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COMPARATIVE ANALYSIS OF HEMATOLOGICAL, BIOCHEMICAL PARAMETERS AND CLINICAL FEATURES IN STEROID RESISTANT AND STEROID SENSITIVE PEDIATRIC NEPHROTIC SYNDROME: A CASE CONTROL STUDY

Maxsuma Xudayberganovna Sultanova
Phd Dotsent 1-Faculty and Hospital Therapy
Tashkent State Medical University Tashkent, Uzbekistan
msultonova@gmail.com

Abstract

Nephrotic syndrome (NS) is a chronic renal pathology, classified into steroid sensitive (SSNS) or steroid resistant (SRNS) groups based on response to corticosteroids. Treatment of choice in pediatric NS is always corticosteroid. Steroid resistant nephrotic syndrome in paediatric patients may account for 20 to 30 % of total NS cases and it is significant concern for paediatricians. Steroid resistant cases are difficult to treat and have higher risk of development of end stage renal disease. A case control study was conducted among 48 children suffering from nephrotic syndrome, aged 2–12 years. Among them, 36 patient showed sensitivity to steroid treatment and 12 showed resistance. Haematological markers (haemoglobin, haematocrit, RBC count) and biochemical markers (albumin, proteinuria, urea, creatinine) were compared. Clinical features (anaemia, hypertension, severity of enema) were also compared. Independent t-tests and chi square tests were used for determining statistical significance ($p < 0.05$ was considered significant). Haemoglobin was

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significantly decreased in SRNS patients (p = 0.0002). RBC count, haematocrit, albumin were reduced in SRNS but not statistically significant. Proteinuria was significantly higher in SRNS (p = 0.001). Presence of hypertension and severity of edema was statistically significant among the case and control groups (p<0.001). SRNS patients showed much reduced haemoglobin and albumin values with more proteinuria than SSNS patients. The findings accentuate the relevance of prompt identification and focused therapy in SRNS. Resistance to steroid has poorer prognosis as significant glomerular damage may injure their kidney and overall health outcomes.

Keywords: Steroid resistant nephrotic syndrome, hypertension, edema, Pediatric nephrotic syndrome, Anaemia, Proteinuria

INTRODUCTION

Nephrotic syndrome is defined by heavy proteinuria (>1 g/m²/day) and hypoalbuminemia (below 3 g/dL). Edema is caused secondary to plasma oncotic pressure loss and hypovolemia and secondary renin-angiotensin-aldosterone axis stimulation with sodium and water retention. Pathogenesis of edema can be varied in some other individuals who exhibit primary sodium retention and intravascular volume expansion. Hypoalbuminemia also induces hepatic lipoprotein synthesis resulting in hypercholesterolemia. Others may have microscopic hematuria and hypertension.

More than 91% of childhood nephrotic syndrome is primary (or idiopathic). Other causes such as amyloidosis, vasculitis, systemic lupus erythematosus, and hepatitis B nephropathy are uncommon.

Nephrotic syndrome in children is broadly categorized into two groups on the basis of corticosteroid responsiveness, steroid-sensitive nephrotic syndrome (80-85% of cases) and steroid-resistant nephrotic syndrome (10-15%) cases. Based on histology, two forms are recognized: (1) A non-proliferative glomerulopathy

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also called minimal change disease or nephrotic syndrome (MCD or MCNS); and (ii) nephrotic syndrome with widespread glomerular lesions. These are focal segmental glomerulosclerosis (FSGS), mesangial proliferative GN, and C3 glomerulopathy; membranous nephropathy is exceptional.

Steroid-sensitive disease (most commonly MCNS) has excellent long-term prognosis. The steroid-resistant form (characteristically with severe glomerular lesions) has poorer course and some patients have progressive renal failure. [1,8,9]. Steroid resistance is diagnosed, if remission does not occur after treatment with prednisolone, in a dose of 2 mg/kg/day ((60mg / (m ^ 2)) / d) for 6 weeks. Nephrotic syndrome that does not respond to corticosteroids from the start is termed initial (primary) steroid resistance, and that which acquires steroid resistance during the management of a relapse, is termed late (secondary) steroid resistance. Steroid-resistance should be assessed in all patients. Histology is typically suggestive of PSGS or MCD. Occasionally, and in older children, extensive lesions such as membranous nephropathy are sometimes found.

Nephrotic syndrome presenting in infancy (<3 months, known as congenital NS) is typically caused by genetic dysplasia of the podocyte, and should never be treated with steroids or other immunosuppressives. Around 20-30% of children with sporadic initial steroid resistance, particularly those with onset in infancy, family history of same in family members, or syndromic presentation, carry homozygous, compound heterozygous or hemizygous NPHS2 (podocin), NPHS1 (nephrin), PLCE1, WT1 and COL4A5 mutations. CD2AP dominant mutations, TRCP6, ACTN4 and INF2 mutations present very infrequently. More than 80 genes are associated with steroid resistance mutations. Next-generation sequencing, by means of targeted gene or whole exome approach, is useful in screening. Patients with a genetic underlying etiology are resistant to immunosuppressive medications, show progressive renal insufficiency, and unlike nongenetic FSGS (which recurs on transplant), show low risk of recurrence. Resistant cases may progress to renal failure with high risk of chronic kidney

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disease (CKD) and end-stage renal disease (ESRD). Hematological and biochemical markers vary markedly in these subtypes. This study aims to compare these markers between SSNS and SRNS patients [2,4,7].

MATERIALS AND METHODS

Study Population

This case control study was conducted on 48 children with nephrotic syndrome of age 2–12 years in Tashkent Medical Academy. Patients were divided into SSNS (36) or SRNS (12) based on response to Prednisone 60 mg/m^2 Body surface area/day within 4 weeks.

Inclusion Criteria

Established cases of primary nephrotic syndrome, No history of prior immunosuppressive therapy other than steroids.

Exclusion Criteria

Secondary nephrotic syndrome (e.g., lupus nephritis), Severe systemic illness with impairment of hematological parameters.

Laboratory Parameters Examine

Hemoglobin, hematocrit, RBC count, Serum albumin, proteinuria, urea, creatinine

Statistical Analysis

Independent t-tests were utilized to compare between groups, and $p < 0.05$ was considered statistically significant. Anemia is diagnosed by critaria determined by WHO as mentioned in table I. Hypertension is determined by American academy of pediatrics guidelines as given in table II.

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WHO CLASSIFICATION OF ANEMIA IN YOUNG POPULATION

Child age	Degree of Anemia (measured by hemoglobin in g/dL)		
	Mild	Moderete	Severe
6 – 59 months	10 -10.9	7-9.9	<7
5-11 years	11-11.4	8-10.9	<8
12-14 years	11-11.9	8-10.9	<8
Above 15 years	11-11.9	8-10.9	<8

HYPERTENSION DIAGNOSIS GUIDELINES

HTN Stages	Children Aged 1to 12 years (Percentile Based)	Adolescents ≥ 13 years (mm Hg based)
Normotensive	< 90th percentile	< 120/<80
Elevated bp	90th percentile or ≥ 120/80 mm Hg (lower) to < 95th percentile	120-129/< 80
Stage 1	≥ 95th percentile to < 95th percentile + 12 mm Hg or 130/80 to 139/89 (lower)	130-139/80-89
Stage 2	2 ≥ 95th percentile + 12 mm Hg or ≥ 140/90 (lower)	≥ 140/90

RESULTS

Forty-eight patients fulfilled the inclusion critaria. twelve subjects in case group (SRNS) and thirty-six patients in control (SSNS) group. Baseline characteristics are shown in table III. The average age and gender distribution shows no significance with p value of 0.76 and 0.738 respectively.

Among the hematological parameters, only hemoglobin was found to be significantly decreased in SRNS group compared to SSNS ($p = 0.0002$). Anemia was more common in SRNS cases, as it was observed in 50.00% of SRNS patients compared to 27.78% of SSNS patients ($p = 0.289$). Hematocrit and RBC counts were found to be lower in the SRNS group, but the differences were not found to be statistically significant ($p > 0.05$) as shown in table IV . Hypertension was also significantly more common in SRNS patients, being present in 100.00% of SRNS compared to 36.11% of SSNS ($p <0.001$).97% of SRNS patients presenting with

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moderate to severe edema compared to 75% in SSNS (p <0.001) as shown in table V.

Proteinuria was significantly higher in SRNS patients, and the mean levels in the two groups were significantly different (p = 0.001) as mentioned in table VII. Albumin was significantly lower in SRNS patients, and the mean difference was statistically nonsignificant (p = 0.15). The serum urea and creatinine levels were not significantly different in the two groups (p = 0.47 and p = 0.38, respectively) as depicted in table VI.

Overall, the study revealed hemoglobin level, hypertension, edema severity, and proteinuria to be significantly associated with SRNS, while age, sex, RBC count, hematocrit, and renal function tests (urea and creatinine) were not statistically significant between the two groups.

BASELINE CHARACTERISTICS OF SUBJECTS

Variable	SRNS (n=12)	SSNS (n=36)	p-value
Age (years)	6.5 ± 2.8	6.8 ± 3.0	0.7619^{a}
Gender (M/F)	7/5	19/17	0.738^{b}

t test analysis

a.

CHI SQUARE TEST ANALYSIS

HEMATOLOGICAL PARAMETERS

Variable	SRNS (n=12) (mean±sd)	SSNS (n=36) (mean±sd)	p-value
RBC (million/µL)	4.1 ± 0.5	4.3 ± 0.6	0.30440^{a}
Hemoglobin (g/dL)	9.8 ± 1.2	11.3 ± 1.1	0.0002^{a}
Hematocrit (%)	34.8 ± 4.1	36.2 ± 3.5	0.2561^{a}
Anemia Prevalence (%)	50.00	27.78	0.289^{a}

a.

t test analysis

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CLINICAL FEATURES

Hypertension	**12 (100%)**	**13(36.11%)**	$< 0.001^a$
No hypertension	0(0%)	23(63.88%)	
Mild edema	3%	25%	$< 0.001^a$
Moderate to severe edema	97%	75%	

chi square test analysis

BIOCHEMICAL MARKERS AND URINE ANALYSIS RESULTS

Parameter	SSNS	SRNS	P-value
Creatinine (mg/dL)	0.52±0.14	0.56±0.12	0.38[a]
Urea (mg/dL)	28.4±7.1	30.1±6.8	0.47[a]
Albumin (g/dL)	2.58± 0.13	2.97 ± 0.15	0.15[a]

t- test analysis

URINE ANALYSIS

Proteinuria (mg/m²/hr)	**3.6 ± 1.2**	**5.1±1.5**	$\mathbf{0.001^a}$

t- test analysis

DISCUSSION

Most common characteristic clinical features of nephrotic syndrome are massive proteinuria, hypoalbuminemia, and edema. The most common etiology among children is Minimal Change Disease (MCD). Children with steroid-resistant nephrotic syndrome (SRNS), however, have non-minimal change histology such as focal segmental glomerulosclerosis (FSGS) [1,2].

Hemoglobin levels were lower among SRNS patients compared to SSNS, meaning that anemia was more present in the steroid-resistant group. Nephrotic syndrome-associated anemia could be due to chronic inflammation, iron deficiency, and dysfunctional production of erythropoietin due to impairment of the renal system. More studies have indicated that anemia is more prevalent in SRNS due to ongoing proteinuria as well as injury to the glomeruli [3]. Hypoalbuminemia produces reduced erythropoiesis due to malfunctioning

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transport of iron. Similar findings were observed other studies, where a higher incidence of anemia was observed in SRNS patients [4,6,7,17]

Hypertension remained a prominent risk factor for SRNS in our study. Steroid-resistant disease was more prevalent in hypertensive children, as described in previous research. Hypertension in SRNS is most likely linked to sodium retention and glomerular injury, compromising renal autoregulation. Persistent proteinuria and endothelial dysfunction are the causes of elevated blood pressure, hence hypertension as a possible predictor of steroid resistance. Our findings agree with recent studies which noted that hypertension was much more common in SRNS patients than in SSNS [3,5].

Edema is characteristic of nephrotic syndrome and occurs when albumin concentration falls below 2.7 g/dL. Mild edema in our study was also highly correlated with SRNS and is evidence for the overfill hypothesis involving primary renal sodium retention as a cause of edema in steroid-resistant disease. Severe edema is the hallmark of SSNS with resultant secondary sodium retention from RAAS activation due to hypoalbuminemia. Our results are in agreement with the reports suggesting SRNS patients to have more chronic but mild edema by virtue of having an intrinsic sodium retention defect of the kidneys [4,9,11,17].

Proteinuria is the characteristic of nephrotic syndrome, and elevated proteinuria levels were highly associated with SRNS in our data. Persistent massive proteinuria is a feature of steroid resistance and signifies ongoing glomerular injury. SSNS children usually go into remission following steroid administration, decreasing proteinuria, while patients with SRNS are unresponsive to this effect whereby they indicated that elevated baseline proteinuria was correlated with unfavourable steroid response [10,12]. Treatment of these resistant patients is quite a challenge since they tend to display highly variable responses to treatment with immunosuppressant drugs. This variability can also be compounded by side effects that accompany long-term treatment regimens and by continued risks of progressive kidney injury despite treatment. Of particular instances are those

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involving patients with steroid-resistant minimal change disease, post-streptococcal glomerulonephritis (PSGN), or mesangioproliferative glomerulonephritis (GN). Treatment regimens that are used in such particular instances tend to be quite similar and follow similar patterns. One of the foremost predictors of kidney outcome in such patients is how they would respond in terms of proteinuria to therapy administered to them and not on the exact underlying kidney histological findings that are noticed. Of key consideration in treatment is not merely to induce and sustain remission of proteinuria optimally, but also to keep side effects of drugs to a minimum through therapy.

Treatment regimens used tend to involve multi drug therapy that includes a treatment with an immunosuppressive agent, usually in conjunction with prednisolone, and with an angiotensin converting enzyme (ACE) blocker. In general terms, tolerable clinical responses to therapy using regimens involving treatment with either cyclosporine or tacrolimus with gradually declining doses of prednisolone and with an ACE blocker such as enalapril or ramipril have been documented. Although optimal therapy is to get to a state of complete remission, it is worth noting that even to get to a state of partial remission with either proteinuria of 1-2+ or spot urine protein to creatinine of between 0.2 to 2 mg/mg is acceptable by clinical standards. In general terms, those patients that are to benefit positively with therapy tend to register with improvements measurable by about the time of around 3 to around 6 months. In those instances that using calcineurin inhibitors is not a viable choice, a treatment regimen using six pulses of intravenous cyclophosphamide with alternate day prednisolone can be used for such patients. However, it is important to highlight that with such a particular treatment strategy, on a general note, the outcomes are less optimal compared to other treatment regimens [1] .

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CONCLUSION

The study results show the contrast in the hematological and biochemical parameters of children suffering from necrotic syndrome with and without resistance to corticosteroids. Age and gender distribution did not show any significant difference indicates that nephrotic syndrome does not show any particular age or gender distribution in pediatric population [13,14,15,16]. Here an approach is made to determine the difference between the steroid resistant and sensitive cases using the help of blood analysis and urine analysis parameters. Hematological analysis shows that hemoglobin level can be an useful indicator in disease prognosis. Proteinuria and hypoalbuminemia levels are significantly correlated with both groups which make them important biomarker for disease monitoring. Hypertriglyceridemia is a consequence of nephrotic syndrome as liver produces more lipid and it has adverse outcome on the patients [15,16,17]. Also, we found more anemic patients in resistant group. Although it was not significant but anemia can have worse health outcomes in the population. Our focus towards the clinical feature difference in both groups especially on hypertension leads to the result that hypertension was significant in resistant group and oedema also more pronounced in that group. This result implies that better monitoring of blood pressure and edema management is particularly very necessary in resistant cases then sensitive [17]. Proteinuria was found to be a significant parameter which corelates in both cases significantly. Loss of protein may lead to worsen outcomes Thus making it an important marker for prognosis. Our target was to compare and contrast between the parameters and determine the potential prognostic marker for resistant and sensitive cases. Nowadays arising cases with resistant nephrotic syndrome is a very difficult challenge because their management is very difficult and the disease diagnosis at proper time is also very necessary.

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