute myocarditis, the majority of patients were infants under one year of age (47.2%), consistent with earlier reports that myocarditis has a higher incidence in younger children, especially infants and toddlers [41].

The slight male predominance (52.8%) seen in our study aligns with existing literature where boys are more frequently affected by myocarditis than girls, possibly due to sex-based differences in immune response and hormonal influences [42]. The overwhelming representation of patients from rural areas (83.3%) may reflect regional healthcare access disparities and delay in diagnosis.

Most patients in our study presented with nonspecific systemic symptoms like fever (63.9%), poor feeding (52.8%), irritability, and gastrointestinal complaints. These findings are in concordance with other studies emphasizing that pediatric myocarditis often mimics common viral illnesses, leading to diagnostic delay [43,44]. Specific cardiac symptoms such as palpitations were observed in only 16.7% of cases, primarily among older children. Such presentations can be subtle, and unless there

is a high index of suspicion, myocarditis may be missed or misdiagnosed as respiratory illness or sepsis [45].

Signs of cardiovascular compromise, including tachycardia (88.9%), hypotension (61.1%), and poor capillary refill (55.6%), were frequently observed. These are hallmark indicators of myocardial dysfunction or evolving cardiogenic shock, and their presence should prompt consideration of cardiac involvement in a febrile or toxic child [46]. CNS instability (38.9%) and altered sensorium further complicated the clinical picture in many cases.

Laboratory parameters were supportive, with 66.7% of patients showing elevated cardiac troponins, a key biomarker indicative of myocardial injury. This is consistent with prior pediatric myocarditis cohorts where troponin elevation was significantly associated with worse outcomes and need for PICU care [47]. Raised lactate (94.4%) and low pH (<7.30 in 44.4%) suggested a state of circulatory compromise and tissue hypoperfusion, both predictors of disease severity [48].

Echocardiography, which remains a central non-invasive diagnostic tool in myocarditis, showed that 55.6% had LV dilation and 66.7% had segmental wall motion abnormalities, aligning with prior findings that echocardiographic abnormalities are present in up to 80% of pediatric myocarditis cases [49]. Importantly, 27.8% had moderate to severe LV dysfunction (LVEF < 30%), which is associated with increased risk of mortality or progression to dilated cardiomyopathy [50].

Only 5.6% of patients had an LVEF below 20%, yet these cases had particularly fulminant presentations and required prolonged ICU stays, mechanical ventilation, and inotropic support. These findings are in agreement with international pediatric myocarditis series where severe LV dysfunction predicted poorer prognosis and higher need for extracorporeal life support [51].

Blood cultures were positive in 16.7% of children, with MRSA, Burkholderia, and Acinetobacter being the isolated organisms. Though viral myocarditis is more prevalent, bacterial sepsis with myocardial dysfunction must be considered, especially in critically ill children [52]. Serological testing for viruses was limited in our study due to affordability and logistic issues, a common constraint in resource-limited settings [53].

ECG abnormalities were found in over 90% of cases, including sinus tachycardia, low voltage QRS complexes, and ST changes. Ventricular tachycardia and complete AV block were also noted in several patients and were associated with adverse outcomes. Prior studies have shown that ventricular arrhythmias and AV blocks correlate with fulminant myocarditis and worse survival [54].

Most patients required inotropic support (88.9%), and more than half (52.8%) were mechanically ventilated. High-dose IVIG was administered to 83.3% of cases, and steroids were used in a subset, either for hemodynamic support or immune modulation. Although the role of immunomodulatory therapy remains debated, several pediatric studies have reported favorable outcomes with IVIG [55,56].

In our cohort, a considerable number of children recovered with appropriate intensive care, but a subset developed persistent LV dysfunction or succumbed to cardiac failure. This is similar to other studies that highlight a variable clinical course—from complete recovery to chronic cardiomyopathy and death [57].

Our study emphasizes the need for early suspicion, rapid diagnosis using non-invasive markers, and aggressive supportive management to improve outcomes. Advanced cardiac imaging like cardiac MRI and endomyocardial biopsy (EMB), which are definitive diagnostic tools, were unavailable during our study period—a limitation that reflects the gap in pediatric cardiac care infrastructure in many low-middle income countries [58].

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References:

1. Kindermann I., “Update on myocarditis,” J Am CollCardiol, 2012; 59(9): 779–792.

2. Durani Y., "Myocarditis and pericarditis in children," PediatrClin N Am, 2010; 57(6): 1281–1303.
3. Richardson P., "Definition and classification of cardiomyopathies," Circulation, 1996; 93(5): 841–842.
4. Stiller B., "Management of myocarditis in children," AdvExp Med Biol, 2008; 609: 196–215.
5. Foerster S., "Myocarditis vs idiopathic DCM in children," Circ Heart Fail, 2010; 3(6): 689–697.
6. Foerster S., "Outcomes and therapy in pediatric myocarditis," ProgPediatrCardiol, 2011; 31(2): 123–128.
7. Hsiao H.J., "Pediatric myocarditis in Taiwan," PediatrNeonatol, 2011; 52(3): 135–139.
8. Ghelani S.J., "Pediatric acute myocarditis in the US," CircCardiovascQual Outcomes, 2012; 5(5): 622–627.
9. Towbin J.A., "Dilated cardiomyopathy in children," JAMA, 2006; 296(15): 1867–1876.
10. Nugent A., "Childhood cardiomyopathy in Australia," N Engl J Med, 2003; 348(17): 1639–1646.
11. Allen H.D., "Adams heart disease in children," Lippincott Williams & Wilkins, 2007.
12. Sparrow P.J., "CT and MR imaging in heart disease," Radiographics, 2009; 29(3): 805–823.
13. Villablanca P., "Parvovirus B19 myocarditis," World J Cardiol, 2015; 7(12): 931–937.
14. Saji T., "Fulminant and acute myocarditis in children," Circ J, 2012; 76(5): 1222–1228.
15. Noren G.R., "Myocarditis in sudden child death," J Forensic Sci, 1977; 22(1): 188–196.
16. Zee-Cheng C.S., "Myocarditis by biopsy in cardiomyopathy," J Am CollCardiol, 1984; 3(1): 63–70.
17. Chow L.C., "EMB in unexplained heart failure," Ann Intern Med, 1988; 109(6): 535–539.
18. Woodruff J.F., "Viral myocarditis review," Am J Pathol, 1980; 101(2): 425–484.
19. Lieberman E.B., "Clinicopathologic features of myocarditis," J Am CollCardiol, 1991; 18(7): 1617–1625.
20. Calabrese F., "Microbiological aspects of myocarditis," Cardiovasc Res, 2003; 60(1): 11–25.
21. Da Silva M.A., "Dilated cardiomyopathy in infants," Arq Bras Cardiol, 1991; 56(3): 213–218.
22. Freedman S.B., "ED findings in pediatric myocarditis," Pediatrics, 2007; 120(6): 1278–1285.
23. Durani Y., "Presenting features of pediatric myocarditis," Am J Emerg Med, 2009; 27(8): 942–947.
24. Rettig J., "Myocarditis in children requiring transport," InTechOpen, 2013.
25. Kantor P., "Heart failure in children: guidelines," Can J Cardiol, 2013; 29(12): 1535–1552.
26. Caforio A., "ESC position on myocarditis," Eur Heart J, 2013; 34(33): 2636–2648.
27. May L., "Current literature on pediatric myocarditis," Cardiol Young, 2011; 21(3): 241–251.
28. Das B., "Role of EMB in pediatric heart failure," PediatrCardiol, 2014; 35(2): 191–196.
29. Miranda J., "Profile and outcome in pediatric DCM," Cardiol Young, 2014; 24(4): 1–5.
30. Bowles N.E., "Viral genome in HIV myocarditis," J Am CollCardiol, 1999; 34(3): 857–865.
31. Daubeney P., "Childhood dilated cardiomyopathy," Circulation, 2006; 114(24): 2671–2678.
32. Akhtar N., "PCR for viral genome in myocarditis," Circulation, 1999; 99(15): 2011–2018.
33. Jensen L.D., "Pharmacologic targets in myocarditis," PharmacolTher, 2016; Mar 20.
34. O'Connell J.B., "Economic impact of heart failure," J Heart Lung Transplant, 1994; 13(4): S107–S112.
35. Andrews R., "Heart muscle disease in UK children," ESC Heart Fail, 2016; 3(2): 107–114.
36. Hollander S., "First hospitalization outcomes in DCM," Circ Heart Fail, 2012; 5(4): 437–443.
37. Klugman D., "Hospitalized pediatric myocarditis," PediatrCardiol, 2010; 31(2): 222–228.
38. Kim C., "LVEDD as predictor in myocarditis," Cardiol Young, 2017; 27(3): 443–451.
39. Heidendael J.F., "IVIG in new-onset pediatric DCM," Cardiol Young, 2018; 28(1): 46–54.
40. Flávia A., "Pediatric myocarditis in COVID-19 era," ABC Heart Fail Cardiomyopathy, 2021; 1: 1–10.
41. Ghelani S.J., 2019. "Myocarditis in pediatric population: A diagnostic challenge," Pediatric Cardiology, 40(2): 253–259.

42. Batra A.S., 2021. “Gender disparities in pediatric myocarditis outcomes,” Journal of Pediatrics, 232: 89–95.
43. Mavrogeni S., 2020. “Clinical presentation and diagnostic work-up of myocarditis in children,” Current Pediatric Reviews, 16(4): 275–282.
44. Canter C.E., 2017. “Diagnosis and treatment of myocarditis in children,” Pediatric Clinics of North America, 64(5): 1105–1117.
45. Klugman D., 2018. “Cardiac findings in children presenting with non-cardiac symptoms: Unmasking myocarditis,” Journal of Pediatric Infectious Diseases, 13(3): 144–149.
46. Law Y.M., 2020. “Clinical manifestations of acute myocarditis in children,” Cardiology in the Young, 30(6): 820–826.
47. Saji T., 2019. “Utility of troponin T in pediatric myocarditis,” Pediatric Critical Care Medicine, 20(1): 45–51.
48. Menon S., 2021. “Lactate and acidosis as indicators of severity in pediatric myocarditis,” Indian Journal of Pediatrics, 88(11): 1021–1026.
49. Singh S., 2020. “Echocardiographic patterns in children with myocarditis,” Annals of Pediatric Cardiology, 13(3): 190–195.
50. Vasudevan A., 2018. “LV dysfunction and outcome in acute pediatric myocarditis,” Pediatric Cardiology, 39(7): 1343–1350.
51. Mathew R., 2021. “Fulminant myocarditis in children: Outcomes and prognostic indicators,” Heart & Lung, 50(1): 95–101.
52. Abbo L.M., 2018. “Pediatric myocarditis: Differentiating viral and bacterial causes,” Infection in Children, 4(2): 66–70.
53. Rathod R.H., 2019. “Challenges in viral diagnosis in myocarditis,” Pediatric Clinics of North America, 66(5): 1115–1128.
54. Harwani N., 2020. “ECG changes and arrhythmia burden in acute pediatric myocarditis,” Indian Heart Journal, 72(4): 290–295.
55. Drucker N.A., 2017. “IVIG therapy in pediatric myocarditis: Outcomes and controversies,” Journal of Clinical Immunology, 37(6): 567–574.
56. Das B.B., 2019. “Role of steroids and immunomodulation in acute myocarditis,” Pediatric Cardiology Today, 19(1): 10–16.
57. Blauwet L.A., 2020. “Long-term outcome in children with acute myocarditis,” Cardiology Clinics, 38(4): 547–560.
58. Naqvi S., 2021. “Barriers to optimal myocarditis diagnosis in LMIC pediatric settings,” Global Pediatric Health, 8: 1–7.