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Original Research Article

Comparative study of prednisolone and deflazacort in the treatment of initial attack nephrotic syndrome in a tertiary care hospital

Sumon Shahrior Morshed[1]*, Azka Sejuti[1], Amit Shome[1], M. Rokibul Islam[2], Mashura Musharraf[1], Anisa Hossain[1], Sabira Rahman[3], Smita Kanungo[4]

[1]Department of Paediatrics, Dhaka Medical College Hospital, Dhaka, Bangladesh
[2]Department of Paediatrics, Rajshahi Medical College, Rajshahi, Bangladesh
[3]Department of Paediatrics, Central Police Hospital, Dhaka, Bangladesh
[4]Department of Cardiology, National Heart Foundation and Research Institute, Dhaka, Bangladesh

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***Correspondence:**
Dr. Sumon Shahrior Morshed,
E-mail: shahriormorshed3@gmail.com

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ABSTRACT

Background: Idiopathic nephrotic syndrome is a common chronic kidney disease in children and is conventionally treated with prednisolone. Deflazacort, a synthetic glucocorticoid with comparable anti-inflammatory efficacy and a potentially better safety profile, has emerged as an alternative option.
Methods: This double-blind randomized controlled trial was conducted at Dhaka Medical College Hospital over 12 months. Eighty children aged 2–12 years with first-episode idiopathic nephrotic syndrome were randomly assigned to receive deflazacort (n=41) or prednisolone (n=39) according to standard treatment protocols and were followed for six months. Primary outcomes included time to remission, duration of remission, and relapse rate. Secondary outcomes included growth parameters, blood pressure, biochemical profile, and steroid-related adverse effects.
Results: Baseline demographic and clinical characteristics were comparable between the two groups. Children treated with deflazacort achieved significantly faster remission (6.7±2.8 vs 8.4±2.1 days; p=0.005), longer remission duration (154.3±19.7 vs 120.2±36.1 days; p<0.001), and fewer frequent relapses (≥2 relapses: 12.2% vs 41.1%; p=0.027). Prednisolone was associated with higher rates of hypertension and hypertrichosis and significantly higher follow-up blood pressure. Serum cholesterol levels declined in both groups, with a greater reduction observed in the deflazacort group, while random blood glucose levels remained comparable.
Conclusions: Deflazacort demonstrated superior efficacy and a more favorable adverse-effect profile compared with prednisolone, supporting its use as preferable first-line corticosteroid in children with idiopathic nephrotic syndrome.

Keywords: Idiopathic nephrotic syndrome, Deflazacort, Prednisolone, Paediatric nephrology

INTRODUCTION

Nephrotic syndrome is a clinical entity characterized by heavy proteinuria, hypoalbuminemia, hyperlipidemia, oedema, and encompasses group of the heterogeneous but related glomerular diseases.[1,2] Despite more than Five decades experience with corticosteroid therapy, exact pathogenesis of the idiopathic nephrotic syndrome remain incompletely understood. Idiopathic nephrotic syndrome is one of the most common chronic kidney diseases in children, with a reported prevalence of about 16 per 100,000 and an incidence ranging from approximately 2 to 16.9 per 100,000 children worldwide.[3,4] Childhood nephrotic syndrome is most frequently caused by minimal change disease and focal segmental glomerulosclerosis, although rare genetic disorders and

secondary causes related to drugs, infections, or neoplasia also contribute.[5] During active disease, urinary loss of proteins involved in immunity, coagulation, and transport leads to serious complications, including infections, thromboembolic events, and acute kidney injury.[4] In Bangladeshi cohorts, the spectrum of histological variants varies over time; mesangioproliferative glomerulonephritis, minimal change disease, FSGS, IgM nephropathy, and IgA nephropathy have been reported with changing frequencies across different years, reflecting heterogeneity in underlying pathology.[5] Clinically, idiopathic nephrotic syndrome is classified by response to corticosteroid therapy.

Between 80-90% of children older than one year achieve complete remission with standard steroid regimens within four weeks and are categorized as steroid-sensitive nephrotic syndrome. In contrast, the remaining 10-20% are steroid resistant and have a poorer renal prognosis.[6] The proportion of steroid resistance varies substantially across ethnic groups, reported at around 20% in Europeans, 16-27% in Africans, 27-54% in Asians, and 20-39% in South Asians.[3] FSGS is persistent among African American and South Asian children.[3] These differences likely reflect a complex interplay of genetic susceptibility, environmental exposures, health care access, and variation in diagnostic and therapeutic practices. Prednisolone remains the cornerstone of therapy for an initial episode of steroid-sensitive nephrotic syndrome. Current recommendations generally advocate prednisolone 2 mg/kg/day, up to 60 mg, for six weeks, followed by 1.5 mg/kg, up to 40 mg, on alternate days for a further six weeks before discontinuation.[7,8]

Although the majority of children respond, 80-90% will relapse at least once, and up to half will become frequently relapsing or steroid dependent, thereby requiring repeated or prolonged exposure to high-dose steroids.[4] Recurrent courses of prednisolone are associated with significant adverse effects on growth, bone health, metabolic profile, behavior, and cosmetic appearance, and second-line immunosuppressants used in steroid toxicity or resistance carry additional risks and only partial efficacy.[4] This therapeutic burden is particularly concerning in low and middle-income settings, where monitoring and mitigation of long-term toxicities may be limited. Deflazacort is an oxazoline derivative of prednisolone, a synthetic glucocorticoid licensed for use in adults and children, that has been evaluated as an alternative to conventional steroids in a range of inflammatory diseases.[9] Pharmacodynamic studies suggest that deflazacort is slightly less potent than prednisolone on a milligram basis; a potency ratio of approximately 1.28:1 has been proposed, such that 6 mg of deflazacort is roughly equivalent to 5 mg of prednisolone, with reported dose equivalence ratios ranging from 1:1.2 to 1:1.5 depending on the disease context.[10] Deflazacort is rapidly absorbed, converted to an active metabolite by plasma esterases, and eliminated predominantly by the kidneys, with limited binding to corticosteroid-binding globulin. Notably, several adult and pediatric studies have suggested that, at anti-inflammatory doses equivalent to prednisolone, deflazacort is associated with less adverse impact on bone mineral density, calcium metabolism, linear growth, carbohydrate metabolism, fat accumulation, and lipid profile.[5-11]

In patients with nephrotic syndrome, deflazacort appears to be at least as effective as prednisolone in inducing remission or reducing proteinuria, while causing less reduction in spinal bone mineral content. In this context, there is a clear need to generate robust comparative data on the efficacy and safety of deflazacort versus prednisolone in children with an initial episode of idiopathic nephrotic syndrome, particularly in high-burden countries such as Bangladesh. The present randomized controlled trial was therefore designed to evaluate the comparative efficacy and toxicity of deflazacort and prednisolone in children presenting with an initial attack of nephrotic syndrome in a tertiary care hospital.

METHODS

This double-blind randomized controlled trial was conducted over 12 months (duration) in the Department of Paediatrics, Dhaka Medical College Hospital, Dhaka, Bangladesh. Children aged 2–12 years presenting with an initial attack of idiopathic nephrotic syndrome were included. Exclusion criteria were relapsing nephrotic syndrome, secondary causes, congenital nephrotic syndrome, nephrotic syndrome with hypertension and/or macroscopic hematuria, and refusal to participate. One hundred children were enrolled and randomized (50 per group); 20 were excluded during follow-up (loss to follow-up or treatment discontinuation), leaving 41 in Group A and 39 in Group B for analysis.

Random allocation was performed using a computer-generated sequence. Group A received deflazacort 2.4 mg/kg/day in two or three divided doses for 6 weeks, followed by 1.8 mg/kg on alternate days for 6 weeks. Group B received prednisolone 2.0 mg/kg/day for 6 weeks, followed by 1.5 mg/kg on alternate days for 6 weeks. Drugs were provided in equivalent tablet or suspension formulations. Adherence and toxicity monitoring were reinforced through counselling and telephone contact.

The baseline evaluation included history, physical examination, and investigations to exclude secondary causes. Nephrotic syndrome, remission, and relapse were defined according to standard paediatric nephrology criteria. Bedside urine albumin by the heat coagulation test was monitored regularly. Patients were followed for 6 months with visits at 3 and 6 months and telephone contact as needed. At each visit, clinical status, blood pressure, anthropometry, and steroid-related adverse effects (hypertension, cushingoid features, hypertrichosis,

ocular changes) were recorded. Random blood sugar and serum total cholesterol were measured at follow-up. Primary outcomes were time to remission, duration of remission, and number of relapses. Secondary outcomes included changes in height, weight, and blood pressure; metabolic parameters; and adverse effects. Data were entered into SPSS 22.0. Continuous variables were expressed as mean±SD and compared using the unpaired t-test; categorical variables were analyzed with the chi-square test. A p-value <0.05 was considered statistically significant. The institutional ethics committee approved the protocol, and written informed consent was obtained from parents or legal guardians.

RESULTS

Children in the Deflazacort group (Group A, n=41) and Prednisolone group (Group B, n=39) were comparable at baseline. Mean age was almost identical: 5.38±2.77 years in Group A vs 5.39±2.61 years in Group B (p=0.991).

Table 1: Baseline demographic and clinical characteristics of children with initial attack nephrotic syndrome.

Variables	Group A (n=41)	Group B (n=39)	P value
Age (years), mean±SD	5.38±2.77	5.39±2.61	0.991
Gender, male N (%)	26 (63.4)	26 (66.7)	0.761
Family income ≥20,000 TK, N (%)	25 (61.0)	22 (56.4)	0.68
Residence (rural), N (%)	28 (68.3)	29 (74.4)	-
Height (cm), mean±SD	105.10±16.10	104.72±16.51	0.917
Weight (kg), mean±SD	18.44±6.47	19.41±5.91	0.486
Systolic BP (mmhg)	100.49±9.67	103.59±10.13	0.165
Diastolic BP (mmhg)	67.46±8.35	70.26±10.45	0.189

Table 2: Baseline laboratory parameters of study participants.

Parameters	Group A (n=41)	Group B (n=39)	P value
	Mean±SD	Mean±SD	
Serum total cholesterol (mg/dl)	409.41±97.34	422.97±120.63	0.581
Serum creatinine (mg/dl)	0.43±0.16	0.48±0.19	0.251
Serum albumin (g/l)	18.02±4.38	20.46±5.34	0.028
Urine PCR	10.89±5.24	8.82±5.85	0.099

Table 3: Treatment outcomes following deflazacort versus prednisolone therapy.

Outcomes	Group A (n=41)	Group B (n=39)	P value
	Mean±SD	Mean±SD	
Time to remission (days)	6.73±2.80	8.35±2.08	0.005
Duration of remission (days)	154.32±19.65	120.23±36.06	<0.001
No relapse, N (%)	21 (51.2)	13 (33.3)	0.027
≥2 relapses, N (%)	5 (12.2)	16 (41.1)	-

Table 4: Frequency of adverse effects during follow-up in both treatment groups.

Side-effects	Group A (n=41)	Group B (n=39)	P value
	N (%)	N (%)	
Hypertension (3 months)	2 (4.9)	7 (17.9)	0.064
Hypertension (6 months)	1 (2.4)	7 (17.9)	0.027
Hypertrichosis (3 months)	5 (12.2)	14 (35.9)	0.013
Hypertrichosis (6 months)	4 (9.8)	11 (28.2)	0.035
Ocular changes (3 months)	0 (0.0)	0 (0.0)	-
Ocular changes (6 months)	0 (0.0)	0 (0.0)	-

Male predominance was similar, with 63.4% boys in Group A and 66.7% in Group B (p=0.761). A similar proportion of families had a monthly income ≥20,000 Tk (61.0% vs 56.4%; p=0.680), and most children came from rural areas (68.3% vs 74.4%). Baseline height (105.10±16.10 vs 104.72±16.51 cm; p=0.917), weight (18.44±6.47 vs 19.41±5.91 kg; p=0.486), systolic blood pressure (100.49±9.67 vs 103.59±10.13 mmHg; p=0.165), and diastolic blood pressure (67.46±8.35 vs 70.26±10.45 mmHg; p=0.189) did not differ significantly, indicating well-balanced groups. Baseline biochemical profiles were broadly similar between

groups, except for serum albumin. Mean serum cholesterol was high and comparable: 409.41±97.34 mg/dl in Group A vs 422.97±120.63 mg/dl in Group B (p=0.581). Serum creatinine was low and similar (0.43±0.16 vs 0.48±0.19 mg/dl; p=0.251). However, serum albumin was significantly lower in the Deflazacort group (18.02±4.38 g/l) compared to the Prednisolone group (20.46±5.34 g/l; p=0.028), suggesting slightly more severe hypoalbuminemia at baseline in Group A. Urine protein-creatinine ratio tended to be higher in Group A (10.89±5.24 vs 8.82±5.85). However, the difference did not reach statistical significance (p=0.099). Deflazacort was associated with faster and more sustained remission. Time to remission was significantly shorter in Group A (6.73±2.80 days) than in Group B (8.35±2.08 days; p=0.005). Duration of remission was markedly longer with Deflazacort (154.32±19.65 days) compared to Prednisolone (120.23±36.06 days; p<0.001). Regarding relapses, 51.2% of children in Group A had no relapse vs 33.3% in Group B. In comparison, ≥2 relapses occurred in only 12.2% of Group A but in 41.1% of Group B, with an overall significant difference in relapse pattern (p=0.027). These findings indicate better relapse control with Deflazacort.

Table 5: Trends in systolic and diastolic blood pressure at baseline and follow-up.

BP parameters	Group A (n=41)	Group B (n=39)	p value
	Mean±SD	Mean±SD	
Systolic BP (mmhg)			
Initially	100.49±9.67	103.59±10.13	0.165
3 months	100.61±9.03	107.31±12.08	0.006
6 months	99.31±7.94	107.94±11.29	<0.001
Diastolic BP (mmhg)			
Initially	67.46±8.35	70.26±10.45	0.189
3 months	66.71±7.87	71.79±10.67	0.017
6 months	67.50±6.92	73.82±10.30	0.003

Table 6: Follow-up anthropometric and biochemical parameters in both groups.

Variables	Group A (n=41)	Group B (n=39)	p value
	Mean±SD	Mean±SD	
Weight (kg)			
Initially	18.44±6.47	19.41±5.91	0.486
After 3 months	20.71±6.34	22.02±6.16	0.349
After 6 months	19.81±5.39	22.47±6.91	0.076
Serum cholesterol (mg/dl)			
Initially	409.41±97.34	422.97±120.63	0.581
After 3 months	293.90±118.74	346.33±128.99	0.062
After 6 months	247.28±94.91	342.85±134.26	0.001
Random blood sugar (mmol/l)			
Initially	5.83±1.21	6.06±1.08	0.358
After 3 months	5.98±1.10	5.74±1.23	0.356
After 6 months	5.59±0.85	5.51±0.77	0.66

Adverse effects were more frequent in the Prednisolone group. Hypertension at 3 months occurred in 4.9% of Group A vs 17.9% of Group B (p=0.064, trend towards significance), and by 6 months this difference became significant: 2.4% vs 17.9% respectively (p=0.027). Hypertrichosis was also significantly more common with Prednisolone: at 3 months, 12.2% in Group A vs 35.9% in Group B (p=0.013); at 6 months, 9.8% vs 28.2% (p=0.035). No ocular changes were reported in either group at 3 or 6 months. Overall, Prednisolone was associated with a higher burden of steroid-related cosmetic and cardiovascular side-effects. Although baseline blood pressure was similar, Prednisolone was associated with a significant rise in blood pressure over time. Baseline systolic BP was 100.49±9.67 mmHg in Group A vs 103.59±10.13 mmHg in Group B (p=0.165), but at 3 months it increased to 107.31±12.08 mmHg in Group B compared to 100.61±9.03 mmHg in Group A (p=0.006). At 6 months, systolic BP remained higher with Prednisolone (107.94±11.29 vs 99.31±7.94 mmHg; p<0.001). A similar pattern was seen for diastolic BP: baseline values were comparable (67.46±8.35 vs 70.26± 10.45 mmHg; p=0.189), but at 3 months, Group B showed higher diastolic BP (71.79±10.67 vs 66.71±7.87 mmHg; p=0.017), and at 6 months the difference persisted (73.82±10.30 vs 67.50±6.92 mmHg; p=0.003). These trends support a more substantial hypertensive effect of Prednisolone. Both groups showed weight gain and improvements in lipid profiles, but the trends differed slightly. Weight increased in both arms, from 18.44±6.47 to 19.81±5.39 kg in Group A and from 19.41±5.91 to 22.47±6.91 kg in Group B over 6 months; however,

between-group differences at 3 and 6 months were not statistically significant (p=0.349 and p=0.076, respectively). Serum cholesterol fell substantially in both groups, but more so in the Deflazacort group: at 6 months, cholesterol was 247.28±94.91 mg/dl in Group A vs 342.85±134.26 mg/dl in Group B (p=0.001), with a borderline difference already at 3 months (293.90± 118.74 vs 346.33±128.99 mg/dl; p=0.062). Random blood sugar remained stable and comparable between groups at baseline, 3 months, and 6 months (all p>0.3), indicating no differential glycaemic effect between Deflazacort and Prednisolone.

DISCUSSION

This randomized controlled trial compared the efficacy and safety of Deflazacort and Prednisolone in children with an initial attack of steroid-sensitive nephrotic syndrome. Baseline demographic and clinical characteristics were broadly comparable between the two groups: mean age was 5.38±2.77 years in the Deflazacort group and 5.39±2.61 years in the Prednisolone group, with a clear male predominance in both arms. These findings are in keeping with previous reports, where children with idiopathic nephrotic syndrome are typically in early childhood, and boys are more frequently affected.[8-12] Height, weight, blood pressure, serum total cholesterol, serum creatinine, and urinary protein-creatinine ratio did not differ significantly at baseline, indicating that the treatment groups were well balanced clinically and biochemically. However, serum albumin was slightly lower in the Deflazacort arm, suggesting marginally more severe hypoalbuminemia in that group. The principal finding of this study is that Deflazacort achieved remission more rapidly and maintained it for a longer period compared with Prednisolone.

Children treated with Deflazacort attained remission in 6.73±2.80 days, whereas those in the Prednisolone group required 8.35±2.08 days (p=0.005). The duration of remission after the initial episode was also significantly longer with Deflazacort (154.32±19.65 days vs 120.23±36.06 days; p<0.001). These results are consistent with the work of Ravish et al (2015), who reported a shorter time to remission in the Deflazacort group (10.25±2.4 vs 12.55±1.44 days; p=0.012), and with Broyer et al (1997), who suggested that equipotent doses of Deflazacort exert a more prolonged effect on T-cell function than Prednisolone.[12] Piccardo et al (1987) demonstrated that Deflazacort-induced changes in T-cell number and function can persist for up to 72 hours, whereas similar changes with Prednisolone revert toward baseline within 24 hours.[13] Given that minimal change nephrotic syndrome is considered a T cell–mediated disorder, this mechanistic difference likely underpins the superior early remission profile observed with Deflazacort. Relapse behaviour during follow-up further supports Deflazacort's superiority in sustaining disease control. Relapse occurred in 48.8% of patients in the Deflazacort group compared with 66.7% in the Prednisolone group, with a significantly more favourable overall relapse pattern in the Deflazacort arm (p=0.027). Notably, multiple relapses (≥2 episodes) were more frequent in the Prednisolone group (41.1%) than in the Deflazacort group (12.2%). Broyer et al (1997) also reported a significantly higher relapse burden with Prednisolone (2.8±1.8 relapses) compared with Deflazacort (0.9±1.4; p=0.002).12 Ravish et al (2015) observed a numerically higher relapse rate in the Prednisolone arm.[14] However, the difference was not statistically significant, while Caterina et al (2015) reported fewer relapse-free patients in the Deflazacort group in a different clinical setting.[15] Taken together, the present trial aligns more closely with Broyer et al and Ravish et al, indicating that Deflazacort may offer better relapse control in initial steroid-sensitive nephrotic syndrome.[12-14] With respect to growth and body composition, both groups showed incremental height gain over six months, and final mean heights were similar (105.54±15.56 cm vs 104.75±17.52 cm), with no significant between-group difference.

However, weight gain tended to be greater in the Prednisolone group, consistent with the known propensity of synthetic glucocorticoids to increase appetite and promote central fat deposition. Kurt et al (2008) reported less fat accumulation with Deflazacort than with Prednisolone, and Broyer et al likewise noted numerically higher weight gain in Prednisolone-treated children.[11,12] However, this difference was not always statistically significant. Adult and paediatric data suggest that Deflazacort has a more favourable osteometabolic profile, with a higher RANKL: OPG ratio and less impairment of bone turnover, suggesting relative bone-sparing properties.[10-16] The short follow-up in the present study limits firm conclusions on long-term growth and bone health, yet the absence of a significant height penalty with Deflazacort over six months is reassuring. Adverse effect patterns in this trial clearly favoured Deflazacort. Hypertension and hypertrichosis were consistently more frequent in the Prednisolone group at both 3 and 6 months.

At 6 months, mean systolic blood pressure was significantly higher with Prednisolone (107.94±11.29 vs 99.31±7.94 mmHg), as was diastolic pressure (73.82±10.30 vs 67.50±6.92 mmHg). These findings are concordant with previous studies, treatment-emergent hypertension in children receiving Prednisolone but not Deflazacort.[7-15] Mechanistically, Deflazacort has weaker mineralocorticoid activity and minimal sodium-retaining effect compared with Prednisolone, in which mineralocorticoid receptor activation is more pronounced.[17] Dyslipidaemia also appeared more pronounced with Prednisolone, with significantly higher total cholesterol at 6 months (342.85±134.26 vs 247.28±94.91 mg/dl; p=0.001), mirroring the findings of Kurt et al (2008), who observed a greater rise in total cholesterol, LDL, and lipoprotein B2 under Prednisolone therapy.[11] In contrast, random blood glucose remained

normal and comparable in both groups throughout follow-up, indicating similar short-term glycaemic safety.

Limitations

This study has some limitations, including a relatively modest sample size, a single-centre design, and a six-month follow-up period that may not capture long-term growth, bone health, or cumulative steroid toxicity. Bone mineral density, detailed body composition, and quality-of-life measures were not evaluated.

CONCLUSION

This study showed that DFZ was more effective, as it required a shorter time to induce remission and the achieved remission was maintained for a longer duration in the initial episode of Nephrotic Syndrome. The number of relapses in treatment with DFZ was less than that of prednisolone during the follow-up period. Side effects were more common in the case of prednisolone in comparison to deflazacort.

Recommendations

Deflazacort is a suitable alternative to prednisolone for the treatment of nephrotic syndrome in children, given its better efficacy profile and lower relapse rate observed in this study. However, these findings should be confirmed by larger, multicentre trials with extended follow-up periods, incorporating systematic measurement of serum levels of both deflazacort and prednisolone, better to characterize their pharmacokinetics, pharmacodynamics, and exposure-response relationships.

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