World Journal of Pharmaceutical and Life Sciences
WJPLS
www.wjpls.org
SJIF Impact Factor: 7.409

DILATED CARDIOMYOPATHY IN CHILDREN: A RETROSPECTIVE STUDY OF 25 CASES

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DOI: https://doi.org/10.5281/zenodo.17224954

Article Received on 06/08/2025 **Article Revised on 26/08/2025** **Article Accepted on 15/09/2025**

ABSTRACT

Background: Dilated cardiomyopathy (DCM) is the most common cardiomyopathy in children and represents a major cause of heart failure and an important indication for cardiac transplantation. **Objective:** To describe the epidemiological, clinical, echocardiographic, etiological, and therapeutic characteristics of pediatric DCM cases managed at the Children's Hospital of Rabat. **Methods:** We conducted a retrospective review of medical records of patients aged 1 month to 16 years diagnosed with DCM in the Department of Pediatrics 4 at the Children's Hospital of Rabat, from January to December 2024. Inclusion criteria were left ventricular (LV) dilatation (end-diastolic dimension z-score > +2) associated with systolic dysfunction (ejection fraction and/or fractional shortening z-score < –2). **Results:** Twenty-five patients were included (14 females, 11 males). The mean age at diagnosis was 37.7 months (range 2–100), with 80% diagnosed before 6 years of age. Clinical heart failure was present in 71% of cases, including 27% with stage IV. Etiology was identified in 44%: myocarditis (20%), Duchenne muscular dystrophy (8%), primary carnitine deficiency (4%), anthracycline-induced (4%), and congenital heart disease with associated myocarditis or hemodynamic overload (20%). In 56%, etiology remained idiopathic. Standard heart failure therapy was administered in all patients. Two deaths occurred during hospitalization, while 10 patients improved, 5 were lost to follow-up, and 8 showed progressive LV dysfunction. **Conclusion:** Pediatric DCM is a heterogeneous disease with high morbidity and mortality. In our setting, myocarditis and genetic or metabolic disorders were leading identifiable causes, but more than half remained idiopathic. Multidisciplinary management, genetic counseling, and long-term follow-up are essential.

KEYWORDS: Dilated cardiomyopathy, Children, Heart failure, Myocarditis, Duchenne muscular dystrophy, Morocco.

INTRODUCTION

Cardiomyopathies comprise a heterogeneous group of myocardial disorders that may impair systolic, diastolic, or both ventricular functions.[1] They represent a major cause of pediatric heart failure and are one of the leading indications for cardiac transplantation.[2] Among them, dilated cardiomyopathy (DCM) is the most common form in children, defined by left ventricular (LV) dilatation associated with systolic dysfunction.[3] Distinguishing primary forms, which involve intrinsic defects of the cardiomyocyte, from secondary forms due to extrinsic factors (infectious, toxic, ischemic, hemodynamic) is crucial.[3]

This study aimed to analyze the epidemiological, clinical, echocardiographic, etiological, and therapeutic aspects of pediatric DCM cases managed at the Children's Hospital of Rabat.

MATERIALS AND METHODS

We retrospectively reviewed archived medical records of patients diagnosed with DCM in the Department of Pediatrics 4, Children's Hospital of Rabat, between January and December 2024.

Inclusion criteria

- Age between 1 month and 16 years,
- Diagnosis of DCM based on LV dilatation (end-diastolic dimension z-score > +2) and LV systolic dysfunction (ejection fraction and/or fractional shortening z-score < –2),
- Complete and traceable clinical data.

Exclusion criteria: incomplete records or duplicated cases.

Of 33 initially identified patients, five duplicates and three not meeting criteria were excluded, leaving 25

cases for analysis. Data collected included epidemiology (age, sex, origin), clinical features, biological, electrocardiographic, and radiological findings, echocardiographic parameters, etiology, treatment, and short-term outcome.

RESULTS

A total of 25 patients were included: 14 females (56%) and 11 males (44%).

- **Age at diagnosis:** mean 37.7 months, median 28 months (range: 2–100).
  - 44% before 24 months, 36% between 24–72 months, and 20% after 72 months.
  - 80% were diagnosed before 6 years.

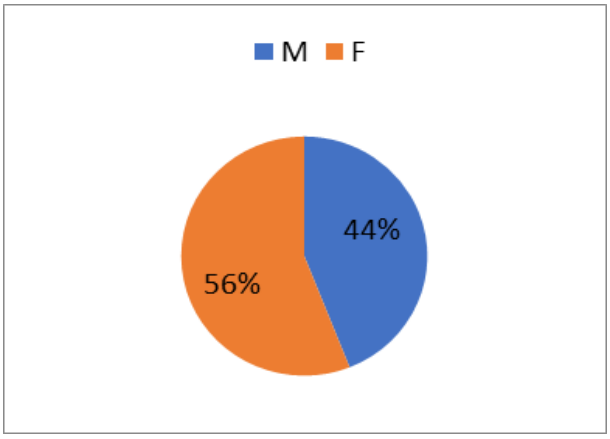

Figure 1: Sex distribution of children with DCM.

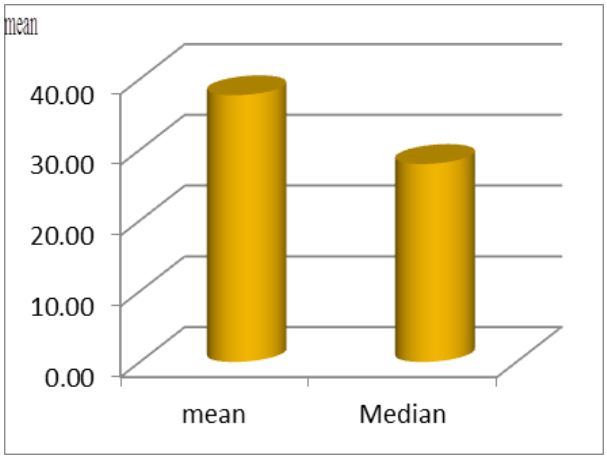

Figure 2: Mean and median age at diagnosis.

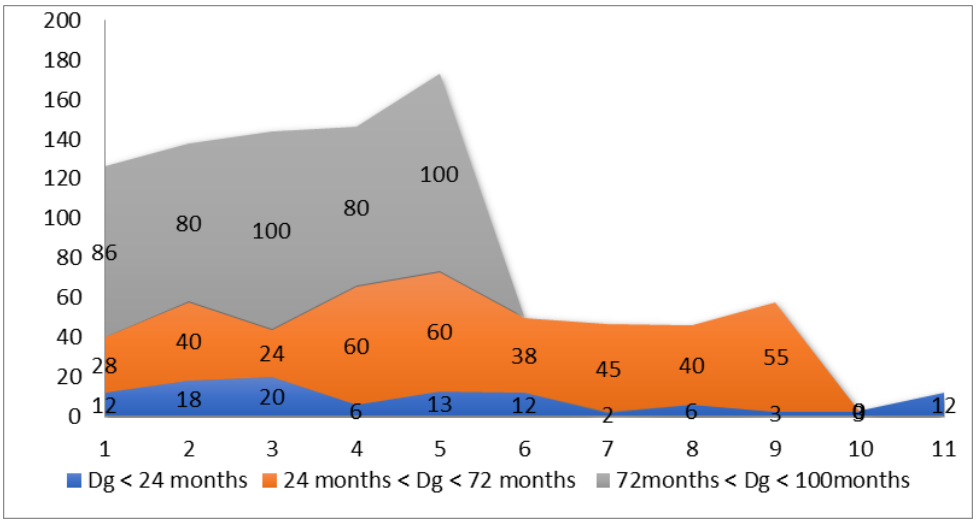

Figure 3: Distribution of cases according to age at diagnosis.

- **Geographic origin:** Rabat–Salé–Kénitra (14 cases), Tanger–Tétouan–Al Hoceïma (5), Fès–Meknès (3), Casablanca–Settat (3).
- **Clinical presentation:** 48% were hospitalized for heart failure, while 52% attended for echocardiographic follow-up. Discovery circumstances were: symptomatic presentation (60%), family screening (20%), incidental finding (16%), and post-cardiac arrest (4%). At diagnosis, 71% had signs of heart failure, including 27% stage IV.

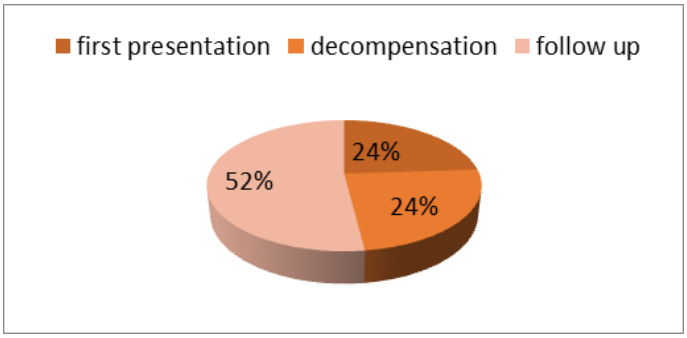

Figure 4: Reasons for consultation during the study period.

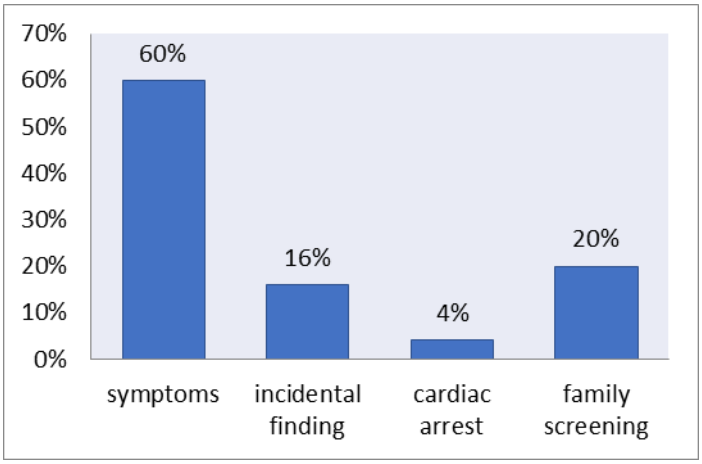

Figure 5: Circumstances of DCM diagnosis.

- **Echocardiography:** Mean LV end-diastolic z-score: 4.1; median EF z-score: –5.4; median FS z-score: –6.0.
- **Etiology:** identified in 11 patients (44%):
  - Myocarditis: 5 (20%),
  - Duchenne muscular dystrophy: 2 (8%),
  - Primary carnitine deficiency: 1 (4%),
  - Anthracycline-induced: 1 (4%),
  - Congenital heart disease with associated myocarditis/hemodynamic overload: 5 (20%).
  - Idiopathic DCM: 14 (56%).

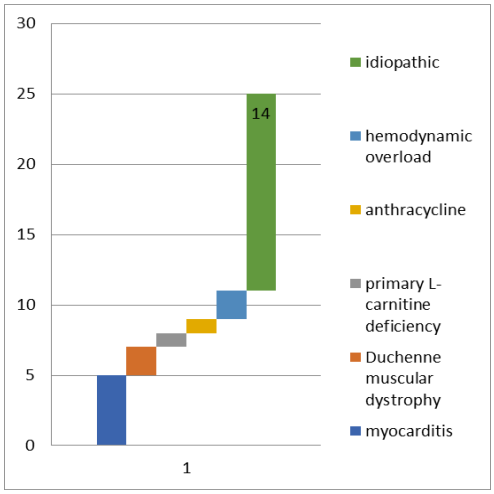

Figure 6: Distribution of DCM etiologies in our series.

- **Treatment:** all received standard heart failure therapy (furosemide n=22, captopril n=21, digoxin n=8, beta-blockers n=5, spironolactone n=7). One patient with cardiogenic shock received dobutamine. Anticoagulation was given to 3 patients with venous thrombosis.
- **Outcome:** 2 deaths during hospitalization; 10 improved with normalized EF within 1 year; 5 lost to follow-up; 8 worsened with recurrent hospitalizations.

DISCUSSION

Cardiomyopathies are heterogeneous disorders that impair myocardial function and can affect both systolic and diastolic performance.[1] DCM is the most common form in pediatrics, a major cause of heart failure, and a leading indication for transplantation.[2,3] It is characterized by LV dilatation with systolic dysfunction.[4–5] Primary forms arise from intrinsic myocardial defects, whereas secondary forms are related to extrinsic insults such as infections, toxins, or overload.[3]

Pediatric DCM is rare but severe, with an estimated prevalence between 0.57 and 1.13 per 100,000 children.[1–6] It predominantly occurs in the first two years of life, with 41% diagnosed before one year.[1–7] Prognosis remains poor, with nearly 40% of affected children requiring transplantation or dying within two years.[1] Management is costly.[8,9] While up to 69% of cases remain idiopathic, genetics play an important role.[10–11]

Diagnosis is based on LV dilatation and systolic dysfunction confirmed by echocardiography.[4] Complementary tools include ECG, cardiac MRI, and sometimes endomyocardial biopsy.[5] MRI is particularly useful for evaluating myocardial fibrosis.[5] BNP and NT-proBNP are helpful biomarkers.[12] Biopsy, although rarely used, can identify specific etiologies, with electron microscopy offering additional insights.[13–14]

Clinically, pediatric DCM is dominated by heart failure, present in over 70% of cases, often with severe forms.[15,16] Symptoms include dyspnea, feeding difficulties, arrhythmias, and occasionally sudden cardiac death. Multiorgan involvement such as renal or hepatic failure and thromboembolic complications may occur.[16]

Etiologies include both secondary and primary causes. Secondary causes encompass pressure/volume overload (valvular disease, coarctation, shunts)[17], coronary anomalies (ALCAPA)[18], arrhythmia-induced cardiomyopathy[3], toxic causes (anthracyclines, radiotherapy)[18–17], and myocarditis.[18–19] Myocarditis is frequent in infants, with viral, bacterial, parasitic, or autoimmune etiologies.[20,21] Primary forms include metabolic diseases (notably primary carnitine deficiency, a reversible cause)[3,18,22], mitochondrial cytopathies[23,24], and genetic forms. Familial/genetic DCM represents up to 20–35% of pediatric cases[2,25], with autosomal dominant, recessive, X-linked, or mitochondrial inheritance.[2] Genes involved include sarcomeric (MYH7, MYBPC3, TNNT2), cytoskeletal (desmin, dystrophin), and nuclear envelope proteins (lamins A/C, emerin).[22–26] Recently, galectin-3 has been implicated in modulating inflammation and fibrosis.[27]

Histologically, DCM is characterized by dilated cavities, fibrotic myocardium, nuclear hypertrophy, and sometimes inflammatory infiltrates, which contribute to remodeling and thromboembolic risk.[28]

Therapy aims to relieve symptoms, slow disease progression, and prevent complications.[29,30] Standard treatment includes diuretics, ACE inhibitors/ARBs/ARNIs, beta-blockers, and mineralocorticoid receptor antagonists.[29–31] Etiology-specific treatments include carnitine supplementation, immunomodulation in myocarditis, and dexrazoxane with anthracyclines.[29,32,31] Arrhythmia management, anticoagulation, and advanced devices (CRT, ICD, mechanical support) are indicated in selected cases.[32,33] Heart transplantation remains the ultimate treatment in refractory cases.[32,33] The American Heart Association staging (A–D) provides guidance for therapeutic stratification.[30–31]

Long-term management requires regular follow-up with clinical exams, ECG, echocardiography, Holter monitoring, exercise testing, and biomarker assessment.[34] Family screening is essential in idiopathic or familial forms.[35–36] Identifying causative mutations supports genetic counseling and targeted screening, especially for high-risk genes such as LMNA.[35–36,32,33] Psychosocial support for families also plays a crucial role.[34]

CONCLUSION

Pediatric DCM is a rare but severe condition with high morbidity and mortality. Despite advances in diagnostics, many cases remain idiopathic. Our findings highlight the predominance of myocarditis, genetic, and metabolic etiologies in Morocco. Comprehensive management combining optimized medical therapy, etiological treatment, family screening, and genetic counseling is essential. Early referral to specialized centers and long-term follow-up can improve outcomes.

ACKNOWLEDGMENTS

The authors thank all the staff of the Department of Pediatrics 4, Children's Hospital of Rabat, and the patients' families for their cooperation.

Conflict of Interest

The authors declare no conflict of interest.

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