Section: Paediatrics

Original Research Article

CLINICAL PROFILE AND SHORT-TERM OUTCOME OF PEDIATRIC DILATED CARDIOMYOPATHY AT A TERTIARY CARE CENTER IN ODISHA, INDIA: A CROSS-SECTIONAL OBSERVATIONAL STUDY

Satyajit Swain[1], Subhashree Kar[2] Sunil Kumar Agarwalla[3]

[1]*Junior Resident, Department of Paediatrics, SCBMCH & SVPPGIP, Cuttack, Odisha, India*
[2]*Assistant Professor, Department of Paediatrics, SCBMCH & SVPPGIP, Cuttack, Odisha, India*
[2]*Professor, Department of Paediatrics, SCBMCH & SVPPGIP, Cuttack, Odisha, India*

Received *: 03/01/2026*
Received in revised form *: 10/02/2026*
Accepted *: 26/02/2026*

Corresponding Author:
Dr. Sunil Kumar Agarwalla,
Professor, Department of Paediatrics, SCBMCH& SVPPGIP, Cuttack, Odisha, India.
Email: sunil_9910@yahoo.com

DOI: 10.70034/ijmedph.2026.1.489

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (1); 2846-2849

ABSTRACT

Background: Pediatric dilated cardiomyopathy (DCM) is a major cause of childhood heart failure and often presents late in resource-limited settings. This study describes the clinical profile and short-term outcome of children with DCM managed at a tertiary care center in Odisha.

Materials and Methods: Hospital-based cross-sectional observational study conducted in the Department of Pediatrics, S.C.B. Medical College and Hospital and SVPPGIP, Cuttack, Odisha, India (January 2024-January 2026). Children aged 0-14 years with echocardiography-confirmed DCM (left ventricular dilatation with impaired systolic function, without congenital/structural heart disease) were enrolled consecutively (n=44). Demographics, clinical presentation, etiology, investigations, echocardiographic parameters, treatment, ICU admission, and one-month outcomes (improved/static/deteriorated/death) were recorded. Categorical variables were summarized as frequency (%) and continuous variables as mean (SD).

Results: Mean age was 7.16±3.92 years; 59.1% were male. Breathlessness/fast breathing (27.3%) was the commonest presenting symptom followed by edema (15.9%) and poor weight gain/failure to thrive (13.6%). Mean symptom duration before presentation was 65.9±32 days. Idiopathic DCM constituted 50.0%, myocarditis 27.3%, nutritional causes 13.6%, and metabolic disorders 9.1%. More than half presented in advanced heart failure (Ross class III-IV: 52.3%) and 77.3% required ICU admission. Mean LVEF was 27.7±8.25% and mean LV dimension was 60±6.81 mm; RV dysfunction was present in 52.3%. At one month, 38.6% improved, 34.1% were static, 22.7% deteriorated, and 4.5% died.

Conclusion: Pediatric DCM in this tertiary-care cohort commonly presented after a prolonged symptomatic period with severe systolic dysfunction and high ICU utilization. Short-term response to standard medical therapy was heterogeneous, underscoring the need for early recognition, etiologic evaluation for reversible causes, and close follow-up.

Keywords: Dilated cardiomyopathy; pediatric; heart failure; myocarditis; echocardiography; India

INTRODUCTION

Dilated cardiomyopathy (DCM) is the most common cardiomyopathy in the pediatric age group and a leading cause of childhood heart failure, hospitalization, and heart transplantation. Unlike adults, in whom ischemic and hypertensive etiologies predominate, pediatric DCM is more often idiopathic, inflammatory (myocarditis), genetic, metabolic, or nutritional in origin. In many low- and middle-income settings, delayed recognition is frequent because early manifestations (tachypnea,

cough, feeding difficulty, poor weight gain) overlap with common respiratory illnesses.[1-10]

Indian data on pediatric DCM remain limited and are mostly from single-center experiences. Describing local clinical patterns, disease severity at presentation, and early outcomes can help clinicians maintain a higher index of suspicion, prioritize etiologic work-up for reversible causes, and counsel families more accurately. This study aimed to assess the clinical profile and one-month outcome of children aged 0-14 years with DCM attending a tertiary care center in Odisha.[11-20]

MATERIALS AND METHODS

Study design and setting: Hospital-based cross-sectional observational study conducted in the Department of Pediatrics, S.C.B. Medical College and Hospital and Sardar Vallabhbhai Patel Post Graduate Institute of Pediatrics (SVPPGIP), Cuttack, Odisha, India.

Study period: January 2024 to January 2026.

Participants: Children aged 0-14 years diagnosed with dilated cardiomyopathy (DCM) were enrolled consecutively (n=44). DCM was defined as left ventricular dilatation with impaired systolic function (with or without right ventricular involvement) on echocardiography, in the absence of congenital heart disease or other significant structural cardiac anomalies. Written informed consent was obtained from parents/guardians.

Data collection: Demographic details (age, sex, socioeconomic status), presenting symptoms and duration, family history, heart failure functional class (Ross), baseline vital signs, ECG, chest radiograph cardiothoracic ratio, and echocardiographic parameters (left ventricular ejection fraction, ventricular dimensions, right ventricular dysfunction) were recorded using a predesigned proforma and compiled using microsoft excel. Etiology was categorized as idiopathic, myocarditis, nutritional, or metabolic based on clinical evaluation and available investigations. All patients received guideline-consistent standard medical therapy as per treating unit protocols.

Outcome assessment: Patients were followed for one month. Outcome was categorized as improved, static, deteriorated, or death based on clinical assessment with supportive investigations where available.

Statistical analysis: Data were analyzed using JAMOVI (version 2.6.44). Continuous variables are presented as mean±SD and categorical variables as frequency (%). Chi-square goodness-of-fit tests were used for selected categorical distributions; $p<0.05$ was considered significant.

Ethics: Institutional Ethics Committee approval was obtained and confidentiality maintained.

RESULTS

A total of 44 children with echocardiography-confirmed DCM were included.

Table 1: Demographic characteristics (n=44)

Variable	Category/Statistic	Value
Age (years)	Mean±SD	7.16±3.92
Age (years)	Median	8.1
Sex	Male	26 (59.1%)
Sex	Female	18 (40.9%)
Socioeconomic status	Lower	15 (34.1%)
Socioeconomic status	Middle	19 (43.2%)
Socioeconomic status	Upper	10 (22.7%)

SD: standard deviation.

Table 2: Presenting symptoms (n=44)

Symptom	N	%
Breathlessness / fast breathing	12	27.3
Edema (facial/pedal)	7	15.9
Poor weight gain / failure to thrive	6	13.6
Cough	5	11.4
Feeding difficulty / poor feeding	4	9.1
Excessive sweating	3	6.8
Palpitations	3	6.8
Easy fatigability	2	4.5
Recurrent respiratory infections	2	4.5

Mean duration of symptoms prior to presentation was 65.9±32 days (median 71 days).

Table 3: Etiological distribution (n=44)

Etiology	N	%
Idiopathic	22	50.0
Myocarditis	12	27.3
Nutritional	6	13.6
Metabolic	4	9.1

Table 4: Clinical severity, investigations, and echocardiography (n=44)

Parameter	Category/Statistic	Value
Family history of cardiomyopathy	Yes	7 (15.9%)
Family history of cardiomyopathy	No	37 (84.1%)
Heart failure class (Ross)	Class I	8 (18.2%)
Heart failure class (Ross)	Class II	13 (29.5%)
Heart failure class (Ross)	Class III	8 (18.2%)
Heart failure class (Ross)	Class IV	15 (34.1%)
Vital signs	Heart rate, mean±SD (beats/min)	138±15.5
Vital signs	Respiratory rate, mean±SD (breaths/min)	36±8.29
Vital signs	SpO2, mean±SD (%)	94.1±3.66
Blood pressure	Systolic, mean±SD (mmHg)	92±12.1
Blood pressure	Diastolic, mean±SD (mmHg)	107±12.1
ECG abnormality	Present	22 (50.0%)
ECG abnormality	Absent	22 (50.0%)
Cardiothoracic ratio	Mean±SD	0.659±0.052
Echocardiography	LVEF, mean±SD (%)	27.7±8.25
Echocardiography	LV dimension, mean±SD (mm)	60±6.81
Echocardiography	RV dysfunction present	23 (52.3%)
ICU admission	Yes	34 (77.3%)
ICU admission	No	10 (22.7%)
Standard therapy	Diuretics + ACEI + beta-blocker	44 (100%)

ACEI: angiotensin-converting enzyme inhibitor; LVEF: left ventricular ejection fraction; LV: left ventricle; RV: right ventricle; SpO2: peripheral oxygen saturation.

Table 5: One-month outcomes (n=44)

Outcome at 1 month	N	%
Improved	17	38.6
Static	15	34.1
Deteriorated	10	22.7
Death	2	4.5

DISCUSSION

This study describes a tertiary-care cohort of children with DCM in Odisha, highlighting delayed presentation, severe ventricular dysfunction, and high intensive care utilization. The mean age (7.16 years) and male predominance are comparable to observations from population-based registries and tertiary-care cohorts.[21-24]

Respiratory symptoms dominated presentation, with breathlessness as the most frequent complaint. The mean pre-diagnosis symptom duration of about two months suggests missed early recognition and potential referral delays. A large proportion presented in Ross class III-IV (52.3%), consistent with the clinical reality that pediatric DCM is frequently recognized only after overt decompensation.[25-28]

Idiopathic DCM was the most common category (50%). Myocarditis accounted for over one-quarter of cases (27.3%), underscoring the importance of considering post-infectious etiologies and the potential for recovery in selected children. Nutritional and metabolic causes together represented 22.7%, emphasizing that treatable etiologies remain relevant and should be actively sought.[29-34]

Echocardiography showed advanced disease (mean LVEF 27.7% with marked LV dilatation), and RV dysfunction was common (52.3%). Despite universal initiation of standard medical therapy, outcomes at one month were heterogeneous: 38.6% improved while 56.8% were static or deteriorated. These findings support close early follow-up and early identification of high-risk children.[35-42]

Limitations include a single-center design, moderate sample size, etiologic classification based on available investigations (with limited genetic/advanced virologic testing), and follow-up restricted to one month.

CONCLUSION

This study demonstrates that pediatric dilated cardiomyopathy in the studied population is characterized by predominant idiopathic etiology, delayed presentation, advanced heart failure, significant ventricular dysfunction, and high requirement for intensive care at diagnosis.

Respiratory distress and heart failure symptoms were the most common presenting features, often persisting for several weeks prior to diagnosis. Echocardiographic findings revealed severe left ventricular systolic dysfunction with marked ventricular dilatation and frequent right ventricular involvement, reflecting advanced myocardial remodeling.

Despite uniform initiation of standard heart failure therapy, early outcomes were heterogeneous, with only a proportion of children showing improvement at one month, while others remained static, deteriorated, or succumbed to the disease.

These findings highlight pediatric dilated cardiomyopathy as a severe, resource-intensive condition with an unpredictable early clinical course. The study emphasize the critical need for early

recognition, timely referral, comprehensive etiological evaluation, and close follow-up, particularly in resource-limited settings. Strengthening early diagnostic pathways and expanding access to specialized pediatric cardiac care may improve outcomes in this vulnerable population

Recommendations

Early suspicion of dilated cardiomyopathy in children with unexplained heart failure, recurrent respiratory symptoms, or cardiomegaly is crucial for improving outcomes as dilated cardiomyopathy is not an uncommon disease in such above scenario.

Prompt echocardiographic evaluation should be performed in all suspected cases, as it is essential for diagnosis, severity assessment, and prognostication.

Efforts should be made to identify potentially reversible etiologies such as myocarditis, nutritional deficiencies, and drug-induced causes.

Early initiation and optimization of standard heart failure therapy (ACE inhibitors, beta-blockers, diuretics, and aldosterone antagonists) is recommended in all patients.

Children presenting with severe ventricular dysfunction require close monitoring, intensive care support, and timely use of inotropes when indicated.

Serial echocardiographic follow-up is essential to monitor ventricular function, remodeling, and disease progression or recovery.

Long-term follow-up with regular counseling of caregivers is necessary, as clinical outcomes vary and late recovery or progression may occur.

REFERENCES

1. Lipshultz SE, Sleeper LA, Towbin JA, et al. The incidence of pediatric cardiomyopathy in two regions of the United States. N Engl J Med. 2003;348(17):1647–1655.
2. Towbin JA, Lowe AM, Colan SD, et al. Incidence, causes, and outcomes of dilated cardiomyopathy in children. JAMA. 2006;296(15):1867–1876.
3. Boucek MM, Waltz DA, Edwards LB, et al. Registry of the International Society for Heart and Lung Transplantation: ninth official pediatric heart transplantation report. J Heart Lung Transplant. 2006;25(8):893–903.
4. Park MK. Dilated cardiomyopathy. In: Park MK, editor. Park's Pediatric Cardiology for Practitioners. 6th ed. Philadelphia: Elsevier; 2014. p. 546–560.
5. Wilkinson JD, Landy DC, Colan SD, et al. The pediatric cardiomyopathy registry and heart failure: key results from the first 15 years. Heart Fail Clin. 2010;6(4):401–413.
6. Hershberger RE, Givertz MM, Ho CY, Judge DP, Kantor PF, McBride KL, et al. Genetic evaluation of cardiomyopathy—a Heart Failure Society of America practice guideline. J Card Fail. 2018;24(5):281–302.
7. Kindermann I, Barth C, Mahfoud F, et al. Update on myocarditis. J Am Coll Cardiol. 2012;59(9):779–792.
8. Reddy KS. Cardiovascular diseases in India. World Health Stat Q. 1993;46(2):101–107.
9. Cox GF, Sleeper LA, Lowe AM, et al. Factors associated with establishing a causal diagnosis for children with cardiomyopathy. Pediatrics. 2006;118(4):1519–1531.
10. Standring S, editor. Gray's Anatomy: The Anatomical Basis of Clinical Practice. 41st ed. London: Elsevier; 2016.
11. Moore KL, Dalley AF, Agur AMR. Clinically Oriented Anatomy. 7th ed. Philadelphia: Wolters Kluwer; 2014.
12. Snell RS. Clinical Anatomy by Regions. 9th ed. Philadelphia: Lippincott Williams & Wilkins; 2012.
13. Anderson RH, Razavi R, Taylor AM. Cardiac anatomy revisited. J Anat. 2004;205(3):159–177.
14. Anderson RH, Ho SY, Becker AE. Anatomy of the human atrioventricular junctions revisited. Anat Rec. 2000;260(1):81–91.
15. Guyton AC, Hall JE. Textbook of Medical Physiology. 13th ed. Philadelphia: Elsevier; 2016.
16. Katz AM. Physiology of the Heart. 5th ed. Philadelphia: Wolters Kluwer; 2011.
17. Zipes DP, Libby P, Bonow RO, Mann DL, Tomaselli GF, editors. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 11th ed. Philadelphia: Elsevier; 2019.
18. Berne RM, Levy MN. Cardiovascular Physiology. 9th ed. Philadelphia: Mosby; 2001.
19. Ganong WF. Review of Medical Physiology. 26th ed. New York: McGraw-Hill; 2018.
20. Opie LH. Mechanisms of cardiac contraction and relaxation. Heart. 2001;86(6):709–714.
21. Towbin JA, Bowles NE. The failing heart. Nature. 2002;415(6868):227–233.
22. Mann DL, Bristow MR. Mechanisms and models in heart failure: the biomechanical model and beyond. Circulation. 2005;111(21):2837–2849.
23. Rutledge J, Bricker JT. Congenital diseases of the heart: clinical-physiological considerations. Tex Heart Inst J. 2001;28(3):237.
24. Lindop G. Pathology of the heart. Surgery (Oxford). 2007;25:189–197.
25. Scott J. Pathophysiology and biochemistry of cardiovascular disease. Curr Opin Genet Dev. 2004;14(3):271–279.
26. Dixon JA, Spinale FG. Pathophysiology of myocardial injury and remodeling: implications for molecular imaging. J Nucl Med. 2010;51(Suppl 1):102S–106S.
27. Li W, Zhu Y, Wang W, He D, Feng L, Li Z. Src tyrosine kinase promotes cardiac remodeling induced by chronic sympathetic activation. Biosci Rep. 2023;43:BSR20231097.
28. Elliott P, Andersson B, Arbustini E, et al. Classification of the cardiomyopathies: a position statement from the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. Eur Heart J. 2008;29(2):270–276.
29. Maron BJ, Towbin JA, Thiene G, et al. Contemporary definitions and classification of the cardiomyopathies: an American Heart Association Scientific Statement. Circulation. 2006;113(14):1807–1816.
30. Weintraub RG, Semsarian C, Macdonald P. Dilated cardiomyopathy. Lancet. 2017;390(10092):400–414.
31. Morales A, Hershberger RE. Genetic evaluation of dilated cardiomyopathy. Curr Cardiol Rep. 2013;15(7):375.
32. Goodwin JF. Cardiomyopathies and specific heart muscle diseases: definitions, terminology, classifications and new and old approaches. Postgrad Med J. 1992;68(Suppl 1):S3–S6.
33. Dungu JN, Langley SG, Hardy-Wallace A, et al. Dilated cardiomyopathy: the role of genetics, highlighted in a family with Filamin C variant. Heart. 2022;108(9):676–682.
34. Wilkinson JD, Landy DC, Colan SD, et al. The pediatric cardiomyopathy registry and heart failure: key results from the first 15 years. Heart Fail Clin. 2010;6(4):401–407.
35. Tsirka AE, Trinkaus K, Chen SC, et al. Improved outcomes of pediatric dilated cardiomyopathy with utilization of heart transplantation. J Am Coll Cardiol. 2004;44(2):391–397.
36. Kothari SS, Dhopeshwarkar RA, Saxena A, Juneja R. Dilated cardiomyopathy in Indian children. Indian Heart J. 2003;55(2):147–151.
37. Patil V, Desai N, Galande C. Clinical and echocardiogram profile of cardiomyopathy at a tertiary care centre. J Cardiovasc Dis Res. 2014;5:34–43.
38. Cohn JN, Ferrari R, Sharpe N. Cardiac remodeling—concepts and clinical implications: a consensus paper from an international forum on cardiac remodeling. J Am Coll Cardiol. 2000;35(3):569–582.
39. Durand JB. Genetic basis of cardiomyopathy. Curr Opin Cardiol. 1999;14(3):225–229.
40. Kemp CD, Conte JV. The pathophysiology of heart failure. Cardiovasc Pathol. 2012;21(5):365–371.
41. Kereiakes DJ, Parmley WW. Myocarditis and cardiomyopathy. Am Heart J. 1984;108(5):1318–1326.
42. Malinow I, Fong DC, Miyamoto M, Badran S, Hong CC. Pediatric dilated cardiomyopathy: a review of current clinical approaches and pathogenesis. Front Pediatr. 2024;12:1404942.