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A case report on Nephrotic syndrome – 2nd relapse

N. Anu Prasad

Doctor of Pharmacy, Arulmigu Kalasalingam College of Pharmacy, Krishankoil, Tamilnadu, India.

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ABSTRACT: Nephrotic syndrome is characterized by permselectivity at the glomerular capillary wall, which results in the inability to limit protein loss in the urine. The nephrotic range proteinuria is characterized as a proteinuria that exceeds 1000 mg/m2 per day, or a urine protein-to creatinine ratio greater than 2 mg/mg. Genetic mutations or congenital infections may be the cause of congenital illness. More people have acquired diseases, which are typically idiopathic. The prognosis of nephrotic syndrome in children is associated with a variety of steroid responsiveness, from steroid-sensitive nephrotic syndrome to steroid-resistant nephrotic syndrome. Many significant hemostatic proteins of comparable sizes are also pathologically discharged in the urine because the primary glomerular defect of nephrotic syndrome causes the leaking of high molecular mass proteins, at least the size of albumin. The most common treatment for children with nephrotic syndrome is oral corticosteroids. The standard preparations are prednisolone.

Corresponding author:
Mr. N. Anu Prasad
Research Scholar
Arulmigu Kalasalingam College of Pharmacy,
Krishankoil, Tamilnadu, India.
Tel: +91-9976977534
E. Mail ID: nmanojprasad6@gmail.com

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INTRODUCTION:

Nephrotic syndrome is a frequent chronic illness defined by changes in permselectivity at the glomerular capillary wall, which results in the inability to limit protein loss in the urine. The nephrotic range proteinuria is characterized as proteinuria that exceeds 1000 mg/m^2 per day, or a urine protein-to creatinine ratio greater than 2 mg/mg on a random basis [1]. Nephrotic syndrome (NS) in children is a common, though relapsing and remitting, renal condition with a wide clinical phenotype ranging from a single episode to infrequently relapsing, frequently relapsing, and steroid-resistant disease [2]. The nephrotic syndrome can be acquired or congenital. Genetic mutations or congenital infections may be the cause of congenital illness. More people have acquired

diseases, which are typically idiopathic. It is categorized as either a steroid sensitive disease or a steroid resistant condition depending on how well it responds to corticosteroid treatment. Additionally, nephrotic syndrome might develop as a result of infections, medications, or neoplasia [3]. The study of disease-related hemostatic derangements has attracted the most interest in pathophysiology in NS. Many significant hemostatic proteins of comparable sizes are also pathologically discharged in the urine because the primary glomerular defect of NS causes the leaking of high molecular mass proteins, at least the size of albumin (about 66 kD) [4]. Although 80 % of these children respond to corticosteroids, approximately half of them suffer from a relapsing or steroid-dependent course, which frequently results in various problems, hospitalizations, or even chronic renal failure [5]. The aim of this case report is to emphasize the need for managing the relapses of nephrotic syndrome in children with steroids according to the individual symptoms of the children.

CASE REPORT:

Clinical features:

A 3 ½ year old male child weighing 15.2 kg was brought by her mother to the outpatient department (OPD) of a tertiary care hospital in Virudhunagar. The child was presented with chief complaints of cough and cold for 8 days and periorbital puffiness for 5 days. The child was apparently normal for 8 days, and then he developed a cough and cold which was associated with a runny nose, dry cough, non - productive and aggravated at night time. He also has complaints of periorbital puffiness in the early morning for the past 5 days, which was reducing during the day. The child has a history of reduced urine output (oliguria) for the past 3 days which was straw coloured, not turbid in nature, not blood stained and abdominal distension since morning. The baby was delivered by C-section and weighed 2.75 kg after birth. The child was already diagnosed with nephrotic syndrome 1 yr back. On examination pitting pedal oedema was present over lower limbs and swelling over face was present on 3rd day of admission in hospital. Based on these clinical features, nephrotic syndrome 2nd relapse was suspected and specific laboratory testing was performed to establish diagnosis.

Past medical and medication history:

- The child is a known case of nephrotic syndrome, which was diagnosed on 18/7/22 and treated with Tab prednisolone 2 mg/kg/day on alternate days for 6 weeks and Tab furosemide 40 mg ½ OD × 1 week.
- 1st relapse of nephrotic syndrome appears at 1/2/23, it was diagnosed in private clinic on 2/2/23 and treated with Tab prednisolone 5 mg OD (6-0-0) for 6 weeks daily (till 15/03/23) then Tab prednisolone dose was reduced to 5 mg OD (4-0-0) for 4 weeks on alternate days.
- The child has a history of previous episodes of admission in hospital for upper respiratory tract infection.
- Known case of simple febrile seizure.

Family history:

- No history of significant illness in the family members.
- K/C/O simple febrile seizures in sibling.

Immunisation history:

- Immunised only up to 9 months, MMR vaccine done. Not immunised for 1½ yrs vaccination. DPT, OPV, MR-2 booster not given.

Developmental delay:

- H/O speech delay - +

Birth history:

- AN scan → Cystic swelling over left side forehead in fetus.
- Birth weight: 2.94 kg

Nutritional history

- Take a salt restricted diet.
- Take a protein rich diet.

Table 1. The Nutritional history.

Parameter	Expected	Actual intake	Inference
Calories (Kcal)	1250	1010	240 Kcal deficit
Protein (g)	20	16	4 g deficit

Child alert, awake, afebrile. Pallor +, B/L puffiness of eyelids, B/L mild pedal edema +.

USG abdomen and pelvis

- Kidney: CMD +
  - LK : 7.2 × 3.4 cm
  - RK : 6 × 2.6 cm
- Bladder: Distended.

Urine routine analysis:

- Sugar – Nil
- Albumin - +++
- Deposits – 2-6 pus cells

Others:

- Spot PCR urine – 3.3
- 24 hours urine protein: 230.8 mg/dL

Table 2. Investigation results.

Parameters	Results	References
Total count	10,900 cells	5,000 – 17,000 cells
Haemoglobin	13.2 g/dL	11-13 g/dL
Red blood cells	4.97 million cells	4.0 – 5.2 million cells
Platelet count	4. 0 Lakhs	1.5 – 4.5 Lakhs
Renal function test		
Urea	38 mg/dL	5-16 mg/dL
Creatinine	0.8 mg/dL	0.3 – 0.7 mg/dL
Serum electrolytes		
Sodium	133 mmol/L	136-145 mmol/L
Potassium	4.4 mmol/L	3.4-4.7 mmol/L
Chloride	106 mmol/L	98-107 mmol/L
Serum lipid profile		
Total cholesterol	386 mg/dL	< 170 mg/dL
TGL	595 mg/dL	< 75 mg/dL
HDL	77 mg/dL	>45 mg/dL
LDL	190 mg/dL	< 110 mg/dL
Liver function test		
Serum bilirubin	0.5 mg/dL	0.1 – 1.2 mg/dL
Total protein	3.9 g/dL	6-8.3 g/dL
Albumin	2.0 g/dL	3.5-5.5 g/dL

Clinical course:

After establishing diagnosis, supportive treatment including injection furosemide 0.5 mg/kg/ml IV twice a day, Tablet Prednisolone 2 mg/kg per day p.o., salt restricted diet, and adequate protein intake were given along with Tablet Amoxicillin 225 mg TDS and T.B-complex. The urine albumin, daily weight and blood pressure were monitored.

Successful control of periorbital oedema with the administration diuresis with furosemide was seen. The child was discharged with Tablet Prednisolone 5 mg, 6 tablets in morning and advised to review within 2 days with urine albumin report.

DISCUSSION:

The prognosis of NS in children is associated with a variety of steroid responsiveness, from steroid-sensitive nephrotic syndrome (SSNS) to steroid-resistant nephrotic syndrome (SRNS). In children, SRNS is the most common acquired cause of end-stage renal disease (ESRD) [6]. In a series of the International Study of Kidney Diseases in Children, minimal-change nephrotic syndrome (MCNS) contributed to 77 % of all cases of paediatric nephrotic syndrome. The long-term outcome of this condition is generally favourable, and prednisone treatment results in complete remission in one-third of patients; nevertheless, 30 % of these children develop a frequently relapsing course (FRNS) [7]. Patients with nephrotic syndrome are vulnerable to a range of problems, including thrombosis, infections, dyslipidemia, and renal impairment [8]. The most common treatment for children with nephrotic syndrome is oral corticosteroids. The standard preparations are prednisolone. Deflazacort is an oxazoline medication, which is a Prednisolone derivative having comparable anti-inflammatory and immunosuppressive action, but it has fewer side effects [1].

CONCLUSION:

We have presented a case of nephrotic syndrome and managed with corticosteroid and furosemide. Based on his past medical history and present clinical features, the actual diagnosis of nephrotic syndrome was made. Our patient was advised to attend monthly follow-up visits.

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