

# Case report on paediatric nephrotic syndrome

Shireen Prince<sup>1</sup>, Kunta Naresh<sup>1</sup>, R. Tulasi<sup>2</sup>

<sup>1</sup> Department of Pharmacy Practice, Malla Reddy Pharmacy College, Dhullapally, Secunderabad, Telangana, India

<sup>2</sup> Department of Paediatrics, Malla Reddy Institute of Medical Sciences, Secunderabad, Telangana, India

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## ARTICLE INFO

### **Corresponding author:**

Kunta Naresh  
Department of Pharmacy Practice  
Faculty of Malla Reddy Pharmacy College  
Dhullapally, Secunderabad-500100  
Telangana  
India  
Phone: 00919908557290  
E-mail: [kuntanaresh785@gmail.com](mailto:kuntanaresh785@gmail.com)

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## ABSTRACT

Nephrotic syndrome (NS) is a glomerular disorder typically characterized by gross proteinuria, hypoalbuminemia, hyperlipidemia, and peripheral oedema. We report the case of a 2-year-old male toddler weighing 15 kg with a 1-week history of swelling around the eyes and both legs, and generalized body swelling. She had a history of fever, cough and decreased urine output. Examination revealed bilateral pedal oedema (pitting type).

Laboratory investigations showed protein in urine, reduced serum albumin (2.0 g/dL) with elevated lipid levels. Although kidney biopsy could not be performed due to economic problem of the family, a diagnosis of idiopathic nephrotic syndrome (NS) was made based on clinical and laboratory findings.

The patient was mainly treated with furosemide, prednisolone and enalapril. Urine I/O charting (Intake/Output chart for assessing fluid intake and ability to pass urine in adequate amounts) was done daily until optimal results were obtained.

## INTRODUCTION

Primary nephrotic syndrome (PNS), also known as idiopathic nephrotic syndrome (INS), is associated with glomerular diseases intrinsic to the kidney and not related to systemic causes. The subcategories of INS are based on histological descriptions, but clinical-pathological correlations have been made (1). Diagnosis is generally based on clinical features and investigations including blood tests, renal imaging, and biopsy (2). The incidence of idiopathic nephrotic syndrome (INS) is 1.15–16.9 per 100 000 children, varying by ethnicity and region. The cause remains unknown but the pathogenesis of idiopathic NS is thought to involve immune dysregulation, systemic circulating factors, or inherited structural abnormalities of the podocyte. Genetic risk is more commonly described among children with steroid-resistant disease. The mainstay of therapy is prednisone for the vast majority of patients who are steroid responsive; however, the disease can run a frequently relapsing course, necessitating the need for alternative immunosuppressive agents. Infection and venous thromboembolism are the main complications of NS with also increased risk of acute kidney injury. Prognosis in terms of long-term kidney outcome overall is excellent for steroid-responsive disease, and steroid resistance is an important determinant of future risk of chronic or end-stage kidney disease (3).

## CASE REPORT

### *Clinical features*

A 2-year-old male toddler weighing 15 kg presented with a history of fever which is high grade continuous type associated with chills and rigors. The patient had cough (wet cough more in amount) whitish colour sputum not foul smelling. Swelling over face was present which initially started around peri-orbital (which is more during morning) and gradually progressed to face which

decreases by evening. The toddler had decreased urine output (oliguria). The baby was delivered by C-section and weighed 2.75 kg after birth. On examination pitting type of oedema was present over lower limbs and swelling over face was present. Based on these clinical presentations, nephrotic syndrome was suspected and specific laboratory testing was performed to establish diagnosis.

### *Laboratory findings*

The urine dipstick indicated for proteinuria, no signs of haematuria. Blood testing showed a significantly depressed C3 level of 0.638 g/L (reference interval 0.9-1.8 g/L) and hypoalbuminaemia of 2.0 g/dL (reference interval 3.5-5.5 g/dL) indicating nephrotic syndrome (NS). The urine creatinine level was – 620 mg/L (reference interval 400-3000mg/L) and APTT was prolonged- 47.7 Sec (reference interval 24-30 Sec). Serologic testing for active infections: anti-streptolysin-O titer was positive. The lipid levels were markedly increased as outlined in the Table 1. LDL was measured and calculated by enzymatic selective protection (Direct). The urine protein/creatinine ratio was found to be high (7.3). Mantoux test was done before administration of steroids which was negative.

### *Clinical course*

After establishing diagnosis, optimal supportive treatment including Enalapril p.o., Prednisolone p.o., intravenous albumin, furosemide, low salt intake, high caloric and protein diet were given along with Ceftriaxone and Ascoril-LS. The urine output and blood pressure was monitored.

Successful control of peripheral oedema with the administration of albumin and diuresis with furosemide was seen. The peri-orbital oedema and leg swelling reduced, and there was a concomitant increase in serum protein levels. The lipid levels also gradually decreased in due course of time without any medication.

**Table 1** Laboratory parameters

Parameters	Result	References
<b>Urine</b>		
Creatinine	620 mg/L	400-3000 mg/L
Protein	4574 mg/L	< 100 mg/L
Protein Creatinine ratio	7.3	< 0.2
<b>Serum electrolytes</b>		
Serum Sodium	131 mmol/L	136-145 mmol/L
Serum Chloride	96 mmol/L	98-107 mmol/L
Serum Potassium	4.6 mmol/L	3.4-4.7 mmol/L
<b>Serum lipid profile</b>		
Total Cholesterol	342 mg/dL	< 170 mg/dL
LDL Cholesterol	196 mg/dL	< 110 mg/dL
Triglycerides	329 mg/dL	< 75 mg/dL
<b>Others</b>		
APTT (plasma)	47.7 sec	24-30 sec
ASO titer (serum)	400 IU/mL	< 200 IU/mL
Serum Albumin	2 g/dL	3.5-5.5 g/dL

## DISCUSSION

The hallmark of INS is massive proteinuria, leading to decreased circulating albumin levels. The initiating event that produces proteinuria remains unknown. However, strong evidence suggests that INS, at least in part, has an immune pathogenesis.

The classical explanation for oedema formation is a decrease in plasma oncotic pressure, as a consequence of low serum albumin levels, causing an extravasation of plasma water into the interstitial space. The resulting contraction in plasma volume (PV) leads to stimulation of the renin-angiotensin-aldosterone axis and anti-diuretic

hormone secretion. The resultant retention of sodium and water by the renal tubules contributes to the extension and maintenance of oedema.

A more recent theory of oedema formation posits that massive proteinuria leads to tubule-interstitial inflammation, release of local vasoconstrictors and inhibition of vasodilation. This leads to reduction in glomerular filtration rate and sodium and water retention<sup>4</sup>.

INS is accompanied by disordered lipid metabolism. Apolipoprotein (apo)-B-containing lipoproteins are elevated, including very-low-density lipoprotein (VLDL), intermediate-density lipoprotein (IDL), low-density lipoproteins (LDL), with resultant increases in total cholesterol and LDL-cholesterol. Elevations in triglyceride levels occur with severe hypoalbuminemia. Also contributing to the dyslipidemia of INS are abnormalities in regulatory enzymes, such as lecithin-cholesterol *acyltransferase*, *lipoprotein lipase*, and cholesterol ester transfer protein (4,5).

Nephrotic syndrome is a hypercoagulable state; the increased risk of thrombosis can be attributed to two basic mechanisms:

1. urine losses of antithrombotic proteins and
2. increased synthesis of prothrombotic factors.

Abnormalities described in INS include decreased antithrombotic factors and increased synthesis of pro-thrombotic factors (6).

Risk of infection may be increased in INS because of low immunoglobulin IgG levels, which do not appear to be the result of urinary losses. Instead, low IgG levels seem to be the result of impaired synthesis, again pointing to a primary disorder in lymphocyte regulation in INS. The medications used to treat INS, such as corticosteroids and alkylating agents, further suppress the immune system and increase the risk of infection (7). The ASO test done in this patient had a positive result.

## CONCLUSION

We have presented a case of idiopathic NS successfully managed with corticosteroid, albumin, furosemide and enalapril. We could not perform kidney biopsy but could make a diagnosis based on clinical features and investigations, and fortunately our patient recovered and attends monthly follow-up visits.

## TAKE HOME MESSAGES/ LEARNING POINTS

- In order to establish the presence of nephrotic syndrome, laboratory tests should confirm nephrotic-range proteinuria, hypoalbuminemia, and hyperlipidemia.
- A 3+ proteinuria on dipstick is highly suggestive of nephrotic syndrome to be confirmed by appropriate laboratory work-up.
- Serologic testing for active infections should be done as the patients with NS are more prone to it.
- Mantoux test [purified protein derivative (PPD)] should be performed prior to steroid treatment to rule out TB infection.



## Contribution of authors

Shireen and Dr. Naresh conceived the idea and wrote and edited the manuscript.

Dr. Tulasi was the paediatrician managing the patient and contributed to the manuscript.



## REFERENCES

1. Pediatric Nephrotic Syndrome: Practice Essentials, Background, Pathophysiology. Available at: <https://emedicine.medscape.com/article/982920-overview>. (Accessed: 31st December 2019).
2. Souvannamethy, P. Management of Nephrotic Syndrome: A Case Report from Lao PDR. in Blood Purification 44, 31–34 (S. Karger AG, 2017).

3. Noone, D. G., Iijima, K. & Parekh, R. Idiopathic nephrotic syndrome in children. *The Lancet* 392, 61–74 (2018).
4. Brenner and Rector's *The Kidney E-Book* – 9th Edition. Available at: <https://www.elsevier.com/books/brenner-and-rectors-the-kidney-e-book/taal/978-1-4557-2304-1>. (Accessed: 31st December 2019).
5. Saland, J. M., Ginsberg, H. & Fisher, E. A. Dyslipidemia in pediatric renal disease: Epidemiology, pathophysiology, and management. *Current Opinion in Pediatrics* 14, 197–204 (2002).
6. Loscalzo, J. Basic implications of clinical observations: Venous thrombosis in the nephrotic syndrome. *N. Engl. J. Med.* 368, 956–958 (2013).
7. Rodríguez-Iturbe, B., Najafian, B., Silva, A. & Alpers, C. E. Akut Postinfectious GN. in *Pediatric Nephrology* (eds. Avner, E. D. et al.) 959–981 (Springer-Verlag, 2016). doi: 10.1007/978-3-662-43596-0.