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REVIEW

The effects of glucocorticoids on cardiac function of patients with Duchenne muscular dystrophy: benefit or not?

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Abstract

Duchenne muscular dystrophy (DMD) is a progressive, incurable X-linked neuromuscular disease caused by mutations in the dystrophin gene, resulting in functional dystrophin deficiency. Currently, cardiovascular complications are the leading cause of death in patients with DMD. Glucocorticoids are considered the gold standard treatment for children with DMD. Long-term glucocorticoid therapy can delay the loss of independent ambulation, improve lung function, and extend lifespan. However, the effects of glucocorticoids on cardiac function in patients with DMD remain controversial. This scoping review aims to summarize and analyze published clinical studies investigating the effects of glucocorticoids on cardiac function in children with DMD. A comprehensive search was conducted using PubMed, Web of Science, and Embase databases with relevant search terms. Abstracts and full texts of retrieved studies were reviewed. The studies were categorized into four themes: glucocorticoid use, Types of glucocorticoids, administration methods, and timing of glucocorticoid initiation. A total of 21 studies were included. Of these, 18 studies investigated the effects of glucocorticoids on cardiac function in patients with DMD, and the study of Koeks et al. reported both effective and non-effective outcomes of glucocorticoids on cardiac function stratified by age group, respectively. One study examined the impact of different glucocorticoid types, one study assessed the effects of glucocorticoid administration methods and one study evaluated the timing of glucocorticoid initiation. Among the 21 studies, 13 studies ($n=1814$ patients) indicated that glucocorticoids could delay the progression of cardiac dysfunction in patients with DMD. Six studies ($n=6294$ patients) reported no significant effects of glucocorticoids on cardiac function, while one study ($n=111$ patients) suggested that early glucocorticoid therapy increased the risk of cardiomyopathy. *Conclusion*: It has been suggested that corticoids may delay the deterioration of cardiac function in patients with DMD. However, limited data exist on the long-term effects of early glucocorticoid therapy on cardiac function, leading to inconclusive findings. Prospective longitudinal studies are needed to determine the optimal timing, dose regimen, and long-term impact of glucocorticoid therapy in patients with DMD.

What is Known:
• *The effects of glucocorticoids on cardiac function in patients with DMD remain controversial.*

What is New:
• *Glucocorticoids can delay the deterioration of cardiac function in DMD patients. However, prospective longitudinal studies are still needed to determine the optimal timing, dose regimen, and long-term effect of glucocorticoid therapy in DMD patients.*

Keywords Duchenne muscular dystrophy · Cardiac function · Glucocorticoids · Effect · Review

Abbreviations

DMD	Duchenne muscular dystrophy
BMD	Becker muscular dystrophy
EF, LVEF	Left ventricular ejection fraction
FS	Fractional shortening
EDVI	End diastolic volume index
EDVI	EDV index
ESVI	ESV index
99 Tcm-MIBI G-MPI	99Tcm-MIBI gated myocardial perfusion imaging (G-MPI)
BNP	Brain natriuretic peptide
LGE	Late gadolinium enhancement

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Extended author information available on the last page of the article

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ECG	Electrocardiogram
MR	Mineralocorticoid receptor
GILZ	Glucocorticoid-induced leucine zipper

Introduction

Duchenne muscular dystrophy (DMD) is a progressive, incurable X-linked neuromuscular disorder caused by mutations in the dystrophin gene, resulting in a lack of functional dystrophin. It affects approximately 1 in 3600 to 6000 male births [1]. DMD causes progressive muscle weakness, motor delays, loss of ambulation, respiratory dysfunction, and cardiomyopathy due to ongoing muscle damage and degeneration. Without intervention, affected individuals typically exhibit abnormal gait by the age of four, require long-term use of a wheelchair or bed rest by approximately 11 years of age, develop cardiomyopathy around 16 years of age, and succumb to circulatory or respiratory failure by the age of 20 [2–4]. However, advancements in multidisciplinary care, assisted ventilation, and early steroid administration have extended the life expectancy of patients with DMD into their late 20 s or early 30 s. Despite these advances, cardiovascular complications account for 40–50% of DMD-related deaths [5–7], posing the greatest threat to long-term survival. In children with DMD, dystrophin dysfunction or loss in myocardial tissue compromises the stability of the myocardial cell membrane, leading to chronic inflammation. Consequently, impaired contractility, fiber necrosis, and limited regenerative capacity contribute to irreversible myocardial dysfunction. early diagnosis and intervention targeting cardiac involvement may slow disease progression and improve outcomes for children with DMD.

Currently, glucocorticoids are the cornerstone therapy for children with DMD [1, 8]. Commonly used glucocorticoids include Prednisone, prednisolone, and deflazacort. Based on the literature, prednisone therapy typically begins at 0.75 mg/kg/day and deflazacort at 0.9 mg/kg/day, with doses adjusted for weight gain and declining motor function. Although the exact mechanisms of glucocorticoids in DMD treatment remain unclear, they are believed to activate target genes such as IκBα, annexin 1, interleukin- 10, and glucocorticoid-induced leucine zipper, thereby exerting broad-spectrum anti-inflammatory effects and reducing muscle fibrosis progression in children with DMD [8–11]. long-term glucocorticoid therapy has been shown to delay the loss of independent ambulation [12–17], improve cardiopulmonary capacity [18], and extend life expectancy [2]. Reflecting these findings, the DMD Care Considerations Working Group recommends initiating glucocorticoid therapy during the motor plateau phase (4–8 years of age) [1].

However, the effects of glucocorticoids on cardiac function in patients with DMD remain controversial. Some studies have suggested that glucocorticoids may protect left ventricular function [7, 19–27], reduce the occurrence of cardiomyopathy, and lower all-cause mortality in children with DMD. Conversely, other studies, including animal models, have reported that glucocorticoid therapy does not significantly improve cardiac function in children with DMD [28, 29], with early use potentially increasing the risk of cardiomyopathy [30]. This increased risk may be attributable to glucocorticoid side effects, such as obesity and hypertension, differences in patient inclusion and exclusion criteria as well as variations in glucocorticoid types, dose regimen, duration of use, length of follow-up, assessed endpoints may explain these inconsistent findings. Consequently, we reviewed the current literature on glucocorticoid use in DMD, aiming to identify relevant studies, highlight potential research questions, and inform the design of future studies to improve cardiac outcomes in children with DMD.

Methods

This study is a scoping review. Final search terms were determined after conducting an initial broad search in PubMed to identify MeSH headings and free-text keywords relevant to the topic. We searched three electronic databases (PubMed, Web of Science, and Embase) and DMD-related websites for articles published between 2000 and 2024. Both MeSH terms and free-text keywords were utilized. Table 1 outlines the inclusion criteria and PubMed search strategy. Equivalent search strings were applied to other databases. Publication date, patient age, sample size, glucocorticoid type, initiation age, duration of use, study design, and medical techniques for evaluating cardiac function were carefully extracted and reviewed by two colleagues. In cases of discrepancies between them, two senior researchers at our center were consulted.

Results

Overview of included studies

Initially, 234 scientific articles were identified. After removing duplicates, 92 articles remained. A further 62 articles were excluded based on the evaluation of their titles and abstracts, and 9 were excluded following a full-text review. Ultimately, 21 articles were included in the present review. The study selection process is illustrated in Fig. 1. Relevant findings from selected studies are summarized in Tables 2, 3, 4, 5, and 6.

Table 1 Inclusion criteria, exclusion criteria and search strategy

Inclusion criteria	• Written in English • Cross-sectional, retrospective or prospective studies on the effect of glucocorticoids on cardiac function in children with DMD • Study date 2000 to 2024
Exclusion criteria	• Review articles, case reports, conference reports, letters, editorial comments and opinions • The study population was less than 10
Search strategy in PubMed	((Duchenne muscular dystrophy[Title/Abstract]) OR (DMD[Title/Abstract])) AND ((((((((((Cardiac[Title/Abstract]) OR (Cardiac function[Title/Abstract])) OR (heart[Title/Abstract])) OR (left ventricular function[Title/Abstract])) OR (left ventricular systolic function[Title/Abstract])) OR (ventricular dysfunction[Title/Abstract])) OR (cardiomyopathy[Title/Abstract])) OR (myocardial fibrosis[Title/Abstract])) OR (fractional shortening[Title/Abstract])) OR (ejection fraction[Title/Abstract])) OR (EF[Title/Abstract])) OR (FS[Title/Abstract])) AND (((((((((Glucocorticoids[Title/Abstract]) OR (Corticosteroids[Title/Abstract])) OR (deflazacort[Title/Abstract])) OR (Prednisone[Title/Abstract])) OR (Prednisolone[Title/Abstract])) OR (steroid[Title/Abstract])) OR (CSs[Title/Abstract])) OR (GC[Title/Abstract]))

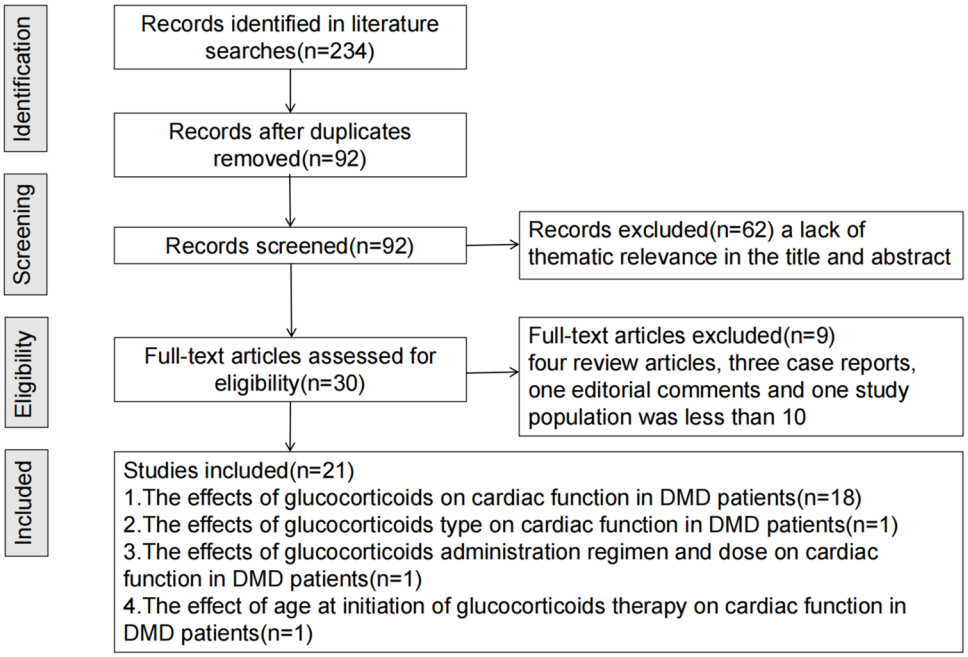

Fig. 1 Screening flowchart of the study

Effects of glucocorticoids on cardiac function in patients with DMD (*n* = 18 studies, *n* = 8108 patients)

Positive findings of glucocorticoids therapy on cardiac function in children with DMD (n = 13 studies, n = 1814 patients)

The primary glucocorticoids currently used in DMD management are prednisone, prednisolone, and deflazacort. Four studies involving 223 patients have reported that deflazacort is effective in delaying cardiac dysfunction in patients with DMD. Silversides et al. [19] conducted a retrospective cohort study in Canada, enrolling 33 patients with DMD to examine the effects of deflazacort on left ventricular function. Of these, 21 patients were treated with deflazacort at an initial dose of 0.9 mg/kg/day. The average age of treatment initiation was 8.4 ± 2.4 years, with an average treatment duration of 5.1 ± 2.4 years. The study found that patients treated with deflazacort had better fractional shortening (FS, 33 ± 7% vs. 21 ± 8%; $p = 0.002$) and smaller left ventricular end-systolic dimensions (30 ± 6 mm vs. 37 ± 8 mm; $p = 0.02$), with a mean age of 15 years at the last follow-up [19]. Biggar et al. [20] also performed a retrospective study on patients with DMD in Canada. Among the 77 patients included, 40 received deflazacort at an initial dose of 0.9 mg/kg/day. The average age at treatment initiation was 7.7 ± 1.2 years, with an average treatment duration of 5.5 years. comprehensive cardiac evaluations were conducted every 12–24 months. The study found that 10% (4/40) of

Table 2 The positive exploration of glucocorticoids therapy on cardiac function in children with DMD

Author Year	Study design/ Number and grouping of DMD patients	Glucocorticoids type and dose	Mean age of initiating glucocorticoids	Glucocorticoids duration	Mean age at study time or end of follow-up	Other cardiovascular drugs	Outcome variable/s	Results	Summative assessment	Height, Weight, BMI, SBP and DPB after treatment
Silversides 2003	Retrospective/ Treated $n=21$ Untreated $n=12$	The initial dose of deflazacort was 0.9 mg/kg/day. At 10 years, the mean dose of deflazacort was 0.76 ±0.19 mg/kg/day; at 15 years 0.61 ±0.20 mg/kg/day; and at 18 years 0.59 ±0.15 mg/kg/day	8.4 ±2.4 years	5.1 ±2.4 years	Receiving deflazacort: 14 ±2 years Not receiving deflazacort: 16±2 years	ACEI or Digoxin	FS, LVESD	Patients receiving deflazacort had better FS and smaller LVESD	After 3 years' follow-up 21 exposed: 6 had LVEF <45%, among them, 2 had HF. 25 still could walk at 12 years old 12 not exposed: 7 had cardiomyopathy, whereas all had stopped walking at the end follow-up	Patients receiving deflazacort had lower SBP(112 ±5 mmHg vs. 106 ±7, $p=0.04$) and shorter height (160 ±8 cm vs. 137 ±9 cm, $p=0.05$)
Biggar 2006	Retrospective/ Treated $n=40$ Untreated $n=34$	The initial dose of deflazacort was 0.9 mg/kg/day. By 10 years of age, the mean dose was 0.8 ±0.18 mg/kg per day, by 15 years it was 0.55 ±0.09 and by 18 years 0.5 ±0.2 mg/kg per day	7.7 ±1.2 years	5.5 years	Receiving deflazacort 15.2 ±2.7 years Not receiving deflazacort 15.2 ±2.5 years	-	FS, EF	Patients receiving deflazacort had a lower incidence of EF <45% and better FS	After 14 years' follow-up 40 exposed: 4 had LVEF <45% at 18 years old, 2 died of HF at 13 and 18 years old 34 not exposed: 20 had LVEF <45% at 18 years old, 12 died (unknown reason). All could not walk at 12 years old	Patients receiving deflazacort had lower height, but weight, SBP, DBP and blood sugar were within normal limits

Table 2 (continued)

Author Year	Study design/ Number and grouping of DMD patients	Glucocorticoids type and dose	Mean age of initiating glucocorticoids	Glucocorticoids duration	Mean age at study time or end of follow-up	Other cardiovascular drugs	Outcome variable/s	Results	Summative assessment	Height, Weight, BMI, SBP and DPB after treatment
Houde 2008	Retrospective/ Treated $n=37$ Untreated $n=42$	Deflazacort was begun at 0.9 mg/kg and was adjusted according to evolution or side effects to a maximum of 1 mg/kg. Mean dose at the most recent this study was 0.69 ± 0.2 mg/kg	7.6 ± 1.7 years	5.5 years	Treated 13.1 ± 3.2 years Untreated 14.5 ± 3.8 years	ACEI	EF, FS	The treated group had higher FS and EF as well as a lower incidence of dilated cardiomyopathy	At the 18 years old 37 exposed: 12 could not walk, 12 boys were cardiomyopathy 42 not exposed: 32 could not walk, 28 boys were cardiomyopathy	Patients receiving deflazacort had lower height and higher obesity rates. There is no difference in SBP and DBP between groups
Markham 2008	Retrospective/ Treated $n=14$ Untreated $n=23$	Prednisone (0.75 mg/kg/day) and Deflazacort (0.9 mg/kg/day)	7.5 ± 0.7 years	4.1 years	Treated 12.0 ± 0.4 years Untreated 12.0 ± 0.6 years	-	FS	Glucocorticoid treatment retards the anticipated development of ventricular dysfunction	After follow-up (12 ± 0.7 years) 14 exposed: 2 had ventricular dysfunction 23 not exposed, 16 had ventricular dysfunction	There is no difference in height, weight, BMI, SBP and DBP between two groups
Mavrogeni 2009	Prospective/ Treated $n=17$ Untreated $n=17$	Prednisone (0.75 mg/kg/day for 10 consecutive days per month) and Deflazacort (0.9 mg/kg/day)	7 years (median age)	7–14 years	Treated 17–22 years Untreated 12–15 years	-	MRI: T2 relaxation time of the myocardium, LVEDV, LVESV and LVEF	Children on deflazacort had better left ventricular systolic function	After 7 years' follow-up 17 exposed: no detailed number documented 17 not exposed: no detailed number documented	-

Table 2 (continued)

Author Year	Study design/ Number and grouping of DMD patients	Glucocorticoids type and dose	Mean age of initiating glucocorticoids	Glucocorticoids duration	Mean age at study time or end of follow-up	Other cardiovascular drugs	Outcome variable/s	Results	Summative assessment	Height, Weight, BMI, SBP and DPB after treatment
Barber 2013	Retrospective/ Treated $n=291$ Untreated $n=171$	Prednisone, Prednisolone, Deflazacort	7.4 ± 2.5 years	4.1 ± 3.4 years	-	-	FS, EF	For every year of glucocorticoid treatment, the probability of developing cardiomyopathy decreased by 4%	At the 14.3 years old 291 exposed: 104 boys developed cardiomyopathy 171 not exposed: 107 boys developed cardiomyopathy	-
Schram 2013	Retrospective/ Treated $n=63$ Untreated $n=23$	Prednisone (ranging from 0.5 to 0.75 mg/kg/day) or Deflazacort (0.9 mg/kg/day)	8.6 ± 3.5 years	-	Treated 19.8 ± 5.5 years Untreated 22.1 ± 5.8 years	ACEI ARB Blocker Digoxin Diuretic	TTE: LVEF, LVSF LVEDD, LVESD	Glucocorticoid therapy is associated with a substantial reduction in all-cause mortality and new-onset and progressive cardiomyopathy	At last follow-up 63 exposed: 7 develop cardiomyopathy, 7 died (unknown reason), 27 had normal LVEF, none developed HF 23 not exposed: 14 develop cardiomyopathy, 5 develop HF, 10 died (unknown reason), 3 had normal LVEF	The glucocorticoid treated group had a significantly lower height, a higher BMI

Table 2 (continued)

Author Year	Study design/ Number and grouping of DMD patients	Glucocorticoids type and dose	Mean age of initiating glucocorticoids	Glucocorticoids duration	Mean age at study time or end of follow-up	Other cardiovascular drugs	Outcome variable/s	Results	Summative assessment	Height, Weight, BMI, SBP and DPB after treatment
Hussein 2014	Self before and after comparison Treated $n = 17$	Prednisone (5 mg/kg/day, twice weekly for 3 months)	3–14 years	-	0.25 years	-	Echo: FS	Three months of intermittent prednisone therapy could improve cardiac function in congenital muscular dystrophy	At last follow-up 17 before exposed, 2 had systolic dysfunction, 9 were affected in motor function 17 after exposed, the 2 showed normalization of the systolic function, 6 were elevated in motor function	-
li Zhang 2015	Self before and after comparison Treated $n = 70$	-	7–10 years	2 years	9–12 years	-	99 Tcm-MIBI G-MPI: SRS	Cardiac function was greatly protected after 2 years of glucocorticoid treatment	After 2 years' follow-up 70 before exposed: no detailed number documented 43 after exposed (other 27 did not record): no detailed number documented	-

Table 2 (continued)

Author Year	Study design/ Number and grouping of DMD patients	Glucocorticoids type and dose	Mean age of initiating glucocorticoids	Glucocorticoids duration	Mean age at study time or end of follow-up	Other cardiovascular drugs	Outcome variable/s	Results	Summative assessment	Height, Weight, BMI, SBP and DPB after treatment
Tandon 2015	Retrospective/ Treated $n=95$ Untreated $n=3$	Prednisone or/ and Deflazacort	7.0 ± 2.5 years	7.6 ± 3.4 years	15.2 ± 4.0 years	-	CMR: LGE + LV segments	Longer steroid treatment duration was associated with a lower age-related increase in myocardial fibrosis burden	At last follow-up 95 exposed and 3 unexposed: 4 died (unknown reason); 1 had non-sustained ventricular tachycardia 1 had atrial fibrillation 8 had non-sustained atrial tachycardia	-
Koeks 2017	Retrospective/ Treated $n=74$ Untread $n=381$ Past treated $n=114$	-	-	-	> 20 years	-	Echo: EF	Cardiomyopathy is less common in DMD patients over 20 treated with glucocorticoids	At last follow-up 74 exposed: 42 developed cardiomyopathy 381 unexposed: 229 developed cardiomyopathy 114 exposed past: 71 developed cardiomyopathy	-
Wittlieb 2020	Retrospective/ Treated $n=319$ Untreated $n=89$	-	-	-	-	ACEI, Beta-blocker, Aldosterone antagonist, Digoxin	Echo: LVEF, FS CMR: LVEF, MDE Cardiac Biomarkers: NT-proBNP, BNP	Glucocorticoid use was lower for those dying of cardiac causes compared to those living	At last follow-up 319 exposed: 2 died of cardiac events 89 unexposed: 6 died of cardiac events	-

Table 2 (continued)

Author Year	Study design/ Number and grouping of DMD patients	Glucocorticoids type and dose	Mean age of initiating glucocorticoids	Glucocorticoids duration	Mean age at study time or end of follow-up	Other cardiovascular drugs	Outcome variable/s	Results	Summative assessment	Height, Weight, BMI, SBP and DPB after treatment
Schiava 2024	Retrospective/ Prednisone/ Prednisolone $n=46$ Deflazacort $n=38$ Untreated $n=28$	Prednisone Prednisolone Deflazacort	7.0 ± 2.3 years	-	23.4 ± 5.2 years	ACEI, ARB	LVEF	For DMD patients who loss of ambulation individuals on glucocorticoids had lower odds of a LVEF < 50% compared to glucocorticoid-naïve individuals;	During the study period: 46 exposed prednisone: 20 reached LVEF < 50% after loss of ambulation and 7 died at late stage 38 exposed deflazacort: 28 reached LVEF < 50% after loss of ambulation and 16 died at late stage 28 unexposed:16 reached LVEF < 50% after loss of ambulation and 24 died at late stage	Overweight is one of the most frequent side effects

Abbreviation: *ACEI*, angiotensin-converting enzyme inhibitor; *FS*, fractional shortening; *LVESD*, left ventricular end-systolic dimensions; *EF*, ejection fraction; *MRI*, magnetic resonance imaging; *BP*, blood pressure; *ARB*, angiotensin receptor blocker; *LVEDD*, left ventricular end diastolic dimension; *LVMi*, left ventricular mass indexed; *SF*, shortening fraction; *mWS*, meridional wall stress; *VCFc*, velocity of circumferential fiber shortening; *VCFdiff*, velocity of circumferential fiber shortening for the given wall stress; *LVEDD*, left ventricular end-diastolic dimension; *99 Tcm-MIBI G-MPI*, 99 Tcm-MIBI gated myocardial perfusion imaging (G-MPI); *SRs*, summed rest score; *EDVi*, end-diastolic volume index; *ESVi*, end-systolic volume index; *CMR*, cardiac magnetic resonance; *LGE*, late gadolinium enhancement; *LGE+*, LGE positive; *LVEF*, left ventricular; *MDE*, myocardial delayed enhancement; *BNP*, brain natriuretic peptide; *NT-proBNP*, N-terminal pro-brain natriuretic peptide; *HF*, heart failure

Table 3 Glucocorticoids therapy has no effect on cardiac function in children with DMD

Author Year	Study design/ Number and grouping of DMD patients	Glucocorticoids type and dose	Mean age of initiating glucocorticoid	Glucocorticoid duration	Mean age at study time or end of follow-up	Other cardiovascular drugs	Outcome variable/s	Results	Summative assessment	Height, weight, BMI, SBP and DPB after treatment
Sheffali 2005	Retrospective/ Treated $n = 24$ Untreated $n = 6$	Prednisolone treatment (0.75 mg/kg) for ten days in each month and 20 days holiday	-	-	10.1 (± 2.3) years	Digoxin, ACEI	Chest x-ray for cardiomegaly 12 lead ECG, echocardiography	No significant association between steroid therapy and cardiac involvement	24 exposed: 22 had EF < 55%, 5 had EF < 50%, 1 died of HF at 9 months of follow-up 6 not exposed: not detailed	-
Kirchmann 2005	Cross-sectional/ Treated $n = 17$ Untreated $n = 25$	Prednisone, Deflazacort	-	-	5.0–22.5 years (median, 12 years)	ACEI, Digoxin, Diuretics, Antiarrhythmics	Echo: FS, ECG, Holter ECG	No differences in any of the cardiac parameters were observed between those patients	17 exposed: 3 developed severe cardiomyopathy and 2 died from respiratory disease at 18 and 20 years old 25 not exposed: not detailed	-
Spurney 2014	Multi-center cross-sectional/ Treated $n = 139$ Untreated $n = 35$	-	-	The majority > 1 year	-	ACEI, Blocker, Digoxin	EF, FS	Glucocorticoid treatment did not significantly impact the presence or absence of cardiomyopathy	139 exposed: 132 with cardiomyopathy had oral glucocorticoid treatment for greater than 3 months 35 not exposed: not detailed	-
Koeks 2017	Multi-center cross-sectional/ Treated $n = 2658$ Untreated $n = 2015$ Past treated $n = 522$	-	-	-	-	-	Echo: EF	Until the age of 20 No significant Effect of glucocorticoid on the development of cardiomyopathy was observed	Not detailed number for disease progression or keeping stable	Until the age of 20 no trend for a negative effect of the use of glucocorticoid was seen

Table 3 (continued)

Author Year	Study design/ Number and grouping of DMD patients	Glucocorticoids type and dose	Mean age of initiating glucocorticoid	Glucocorticoid duration	Mean age at study time or end of follow-up	Other cardiovascular drugs	Outcome variable/s	Results	Summative assessment	Height, weight, BMI, SBP and DPB after treatment
Butterfield 2021	Prospective study (retrospectively identified and prospectively followed) Stopped use within 1 year of LOA: $n=104$ Use > 1 y beyond LOA: $n=101$ None = 193	-	-	Stopped use within 1 year of LOA:3.69 ±2.95 years Use > 1 y beyond LOA:8.73 ±4.44 years	-	-	EF, SF	Left ventricular dysfunction onset of median age was not significantly different between the three groups	At last follow-up beyond LOA 334 enrolled analysis 88 exposed: 48 had abnormal LV function 155 not exposed: 86 had abnormal LV function 91 past exposed: 41 had abnormal LV function	-
Conway 2024	Retrospective/ Treated $n=227$ Untreated $n=228$	-	7 years	5.5 years	13.8 years	ACEI, ARB, Blocker, MRA	Echo: EF, SF	Compared to no treatment, only continuous corticosteroids were not cardioprotective	Not detailed number for disease progression or keeping stable	-

Abbreviation: *ACEI*, angiotensin-converting enzyme inhibitor; *Echo*, echocardiography; *EF*, ejection fraction; *SF*, shortening fraction; *ECG*, electrocardiogram; *LOA*, loss of ambulation; *HF*, heart failure

Table 4 Effects of glucocorticoid type on cardiac function in patients with DMD

Author Year	Study design	Number and grouping of DMD patients	Glucocorticoids type and dose	Mean age of initiating glucocorticoid	Glucocorticoid duration	Mean age at study time or end of follow-up	Other cardiovascular drugs	Outcome variable/s	Results	Height, Weight, BMI, SBP and DPB after treatment
Markham 2005	Retrospective study	Treated $n=48$ Untreated $n=63$	Prednisone, Deflazacort	6.7 ± 2.5 years	3 ± 2.5 years	Treated 11 ± 4 years Untreated 12 ± 5 years	-	LVFS, LVEDD	Glucocorticoid therapy can delay the decline of FS, which is independent of glucocorticoid type and the effect goes beyond the treatment time	Patients receiving glucocorticoid had lower height. There was no difference in weight, SBP and DBP between groups

Abbreviations: *LVFS*, left ventricular fractional shortening; *LVEDD*, left ventricular end diastolic dimension; *FS*, fractional shortening

Table 5 Effects of glucocorticoids administration regimens and dose on cardiac function in patients with DMD

Author Year	Study design	Number and grouping of DMD patients	Glucocorticoids type and dose	Mean age of initiating glucocorticoid	Glucocorticoid duration	Mean age at study time or end of follow-up	Other cardiovascular drugs	Outcome variable/s	Results	Height, weight, BMI, SBP and DPB after treatment
Trucco 2020	Single site retrospective longitudinal study	Treated $n=208$ (glucocorticoids-daily $n=52$ glucocorticoids-intermittent $n=156$) Untreated $n=21$	Prednisolone (0.9 mg/kg), Deflazacort (0.75 mg/kg)	6.3 ± 1.8	-	11.9 ± 4.2	ACEI, ARB	FS	Glucocorticoids irrespective of their regimen significantly delayed cardiomyopathy	-

Abbreviaion: *ACEI*, angiotensin-converting enzyme inhibitor; *ARB*, angiotensin receptor blocker; *FS*, fractional shortening

Table 6 Effect of age at initiation of glucocorticoid therapy on cardiac function in patients with DMD

Author Year	Study design	Number and grouping of DMD patients	Glucocorticoids type and dose	Mean age of initiating glucocorticoid	Glucocorticoid duration	Mean age at study time or end of follow-up	Other cardiovascular drugs	Outcome variable/s	Results	Height, Weight, BMI, SBP and DPB after treatment
Kim 2017	Multicenter observation study	Early treated $n = 47$ Late treated $n = 218$ Untreated $n = 216$	Deflazacort, prednisone, prednisolone	Early treated 4.2 years Late treated 7.6 years	5.9–6.4 years (there is no difference between early and late groups)	Early treated 15.5 years Late treated 18.0 years Untreated 16.8 years	-	FS, EF	DMD initiated corticosteroid treatment in early childhood had a higher risk of earlier onset cardiomyopathy	-

Abbreviation: *FS*, fractional shortening; *EF*, ejection fraction

treated patients exhibited moderate or severe left ventricular systolic failure (ejection fraction [EF] < 45%), compared to 58% (20/34) of untreated patients. Additionally, treated patients demonstrated a mean FS of 33 ± 7% compared to 21 ± 8% in untreated patients ($p < 0.002$) [20]. Subsequently, Houde et al. (Canada) conducted an 8-year follow-up study on patients with DMD treated with deflazacort in 2008. Similar to the studies mentioned above, deflazacort was initiated at a dose of 0.9 mg/kg/day, although the protocol for dose adjustment in patients over 10 years of age was not clearly described. The study included 37 treated individuals with an average age of 7.6 ± 1.7 years at treatment initiation and a mean treatment duration of 5.5 years. Follow-up data indicated that the treated group demonstrated better left ventricular ejection fraction (LVEF; 52.9 ± 6.3% vs. 46 ± 10%), (FS; 30.8 ± 4.5% vs. 26.6 ± 5.7%, $p < 0.05$), and a lower prevalence of dilated cardiomyopathy (34% vs. 58%) compared to the untreated group [21]. Additionally, Mavrogeni et al. [22] conducted a study in Greece involving 17 patients with DMD (aged 17–22 years) who had been taking deflazacort at 0.9 mg/kg/day for at least 7 years and 17 sex-matched patients with DMD (aged 12–15 years) who had not received any medication. Cardiac function was assessed using magnetic resonance imaging (MRI). The study found that the treated group exhibited higher T2 relaxation time values of the heart (47 ms vs. 33 ms, $p < 0.001$) and better left ventricular systolic function (LVEF, median [range]: 53% [51–57] vs. 48% [42–51], $p < 0.001$) compared to the untreated group [22].

Several studies have demonstrated that glucocorticoids, regardless of type, can improve the cardiac function of patients with DMD. In 2008, Markham et al. conducted a study involving 23 untreated patients with DMD and 14 glucocorticoid-treated patients with DMD who underwent baseline cardiac evaluations between 1998 and 2006 in USA. The study aimed to determine whether initiating steroid treatment prior to the onset of ventricular dysfunction could preserve cardiac function. Glucocorticoid therapy (prednisone 0.75 mg/kg/day or deflazacort 0.9 mg/kg/day) was initiated at a mean age of 7.5 ± 0.7 years, with patients reaching 12 ± 0.7 years at the final follow-up. Results indicated that 93% of treated patients did not develop ventricular dysfunction, compared to 53% of untreated patients after 1500 days [23]. In 2013, Barber et al. analyzed data from 462 patients with DMD across five sites in USA. Of these, 291 patients received glucocorticoid therapy with prednisone, prednisolone, or deflazacort. The average age of treatment initiation was 7.4 ± 2.5 years, with an average therapy duration of 4.1 ± 3.4 years. The study concluded that glucocorticoids delayed the onset of cardiomyopathy and reduced the risk of developing cardiomyopathy by 4% per year among treated patients [24]. In 2013, Schram et al. also conducted a single-center retrospective observational

cohort study involving 86 patients with DMD in Canada. Sixty-three patients received glucocorticoid therapy (prednisone 0.5–0.75 mg/kg/day or deflazacort 0.9 mg/kg/day), with an average treatment initiation age of 8.6 ± 3.5 years and a mean treatment duration of 11.0 ± 4.8 years. Glucocorticoid therapy resulted in a slower decline in (LVEF; − 0.43% per year vs. − 1.09% per year, $p = 0.0101$) and (FS; − 0.32% per year vs. − 0.65% per year, $p = 0.0025$). Additionally, the treated group exhibited less pronounced left ventricular end-diastolic enlargement (+ 0.47 mm per year vs. + 0.92 mm per year, $p = 0.0105$). Patients receiving steroid therapy also showed significantly higher survival rates at 10 years (98% vs. 72.1%) and 15 years (78.6% vs. 27.9%; $p = 0.0005$) compared to those who did not receive steroids. Furthermore, the study also found a lower mortality rate (HR: 0.24; 95% CI: 0.07–0.91; $p = 0.0351$), fewer heart failure-related deaths (0% vs. 22%; $p = 0.0010$), and a lower incidence of new-onset cardiomyopathy (HR: 0.38; 95% CI: 0.16–0.90; $p = 0.0270$) among patients receiving steroid therapy [25]. One year later, Hussein et al. (Egypt) conducted a prospective self-before-and-after comparison study involving 17 patients with DMD aged 3–14 years who were administered prednisone (5 mg/kg/day, twice weekly for three months). After treatment, FS (32 ± 8.6% vs. 36.8 ± 6.8%) and left ventricular mass (88.4 ± 44.6 g vs. 111.6 ± 59.8 g) significantly improved [31]. In 2015, Zhang et al. (China) conducted a single-center before-and-after comparison of cardiac function using 99 Tcm-MIBI G-MPI imaging to evaluate effect of glucocorticoid treatment. The study analyzed follow-up results in age groups 7 years ($n = 10$ patients), 8 years ($n = 12$ patients), 9 years ($n = 11$ patients), and 10 years ($n = 10$ patients), respectively. Results revealed that myocardial perfusion significantly improved in all age groups after 2 years of glucocorticoid treatment when compared to baseline levels of them ($p < 0.05$ for all groups). However, no deterioration in LVEF, end-diastolic volume index, or end-systolic volume index was observed among the above patients when compared with the cardiac function of before treatment [26]. At the same time, Tandon et al. (USA) examined the longitudinal relationship between myocardial fibrosis and ventricular dysfunction using cardiac MRI in 98 patients with DMD in 2015. Among these, three patients did not receive glucocorticoid therapy. The mean age of glucocorticoid initiation was 7.0 ± 2.5 years, with a mean treatment duration of 7.6 ± 3.4 years. Their investigation revealed that although late gadolinium enhancement + left ventricle (LV) segments increased with age, longer glucocorticoid therapy reduced age-related myocardial fibrosis in the LV [27]. In 2017, Koeks et al. (Netherlands) conducted a large multinational cohort study on corticosteroid effects in DMD. Among patients aged 20 and older, those receiving steroids ($n = 31$) had less cardiomyopathy than past users ($n = 71$) or never-treated patients ($n = 229$). The study also showed that patients with a single exon 45 deletion lost ambulation later than those with deletions in exons 51, 44, 52, or 50 (mean age 15 vs. 11 years; $p = 0.027$). Corticosteroid treatment delayed ambulation loss for patients with exon 44 skipping or single exon 50 and exon 51 deletions [32]. Recently, Wittlieb et al. (USA) conducted a multi-center retrospective cohort study with 408 children with DMD. After 1 year of follow-up, children were grouped into non-death, cardiac death, and non-cardiac death categories, revealing that absence of glucocorticoid treatment was a significant risk factor for all-cause mortality [7]. In the most recent study, Schiava et al. (England) performed a retrospective single-center analysis of 112 patients with DMD to assess glucocorticoid use after loss of ambulation and late-stage outcomes between 1986 and 2022. The mean age was 23.4 ± 5.2 years, and mean follow-up was 18.5 ± 5.5 years. The results showed that glucocorticoids use after loss of ambulation reduced the odds of a LVEF < 50% at last assessment compared to individuals who did not receive glucocorticoids, and also delayed wheelchair balancing issues and loss of hand-to-mouth function [33] (details are summarized in Table 2).

In summary, the dose of prednisone commonly ranged from 0.5 to 0.75 mg/kg/day, while deflazacort was typically initiated at 0.9 mg/kg/day in the studies reviewed. The median age of initiation ranged from 7 to 8.6 years, with therapy duration of 2–14 years, and follow-up periods of 12–22 years. And across the above studies, we found that a total of 1142 patients were exposed to corticosteroids while 937 did not receive corticosteroids. Except three studies ($n = 202$ patients) did not illustrate how many treated and untreated patients showed disease progression, the leaving eleven studies ($n = 1612$ patients) showed detailed effect of corticosteroids treatment. Among treated patients ($n = 960$ patients), 236 patients showed disease progression. Among untreated patients ($n = 917$ patients), 534 patients showed disease progression. Additionally, only one study conducted by Hussein et al., who illustrated that the FS of 5 patients with DMD were less than 28% before starting the prednisone therapy and all these patients improved as well as motor function was also improved.

Glucocorticoid therapy has no effect on cardiac function in children with DMD (n = 6 studies, n = 6294 patients)

In 2005, Shefali et al. (India) retrospectively studied 30 patients with DMD (mean age: 10.1 ± 2.3 years), of whom 24 received prednisolone (0.75 mg/kg) for 10 days per month. No significant association was observed between steroid treatment and cardiac involvement [34]. In the same time, Kirchmann et al. (Germany) retrospectively analyzed echocardiograms and electrocardiograms of 42 children with DMD (median age 12 years; range 5.0–22.5 years),

including 17 patients treated with prednisone or deflazacort in 2005. Multiple regression models revealed no association between FS, ECG, Holter ECG, heart rate variability, and glucocorticoid usage, with no significant differences in cardiac parameters between glucocorticoid-treated and non-treated groups ($p > 0.05$) [28]. In 2014, Spurney et al. (USA) conducted a multicenter retrospective study with a total of 340 children with DMD aged from 2 to 28 years. Results indicated that age was a significant predictor of cardiomyopathy, but glucocorticoid use as a covariate did not alter the association between age, clinical stage, and cardiomyopathy ($p > 0.68$) [29]. Subsequently, in 2017, Koeks et al. (Netherlands) observed the highest corticosteroid usage among patients aged 6–14 years but found no significant effect on cardiomyopathy development until age 20 ($p = 0.94$) [32]. In 2021, Butterfield et al. (USA) prospectively followed 398 patients with DMD after loss of ambulation to assess whether corticosteroids improved cardiac function. Groups included 193 who never used corticosteroids, 104 who discontinued use within 1 year of loss of ambulation, and 101 who continued use for ≥ 1 year beyond loss of ambulation (mean treatment duration: 8.73 ± 4.44 years). The results showed no significant difference in the age of LV dysfunction onset (EF < 55% or FS < 28%) between groups [35]. Recently, Conway et al. (USA) retrospectively studied 455 patients with DMD to determine if glucocorticoids delayed LV dysfunction onset. Over half of the patients used glucocorticoids (mean therapy duration: 5.5 years). However, continuous glucocorticoid use (HR = 1.01; 95% CI: 0.66–1.53) was not cardio-protective compared to non-treated individuals [36] (details are summarized in Table 3).

Globally, five of the six studies did not provide detailed descriptions of glucocorticoid initiation and duration in children with DMD. These studies are analyzed individually below. In the study of Sheffali et al., 30 patients beyond 6 years of age were enrolled and around one-third patients had cardiomegaly. LVEF < 55% was observed in 64.2% and LVEF < 50% in 17.8%. However, no cardio-protective effect of steroids in that study without specific involved numbers of treated and untreated groups. In Kirchmann's analysis, the wide age range of participants (5–22.5 years, median 12 years) and a lower FS threshold (< 25%) compared to other studies (28%) may have contributed to differing results. In Spurney et al.'s study, 76% (257/340) of patients received glucocorticoids for more than 1 year. Koeks et al. conducted the largest genetically confirmed DMD cohort study, highlighting the role of genetic modifiers in clinical outcomes. They found no clear cardio-protective effect of glucocorticoids before age 20 but observed positive effects in older patients. Butterfield et al. did not observe differences in time to abnormal LV function onset between corticosteroid use groups. Conway et al. reported no cardio-protective effect of glucocorticoids in patients with DMD.

Across the above six studies, there were 3166 patients exposed to corticosteroids and 3128 patients did not receive corticosteroids treatment. Among treated patients, four studies ($n = 644$ patients) provided detailed numbers of disease progression, which included approximately 207 treated patients. And only one study ($n = 398$ patients) provided specific number of disease progression in untreated patients ($n = 155$ patients), which 38 untreated patients showed disease cardiomyopathy in that study. And no study showed the detailed number of disease improvement or keeping stable.

Effects of glucocorticoid type on cardiac function in patients with DMD (n = 1 study, n = 111 patients)

In 2005, Markham et al. (USA) retrospectively studied 111 patients with DMD, of whom 48 received prednisone or deflazacort (mean initiation age: 6.7 ± 2.5 years; mean duration: 3.0 ± 2.5 years). untreated participants had a 4.4–15.2 times higher risk of FS dropping below 28%. Among treated patients, 29 received prednisone and 19 deflazacort, with similar FS outcomes between groups. Ten patients (mean age: 16.3 ± 3.0 years; treated for 4.2 ± 1.6 years) discontinued glucocorticoids due to side effects but still had higher FS values (35% ± 6%) than untreated subjects, even after discontinuing therapy (6 ± 4 years; $p < 0.001$) [37] (details are summarized in Table 4).

In summary, this study suggested that glucocorticoid therapy delays FS decline in children with DMD, with sustained benefits beyond treatment duration and independent of glucocorticoid type.

Effects of glucocorticoids administration regimens and dose on cardiac function in patients with DMD (n = 1 study, n = 229 patients)

In 2020, Trucco et al. conducted a single-center longitudinal study with 229 patients with DMD to examine the effects of long-term glucocorticoid therapy and regimens (prednisolone 0.9 mg/kg or deflazacort 0.75 mg/kg) over 5 years. FS declined by 0.53% per year in glucocorticoid-treated patients, significantly slower than in glucocorticoid-naïve patients (1.17% per year, $p < 0.01$). Cardiomyopathy onset occurred later in treated patients (16.6 years) compared to untreated ones (13.9 years; $p < 0.05$). Though the study concluded that glucocorticoids significantly retard cardiomyopathy progression, regardless of regimen (daily or intermittent), the age at the initiation of glucocorticoid usage and the unequal distribution of participants across subgroups did not provide [38] (details are summarized in Table 5).

Effect of age at initiation of glucocorticoid therapy on cardiac function in patients with DMD (n = 1 study, n = 481 patients)

In 2017, Kim et al. (USA) conducted a multicenter observational analysis of 481 patients with DMD to explore the relationship between corticosteroid initiation age and severe clinical outcomes. Participants were categorized by initiation timing: early childhood (≤ 5 years) or late childhood (> 5 years). Results showed that early initiation increased the risk of early-onset cardiomyopathy compared to late initiation or no glucocorticoid treatment (HR = 2.0; 95% CI: [1.2, 3.4]) [30] (details are summarized in Table 6).

This was the only study to examine the impact of glucocorticoid initiation age on cardiac function and to categorize treatment groups by age. The mean initiation age for the early group was 4.2 years, significantly younger than in other studies. Nevertheless, the study's reliability was compromised by confounding indications, as sicker patients were more likely to be prescribed therapy earlier, and no clinical cardiac evaluation was conducted prior to treatment. Additionally, the substantial disparity in sample sizes between the two groups ($n = 59$ vs. $n = 248$) further undermined the strength of the study's conclusions.

If early glucocorticoid use in children with DMD increases cardiomyopathy risk, what might be the mechanism? Although the exact cause is unknown, several studies suggest that glucocorticoids can cause damage to cardiac function. One animal study found that excessive glucocorticoid exposure induces pathological myocardial remodeling and pathophysiological changes via the angiotensin II signaling pathway [39]. Another study using the mdx mouse model of DMD reported that while prednisolone improved skeletal contractile function, it worsened cardiac histological damage [40]. Similarly, a study in children with DMD suggested that effective skeletal muscle therapy might exacerbate cardiac disease [41]. long-term glucocorticoid use is known to cause side effects such as bone fragility, immunosuppression, metabolic disorders, atherosclerosis, and heart failure, all of which may accelerate cardiomyopathy progression in patients with DMD. A promising alternative is vamorolone, a new glucocorticoid that shares prednisone's pharmacokinetic and metabolic characteristics but avoids many of its side effects [42–44]. Animal studies indicate that vamorolone acts as a mineralocorticoid receptor antagonist, providing anti-inflammatory and therapeutic effects on cardiomyopathy while safely inhibiting inflammation [45]. Based on its mechanism and properties, long-term vamorolone treatment may protect motor, respiratory, and cardiac functions in children with DMD. However, whether early vamorolone use increases cardiomyopathy risk remains unexplored.

Discussion

According to current research, glucocorticoids can delay the deterioration of cardiac function in patients with DMD [7, 19–27, 31–33]. However, some studies have found glucocorticoids to be ineffective in improving cardiac function in children with DMD [22, 28, 29, 32, 36], and one study even hypothesized that early glucocorticoid use may increase the risk of cardiomyopathy [30]. These contradictions may be attributed to variations in study populations and groups, differences in glucocorticoid initiation age and therapy duration, total doses administered, outcome endpoints, and the presence of confounding factors and biases in studies with purely observational designs. In addition, patients with unfavorable genotypes, corticosteroid resistance, use of various cardiac medications or intolerable side effects likely discontinued therapy after loss of ambulation, potentially skewing results.

Among the 21 studies reviewed, only 8 statistically analyzed confounding indicators related to glucocorticoid side effects that could potentially worsen cardiac function, such as height, weight, BMI, and blood pressure. These studies reported that glucocorticoid treatment can lead to growth restriction, obesity or increased BMI. However, the relationship between obesity or BMI and cardiac function was not investigated. Therefore, future research on the effects of glucocorticoids on cardiac function in children with DMD must address several critical questions. First, when is the optimal age to initiate glucocorticoid therapy? One study suggested that early initiation (≤ 5 years of age) may increase the risk of cardiomyopathy [30], while current recommendations propose starting treatment between 4 and 8 years of age [1]. If glucocorticoid therapy initiated before the age of 5 does increase cardiomyopathy incidence, this raises the question of whether the recommended age should be re-evaluated. Second, which type, regimen, and dosage of glucocorticoids offer the greatest benefit to children with DMD? At present, there are limited comparative studies on the different types and regimens of glucocorticoids. One study indicated that intermittent and daily glucocorticoid administration have comparable effects on cardiac function in children with DMD [38]. Despite vamorolone's pharmacokinetics and metabolic pathway being similar to those of prednisone, it does not exhibit the side effects associated with traditional glucocorticoids. Future RCTs could explore whether the daily use of glucocorticoids has similar effects like glucocorticoids intermittent administration on the cardiac function of children with DMD and whether patients with DMD might benefit from treatment with vamorolone [42–44]. Another critical question pertains to the maximum duration of glucocorticoid therapy. While long-term glucocorticoid use can provide early protective effects on cardiac function,

it is associated with side effects such as obesity and hypertension, which may adversely impact cardiac function over time. Determining the optimal treatment duration is essential to maximize efficacy while minimizing adverse effects. Additionally, although glucocorticoids may protect cardiac function in the early stages of DMD, prolonged use and the accumulation of side effects could exacerbate cardiac damage. Most follow-up studies on glucocorticoids and cardiac function in children with DMD span only 4–7 years [19–21, 23, 24, 27], with the longest extending to approximately 16 years [33]. Controlled studies investigating long-term glucocorticoid use remain scarce. Consequently, the dynamic effects of extended glucocorticoid use on cardiac function in children with DMD remain unclear. Future research should address the potential side effects of glucocorticoids on cardiac function, develop evidence-based dosing strategies and determine the optimal timing for therapy initiation. At the same time, a dose rationale should be developed for this population and indication (i.e., population pharmacokinetics and pharmacokinetics-pharmacodynamics models) to achieve an optimal balance between therapeutic effects and side effects. Such models would allow exploration of a broader range of scenarios in a larger virtual cohort than is feasible with multiple-arm RCTs. Future studies should also consider the impact of DMD genotype, cardiac drug usage, physical exercise, and respiratory function on cardiac outcomes in patients with DMD. Additionally, it is important to acknowledge that standard echocardiography, as a late marker, is sub-optimal for investigating DMD-associated dilated cardiomyopathy. Advanced echocardiographic techniques (e.g., regional LV motion analysis, speckle tracking and strain analysis) and, more importantly, cardiac MRI (assessing dimensions, function, water content, and fibrosis) are currently recommended for early detection of cardiac involvement in studies on DMD-associated cardiomyopathy [46].

Limitation

However, several limitations were identified. First, most studies were retrospective, with only one prospective study. Second, the influence of DMD genotype and cardio-protective drugs was inadequately considered. Only one study examined glucocorticoid effects on the limited different genotype, and five studies used cardio-protective drugs, while the remaining seven did not report on this factor. Third, echocardiography was the most commonly used tool in the majority of studies to evaluate cardiac function. However, it is important to note that the measurement of LVEF and FS can be easily influenced by variability among analyst. More importantly, the potential effects of chest wall deformities on cardiac morphology and function further complicate accurate assessment. Additionally, treatment and follow-up durations were relatively limited. Current guidelines recommend starting corticosteroid at 4–8 years of age, yet the reviewed studies initiated therapy at 7–8.6 years. Furthermore, while DMD survival has extended to about 30 years, most studies did not evaluate glucocorticoid effects on cardiac function in children with DMD beyond 23 years of age. Confounding factors such as genotype variations, concomitant medications, and follow-up duration should also be addressed. To overcome these limitations, prospective, randomized controlled trials (RCTs) with strict inclusion and exclusion criteria are needed to evaluate the long-term effects of glucocorticoid therapy on cardiac function in patients with DMD.

Conclusion

It is currently assumed that glucocorticoids can delay the deterioration of cardiac function in patients with DMD. However, there is a lack of studies addressing the long-term effects of glucocorticoid therapy and the implications of early glucocorticoid initiation on cardiac outcomes, resulting in inconclusive findings. To address these gaps, prospective longitudinal studies are essential to determine the optimal timing, dose regimens and long-term effects of glucocorticoid therapy in patients with DMD. Such studies will provide a more comprehensive understanding of the role of glucocorticoid therapy in managing cardiac function and improving outcomes for patients with DMD.

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Declarations

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