Case Report

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Case Report on Nephrotic Syndrome

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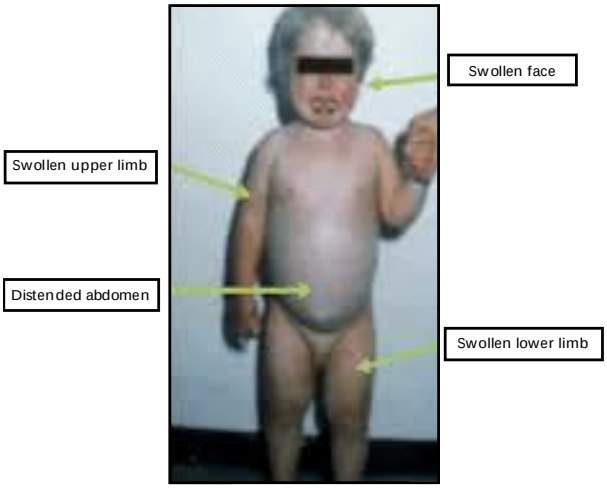
Abstract

Nephrotic syndrome is a nonspecific kidney disorder characterized by three signs of disease: large proteinuria, hypoalbuminemia, and edema. By obtaining a complete medical history, physical examination and a series of biochemical tests are required in order to arrive at an accurate diagnosis that verifies the presence of the illness. The incidence are high as it includes many types.

The types of primary nephrotic syndrome such as minimal change nephropathy, membranous nephropathy, and focal segmental glomerulosclerosis nephropathy remains challenging in treatment and nursing management. Three pediatric patients are selected randomly from the paediatric ward AIIMS Rishikesh, who were diagnosed with Nephrotic syndrome are managed with adequate medical and nursing care. The prognosis showed the improvement in resolving the syndrome specially on edema reduction and urine output .

Keywords: Nephrotic Syndrome; Kidney Disorder; Proteinuria; Hypoalbuminemia; Edema and Urine Output.



Introduction

Nephrotic syndrome is a nonspecific kidney disorder characterized by three signs of disease: large proteinuria, hypoalbuminemia, and edema [1]. Essentially, loss of protein through the kidneys (proteinuria) leads to low protein levels in the blood (hypoalbuminemia), which causes water to be drawn into soft tissues (edema). Very low hypoalbuminemia can also cause a variety of secondary problems, such as water in the abdominal cavity (ascites), around the heart or lung (pericardial effusion, pleural effusion), high cholesterol (hence hyperlipidemia), loss of molecules regulating coagulation (hence increased risk of thrombosis). Nephrotic syndrome has many causes and may either be the result of a glomerular disease that can be either limited to the kidney, called *primary* nephrotic syndrome (primary glomerulonephritis), or a condition that affects the kidney and other parts of the body, called *secondary* nephrotic syndrome. Along with obtaining a complete medical history, a series of biochemical tests are required in order to arrive at an accurate diagnosis that verifies the presence of the illness.

The treatment of primary nephrotic syndrome such as minimal change nephropathy, membranous nephropathy, and focal segmental glomerulosclerosis nephropathy remains challenging. Whilst most cases of idiopathic

nephrotic syndrome respond to steroid therapy and experience a limited number of relapses prior to complete remission, some cases suffer from frequent relapses and become steroid dependent or are primarily steroid resistant. Treatment options are limited to immunosuppressive drugs with significant side effect profiles. This present case study discusses the disease process and prognosis of the 3 children with various type of Nephrotic Syndromee.

For the comparative study, 3 patients are selected randomly from the paediatric ward AIIMS Rishikesh, who were diagnosed with Nephrotic Syndrome.

The Details of the Patients are Followed

Bio demographic data	Patient AX	Patient BY	Patient CZ
Age	6 years	6 years	6 years
Sex	Male	Female	Male
Address	Jwalapur, haridwar	Tehri garhwal	Lalpur balawala bijnor
IPD No.	456054/01/16	37746/09/15	134578/1015
Education	1st class	Kinduganden	play school
Religion	Hindu	Hindu	Muslim
Date of admission	29/01/16	9/10/15	8/10/15

Definition

Nephrotic syndrome is a syndrome characterized by edema and Lange amounts of proteins in the urine and usually increased blood cholesterol, usually associated with glomerulonephritis or within a complication of systemic disease.

Incidence

Incidence of the condition is 2-7 per 1000 children most common in male. Mean age of occurrence is about 2-5 years.

Classification

Book picture	Patient picture		
	Patient AX	Patient BY	Patient CZ
TYPE I- Idiopathic nephritic syndrome/Primary glomerulonephrosis • Approximately 90% of children with nephritic syndrome have idiopathic nephritic syndrome, idiopathic nephritic syndrome is anointed with primary glomeular disease without evidence of a specific systemic cause. • Idiopathic nephritic syndrome includes multiple histological types: minimal change disease, mesangial proliferation, focal segmental glomerulosclerosis, and membranous nephropathy. **Type- II secondary nephritic syndrome** • Nephritic syndrome can occur as a secondary feature of many form of glomerular disease. • This may be associated with membranous nephropathy, membranous proliferative glomerulo nephritis, lupus nephritis, malaria, schistoso-miaris, malignancy and therapies with numerous drugs and chemicals **Type III. Congenital nephrotic syndrome** • **Congenital** nephrotic syndrome is defined as nephoitic syndrome manifesting at birth or within first 3 month of life congenital nephrotic syndrome may be primary or secondary	Idiopathic nephritic syndrome	Secondary nephritic syndrome Patient BYwas diagnosed previously to have septicaemia	Idiopathic nephritic syndrome. Minimal changes disease

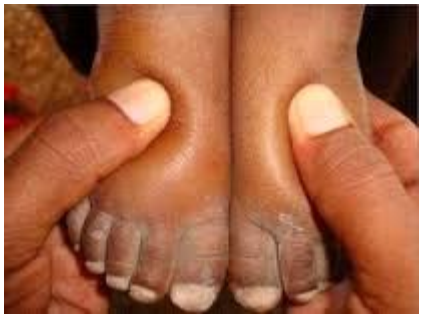
Etiology

Book picture	Patient picture		
	Patient AX	**Patient BY**	**Patient CZ**
the etiological or risk factor are divided into 2 types:- a. Primary glome rulo nephritis b. Secondary glome rulo nephritis **Primary glomerulonephrities**- caused by any glomerulor disease limited to kidney only i. Minimal change disease-cause due to minimal changes in glomerulus ii. Focal segmental glome rulosis-caused by tissue scanning in glomeruli iii. Membranous glomerulonephritis-infiammation of glomeular membrane iv. Membranoproliferative glomerulo nephritis - infiammation of glomeruli along tantibodies deposition in membrane v. Rapidly progressive glomerulo nephritis -glomeruli are in moon shaped. - GFR decreased by 30% **Secondary glomerulonephritis-** caused by any glomeruloe disease that affect the whole kidney as well as other parts of body i. Diabetic nephropathy-complication of diabetes ii. Systemic lupus erythematosis - it is an autoimmune disease that can affect many organs. iii. Sarcoidosis-accumulation of infiammatory granules in kidney. iv. Syphilis v. Hepatitis vi. Sjoguevis syndrome vii. HIV / AIDS viii. Amyloidosis - Deposition of amyloidal substance in glomeruli modifying thin shape and function ix. Multiple myeloma - cancerous cell in kidney x. Genetic disease xi. Drugs- penicillin gold salt, captopril etc.	The etiologic in patient AX was minimal change disease which results in the abnormal kidney function (primary glomerulo nephrities)	The etiologic in patient BY was minimal change disease which results in the abnormal kidney function (primary glomerulo nephrities)	The etiologic in patient CZ was minimal change disease which results in the abnormal kidney function (primary glomerulo nephrities)

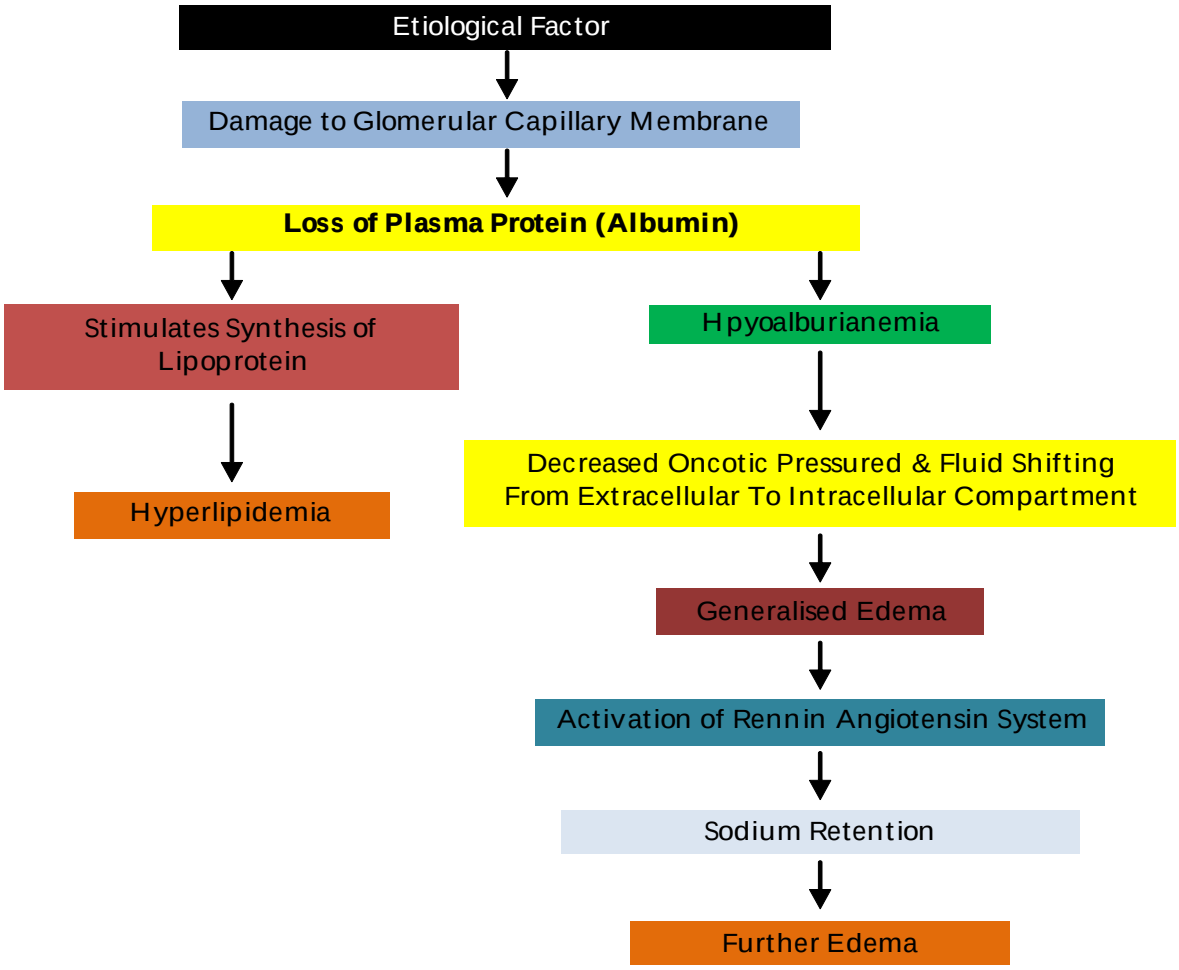
Clinical Manifestation

Book picture	Patient picture		
	Patient AX	**Patient BY**	**Patient CZ**
The onset of the disease is usually gradual or may be acute. I. The child may present with peri orbital puffiness II. Edema may be minimal or massive III. Profound weight gain within a short period of days or week is found IV. Dependent edema develops in the ankle, feet genital (scrotum) and hands. V. Striae may appear on the skin due to overstretching by edema VI. Fluid accumulation in body spaces a. Ascites b. Pleural effusion	• Periorbital puffiness • Oedema • Wt gain (28.5kg) • Ascetics present • Generalized edema • Urine output reduced • Fatigue • lethargy • Irritability • Wasting of muscle • Proteinuria	• Periorbital puffiness • Oedema • Plural effusion present • Generalized Oedema • Fatigue • Lethargic • Irritable • Hematuria • Proteinuria	• Oedema • Ascetic • Wt gain • Proteinuria • Fatigue • Lethargic • Irritability

VII. Generalized edema (anasarca)
VIII. Urine output reduced
IX. Concentrated & frothy appearance of urine.
X. GT disturbances usually found as vomiting, loss of appetite & diarrhoea
XI. Other features includes like:- fatigue, lethargy, pallor, irritability.
XII. Hypertension, hematuria, hepatomegaly and wasting of muscle may found in some cases



Pathophysiology



Diagnostic Measure

Book picture	Patient picture		
	Patient AX	**Patient BY**	**Patient CZ**
• History of illness and physical examination to exclude clinical features help to diagnose the condition clinically. • Laboratory investigations to confirm the diagnosis may includes the followings: - • Urine examination shows gross proteinuria (2 to 20 g 1 day), presence of cast, slight hematuria and increased specific gravity. • Blood examination demonstrates reduced total protein, albumin less than 2.5 gl / dl and cholesterol more than 200 mg / dl. • Lipoproteins and BUN (blood urea nitrogen) are increased. • Serum albumin and globulin ratio is reversed • Hypogammaglobulinemia, hypomagnesemia and low-ceatinine level • Renal biopsy is indicated in case of poor response to steroid therapy • Other investigation show low ASO titer and IgM, raised IgC & IgE, serum complements is normal	• Serum total protein =3.9 gm / dl • Serum albumin=1.2 gm / dl • Serum globulin= 2.7 gm / dl • A.G ratio =0.4 ➢ Urine examination • Protein =+ve appro. 500mg / dl • Leucoogtis= tve • Casts = granulan cast present ➢ Hematological report • TLC = 13400 cells / cumm. ➢ Other investigation are normal ➢ Renal biopsy is not indicated	➢ Biochemisty examination • serum total protein = 3.5 gm / dl • serum albumin=1.5 gm / dl • serum globulin= 2.9 gm / dl • A.G ratio = 0.5 ➢ Urine examination • Protein =+ve appro. 400 mg / dl • Costs= present ➢ Haematological report • Neutrophitis =31.8% • Eosinophilis = 6.8% • MCH= 24.9 pg ➢ Lipid profile • Total cholesterol 446.0 mg / dl • Serum triglycerides 229 mg / dl ➢ Other investigation are normal ➢ Renal biopsy is not done	➢ Biochemistry examination • Serum total protein = 3.0 gm / ldl • Serum albumin= 1.0 gm / dl • Serum globulin= 2.0 gm / dl • A. G ration = 0.5 ➢ Urine examination • Protein= +ve • Appro. 500 mg / dl • Blood = present • Leukucyte= present ➢ Haematological report • Hb= 9.6 gm / dl • RBC = 3.92 million cells cumm • Lymphocytes =47% • Hematocrit = 28.6% ➢ Lipid profile Total cholesterol= 320.0 mg / dl

Medical & Nursing Management

Book picture	Patient picture		
	Patient AX	**Patient BY**	**Patient CZ**
➢ Bed rest and high protein diet with restriction of fluid intake are important aspects of management ➢ Steroid therapy with oral predni solone is the most significant aspect of management of nephritic syndrome. It is given 2 mg 1 kg iday in 2to 3 divided doses f or at teat 4 to 6 weeks and then gradually tapered off or abruptly stopped, after another 4 to 6 weeks. ➢ Antacid is given along with prednisolone to prevent gastic complication ➢ antibiotic therapy is indicated in the presence of any infection ➢ Diuretics are prescribed in the presence ascites frusemide 1 to 3 mg 1kg 1 day in 2 divided doses in given ➢ Rapid fluid loses should not be attempted in 8 to 12 hours ➢ Potass ium supplementation to be given along with diuretics ➢ Albumin infusion (1g 1 kg 1 day) may be given in case of masive	• Bed rest & high protein diet is recommender to client • Antibiotic therapy i e. Cefexime & augmentine is prescribed to the patient • Lasix (furosemide) is prescribed to patient • Low sodium diet is recomemded.	• Bed rest & high protein diet is recommended to client • Antibiotic therapy IV metrogyl 15mg TDS, oral is prescribed to the patient • Wysolone (prednidolone) 10mg, BD, oral is prescribed to patient • Syp gelusil (magnesium hydrochloride) 10 ml OD, oral is prescribed • Furosemide (lasix) 21mg 18 hourly 1 oral is prescribed to patient • Fluid intake restriction • low sodium diet	• Bed rest & high protein diet i.e. 1.2g ml / kg / day is recommended to patient • Antibiotic therapy i.e. cetixine 200gm / orally / TDS and augmentin 375mg / orally / BD i s prescribed by doctor • Lasix (furose mide) is prescribed 20mg / orally / BD. • Input / output chart should be maintain • Albumin 600gm / IU / TDS is administered to patient

edema & ascetics. It helps to shift the fluid from interstitial space into the vascular system.
- Blood transfusion or plasma may be given is some cases to treat hypoalbuminemia.
- Immunasuppresive drugs (leuamisole , methotrexate, cyclophosphamide, cyclosporine, chlorambucil) may be administered along with prednisolone in case of frequent (4 or more per year) relapse and in steroid dependent cases.
- Renal transplantation is indicated in end stage failure

Prognosis

Book picture	Patient picture		
	Patient AX	**Patient BY**	**Patient CZ**
➢ Generally good although this depends on the underlying cause, the age of the patient and their response to treatment.	• The child is 6 years old. Enema was reduced, child showed adequate urine output. Childs	• Child was referred to other hospital with reference note.	• Urine out put was moderaqtely adequate, weight was reducing little. Periorbital Oedema reduced.

Complications

Book picture	Patient picture		
	Patient AX	**Patient BY**	**Patient CZ**
• Thromboembolic disorders • Infections: • Acute kidney failure . • Pulmonary edema: • Hypothyroidism • Hypocalcaemia: • Iron deficiency anaemia: • Protein malnutrition: • Growth retardation: • Vitamin D deficiency • Cushing's Syndrome	• Iron deficiency anaemia • Protein energy malnutrition • Growth restriction	• Iron deficiency anaemia. • Growth retardation	• Growth retardation • Iron deficiency anaemia.

Discussion

The nephritic syndrome becomes common renal disease among children now days. The causes are idiopathic for most of the children. And this leads to secondary nephritic syndrome. The children with nephritic syndrome admit in the paediatric ward very often with recurrence. In the above 3 cases baby BY admitted 3rd time in the paediatric ward with recurrence. The treatment of choice is depended upon age and type of nephritic syndrome. Steroid therapy is proved to be affective management in treating nephritic syndrome. Baby BY was treated with hydrocortisone because of the recurrent attract of the same disease but not other two babies were not received steroids. Master AX and BY discharged form hospital once they started to show progress were as master CZ got discharged against medical advice.

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