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Insights into Nephrotic Syndrome in Pediarics: Case Series Analysis

Vankodoth Sireesha [a++*], Asiya Sultana [a#], P. Ramya Sri [a#], Ayesha Mateen [a#], M. Shiva [a#] and T. Rama Rao [b†]

[a] Department of Pharm D, CMR College of Pharmacy, Medchal, Hyderabad, Telangana, India.
[b] CMR College of Pharmacy, Medchal, Hyderabad, Telangana, India.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Report

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ABSTRACT

Nephrotic syndrome (NS) is one of the most common childhood kidney diseases. NS can affect children of any age from infancy to adolescence and predominantly occurs in the age group of 1–6 years. It is an illness caused by idiopathic diseases like minimal change nephrotic syndrome (MCNS) and focal segmental glomerulosclerosis (FSGS), membrane proliferative Glomerulonephritis, membranous Glomerulonephritis and is characterized by increased permeability across the glomerular filtration barrier. NS is classified as primary, secondary, and congenital. It consists of four clinical features like severe proteinuria, edema, hyperlipidaemia, hypoalbuminemia. The main cause of nephrotic syndrome are diseases associated with drugs and rarely genetic disorders. The pathogenesis of the disease affects various biological functions linked to loss of proteins negatively, which results in systemic complications which may be disease and

[++] Assistant Professor;
[#] Pharm-D PB;
[†] Principal and Professor;
**Corresponding author: E-mail: sireeshaganesh59@gmail.com; Int;*

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drug-associated complications. Using drugs to treat NS leads to several complications which include improper growth, metabolism, behavior change inpatient. Itis a chronic relapsing disease for most of the steroid response drugs for treatment of nephrotic syndrome. Glucocorticoids, immunosuppressants and biological agents are used for the treatment of the diseases. In this case series, we presented a case series of nephrotic syndrome in pediatrics. Total 4 patients were taken into considerations. The patients came with a chief complaints of swelling of face, foamy urine, abdominal pain, dark coloured urine with decreased urine output, joint pains. The laboratory investigations for all the 4 patients showed that there were decrease in albumin levels (hypoalbuminemia), decrease in serum creatinine levels and protein in urine, although kidney biopsy couldn't be perform but the diagnosis was made based on clinical and laboratory findings. The patients were mainly treated with prednisolone, pantoprazole, enalapril, nifedipine, amoxiclav, and even albumin transfusion were done. NS was successfully managed and fortunately the patients recovered.

Keywords: Corticosteroids; immunosuppressants; congenital; proteinuria; nephrotic syndrome; glomerulonephritis.

1. INTRODUCTION

Nephrotic syndrome (NS) is one of the most common renal diseases in children. NS can affect children of any age from infancy to adolescence and predominantly occurs in the age group of 1–6 years [1]. Worldwide the prevalence of childhood NS is approximately 16 casesper100,000 children with anin cadence of 5 per 100,000 children. Pediatric nephrotic syndrome (NS) is characterized by a 4 findings such as severe proteinuria, hypoalbuminemia, hyperlipidemia and edema. These are caused by the highly increased permeability of glomerular filtration barrier to plasma protein and severe loss of large amount of plasma protein from urine [2]. Proteinuria is characterized by presence of urine protein to creatinine ratio greater than 2 gm/*m2 per day* in children and greater than 3gm/*m2 per day in Adults.* plasma albumin less than 2.5g/dl indicates hypoalbuminemia [3]. Nephrotic syndrome is classified into idiopathic (INS) and congenital depending on the cause and timing of proteinuria. INS accounts for 90% of NS in children and 80% of NS in children is minimal change NS (MCNS). Focal segmental glomerulosclerosis (FSGS) is the second most common disease in paediatric after MCNS [4]. NS can be congenital or acquired. Congenital NS may be due to a genetic mutation. Acquired is more common and is usually due to infections, pharmacological agents. The Common primary causes of nephrotic syndrome are intrinsic kidney diseases such as membranous nephropathy, minimal-change nephropathy and focal glomerulosclerosis. Secondary causes of nephrotic syndrome includes Diabetes mellitus, immune disorders like lupus erythematosus, glomeruli acute post-infectious nephritis, membrane proliferate glomerulonephritis, Infectious diseases like human HIV, hepatitis B virus and hepatitis C, Leprosy, Syphilis few Drugs may also cause NS such as NSAIDs, Penicillamine, Bisphosphonates, rifamycin, heroine, Lithium [5].

The clinical manifestation of NS includes -Severe swelling (edema), specially around your eyes, ankles, fingers and feet, Foamy urine (heavy proteinuria), Weight gain (due to fluid retention), Fatigue, Loss of appetite, Hypoalbuminemia, Hyperlipidemia, airway hyper responsiveness, skin pruritus, repeated eczema, skin allergy [2]. The metabolic consequences of the NS include- Hypocalcemia and bone abnormalities, atherosclerosis, Hypercoagulability. Additional consequences include Hypertension due to reduced kidney function, Sepsis, Thromboembolism [5]. The pathophysiology of NS explains the mechanism involved during the disease process- Proteinuria and Hypoalbuminemia: Proteinuria occurs due alterations in the glomerular filtration barrier which is composed of 3 layers, the endothelium, the glomerular basement membrane, the visceral glomerular epithelium comprised of podocytes and their slit diaphragms. Podocytes play a passive role in the process of glomerular filtration but the concept changed with the discovery that mutation of a protein located at the slit diaphragm known as nephrin, which causes congenital nephrotic syndrome. A number of acquired and inherited diseases now are attributed to defects in the slit diaphragm protein complex thus leading to hypoalbuminea and Proteinuria. Edemais caused due to decreas in plasma pressure due to loss of serum albumin results in edema causes water to efflux into the interstitial space such movement reduces the intravascular volume leading to renal hypoper

fusion and stimulation of there in-angiotensin-aldosterone (RAA) system. Aldosterone increases reabsorption of sodium, particularly at the level of the distal segments of the nephron. Plasma volume has been shown to be decreased only in some children who have minimal change nephrotic syndrome (MCNS), particularly during the initial phase of a relapse. Hyperlipidaemia occurs due to decreased osmotic pressure, so inorder to maintain this pressure, there is Increased concentrations of very low-density lipoprotein (VLDL),intermediate-density lipoprotein (IDL), and low- density lipoprotein (LDL) result in increased serum cholesterol and triglycerides concentrations thus causes Lipiduria and results in fatty cast [6]. The loss of proteins negatively affects various biological functions which can result in complications. Complications of childhood NS are divided into two types-disease and drug associated complications. Disease associated complications include Hypercoagulation which occurs due to loss of protein C and protein S and Anti Thrombin III in urine of the pateints with NS, Increases the riskof infection due to loss of immunoglobulins in the urine , Hypothyroidism due to loss of Thyroid bindinggloubulins, Vitamin DDeficeincyisdueto loss of cholecalciferol binding protein. Drug-associated complications include sensitivity to steroids, steroid treatment is associated with severe side effects such as growth retardation, hypertension, osteoporosis, and bone fractures and is also linked with psychological stress.

The diagnostic procedures for Nephrotic Syndrome are Urine examination includes proteinuria and Lipiduria, the presence of free lipid or lipid within tubular cells. Blood tests includes serum albumin level is classically low in NS. Ultrasonography and Renal biopsy are also carried out in some patients which shows the presences of sclerosis and effacement of foot process of the podocyst levels [5]. In most patients the treatment and management strategies carriedout in children with NS includes Specific treatment in children-For children with INS corticosteroids are the firstline treatment. Alternative immunosuppressive agents are used (eg: cyclophosphamide, calcineurin inhibitors). Rituximab, an antibody against B-cells, has proved an effective steroid-sparing agent in children with steroid-dependent INS as there is a chance of relapse with in 2 weeks of cessation. Congenital nephrotic syndrome (eg: from mutations in genes *NPHS*or*WT1*) is often treated with bilateral nephrectomy followed by dialysis and transplantation. Acute Nephrotic Syndrome in Childhood is treated by a good parental, patient education and close outpatient follow-up care. Hospitalization should be considered if any of the following are present such as Generalized edema, bacterial sepsis, pneumonia, thromboembolism, Diureticsare also prescribed if needed. Furosemide (1mg/kg/day) and spironolactone (2mg/kg/day) will help in fluid retention if severe, provided no signs of acute kidney injury or volume contraction are evident. To prevent infection, oral penicillin can be prescribed for children. Acyclovir should be given if the patient develops skin allergy. Diet and Activity includes adequate protein intake (1g/kg/day). A low-salt diet helps to limit the fluid retention and edema that occurs in NS. Quantify dietary intake of sodium down to 2 g (88 mEq)/day or less. Increases daily physical activities helps to reduce the risk of blood clots. Long-Term Monitoring includes Ongoing use and adjustment of diuretics and angiotensin antagonists are done according to the amount of edema and proteinuria that a patient has. Follow-up care in patients with NS also includes immunizations and monitoring for corticosteroid toxicity. Supplemental calcium and vitamin D may help in maintaining bone loss [7,8]. Categorization is based on the reaction to corticosteroid treatment, dividing it into steroid-sensitive or steroid-resistant disease, while many patients exhibit a positive response to steroid therapy, leading to a favorable prognosis, however, treatment duration influences psychosocial factors like internal struggles and anxiety/depression, potentially diminishing quality of life [1,3].

In this case series, we presented a case series of nephrotic syndrome in pediatrics, aiming to discuss the diagnosis, treatment and follow-up of such cases. We choose this case series because there is no clear guidelines that favor one treatment over another in management of NS in pediatrics.

The cases were collected from the in-patients Department of Pediatrics. Gandhi hospital, Secunderabad, 500003, Telangana state.

2. CASE PRESENTATION

2.1 Case 1

A 7-year-old-male patient with the weight of 12kg presented with the chief complaints of swelling of face since 3 days as shown in the Fig. 1, slight weigh gain & foamy urine with decrease urine

output. The Table 1 shows the diagnostic clinical investigational results which shows that the patientis was alert, stable, afebrile with the BP of90/60mmHg, pulserate-110bpm, RR-27/min, CVS-S1S2+, Bae+clear, P/A-soft, and Immunization taken upto 14 months, patient had good developmental history, physical examination confirmed puffiness of face.Upon laboratory investigations of nephritic components,the CUE test (dipsticktest) confirmedasproteinuria+3 (Thedipstickshows 3+ or more of protein in the urine for 3 days in a row, this means the child is having a relapse. urine/urine creatinine-both of the irrational came to be 1.5,Albumin-2g/dl hypoalbuminemia (decrease in these levels indicate loss of proteins in urine) no haematuria and hyperlipidaemia recorded, WBC-7.3 cumm, RBC-5.45 million cells per microlitre,HGB-14.8g/dl, HCT- 43.3%, MCV-79.4fl, MCH-27.2pg, MCHC-34.2g/dl, PLT-358 ul, NEUT-42.2%, LYMP-51.1%, MONO-6.6%, ESINO-0, BASO-0.1, UREA-15mg/dl, CR-0.1mg/dl, ALT-9U/l, ALPO4-187, GLOB-2.72, SODIUM-119(low), Potassium 4.6, Chloride -98. Based on the above findings, the patients diagnosed with Nephrotic syndrome with Grade3 Hypertension with first relapse which means now it's a second relapse.The Table 2 indicates the management strategies of the patient which was carried out ,initially the patient was managed with tab Prednisolone - 1.5mg/kg/alt (1 ½ alt day) it acts by lowering the immunity. Most children, with nephrotic syndrome respond to corticosteroid drugs (prednisone, prednisolone) reducing the risk of serious infection. However they usually have repeat episodes, which are often triggered by viral infections.,TabEnalapril-0.4mg/kg/day (1tab/po/OD) which is an ACE inhibitor it acts by lowering hypertension, Tab Pantoprazole 40mg (1/4tab/po/OD) BBF. It is a proton pump inhibitor (PPI) helps in reducing gastric acid secretion thereby producing reflux relief., syrup Calcium P- 7ml/kg/day (50mg/po/OD) to treat calcium deficiencies (likehypocalcaemia, prevention of bone abnormalities, reducing the risk of fracture, supporting overall health. Tab coenzyme Q10 (1tab-30mg). Monitoring the vitals is necessary. However, a high-protein diet isn't recommended for nephrotic syndrome. Too much protein is dangerous because it can damage nephrons and cause renal insufficiency. Low to moderate protein intake is recommended, depending on the condition of the kidneys. However Patient vital signs began improving after 4-5 days of initial treatment. All investigation were normal after 3 days. However patient was given supportive care as mentioned above, until the patient was discharged with maximal improvement [9-12].

2.2 Case 2

A 14-year-old male patient with the weight of 28kg, presented with complaints of abdominal pain, dark coloured urine with decreased output. The child was reported with known case of hypertension and post streptococcal glomerular nephritis (PSGN). patient was treated for hypertension in past. The Table 1 shows the diagnostic clinical examination results of the patient which indicates that the patient is active and afebrile, with the pulse rate of 112 beats per minute, blood pressure was found to be 140/100 mmHg, P/Awas found to be soft, non-tender, No organomegaly. On investigations the results of nephritic components were determined-complete urine examination with spot urine creatinine-22mg%, spoturineproteins-23mg%, Urea - 23mg\dl, Creatinine-0.39mg/dl, Albumin-4.07g/dl with normal levels, Total protein-6.59 mg/dl(proteinuria) no haematuria recorded,magnesium-2.44mg/dl,Globulin-2.52g/dl, sodium-140mEg/L, Potassium-5.8mEg/L, Chlorine-109mEg/L, ALT-24 U/L, AST-34 U/L, ALP-880 IU. Ultrasound: right renal calculi with PCO2-27.00mmHg(↓)PO2-42.9mmHg(↑)Renal Angiography:6mm calculi noted in pelvis, Mild dilation of pelvic alyceal system noted, Atelectatic band opacitieswere noted in antero-basal segment of right lower lobe, Renal artery Doppler ratio:0.59.Based on findings the patient diagnosed with Nephrotic syndrome with non obstructive renal calculi.

The Table 2 shows the management strategies carried out to the patient, was managed with (Nifedipine-0.25mg) Anti-hypertensive drug in past as the history of hypertension by maternal grand father of the patient. Initially managed with Antibiotics(syrup-Amoxyclav-3ml twice a day) for trating infections, Proton pumpdue to fluid retention inhibitor (T.Lanazol-7.5mgBBF) helps inreducing gastric acid secretion thereby producing reflux relief, dietary supplement (syrup-Zinc-5mloncea day) . Patient was suggested to have plenty of fluids, oral rehydrated salts regularly. The patient was subsequently given supportive care with normal saline. As the urine colour was lighter than earlier and the abdominal pain was cured the patient showed good response with the medications and tends to discharged with the improvement [13].

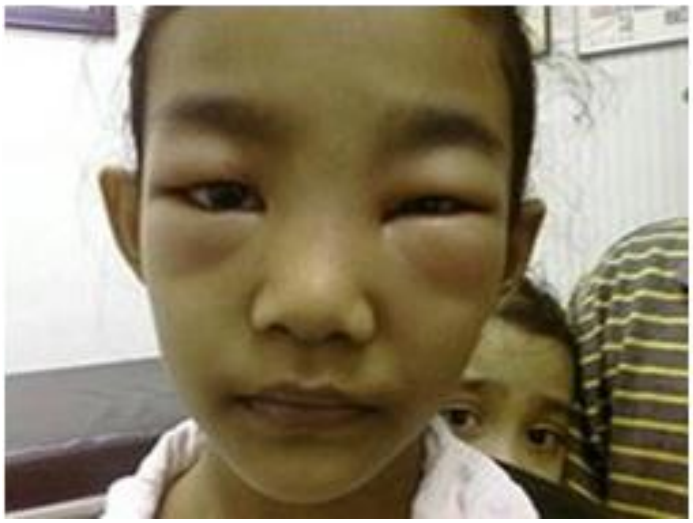

Fig. 1. Swelling (edema)

Fig. 2. Swelling of fingers-Edema due to hypoalbuminemia

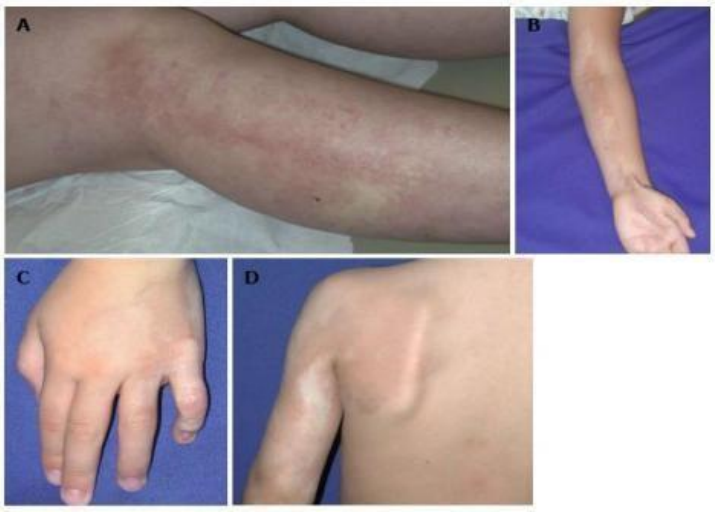

Fig. 3. Connective tissue disorder of the skin

2.3 Case 3

A 10 –year old male patient with the weight of 25 kg was admitted in the hospital with complaints of facial swelling, abdominal distension, swelling of fingers and lower limbs since three days as shown in the Fig. 2 and decreased urine output since two days. On examination patient was alert, active, afebrile and the Table 1 shows the clinical investigation results which shows that blood pressure was found to be 120/100mmHg, pulse rate was found Tobe 104beatsper minute, P/A was found to be soft and distended, CVS was found to be S1S2+, no murmur, complete blood picture i.e hemoglobin (Hb) - 11.8g/dL, Platelet count (PLT) – 2.7 lakhs, White Blood count (WBC) - 4, 750/cumm with Neutrophils-65.6%, Lymphocytes-25.9%, Monocytes- 7.8%, nephritic components recorded as decreased urine output volume (↓)-3ml, PH-0.6, serum creatinine was found to be 0.27, albumin-1.24g/dL(↓) (hypoalbuminemia), totalprotein-6.2 mg/dl (proteinuria), no hematuria recorded. Based on findings the patient was diagnosed with NEPHROTIC SYNDROME with decreased albumin levels.

The Table 2 shows the management strategies carried out to the patient, initially the patient was managed with Third-generation cephalosporin (Inj.Cefotaxime-100mg/kg/day i.e.835mg) thrice in a day. Calcium channel blockers (Tab.Nifedipine-0.25g/kg/day i.e.3.125mg) thrice in a day. And also managed with ALBUMIN transfusion twice in a week because the patients suffering from hypoalbuminemia, by monitoring vitals: PR, RR, BP etc, patient was advised to restrict salt diet, to take plenty of fluids.as the patient was maintained with albumin-3.23g/dl after albumin transfusion and preferably responded to the management, the patient was discharged with maximum improvements.

Table1.Clinical findings in nephrotic syndrome of pediatrics

Clinicaltests	Case1	Case2	Case3	Case4
Blood Pressure	90/60mmHg	140/100mmHg	120/100mmHg	100/60mmHg
Saturation	97%	90%	98%	98%
Pulse Rate	110bpm	112bpm	104bpm	98bpm
Temperature	Afebrile	Afebrile	Afebrile	febrile
CVS	S1S2+	S1S2+	S1S2+	S1S2+
GRBS	-	-	-	-
Heamoglobin	14.8g/dl	12.1g/dl	11.8g/dl	10.4g/dl
RBC	5.45 /mcl	-	6.1 /mcl	4.04/mcl
WBC	7,300/cumm	-	4,750/cumm	83,000/cumm(↑)
Platelets	3lakhs	-	2.7 lakhs	2.09lakhs
Neutrophils	42.2%	-	65.6%	4.54%
Lymphocytes	51.1%	-	25.9%	3.02%
Monocytes	6.6%	-	7.8%	6.9%
Albumin	2g/dl(↓)	4.07g/dl(↓)	1.24g/dl(↓)	2.04g/dl(↓)
Urea	15mg/dl (dipstick+3)	23mg/dl(↑)	17mg/dl	28mg/dl(↑)
ALT	9U/L	24U/L	19U/L	-
AST	-	34U/L	28U/L	72U/L
SR.CREATININE	0.1mg/dl(↓)	2.52g/dl	0.62g/dl(↓)	0.77g/dl(↓)
Bilirubin	-	-	-	0.19g/dl(↓)
GFR	-	-	-	-
Phosphate	-	-	4.4	4.8
Calcium	-	-	7.5	-
Magnesium	-	-	2.25	-
Potassium	4.6	-	4.1	3.8
Chlorine	98	-	110	106
Sodium	119	-	134	147
MCH	27.2	-	19.3	26.2
MCV	79.4	-	65.6	85.4
MCHC	32.2 g/dl	-	29.5 g/dl	30.7g/dl

Table 2. Management in nephrotic syndrome of pediatrics

Categories	Case1	Case2	Case3	Case4
Corticosteroids	Predisolone 1.5mg/kg/altday	-	-	-
Proton Pump Inhibitor	Pantop 40mg/OD	Lanazol 7.5mg/OD	-	Pantop20mg/OD
Ace Inhibitor	Enalapril 0.4mg/kg/OD	-	-	-
Calcium Channel Blockers	-	Nifedipine 0.25g/kg/OD	Nifedipine 0.25g/kg/OD	Nifedipine 0.1g/kg/OD
Vit.d	-	-	-	Calciferol 60000IU/week
Supplements	Calcium syrup 7ml/kg	Zinc syrup 5ml/OD	-	Calcium,MVT,B-complex
Antibiotic	-	Amoxyclav 3ml/BD	Cefotaxime 100mg/kg/OD	Ceftriaxone 100mg/kg/OD
Said's	-	-	-	Naproxen 10mg/kg/OD
Anti-in flammatory & anti - thrombotic	-	-	-	Hydroxychloroquine 200mg/OD
Anti-pyretic & analgesic	-	-	-	PCMsyrup9ml/OD
Antibacterial	-	-	-	Mupirocinointment
Albumintrasfusion	-	-	Transfused twiceinaweek	-

2.4 Case 4

A 7 year old male patient presented with complaints of joint pain since 20 days. Child was apparently asymptomatic few days ago, then he developed fever, which was acute in onset, low grade not associated with chills and skin allergy due to connective tissue disorder as shown in the Fig. 3. Following which child developed pain in both knee joints, which was acute in onset, later progressed to other joints to elbow, small and large joints. Child has a past medication history of severe anaemia at 3 years of age, and r/o proteinuria in 5 years of age and also have h/o simple febrile seizures at 11 months of age.

The Table 1 shows the clinical investigation results, which indicates that the child was active, alert with PR 90/min, RR-26/min and blood pressure was 100/60mmHg, haemoglobin (Hb)-10.4 g/dl, platelet count (PLT) -2.09 lakh, red blood cell count(RBC)-4.04/mcl, Whitebloodcount (WBC)-83,000/cumm with netrophills-4.54%, lymphocytes-3.02%, basophillis-0.08%. Upon urine examination urea and creatinine was 28 and0.77mg/dlwithALP-104,AST-72u/l, Albumin-2.04g/dl[↓] (hypoalbuminemia) and totalbilirubin-0.19g/dl[↓] (hyperbilirubinemia).The INR was 1.3, PT was 17.0 and APTT was 34. The USG abdomen revealed that left kidney was bulky. The patient was determine to have polyarthritis decrease evaluation with connective tissue disorder and it was a known case of SLE lupus nephritis a nephrotic syndrome. The Table 2 indicates the management strategies that is carried out to the patient, initially managed with syrup PCM 9ml and after investigations he was managed with tablet MVT/B complex, an antibacterialointment-Mupirocin which is used to treat secondary skin infections caused by any bacteria was prescribed, cholecalciferol vitamin D3 (vit danalogue) sachets 60000IU/week which works by helping the body to use more ofcalcium found in foods or supplement, antacid tablet pantop 20mg was prescribed, antibiotic injection ceftriaxone 100mg/kg/day was given in order to decrease the susceptible bacterial infection, tablet Naproxen which is an non- steroidal anti-inflammatory drug with dose- 10mg/kg/day was given to treat joint pains, calcium channel blocker Nifedipine 0.1g/kg/day, and tablet Aspirin 75mg was prescribed as an altered tablet with naproxen at day 7, tablet Hydroxychloroquine which is an anti- inflammatory and anti-thrombotic agent was prescribed with dose 200mg given to treat SLE/lupus nephritis, which is also used to treat arthritis. The patient was managed with the above mentioned medications

until patient is discharged with maximum improvements.

3. DISCUSSION

Nephrotic syndrome (NS) is the most common childhood renal diseases. NS can affect children of any age from infancy to adolescence and predominantly occurs in the age group of 1–6 years. Paediatric nephrotic syndrome (NS) is a group of clinical syndrome which can be characterised by severe proteinuria, hypoalbuminemia, hyperlipidaemia, and edema. It is characterized by urine protein to creatinine ratio greater than 200mg/mmol indicates proteinuria and plasma albumin less than 25g/dl indicates hypoalbuminemia. The loss of proteins negatively affects various biological functions which can result in complications which may have high risk of infection, blood clot, hypertension, high cholesterol and acute AKI [1]. The clinically identified symptoms of NS includes-Severe swelling (edema), specially around your eyes, ankles and feet, fingers, Foamy urine (heavy proteinuria), Weight gain (due to fluid retention), Fatigue, Loss of appetite, Hypoalbuminemia, Hyperlipidaemia, airway hyperresponsiveness, skin pruritus, repeated eczema or skin allergy [2]. Most patients with NS have a good response to steroid therapy and consequently show good prognosis [1].

We have observed a series of 4 different patients diagnosed with NS, with different age groups and co-morbid conditions. In this series, the patients were presented with symptoms such as swelling of face, weight gain, foamy urine,abdominal pain, dark coloured urine, swelling of lower limbs, decreased urine output, fever, pain in both knee joints and upon undergoing laboratory investigations the values for serum creatinine and albumin where found to be low which is less than 25 g/dl and the urea values where found to be increased when compare with the normal valuesi.e20mg/dl. Incase1no radiological investigational test was performed but the patient was managed with prednisolone medication by considering the laboratory findings and the symptoms were improved. The radiological investigations in case 2 Ultrasound shows right renal calculi with PCO2-27.00mmHg (↓) PO2-42.9mmHg(↑) Renal Angiography with 6mm calculi noted in pelvis, Mild dilation of pelvicalyceal system noted, Atelectatic band opacitieswere noted in antero-basal segment of right lower lobe, Renalartery Dopplerratio was 0.5 and managed with nifedipine, the symptoms were improved. In case 3 the radiological investigation shows decrease levels of albumin (1.24g/dL(↓)), managed with ALBUMIN transfusion twice in a week, and the patient was monitored for vitals PR,RR,BP as the patient was managed with increase in albumin(3.23g/dl). The radiological investigation carried out in case 4, reveals USG abdomen shows that left kidney was bulky due to heavy proteinuria. The patient was determine to have polyarthritis decrease evaluation with connective tissue disorder and managed with tab. calcium, hydroxychloroquine and mupirocin ointment, the patient was recovered after the management [14,15].

4. CONCLUSION

Nephrotic syndrome is the most common kidney diseases in children. Patients with NS sufferrom loss of proteins that negatively affects various functions during disease and can result in various complications. The children also suffer from severe and prolonged proteinuria, which has been linked to a higher risk of systemic complications like HTN, DM, CVS, atherosclerosis, vit D etc. These complications need the attention of health-care providers to prevent worsening of patients' condition. Further it may also cause psychological and developmental problems that may decrease the quality of life. Thus we have presented a casesof NS successfully managed with corticosteroid, albumin, Nifedipine and enalapril. Kidney biopsy procedure was not carried by professionals but could make a diagnosis based on clinical features and investigations, and fortunately patients recovered and attend smonthly follow-up visits.

CONSENT

As per international standards, parental written consent as been collected and preserved by the author(s).

ETHICALAPPROVAL

As per international standards for university standards written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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