

# Steroid use and lipid abnormalities in children: assessing the risk in chronic disease management

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## Abstract

**Introduction:** Although steroid-induced dyslipidemia is well-documented in adults, its impact on children remains underexplored. Given the long-term cardiovascular risks associated with dyslipidemia, understanding its link with steroid use in children is crucial.

**Aim:** This study aimed to evaluate the association of steroid therapy, specifically its dose, duration, and type with the development of dyslipidemia in children with chronic diseases.

**Materials and methods:** A retrospective cross-sectional study was conducted from January 2022 to January 2024 in a tertiary hospital in Medan, Indonesia. Medical records of children receiving steroid therapy for at least six weeks were reviewed. Steroid doses were converted to prednisone equivalents. Dyslipidemia was defined according to the Expert Panel Guidelines. Data distribution was assessed using normality tests and appropriate statistical tests were selected based on the distribution of each variable.

**Results:** The study included 63 children, 54 (85.7%) of whom had dyslipidemia. A significant association was found between higher steroid dose and dyslipidemia ( $p=0.002$ ), especially for LDL and total cholesterol levels ( $p=0.005$  and  $p=0.017$ , respectively). Although the association between dyslipidemia and steroid duration was borderline ( $p=0.050$ ), children treated for 6–24 weeks exhibited significantly higher LDL ( $p=0.035$ ) and total cholesterol ( $p=0.010$ ) compared to those treated longer. No significant differences in lipid parameters were observed across steroid types.

**Conclusion:** Steroid use in children with chronic diseases is significantly associated with dyslipidemia. A higher steroid dose was associated with abnormal lipid profiles. These findings support the recommendation for routine lipid monitoring and careful dose consideration to help mitigate long-term cardiovascular risk.

## Keywords

corticosteroids, dyslipidemia, hyperlipidemia, pediatrics

## Introduction

Steroids are indispensable in managing pediatric chronic diseases, yet their long-term metabolic consequences remain a growing concern. While widely studied in adults,

the effects of steroid therapy on pediatric lipid metabolism remain underexplored. Steroids serve as a cornerstone treatment for numerous childhood conditions due to their potent immunosuppressive and anti-inflammatory properties. However, their prolonged use is linked with signif-

icant adverse effects that may outweigh their therapeutic benefits. Current estimates suggest that approximately 1% of children under the age of 20 use oral steroids monthly, highlighting the widespread nature of exposure to these medications.<sup>[1,2]</sup>

Steroids influence lipid metabolism by increasing lipolysis, enhancing lipoprotein lipase (LPL) activity, and altering adipokine levels.<sup>[3,4]</sup> These metabolic changes can contribute to dyslipidemia, a major risk factor for early-onset cardiovascular disease.<sup>[5,6]</sup> While steroid therapy is essential for managing chronic conditions in children, its prolonged use raises significant concerns about long-term metabolic complications. Although extensive research has established the link between steroid therapy and lipid abnormalities in adults, data on pediatric populations remain limited. The specific impact of steroid dose, duration, and type on dyslipidemia risk in children remains unclear, highlighting a critical gap in knowledge. Given the well-documented metabolic effects of steroids, we hypothesize that their use similarly alters lipid profiles in children, presenting an overlooked risk in pediatric care.

## Aim

Our study investigates the effects of steroid therapy considering its dose, duration, and type on lipid profiles in children with chronic diseases.

## Materials and methods

This retrospective cross-sectional study analyzed medical records from the Endocrinology Outpatient Clinic of a tertiary hospital in Medan, Indonesia, from January 2022 to January 2024. A consecutive sampling method was used to include all pediatric patients (0–18 years) who met the inclusion criteria. For this study, a chronic disease was operationally defined as a non-communicable condition requiring steroid therapy for at least six weeks to manage inflammation or suppress the immune system. Eligible underlying diagnoses included idiopathic thrombocytopenic purpura (ITP), systemic lupus erythematosus (SLE), acute lymphoblastic leukemia (ALL), aplastic anemia, and autoimmune hemolytic anemia (AIHA). Children with pre-existing conditions such as dyslipidemia, nephrotic syndrome, obesity, or diabetes mellitus diagnosed prior to the initiation of steroid therapy were excluded. The minimum sample size was determined using the formula:

$$n = (Z^2 \alpha / 2 \times P \times Q) / d^2$$

where  $Z^2 \alpha / 2 = 1.96$  (for  $\alpha = 5\%$ ),  $p = 0.5$ ,  $Q = 0.5$ , and  $d = 0.17$ . A 95% confidence interval ( $\alpha = 5\%$ ) and a conservative estimate of disease prevalence ( $p = 0.5$ ) were used to ensure adequate power. The calculated minimum sample size was 34.

Dyslipidemia was diagnosed according to the Expert Panel Integrated Guidelines for Cardiovascular Health and

Risk Reduction in Children and Adolescents. The criteria were as follows: total cholesterol  $\geq 200$  mg/dL or low-density lipoprotein (LDL)  $\geq 130$  mg/dL, or high-density lipoprotein (HDL)  $< 40$  mg/dL, or triglyceride (TG)  $\geq 100$  mg/dL (for 0–9 years old),  $\geq 130$  mg/dL (for 10–19 years old).<sup>[7]</sup> Lipid profile results were obtained for each patient from medical records after a minimum of six weeks of continuous steroid therapy.

The steroid preparations used in this study were administered orally. We converted the steroid dose from other types of steroid equivalent to prednisone. The conversion rate for methylprednisolone to prednisone was 4:5.<sup>[8]</sup> The conversion rate for dexamethasone to prednisone was 0.75:5.<sup>[9]</sup> A low dose of steroids was defined as equivalent to prednisone  $< 10$  mg/day, moderate dose of 10–20 mg/day, and high dose of  $> 20$  mg/day.<sup>[1]</sup> The conversion of steroid doses to prednisone equivalents allows for a standardized comparison across different steroid types, ensuring consistency in dose-response analysis.

All statistical analyses were performed using SPSS Statistics version 29.0, with a 95% confidence interval. Data distribution was assessed using normality tests and appropriate statistical tests were selected based on the distribution of each variable.

The chi-square test was used to determine associations between dyslipidemia and categorical variables. Comparisons of lipid profiles based on steroid treatment duration were conducted using the Mann-Whitney U test, while differences in lipid profiles across steroid types and dosage categories were analyzed using Kruskal-Wallis test. For significant Kruskal-Wallis test results, post-hoc pairwise comparisons were conducted using the Mann-Whitney U test with a Bonferroni correction applied to the  $p$ -value. A  $p$ -value of  $< 0.05$  was considered statistically significant.

This study was approved by the Ethics Committee of Universitas Sumatera Utara (No. 1011/KEPK/USU/2023) on October 6, 2023. Written informed consent was obtained for participation in the study and the use of patient data for research and educational purposes. The procedures in the study followed the guidelines laid down in the Declaration of Helsinki 2013.

## Results

A total of sixty-three children were included in this study, consisting of 45 girls (71%) and 18 boys (29%). The median age was 14 years (range: 2–17 years) in both the dyslipidemia and non-dyslipidemia groups, with no significant difference between them ( $p = 0.737$ ). The demographic characteristics and key variables of the study cohort are first presented, followed by a detailed analysis with steroid dose, type, and duration (**Table 1**). SLE and ITP were the most common underlying conditions, followed by ALL and AIHA. The occurrence of dyslipidemia did not vary significantly by disease type ( $p = 0.971$ ).

Dyslipidemia was identified in 54 out of 63 patients

**Table 1.** Characteristics of subjects

Characteristics		Dyslipidemia		p-value
		Yes (n=54)	No (n=9)	
Sex, n (%)	Female	38 (84.4)	7 (15.6)	0.649 <sup>†</sup>
	Male	16 (88.9)	2 (11.1)	
Age, years (median (min-max))		14 (2-17)	14 (5-17)	0.737 <sup>‡</sup>
Weight, kg (median (min-max))		47.35 (11-107)	39.6 (18-61.7)	0.280 <sup>‡</sup>
Height, cm (median (min-max))		148 (85-172)	141 (105-156)	0.798 <sup>‡</sup>
Disease, n (%)	ITP	12 (84.6)	2 (15.4)	0.971 <sup>†</sup>
	SLE	24 (82.7)	5 (17.3)	
	ALL	11 (91.7)	1 (8.3)	
	AIHA	7 (87.5)	1 (12.5)	
Type of steroid, n (%)	Prednisone	14 (100)	0 (0)	0.176 <sup>†</sup>
	Methylprednisolone	32 (80.0)	8 (20.0)	
	Dexamethasone	8 (88.8)	1 (11.2)	
Steroid dose (mg/day)		30 (1.5-85)	12 (4-16.7)	0.002 <sup>‡</sup>
Steroid dose category, n (%)	Low	11 (78.6%)	3 (21.4)	0.002 <sup>†</sup>
	Moderate	11 (64.7)	6 (35.3)	
	High	32 (100)	0 (0)	
Duration, n (%)	6-24 weeks	31 (93.9)	2 (6.1)	0.050 <sup>†</sup>
	>24 weeks	23 (76.7)	7 (23.3)	

† chi-square/Fisher's test; ‡ Mann-Whitney U test; ITP: immune thrombocytopenic purpura; SLE: systemic lupus erythematosus; ALL: acute lymphoblastic leukemia; AIHA: autoimmune hemolytic anemia

(85.7%). A significant association emerged between steroid dosage and dyslipidemia status ( $p=0.002$ ), with all individuals in the high-dose group affected. In comparison, the prevalence was lower among those receiving moderate-dose (64.7%) and low-dose therapy (78.6%). The median daily corticosteroid dose was notably greater in those with dyslipidemia than in those without (30 mg/day vs. 12 mg/day,  $p=0.002$ ). The duration of steroid therapy between 6 and 24 weeks was associated with a higher dyslipidemia rate (93.9%) relative to durations exceeding 24 weeks (76.7%), although this finding approached statistical significance ( $p=0.050$ ) but did not reach it.

Lipid parameters did not differ significantly across steroid types (Table 2). However, children given methylprednisolone or dexamethasone tended to have higher triglycer-

ide, LDL, and total cholesterol concentrations than those treated with prednisone, despite the lack of significance.

When stratified by treatment duration, LDL and total cholesterol levels were significantly elevated among those treated for 6-24 weeks compared to those with longer exposure (LDL: 138 mg/dL vs. 105 mg/dL,  $p=0.035$ ; total cholesterol: 203 mg/dL vs. 182 mg/dL,  $p=0.010$ ), as shown in Table 3. While HDL and triglyceride levels were also increased in the shorter-duration group, these trends did not reach statistical significance ( $p=0.086$  and  $p=0.158$ , respectively).

A comparative analysis of lipid profiles by steroid dose category showed a statistically significant difference among the low, moderate, and high-dose groups for LDL ( $p=0.005$ ) and total cholesterol ( $p=0.017$ ), as detailed in

**Table 2.** Comparison of lipid profile outcomes by steroid type

	Type of steroid			p-value <sup>†</sup>
	Prednisone	Methylprednisolone	Dexamethasone	
HDL (median (min-max))	47 (26-108)	44 (23-106)	42 (18-114)	0.762
LDL (median (min-max))	139 (44-166)	116 (65-760)	111 (42-215)	0.718
Total cholesterol (median (min-max))	198 (81-263)	201 (109-872)	197 (92-321)	0.584
Triglyceride (median (min-max))	125 (85-207)	169 (68-1184)	199 (72-302)	0.355

† Kruskal-Wallis test; HDL: high density lipoprotein; LDL: low density lipoprotein

**Table 3.** Association between duration of steroid treatment and lipid profiles

	Duration of steroid treatment		p-value <sup>†</sup>
	6–24 weeks	>24 weeks	
HDL (median (min-max))	48 (24–108)	43 (18–114)	0.086
LDL (median (min-max))	138 (65–760)	105 (42–760)	0.035
Total cholesterol (median (min-max))	203 (109–872)	182 (81–872)	0.010
Triglyceride (median (min-max))	194 (68–552)	133 (72–1184)	0.158

<sup>†</sup> Mann-Whitney U test; HDL: high density lipoprotein; LDL: low density lipoprotein

**Table 4.** HDL and triglyceride levels did not differ significantly across the groups. To identify the specific group differences, post-hoc pairwise comparisons were performed using the Mann-Whitney U test with a Bonferroni-corrected significance level of  $p < 0.0167$ . This analysis revealed that the high-dose group had significantly higher LDL (median 142 mg/dL) and total cholesterol (median 210.5 mg/dL) levels compared to the moderate-dose group ( $p = 0.008$  and  $p = 0.002$ , respectively). No other pairwise comparisons reached statistical significance.

## Discussion

Steroids are widely used in pediatric chronic disease management, but their long-term metabolic effects remain a concern.<sup>[5]</sup> In our study, 88.7% of children developed dyslipidemia following corticosteroid therapy, a prevalence notably higher than the 68.8% reported in a prior study on pediatric rheumatic diseases.<sup>[10]</sup> The dose-dependent relationship between corticosteroid use and dyslipidemia observed in our cohort aligns with Rodrigues et al.<sup>[10]</sup> who reported a similar association between cumulative steroid dose and increased lipid profile levels in children with juvenile SLE, reinforcing the pharmacological impact of corticosteroids on lipid metabolism.

Corticosteroids are known to influence hepatic lipid metabolism, including enhanced VLDL production and suppressed lipoprotein lipase activity, thereby increasing the risk of dyslipidemia.<sup>[11–14]</sup> In our cohort, steroid dose significantly affected LDL and total cholesterol concentrations. Clinical studies further support this mechanism. In

adult patients with SLE, higher prednisone doses have been associated with elevated serum triglycerides, LDL, and apolipoprotein B, and reduced HDL.<sup>[15]</sup> Dolatabadi et al.<sup>[16]</sup> and Atik et al.<sup>[17]</sup> also reported dose-dependent increases in total cholesterol with glucocorticoid therapy. Moreover, Basu et al.<sup>[18]</sup> identified anti-LPL antibodies in pediatric SLE, further implicating impaired lipid clearance in children.

Although corticosteroids differ in potency, half-life, and pharmacodynamics, our findings suggest that lipid profile disturbances are not strongly influenced by the specific agent used. While children receiving methylprednisolone exhibited numerically higher LDL and total cholesterol levels, these differences were not statistically significant. This aligned with findings by Malynda et al.<sup>[19]</sup> who observed rapid increases in these lipids following high-dose methylprednisolone in pediatric lupus nephritis. However, those effects were observed within days of therapy and may not reflect steady-state changes during chronic use. Although methylprednisolone was associated with numerically higher lipid values in our study, the absence of significant differences across steroid types suggests that dyslipidemia risk may be inherent to corticosteroid exposure itself, rather than specific to individual agents.

Prior literature suggests that long-term corticosteroid use increases the risk of metabolic complications.<sup>[20,21]</sup> For instance, Ericson-Neilsen et al.<sup>[21]</sup> found that up to 90% of patients using corticosteroids for more than 60 days experienced adverse effects. Notably, in our cohort, LDL and total cholesterol levels were significantly elevated among those treated for 6–24 weeks compared to those with longer exposure, suggesting that metabolic disturbances may develop earlier than commonly assumed. This contrasts with the

**Table 4.** Association between steroid dose and lipid profiles

	Steroid Dose			p-value <sup>†</sup>
	Low	Moderate	High	
HDL (median (min-max))	47 (28–77)	42 (18–106)	43.5 (23–114)	0.201
LDL (median (min-max))	107 (79–760)	98 (42–760)	142 (44–301) ‡	0.005
Total cholesterol (median (min-max))	190 (140–872)	151 (92–872)	210.5 (81–427) ‡	0.017
Triglyceride (median (min-max))	178.5 (75–1184)	118 (72–278)	184 (68–552)	0.257

<sup>†</sup> Kruskal-Wallis test; ‡ Post-hoc analysis (Mann-Whitney U test with Bonferroni correction,  $p < 0.0167$ ) revealed a significant difference for: LDL; moderate vs. high ( $p = 0.008$ ) and total cholesterol; moderate vs. high ( $p = 0.002$ ). HDL: high density lipoprotein; LDL: low density lipoprotein

expectation that longer exposure would result in greater dysregulation and may reflect higher initial dosing during early therapy phases. Lau et al.<sup>[22]</sup> similarly observed steroid withdrawal led to lower total cholesterol and LDL, suggesting that prolonged steroid use might stabilize lipid profiles over time. This could imply that the initial phase of steroid therapy (6–24 weeks) sees higher lipid elevations, possibly due to higher initial doses, with potential adaptation or tapering in longer durations.

This study has several important limitations. The primary limitation is its retrospective, cross-sectional design, which prevents the establishment of a definitive causal link between steroid therapy and dyslipidemia. Because baseline (pre-therapy) lipid measurements were unavailable, we could not analyze the change in lipid profiles within individual patients over time. Consequently, our findings demonstrate a strong association rather than a proven causal effect.

This association may be significantly confounded by the underlying disease activity itself, a factor known to drive dyslipidemia independent of corticosteroid use. For instance, inflammatory cytokines like TNF- $\alpha$  and IL-6 can upregulate LPL activity and enhance hepatic lipogenesis in pediatric SLE and JIA.<sup>[23,24]</sup> Dyslipidemia is also thought to develop early in disease progression. Mogensen et al.<sup>[25]</sup> also support this notion as 99% of pediatric ALL cases in their cohort presented with dyslipidemia prior to any corticosteroid exposure suggesting a significant disease-driven metabolic burden. Furthermore, other key variables were not assessed in this study, including disease severity, concurrent medications, pubertal status, diet, and physical activity, all of which could have influenced the observed lipid patterns. Additionally, our analysis examined the effects of steroid dose, duration, and type in isolation. We did not perform a multivariable analysis to test for potential interaction effects, and it is possible that the impact of one factor (e.g., duration) depends on another (e.g., steroid type). Finally, while providing valuable region-specific data, our single-center design and modest sample size may limit the generalizability of the findings.

Future prospective, multicenter studies with larger, stratified cohorts are therefore essential to address these limitations. Such studies should include baseline lipid measurements and employ multivariable models to clarify long-term outcomes and support the development of individualized care strategies for this vulnerable population.

## Conclusion

Steroid therapy in children with chronic diseases is significantly associated with dyslipidemia. In our cohort, a higher steroid dose was a key factor associated with dyslipidemia, particularly with elevated LDL and total cholesterol levels, while no significant association was found with steroid type. Additionally, dyslipidemia was also observed in patients with shorter (6–24 weeks) treatment durations, suggesting that metabolic changes may occur early. These

findings highlight the need for proactive lipid monitoring in children undergoing steroid therapy. Future prospective studies are needed to confirm these associations, evaluate long-term outcomes, and inform the development of preventive strategies to mitigate cardiovascular risk.

## Ethical approval

This study was conducted in accordance with the Declaration of Helsinki (2013) and approved by the Ethics Committee of Universitas Sumatera Utara (No. 1011/KEPK/USU/2023) on October 6, 2023.

## Ethical statements

- The authors declared that no clinical trials were used in the present study.
- The authors declared that written informed consent had been obtained for participation in the study and for the utilization of patient data for research and educational purposes.
- The authors declared that certain experiments on humans or human tissues were performed for the present study.
- The authors declared that no experiments on animals were performed for the present study.
- The authors declared that no commercially available immortalized human and animal cell lines were used in the present study.

## Conflict of interest

The authors declare that they have no conflict of interest.

## Use of AI

The authors declare that no AI tools were used.

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## Author contributions

SML: conceptualization, data curation, formal analysis, methodology, project administration, resources, supervision, validation, visualization, writing—original draft, writing—review and editing; MD: investigation, methodology, project administration, resources, supervision, validation, review and editing; MQ: investigation, methodology, project administration, writing—original draft, writing—review

and editing; SI: investigation, methodology, writing–original draft, writing–review and editing. All authors contributed to the study design, data analysis, and manuscript preparation. They have reviewed and approved the final version of this manuscript and take full responsibility for its content.

## Data availability

All data used are referenced or included in the article.

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