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A CASE REPORT ON NEPHROTIC SYNDROME WITH INFREQUENT RELAPSE

[1]S.Athaur Rahiman*, [1]Dr. E.Alekhya Goud, [1]S.Nouhin, [2]Dr. Sai Vani, [2]S.Jagadeesh

*[1]Department of Pharmacy Practice, Santhiram College of Pharmacy, Nandyal, Kurnool DT, Andhra Pradesh, **INDIA***

*[2]Santhiram Medical College & General Hospital, Nandyal, Kurnool DT, Andhra Pradesh, **INDIA***

Abstract

Nephrotic Syndrome with infrequent relapse is a most common Glomerular condition that damage cluster of blood vessels in kidneys, this is represented by severe edema, foamy urine, anorexia & proteinuria.In most of the cases it is diagnosed by lipid profile, S.creatnine, BUN, urine analysis & biopsy. Steroids are the initial mainstay of the therapy. A 13 year old male patient admitted to nephrology ward having previous history of nephrotic syndrome with infrequent relapse. The patient was experiencing with Periorbital Odema, abdominal swelling, and facial puffiness. On examination it showed massive urinary protein, low total protein and albumin levels. The patient was diagnosed with nephrotic syndrome and was treated with corticosteroids, antibiotics, antacids and multivitamins.

Keywords: NSIR (Nephrotic syndrome with infrequent relapse), Corticosteroids, Kidney biopsy, anorexia.

Corresponding Author:

S.Athaur Rahiman

Department of Pharmacy Practice

Santhiram College of Pharmacy, Nandyal,

*Kurnool DT, Andhra Pradesh, **INDIA***

***Email:** sarahiman1997@gmail.com*

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INTRODUCTION

Nephrotic Syndrome is a kidney disorder that result increase permeability of glomerular filtration barrier . It is caused by increased permeability through the basement membrane in the glomerulus. The first recorded description of nephrotic syndrome dates to the 15th century. Later, Volhard and Fahr popularized the term nephrosis [1].

Minimal change disease (MCD)

1) Characterised as childhood NS, prevalent in 77%-85% of cases.
2) Idiopathic in nature, some reports in adult cases showed an association with Hodgkin lymphoma.
3) Renal biopsy samples when subjected to light microscopy showed no change.
4) On electron microscopy, tissue thinning can be seen.
5) Staining via florescence for immune complexes was negative.

Focal segmental glomerulosclerosis

1) Prevalent in 10%-15% of cases.
2) Characterised as adulthood NS.
3) Light microscopy of renal biopsy sample showed sclerosis in portions of selected glomeruli, which can progress into global glomerular sclerosis.
4) Tissue thinning was seen on electron microscopy, which was negative in most cases [2].

Nephrotic syndrome is caused by infections like Hepatitis B, C , Human immune deficiency virus , Malaria , Toxoplasmosis , Syphilis and some drugs like Non-steroidal anti-inflammatory drugs, Pamidronate , Interferon , Heroin , Lithium. Some Malignancies like Lymphoma , Leukemia. Miscellaneous are Systamic lupus erythematosus, Mesangio proliferative glomerulo nephritis and Diabetes mellitus [3,4] .

NS is having high amount of the protein in urine and low serum albumin, abundant albuminuria, generalized edema, and hyperlipidemia. During this disease the loss of proteins for various functions can result in complication such as AKI [5].

NS with inherited causes involves autosomal recessive type, NS type I , NS type II and isolated diffuse mesangial sclerosis p6]. Glomerular filtration barrier has three layers that is fenestrated endothelium, glomerular basement membrane, visceral glomerular epithelium containing podocytes (Figure 1). In NS, disturbance of the normal filtration process of glomerulus occurs, thus protein passage through filtration barrier due to three reasons including glomerular basement

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membrane's defects, T-cells in the damage to podocytes leading to MCD of NS, and pathology changes according to the type of NS [7,8] (Figure 2).

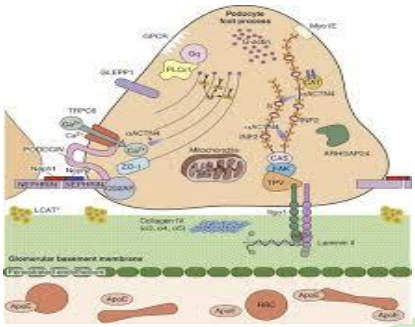

Fig.1. Pathophysiology of NS

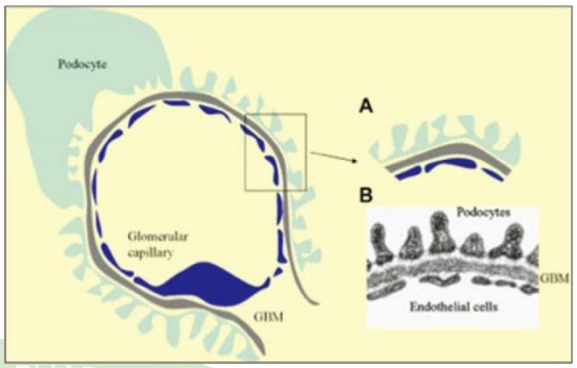

Fig. 2. Glomerular filtration barrier

New-onset edema, particularly in the lower extremities, is the most common presenting symptom of NS. Depending on disease severity, patients may have edema extending to the proximal lower extremities, lower abdomen, or genitalia. Ascites, periorbital edema, hypertension, and pleural effusion are also possible presenting features. Patients may report foamy urine, exertional dyspnea or fatigue, and significant fluid-associated weight gain [9,10].

Confirmation of proteinuria via 24-hour urine collection is cumbersome for patients, and the specimen can be collected incorrectly. The protein-to-creatinine ratio from a single urine sample is commonly used to diagnose nephrotic-range proteinuria. Although this spot test has limited accuracy in patients who exercise heavily, are gaining or losing muscle mass, or have similar factors, in general, it is sufficient for diagnosing heavy proteinuria [11].

Further diagnostic assessment of patients with NS has three goals: to assess for complications, identify underlying disease, and potentially determine the histologic type of idiopathic NS. The role of renal biopsy in patients with NS is controversial, and there are no evidence-based guidelines regarding indications for biopsy. Whether biopsy is performed often depends on the preferences of consulting nephrologists. In patients with NS from a known secondary cause and who are responding to treatment appropriately, biopsy will likely add little to treatment. Biopsy may be more useful for treatment and prognosis in patients with idiopathic NS of an unknown histologic disease type or with suspected underlying systemic lupus erythematosus or other renal disorders.

Nephrotic syndrome should be treated with Immunosuppressive drugs to overcome relapses. However, recent experiments have shown that steroids and cyclosporine, may also act directly on the podocyte to stabilize its structure [12,13]

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CASE REPORT

A 13years old male patient was admitted in nephrology department with IP NO-SR 19001888 having chief complaints of periorbital swelling, burning micturation abdominal distention, red colour urine, viral fever with cough and cold. He had previous episode of nephrotic syndrome, currently on examination the laboratory investigations shown elevated urine protein, albumin & creatinine levels. The radiological and histopathological reports shown a bilateral thicked renal parenchyma with grade-1 changes.

Patient was treated with:

Tab.Augmentin (Amoxycillin+ Clavulanic Acid) 625 mg twise a day

Tab.Omnacortil (Prednisolone) 40 mg once a day

Tab. Rantac (Ranitidine) 150 mg Once a day

Tab.Shelcal HD (Calcium with vitamin D3) 500 mg+500 IU Once a day

Tab. Supradyn (Multi Vitamin) Once a day

DISCUSSION

Nephrotic Syndrome is a kidney disorder that result increase permeability of Glomerular filtration barrier. It is caused by increased permeability through the basement membrane in the Glomerulus the result of an abnormality of Glomerular permeability may be started with disease specific to the kidneys such as diabetes, SLE, Neoplasia. The major characteristics of clinical diagnosis are Proteinuria, hyperlipidemia, edema. During this disease the loss of proteins for various functions can result in complication such as AKI. The abnormalities present in Nephrotic Syndrome includes, 24 hours urine protein, Albumin increased, the clinical history of biopsy report SSNS now relapse NS with hematuria to rule out FSGS Sr.creatinine 0.56 mg/dl, Proteinuria 3+++, 1 g /24 hours, RBC — 8-10, WBC — 4-6 hpf the microscopic description entered single core measuring 0.7 cm long received for IF study, section shows a core of renal cortex with up to 18 glomeruli, the Glomerular appear unremarkable with no increasing cellularity, basement membrane thickening, segmental region. If study shows the 8 glomeruli with insignificant Mesangial deposits of IGM & C3C, the finally diagnosis is near normal Unlight microscopy with no immune deposits consisting with minimal change disease. In this case the radiological report examined as bilateral thickened parenchymal grade-II changes to rule out infection etiology and biopsy was done. Treatment was Steroid therapy is applied to all children whatever the histopathology.

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Initial prednisone therapy consists of 60 mg/m2 administered daily for four weeks (maximum dose, 60 mg/day), 40 mg/m2/on alternate days for four weeks, reduce dose by 5 mg/m2 to 10 mg/m2 each week for another four weeks then stop. Relapse: Prednisolone should be restarted once a relapse has been diagnosed: 2 mg/kg daily (maximum 60 mg) until the urine is negative or trace for three days, then 40 mg/m 2 /on alternate days for 4 weeks then stop or taper the dose over 4 to 8 week [14,15].

CONCLUSION

Patients having less than two relapses within 6 months or less than four relapses within 12 months of response are called as Nephrotic syndrome infrequent relapse. The standard treatment for this disease includes Immunosuppression drugs are used to prevent relapses, recent experiments shows that Oral corticosteroid act directly on the Podocyte to stabilize its structure.

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