A STUDY ON BACTERIOLOGICAL PROFILE AND ANTIMICROBIAL SUSCEPTIBILITY PATTERN OF INFECTIONS IN HOSPITALISED CHILDREN WITH NEPHROTIC SYNDROME

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for the award of the degree of

M.D.MICROBIOLOGY BRANCH – IV

DEGREE EXAMINATION

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY
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CERTIFICATE

This is to certify that this dissertation titled **"A STUDY ON BACTERIOLOGICAL PROFILE AND ANTIMICROBIAL SUSCEPTIBILITY PATTERN OF INFECTIONS IN HOSPITALISED CHILDREN WITH NEPHROTIC SYNDROME"** is a bonafide record of work done by **DR. S.MEENAKSHI ,**during the period of her Post Graduate study from 2014 to 2017 under guidance and supervision in the Institute of Microbiology, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai- 600003, in partial fulfillment of the requirement of **M.D MICROBIOLOGY** degree Examination of **The Tamilnadu Dr. M.G.R Medical University to be held in April 2017.**

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DECLARATION

I, **DR. S.MEENAKSHI** declare that the dissertation entitled **"A STUDY ON BACTERIOLOGICAL PROFILE AND ANTIMICROBIAL SUSCEPTIBILITY PATTERN OF INFECTIONS IN HOSPITALISED CHILDREN WITH NEPHROTIC SYNDROME"** submitted by me for the degree of M.D. is the record work carried out by me during the period of **September 2015 –August 2016** under the guidance of **Prof. Dr. R.VANAJA, M.D.,** Institute of Microbiology, Madras Medical College, Chennai. This dissertation is submitted to The **Tamil Nadu Dr.M.G.R. Medical University, Chennai,** in partial fulfillment of the University regulations for the award of degree of M.D., Branch IV (Microbiology) examination to be held in April 2017.

Place: Chennai **Signature of the candidate**

Date: **(Dr. S.MEENAKSHI)**

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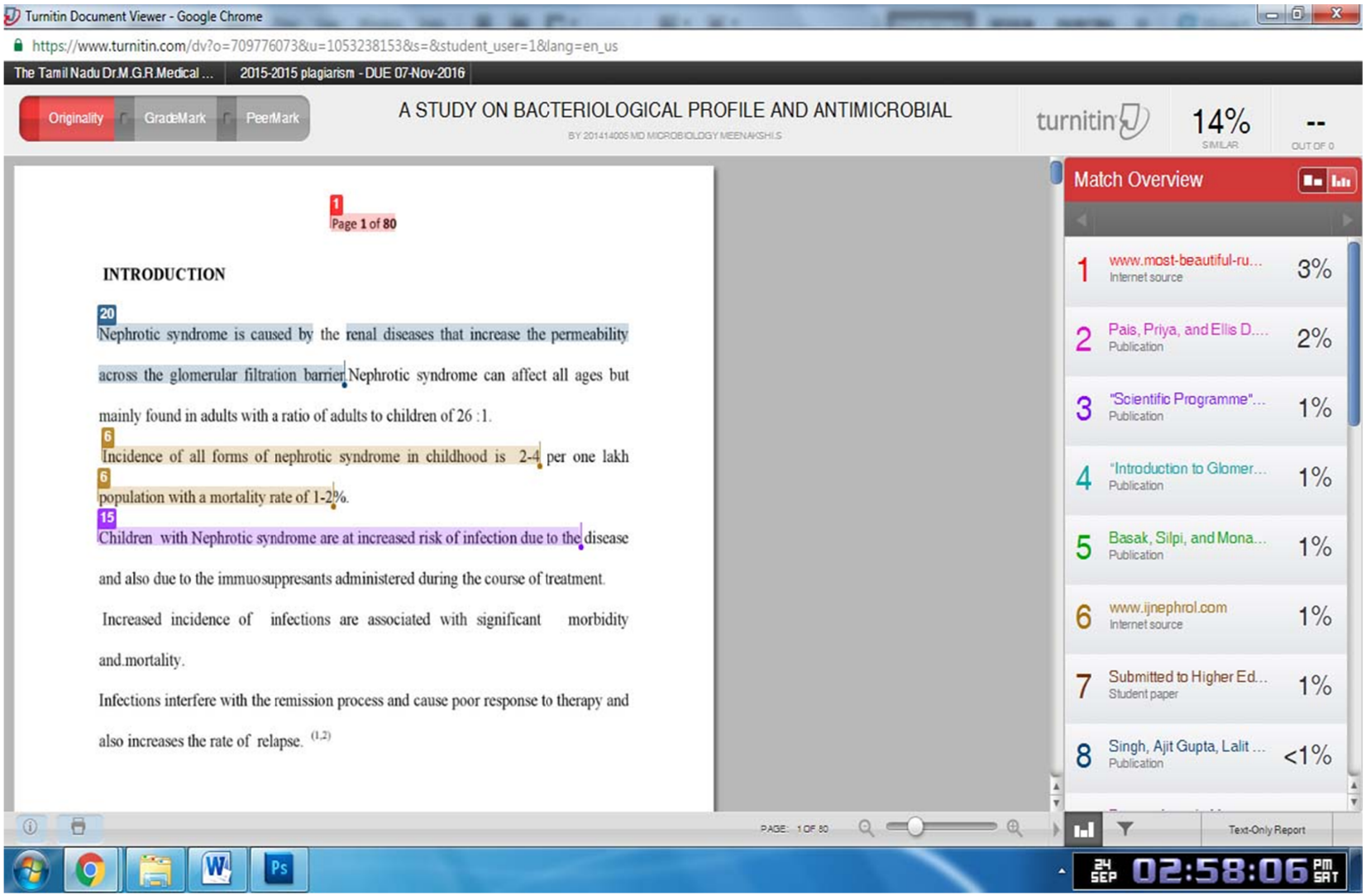
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INTRODUCTION
Nephrotic syndrome is caused by the renal diseases that increase the permeability across the glomerular filtration barrier.Nephrotic syndrome can affect all ages but mainly found in adults with a ratio of adults to children of 26 :1.
Incidence of all forms of nephrotic syndrome in childhood is 2-4 per one lakh population with a mortality rate of 1-2%.
Children with Nephrotic syndrome are at increased risk of infection due to the disease and also due to the immuosuppresants administered during the course of treatment.
Increased incidence of infections are associated with significant morbidity and.mortality.
Infections interfere with the remission process and cause poor response to therapy and also increases the rate of relapse. (1,2)
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Introduction

INTRODUCTION

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Increased incidence of infections are associated with significant morbidity and mortality.

Infections interfere with the remission process and cause poor response to therapy and also increases the rate of relapse. [1,2]

The most common infections seen in nephrotic syndrome are urinary tract infection, acute respiratory tract infection,skin and soft tissue infection, peritonitis, etc.

Nephrotic syndrome if not treated properly causes glomerular damage which results in reduction of glomerular filtration rate. [1,2]

The incidence of UTI in general population is 1% in male children and 3% in female children. But in children with Nephrotic syndrome the incidence of UTI is high which is13.2%. [1,2]

UTI is the most significant infection of all because it may produce various sequelae like renal damage and relapse after remission.

In 1996 **Gulaty** et al reported that UTI is the most common infection in nephrotic syndrome children and it is associated with significantly lower level of serum albumin and higher level of serum cholesterol concentrations than the nephrotic syndrome children without infections[1,2]. Because of this common clinical features like fever and other many physical findings are masked in children with infections. [3,4]

Several studies regarding the infections in Nephrotic syndrome were done mostly in developed countries.So, it is necessary for more studies in our country and also to know the spectrum of infections and antibiotic susceptibility of organisms causing the infection.

Aims & Objectives

AIMS AND OBJECTIVES

1. To study the spectrum of bacterial Infections in children with Nephrotic syndrome
2. To isolate and identify the bacteria causing infections in Nephrotic syndrome
3. To determine the Antibiotic susceptibility pattern of the isolated pathogens.

Review of Literature

REVIEW OF LITERATURE

Nephrotic syndrome affects 1-3 per one lakh children who are under 16 years of age.

Nephrotic syndrome manifests due to glomerular diseases.Most common type of nephrotic syndrome in children is of Primary or Idiopathic Nephrotic syndrome. [3]

Idiopathic Nephrotic syndrome is more common in male child than female child (2:1) and is common in age group between 2-6 yrs. It has also been reported earlier in six months of age and throughout childhood.Minimal Change Nephrotic Syndromeis present in “85-90%” of patients with less than 6 yr of age.The first episode of idiopathic nephrotic syndrome, and also subsequent relapses, usually follows “minor infections” and, rarely follows reactions due to “insect bites, beestings, or poison ivy”.

DEFINITIONS

NEPHROTIC SYNDROME

Nephrotic range proteinuria is defined as“ proteinuria >3.5 g /24 hrs or a urine protein : creatinine ratio > 2”. The “triad of clinical findings” associated with nephrotic syndrome arising from the large urinary losses of protein are “hypoalbuminemia (≤ 2.5 g/dl), edema, and hyperlipidemia (cholesterol >200 mg/dl).” [3]

REMISSION OF NEPHROTIC SYNDROME

Remission of Nephrotic syndrome is defined as “urine albumin nil or trace or proteinuria < 4 mg/m2/h for three consecutive early morning specimens”. [3,5]

RELAPSE OF NEPHROTIC SYNDROME

Relapse of nephrotic syndrome is defined as “urine protein : creatinine ratio of > 2 or urine albumin ≥3+ protein on urine dipstick testing for 3 consecutive days having been in remission”. [3,5]

FREQUENT RELAPSES

Frequent Relapses are defined as “two or more relapses in initial 6 months or more than 3 relapses in any 12 months”. [5]

STEROID DEPENDENT NEPHROTIC SYNDROME

Steroid dependence is defined as “two consecutive relapses when the patient is on alternate day steroid or within 14 days of discontinuation of steroids”. [5]

STEROID RESISTANCE NEPHROTIC SYNDROME

Steroid resistance is defined as“ the failure to achieve remission after 4 wk of prednisolone therapy at a dose of 2 mg/kg/ day”. [3,5]

HISTORY

“Dropsy” is the term used earlier for edema from the times of “Hippocrates” .This term was first recorded at 1290 AD. It denotes edema caused by heart disease,liver disease,renal disease and in nutritional disorders. [6,7,8,9]

“Hippocrates” said that , “when bubbles settle on the surface of the urine of the patient, it indicates a disease of the kidney and that the disease will be protracted”.Cornelus Roelans of Belgium described in 1484 a child with nephrotic syndrome and “whole body swelling.” He went on to recommend the treatment as follows: “take the tops of elder plant and daneswort, cook in white wine and wrap the child in hot clothes by applying the poultice in whole or in part, and so cure him” .

Theodore Zwinger of Basel in 1722 gave an accurate description of Nephrotic Syndrome in children.He said that “obstruction and compression of the tubules of the kidney” leads to reduced urinary output children .Morgagni’s disciple William Heberden went on to say: “Dropsy is very rarely an original distemper, but generally a symptom of some other which is too often incurable”. Several observers like, “Cotugno , Cruikshank , Wells , and Brande” found coagulability of urine in the patients. [6,7,8,9]

Richard Bright, who was working at Guy’s Hospital in London, said that kidney disease could cause dropsy. In 1827 Richard Bright (1789–1858) finally was able to compile together the triad of “ generalized edema, proteinuria, and kidney disease”, as presenting features of this disease. Glomerulonephritis was for over a century called as Bright’s Disease. Several post-mortem analysis of kidney disease were done. In 1840 Bright explained three varietiesof postmortem appearance of the kidneys, Chritison seven. Pierre Rayer, and Carl Rokitansky explained not less than eight varieties in 1846, but also “specknierre” or bacon

kidney,which was later recognized as amyloidosis .Klebs coined the term "glomerulonephritis" in 1872 to describe the exudative glomerular changes that is seen under the microscope . [6,7,8,9]

In 1905 German Pathologist Friedrich von Muller introduced the term 'Nephrosis', which means a non-inflammatory kidney disease, that is to be distinguished from Nephritis, which is the inflammatory type of Bright's disease. The concept of "nephritis in contrast to nephrosis" was further popularized by F.Volhard, T. Fahr, and C. Munk. [6,7,8]

Between 1930 and the 1950s the term "Nephrotic Syndrome" largely replaced the term "Nephrosis". The concept that "glomeruli changes are responsible for protein leak and not the changes in tubules" only became widely accepted in the 1940s.

In 1949-50 the first reports of a successful treatment for nephrotic syndrome were appearing, with the help of newly synthesised steroid hormones. Other treatment with the mercurial diuretics, which was invented in the 1920s, were weak and toxic, and was ineffective in nephrotic syndrome. [6,7,8,9]

The Indian Paediatric Nephrology Group in 2001 formulated guidelines for the management of children with Nephrotic syndrome and Revised guidelines for management of "Steroid –Sensitive Nephrotic Syndrome" which was published on 2008.

ANATOMY OF GLOMERULUS

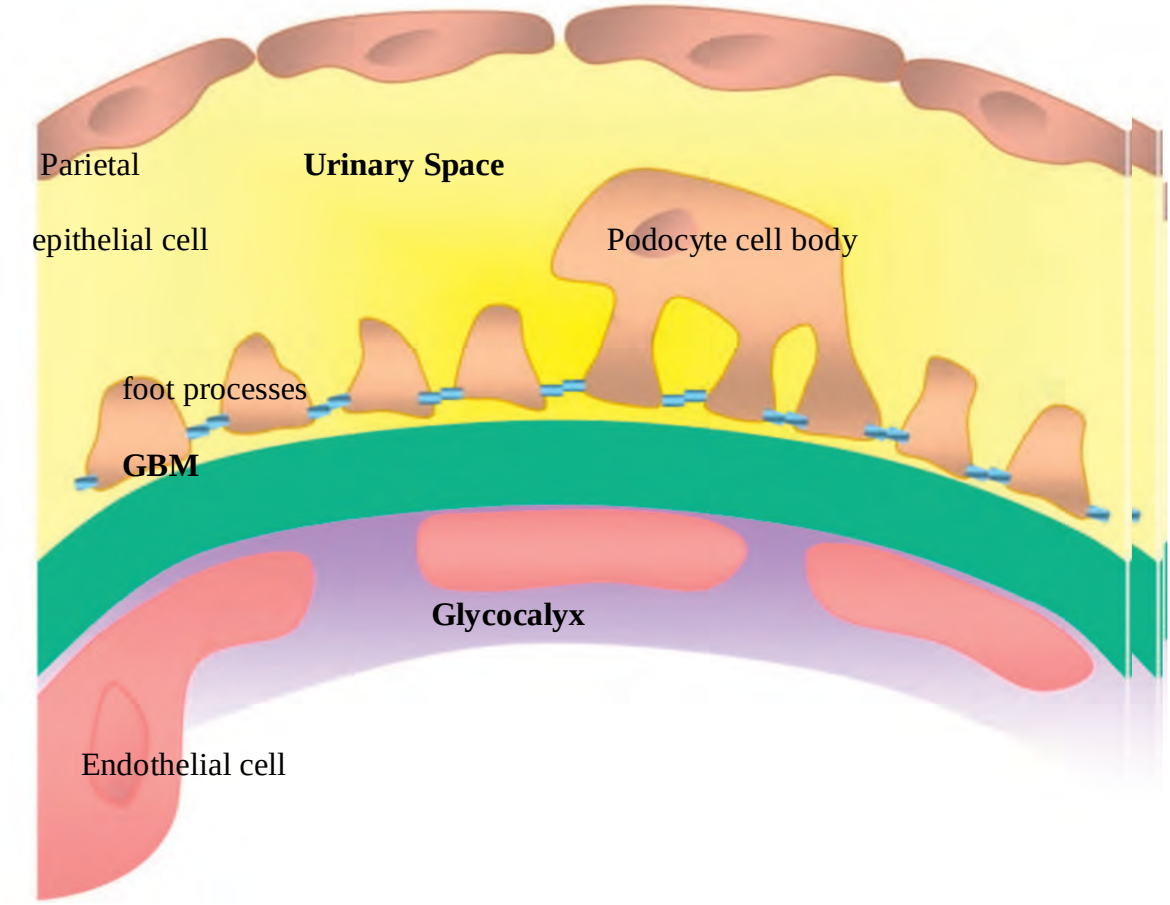
The glomerular apparatus consists of specialized capillaries which acts as the filtering mechanism of the kidney. The glomerular capillary wall contains three layers namely are glomerular endothelial cell, glomerular basement membrane and podocyte. These three layers act as the glomerular filtration barrier and thus prevents passing of proteins and large molecules from the capillary lumen into the urinary space.The urinary space contains the podocyte cell body.Through the foot processes the podocyte cell is attached to the glomerular basement membrane.each foot processes are separated by filtration slit. The slit-pore membrane forms a filter for plasma water and solute by the synthetic interaction of"nephrin, annexin-4, CD2AP, FAT, ZO-1, P-cadherin, podocin, TRPC6, PLCE1, and neph 1–3 proteins".Mutations of many of the above proteins also result in proteinuria. [3,4]

The Glomerular basement membranes of consist three layers namely

1. A central electron-dense lamina densa;
2. The lamina rara interna, which lies between the lamina densa and the endothelial cells;
3. The lamina rara externa, which lies between the lamina densa and the epithelial cells. [3,4]

Any Disruption in the Glomerular filtration barrier causes the passage of protein across the capillary wall, which leads to proteinuria.

Glomerular proteinuria may range widely from "<1 g to >30 g" of protein in a 24 hr period. In most glomerular diseases the cell which is predominantly injured is the podocyte which causes proteinuria . [3,4]



PHYSIOLOGY OF GLOMERULUS

The Glomerular capillary wall has a charge and size selective property. These property does not allow albumin, globulin and large plasma proteins into the urinary space.

Low molecular proteins which crosses the capillary wall are reabsorbed by the proximal tubule.The proteins which are normally excreted are Tamm-Horsfall protein (uromodulin), a protective glycoprotein are secreted by the tubules. [3,4]

The effectiveness of the glomerular capillary wall which acts as a filtration barrier is assessed by absence of plasma proteins larger than the size of albumin in the glomerular filtrate. Factors which restricts the filtration are the size and ionic charge of the macromolecules.The cells which possess negative ionic charges are the endothelial cell, basement membrane, epithelial cell of the glomerular capillary wall and also heparin sulphate and glycoproteins which contains sialic acid.

Proteins usually have a relatively low isoelectric point and carry a net negative charge. Hence, proteins are repelled by the negatively charged sites of the glomerular capillary wall.These property of proteins restrict their filtration. [3,4]

CAUSES OF NEPHROTIC SYNDROME [3,4]

Causes are mainly classified into

1. IdiopathicNephrotic syndrome
2. Genetic disorders with Nephrotic syndrome
3. Secondary causes of Nephrotic syndrome

IDIOPATHIC NEPHROTIC SYNDROME

- Minimal Change Disease
- Focal Segmental Glomerulosclerosis

- Membranous Nephropathy
- Glomerulonephritis associated with nephrotic syndrome–

  Membranoproliferative Glomerulonephritis

  Crescentic Glomerulonephritis

  Immunoglobulin A nephropathy. [3,4]

GENETIC DISORDERS ASSOCIATED WITH PROTEINURIA OR NEPHROTIC SYNDROME

1.Nephrotic Syndrome (Typical)

- Finnish-type Congenital Nephrotic syndrome ; (absence of nephrin)
- Focal Segmental GlomeruloSclerosis ; (mutations in nephrin, podocin, *MYO1E*, α-actinin 4, TRPC6)
- Diffuse mesangial sclerosis; (mutations in lamininβ2 chain)
- Denys-Drash syndrome ; (mutations in WT1 transcription factor)
- Congenital nephrotic syndrome with lung and skin involvement; (integrin α-3 mutation)
- Mitochondrial disorders)

2.Proteinuria With or Without Nephrotic Syndrome

- Nail-patella syndrome; (mutation in LMX1B transcription factor)
- Alport syndrome; (mutation in collagen biosynthesis genes)

3.Multisystem Syndromes With or Without Nephrotic Syndrome

- Galloway-Mowat syndrome
- Charcot-Marie-Tooth disease

- Jeune syndrome
- Cockayne syndrome
- Laurence-Moon-Biedl-Bardet syndrome

4.Metabolic Disorders With or Without Nephrotic Syndrome

- Alagille syndrome
- α1-Antitrypsin deficiency
- Fabry disease
- Glutaricacidemia
- Glycogen storage disease
- Hurler syndrome
- Partial lipodystrophy
- Mitochondrial cytopathies
- Sickle cell disease. [3,4]

SECONDARY CAUSES OF NEPHROTIC SYNDROME

1.Infections

- Infectious mononucleosis
- Malaria
- Syphilis ;Congenital and Secondary
- Toxoplasmosis
- Schistosomiasis
- Filariasis
- Endocarditis

- Hepatitis -B, Hepatitis- C
- HIV-1

2.Drugs

- Non Steroidal Antiinflammatory drugs
- Pamidronate
- Interferon
- Mercury
- Heroin
- Lithium
- Captopril
- Penicillamine
- Gold

3.Immunologic or Allergic Disorders

- Bee sting
- Food allergens
- Serum sickness
- Vasculitis syndromes
- Castleman disease
- Kimura disease

4. Associated With Malignant Disease

- Leukemia
- Solid tumors

5.Glomerular Hyperfiltration

- Oligomeganephronia
- Morbid obesity
- Adaptation to nephron reduction. [3,4]

PATHOPHYSIOLOGY

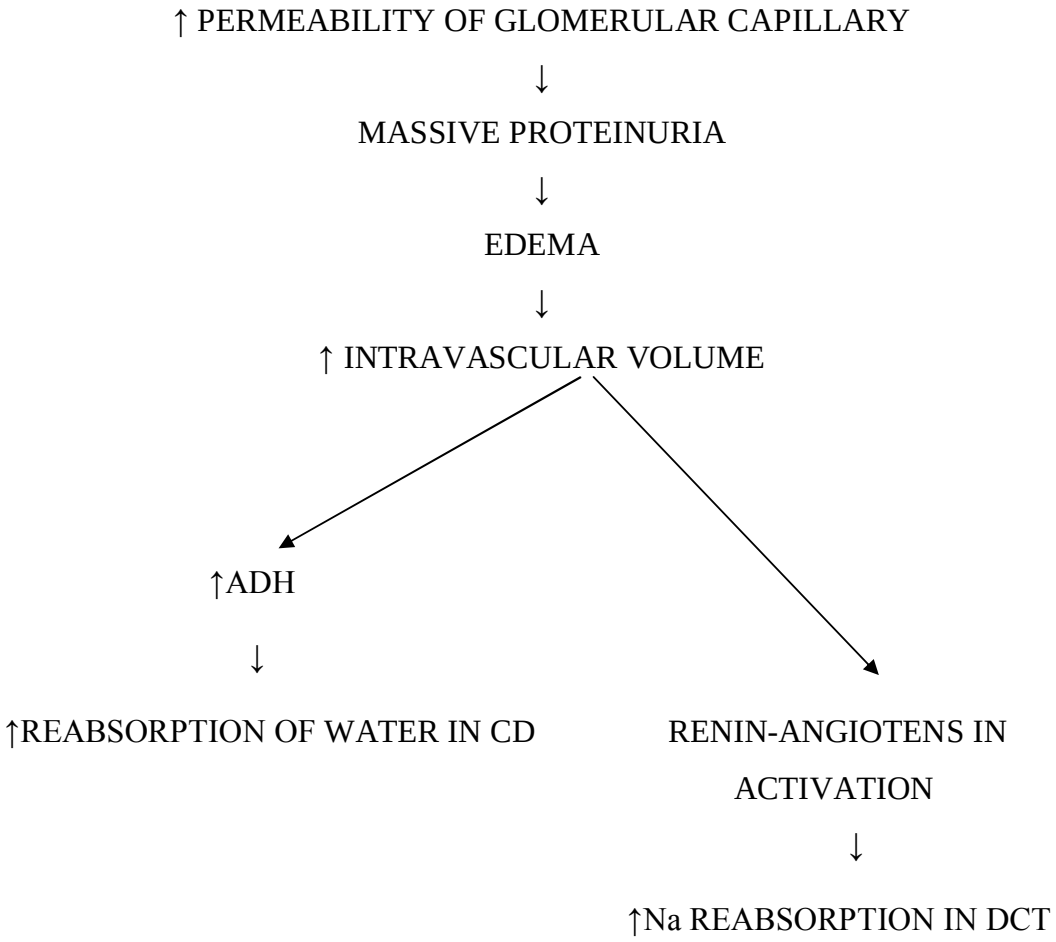
The main abnormality in Nephrotic syndrome is the increased permeability of the glomerular capillary wall.This leads to massive proteinuria and hypoalbuminenia. [3,4]

The podocytes located outside the glomerular capillary loop is a highly differentiated epithelial cell. It plays a main role in the development of proteinuria which may lead to progression of glomerulosclerosis. The podocyte has foot processes which terminate on the basement membrane.The podocyte is involved in synthesis and also in the repair of the glomerular basement membrane. It plays a major role in the membrane filtration barrier and also functions as structural support of the capillary loop.Slit diaphragms consists of numerous proteins which contribute to complex signalling pathways.These complex signaling pathways play a major role in podocyte function. [3,4]

The slit diaphragm consists of important component Proteins. These proteins include nephrin, podocin, CD2AP,and α-actinin 4. Podocyte injury or genetic mutations of genes produces podocyte proteins.These may cause nephrotic-range proteinuria .

There are immune and nonimmune insults to the podocyte in idiopathic, hereditary, and secondary forms of nephritic syndrome.These insults cause foot process effacement of the podocyte which leads to decrease in number of functional podocytes and alterationof integrity of the slit diaphragm. Finally there will be increased protein “leakiness”, across the glomerular capillary wall into the urinary space. [3,4]

PATHOPHYSIOLOGY



PATHOPHYSIOLOGY OF VARIOUS TYPES OF NEPHROTIC SYNDROME UNDER MICROSCOPY

1.MINIMAL CHANGE DISEASE(75%)

LIGHT MICROSCOPY

- It is mostly normal in this disease.
- Rarely there is minimal increase in mesangial cell and matrix.

ELECTRON MICROSCOPY

- There will be effacement of epithelial foot process

2.FOCAL SEGMENTAL GLOMERULOSCLEROSIS :(10%)

LIGHT MICROSCOPY

- There will be mesangial cell proliferation
- There will be segmental scarring

IMMUNOFLUORESCENT MICROSCOPY

- "IGM and C3" are seen in areas of segmental scarring

ELECTRON MICROSCOPY

- Shows obliteration of glomerular capillary lumen

3.MESANGIAL PROLIFERATION(5%)

LIGHT MICROSOPY

- There is diffuse increase in mesangial cells and matrix

IMMUNOFLUORESCENT MICROSCOPY

- Staining of Ig M and Ig A will be seen

ELECTRON MICROSCOPY

(i) There will be increase in number of mesangial cells and matrix

(ii) There will be effacement of epithelial cell foot processes.[3,4]

CLINICAL SEQUENCES OF NEPHROTIC SYNDROME

1.EDEMA

The most common presenting symptom of children with nephrotic syndrome is edema.. There are two opposing theories for the mechanism of edema formation, They are

1. The "*underfill hypothesis"*

2.The "*overfill hypothesis"*,

These two hypothesis have been proposed as mechanisms causing edema in Nephrotic syndrome.

The "*underfill hypothesis"* is based on the thing that "nephrotic-range proteinuria leads to a fall in the plasma protein level with a corresponding decrease in intravascular oncotic pressure". Because of this there is leakage of plasma water into the interstitium, which generates edema. Due to reduced intravascular volume, there is an increased secretion of vasopressin and atrial natriuretic factor. These increased secretion along with aldosterone leads to increased sodium and water retention by the tubules. Due to intravascular volume depletion there is retention of sodium and water. However this hypothesis does not fit for some patients who have intravascular volume overload and not volume depletion. These patients should be treated by not only with albumin but

also by diuretics. If mineralocorticoid receptor antagonists used for reducing the renin– aldosterone axis it does not produce increase in excretion of sodium. Usually in remission of Minimal change Nephrotic syndrome, first there is a increased urine output in many children before urinary protein excretion is measurably reduced. [3,4]

The *"overfill hypothesis"* postulates that nephrotic syndrome is usually associated with primary retention of sodium.These retention is associated with subsequent volume expansion and also there is leakage of excess fluid into the interstitium. In nephrotic syndrome the epithelial sodium channel which is present in the distal tubule play a major role in sodium reabsorption .[3,4]

The main drawback of this hypothesis is that many nephrotic patients clinically manifests with intravascular volume depletion with symptoms of low blood pressure, tachycardia, and increased hemoconcentration. Also, an epithelial sodium channel blocker namely amiloride, used alone does not produce adequate diuresis. [3,4]

2.HYPERLIPIDEMIA

Children with nephrotic syndrome have an altered lipid profile when compared to others..There is an increase in level of "cholesterol, triglycerides, low density lipoproteins, and very low density lipoproteins" except for the high density lipoprotein level which remains the same or is decreased. There is a decrease in the level of lipoprotein lipase .It is actually thought that

Hyperlipidemia is due to the result of increased synthesis of lipids and also due to decreased catabolism of lipids. [3,4]

3.INCREASED SUSCEPTIBILITY TO INFECTIONS

Nephrotic syndrome children are more susceptible to infections. The most common are urinary tract infections ,acute respiratory tract infections, cutaneous infections, spontaneous bacterial peritonitis, and bacteremia.

Viral infections and allergen challenges may cause Minimal change nephrotic syndrome .In children with Hodgkin lymphoma and T-cell lymphoma Minimal change nephrotic syndrome occur.

Nephrotic syndrome also occurs during immunosuppression which occurs with drugs such as corticosteroids and cyclosporine .By overall analysis it is clear that immune system plays a major role in the pathogenesis of the nephrotic syndrome . [3,4]

These infections occur due to many factors.They are

1. Urinary losses of immunoglobulin (Ig) G which leads to hypoglobulinemia.
2. Urinary loss of complement factors mostly C3 and C5 which produce defect in complement cascade.
3. Loss of factors B and D which participates in alternative pathway that leads to impaired opsonisation of micro organisms. [3,4]

Children with nephrotic syndrome are at high risk of infection with encapsulated bacteria.

4.HYPERCOAGULABILITY

Nephrotic syndrome is a hypercoagulable state.These state is due to many factors. They are

1. Hemoconcentration
2. Intravascular volume Depletion
3. Increased number of platelets and its aggregability
4. changes in levels of coagulation factors-increase in clotting factors i,ii,vii,x,fibrinogens.
5. There will be an increase in hepatic production of fibrinogen
6. urinary losses of antithrombin III and protein S which are anti-thrombotic factors

These all factors produce vascular stasis.Due to stasis deep venous thrombosis may occur in many venous bed. [3,4]

COMPLICATIONS

Complications are the main cause of morbidity and mortality in Nephrotic syndrome. They are mainly due to drugs and due to disease.

COMPLICATIONS DUE TO DRUGS

The common drugs which produce toxicity are

a) STEROIDS
b) CYCLOPHOSPHAMIDE
c) FRUSEMIDE
d) SPIRONOLACTONE

COMPLICATIONS DUE TO DISEASE

1. DUE TO PROTEIN LOSS
    - edema
    - hypothyroidism
    - infections
    - hypocalcemic tetany
    - hypercoagulable states
    - anemia

2. DUE TO HYPOVOLEMIA
    - acute renal failure
    - shock
    - renal vein thrombosis

3. DUE TO HYPERCOAGULABILITY
    - renal vein thrombosis
    - cerebral vein thrombosis
    - peripheral vein thrombosis

4. DUE TO LOSS OF IMMUNOGLOBULINS
    - infections. [3,4,10]

INVESTIGATIONS:

The following parameters were seen:

- **Urine complete**

  usually albumin will be 3+ or 4 +

- **Spot urine Protein / Creatinine ratio**

  this will be more than 2.

- **24 hr urine protein**

  it will be proteinuria/>50mg / kg /24 hr

- **Serum. Albumin**

  will be <2.5g/dl.

- **Serum. Cholesterol**,

  will be elevated

- **Serum.Triglycerides**

- **Serum. Creatinine,**

  usually will be normal but elevated if renal perfusion is reduced

- **Serum.Electrolytes.** [3,4]

OTHERS

- **Urine Culture & Sensitivity** to rule out urinary tract infection
- **Blood- CBC,**
- **Hematocrit**
- **Blood urea Nitrogen**

Parameters for evaluation of secondary forms of Nephrotic syndrome

- Complement C3 level,
- Anti-Nuclear Antibody,
- Double-Stranded DNA
- Hepatitis B and C,
- HIV in high-risk populations;
- Kidney biopsy (for children ≥12 yr, who are less likely to have MCNS).[3,4]

TREATMENT

CHILDREN TREATED AS OUTPATIENT

Children with first episode of nephrotic syndrome and who have mild to moderate edema can be managed as outpatients.

CHILDREN REQUIRING HOSPITALISATION

- Severe edema compromising respiration / ambulation
- Unstable vital signs,
- Fever
- Urine output<1ml/kg/hr
- Severe Hemoconcentration evidenced by hematocrit>48%

TREATMENT REGIMEN

International Study for Kidney Diseases in children recommends a regimen Comprising of 4 weeks.

For the treatment of the Initial Episode of Nephrotic Syndrome and with children who have minimal change Nephrotic syndrome prednisolone should be administered as a "single daily dose of 60 mg / m^2 /day or 2 mg/kg/day to a maximum of 60 mg daily for 4-6 weeks" followed by "alternate-day prednisolone (starting at 40 mg /m^2 qod or 1.5 mg/kg qod) for a period ranging from 8 weeks to 5 months, with tapering the dose" [3,4,5]

STEROID TOXICITY:

i. Cushingoid appearance
ii. Behavioural changes
iii. Secondary hypertension
iv. Obesity
v. Glucose intolerance
vi. Cataract
vii. Osteopenia
viii. Decreased growth ratio

TREATMENT PLAN FOR "STEROID SENSITIVE NEPHROTIC SYNDROME (RELAPSE)" [3,4,5]

Daily predinisolone 60mg/m^2/day

until urine protien negative for 3 days

↓

Alternate day predinisolone 40mg/m^2/day for 4 weeks

↓

Predinisolne tapered to lowest dose that keeps child0ren remission for 6-9 months

↓

For frequent relapsers

↓

Steroid tapered to alternate day schedule and addlevamisole 2-2.5mg/kg on alternate days for 6-12 months on which steroids are not given (steroid 0.5mg/kg on 6^{th} month & 0.25mg/kg on 12^{th} month)

↓

Levamisole continued for one more year

If levamisole fails next line is alkylating agents

↓

After induction steroids is slowly tapered to alternate day schedule and then add daily cyclophosphamide 2mg/kg/day for 12 week course

↓

If drug resistance do renal biopsy

↓

1.If FSGS prolonged course of I.V Methlyprednisolone or Cyclophosphamide

2.If MCNS give cyclosporin a 6mg/kg/day for 1-2 years

↓

If protienuria not controlled then treated with

NSAID's(Indomethacin 3mg/kg/ day in 3 divided doses)/ ACE↓(Enalapril 0.25mg/kg/day in 2 divided doses) should be given.

↓

If failed Dialysis & Renal transplantation

TREATMENT PLAN FOR "STEROID RESISTANT NEPHROTIC SYNDROME" [3,4,5]

Aim→ control of proteinuria, prevention &management of complications.

- I.V.Methylprednisolone30mg/kg/day x 5days+

I.V.Cyclophophamide500mg/m^2/dose-6^{th} day

(repeated every month for 5 months)

(older–oralcyclophosphamide2mg/kg/day x 12

wks+oralprednisolone0.5mg/kgday x 12-16wks)

↓

maintanence oral prednisolone

1mg/kg/day alternate days x 1yr

↓

If treatment failedantiproteinuric drugs

(NSAIDs,ACE↓)

↓

If failed to respond to drugs dialysis & renal transplantation

IMMUNOMODULATORS

1.LEVAMISOLE

- DOSE → 2-2.5mg/kg
- used in steroid sparing effect,not to induce remission
- used in frequent relapse associated with steroid sensitive ns.
- after inducing remission steroid tapered to lowest dose in alternate days & levamisole given in alternate days
- levamisole continued for one more year

2.ALKYLATING AGENTS (CYCLOPHOSPHAMIDE / CHLORAMBUCIL)

- if levamisole fails→next line of treatment is this
- both agents can induce remission→long lasting
- either of these can be started on a daily therapy after induction of steroid therapy,then steroids are slowly tapered to alternate day schedule
- cyclophosphamide 2mg/kg/day .cyclophosphamide should not exceed 168mg/kg/course.
- cyclophosphamide toxicity -bone marrow supression, allopecia, hemorrhagiccystitis, gonadtoxicity, rarely malignancy.
- WBC count should be monitered regularly
- If WBC<4000cells/mm^3 → stop dose & if > 4000cells / mm^3 restart dose
- post pubertal female child is at a greater risk of toxicity than pre-pubertal female child

3.CYCLOSPORIN A

- DOSE 6mg/kg/day
- induce remission for 1-2years
- 12-18 months of cyclosporin allows resolution of steroid toxicity
- Toxicity -hirsuitism,(regress when drug is discontinued)

  -gingival hypertrophy,

  -hypertension,

  -hypomagnesemia,

  -hepatotoxicity,

  -nephrotoxicity-cannot be appreciated simply by serum creatinine→follow up kidney biopsy after a year of therapy " [3,4,5]

SUPPORTIVE CARE

MONITORING:

- Fluid intake & output
- Daily blood pressure
- Daily weight
- Daily urine examination for albumin
- Daily abdominal circumference for edema & ascites

DIET:

- Sodium restriction only during times of massive edema

  Sodium promotes edema formation

- Sodium requirement for age<5 →1 to 1.5gm/day

  For school age→2gm/day

  adolescent→3-4gm/day

FLUID RESTRICTION

- In severe edema→intake of fluid= insensible water loss

  below 6yrs →30ml/kg/day

  above 6 yrs→20ml/kg/day

- In moderate edema→fluid intake equals previous day urine output + insensible water loss in mild edema→fliud is restricted minimally

PROTIEN INTAKE:

- advise to take normal protien intake
- high protien diet will only ↑ urinary protien loss and contributes to glomerular injury

ACTIVITY:

- allowed to do normal activity & schooling

DIURETICS:

- helpful in children with moderate edema/oliguria
- useful in steroid resistant disease
- serum potassium level should be monitored

- can precipitate hypotension,thrombosis, ATN→so intravascular volume should be monitored
- albumin and diuretics can be used in marked ascites compromising pulmonary .function.,pleural effusion,scrotal or labial edema,severe peripheral edema with skin breakdown
- protects BP& renal perfusion in septic child with Nephrotic syndrome.
- 1gm/kg upto 25gms infused over 2 hours

DIURETICS DOSAGE:

- Furosemide(most common used)- 1to 2mg/kg/dose oral/i.v
- Spironolactone-1.5-3.5mg/kg/day 6-8thhrly
- Thiazide-1-2mg/kg/day

Metalazone-0.5mg/kg/day(useful in furosemide refractory Nephrotic syndrome) " [3,4,5]

IMMUNIZATION

Patients who are on steroid therapy for more than 14 days are immunocompromised. They should not receive Live attenuated vaccines.

- Pneumococcal vaccine can be given after 2yrs during the remission phase. pneumococcal vaccine should be given to all children with Nephrotic syndrome.
- OPV replaced by IPV (if not given)

- DPT, H.INFLUENZA B, HEP-B vaccines administered as usual schedule
- Live vaccine(Varicella,Measles,MMR) can be given if necessary. During the epidemics vaccines should be administered when child is in remission phase for 3 months" [3,4,5]

Materials & Methods

MATERIALS AND METHODS

This study on Bacteriological profile and Antimicrobial susceptibility pattern of infections in hospitalised children with nephrotic syndrome was carried out in the Institute of Microbiology, Madras Medical College ,Rajiv Gandhi Goverrntment General Hosital, Chennai in association with Department of Nephrology, Institute Of Child Health, Madras Medical college, Chennai.

STUDY DESIGN

Cross-Sectional Study

STUDY PERIOD:

One year September 2015 –August 2016

STUDY POPULATION

A total number of 100 children with Nephrotic syndrome hospitalised in the paediatric Nephrology ward, Department of Nephrology, Institute Of Child Health, Madras Medical college, Chennai.

ETHICAL CLEARANCE

Before starting the study approval was obtained from the Institutional Ethics Committee. Informed consent was obtained from the parents / guardian of children with Nephrotic syndrome who satisfied the inclusion criteria.

INCLUSION CRITERIA

- Nephrotic Syndrome children of age between1 to 12 years.

- Children with Nephrotic Syndrome hospitalised for reasons like relapse,reevaluation,non response to therapy,cyclophosphamide therapy and with infectious complications

EXCLUSION CRITERIA

- Children with urogenital anomalies
- Children diagnosed as a case of acute renal failure or chronic renal failure .

COLLECTION OF DATA

Data were collected from children with Nephrotic syndrome who satisfied the inclusion criteria. Demographic details like name, age, sex, address, date of admission, clinical data like presenting complaints, past history, treatment history, details of clinical diagnosis and investigations were collected.

METHODOLOGY

SAMPLE COLLECTION

Samples were collected after getting informed consent from the parents/ guardian of the children.

Following samples were collected

1.Urine

2.Blood

3.Throat swab

COLLECTION OF URINE

The child / parent was given a sterile container which has wide mouth and also leak proof.

The child / parent was asked to collect 10 ml of clean catch Mid-Stream urine.

GUIDELINES TO COLLECT URINE

For female children first the parent is asked to wash their hands.Then asked to clean the genitalia with clean water and dried with gauze pad which is sterile. Then asked to collect Mid-stream urine after separating the labia.

For male children first the parent is asked to wash their hands.Then asked to Retract the prepuce and clean that area with clean water and dried with gauze pad which is sterile.Then asked to collect Mid-stream urine.

The collected sample was processed within 2 hours of collection. [11]

COLLECTION OF BLOOD

Blood was collected with sterile precautions by disinfecting the venepuncture site with 70% alcohol and let it dry.Then the site was disinfected with 1% povidone-iodine .with sterile disposable syringe and needle 5 ml of blood was collected. After collection of blood it was immediately transferred into the 25 ml of Brain-Heart Infusion broth in the blood culture bottle.After collection of blood the punctured site is again cleaned with 70% alcohol [11]

COLLECTION OF THROAT SWAB

A sterile swab was inserted through the mouth and specimen collected from posterior pharyngeal wall .The collected swab was processed within 2 hours by plating it in BAP. [11]

PROCESSING OF SPECIMENS

PROCESSING OF URINE SAMPLE

DIRECT GRAM STAIN

A drop of urine was transfered on a clean glass slide and was spread as a thin smear. It was allowed to air dry and then it was heat fixed. Gram staining was done and the smear was examined with 100x oil immersion objective.

Gram stain morphology and presence of any pus cells ,bacteria were documented.

CULTURE

A 4mm diameter of calibrated Nichrome loop 0.01 ml of urine was taken and streaked on to MacConkey agar plate and Blood agar plate. The plates were then kept at 37˚C in the incubator for overnight and observed for any growth after overnight incubation and at 48 hrs. [11]

A colony count of more than 1,00, 000/ml (10^5/ml) is considered as significant bacteriuria. [13]

PROCESSING OF BLOOD SAMPLE

CULTURE

The blood sample collected in Brain-Heart infusion broth was incubated at 37˚C for 48 hours. After 48 hours the BHI broth containing the blood was streaked on to MacConkey agar plate and Blood agar plate. The plates were then kept at 37˚C in the incubator for 24 hours. . [11]

PROCESSING OF THROAT SWAB

DIRECT GRAM STAIN

A evenly spread thin smear was made with the swab. It was allowed to air dry minutes and heat fixed. Gram staining was done and the smear was examined with 100x oil immersion objective.

Gram stain morphology and presence of any epithelial cells ,pus cells and bacteria were documented.

CULTURE

The swab was inoculated on to MacConkey agar plate and Blood agar plate. The plates were then kept at 37˚C in the incubator for 24 hours in a candle jar which is carbon-di-oxide enriched. . [11]

IDENTIFICATION OF ISOLATES [12]

All the bacterial isolates obtained from the samples were identified by standard bacteriological techniques.

If gram negative bacilli were seen the colonies were subjected to the following test using standard microbiological techniques and relevant biochemical reactions.

1. Catalase test
2. Oxidase test
3. Hanging drop for testing motility
4. Indole test
5. Citrate test
6. Urease test
7. Triple sugar iron test
8. Sugar fermentation test.

If the Grams stain morphology showed gram positive cocci in clusters following tests were done

1. Catalase
2. Coagulase test – slide and tube
3. Bacitracin sensitivity using 0.04 unit disk
4. Urease test
5. Mannitol fermentation test

IDENTIFICATION OF *Staphylococcus aureus* [13]

The following bacteriological techniques given below are used to identify *Staphylococcus aureus.*

Gram stain	Gram positive cocci in clusters
Blood agar	Beta haemolytic and golden yellow pigmented colonies
catalase	positive
Slide coagulase	positive
Tube coagulase	positive
urease	positive
mannitol	fermented
Methyl –red test	positive
Voges Proskauer test	positive
Phosphatase test	positive

IDENTIFICATION OF *Staphylococcus epidermidis* [13]

The following bacteriological techniques given below are used to identify *Staphylococcus epidermidis.*

Gram stain	Gram positive cocci in clusters
Blood agar	White opaque colonies
catalase	positive
Slide coagulase	negative
Tube coagulase	negative
Phosphatase test	positive
mannitol	Not fermented
Novobiocin sensitivity	sensitive
Polymyxin –B sensitivity	resistant

IDENTIFICATION OF *Micrococci* [13]

The following bacteriological techniques given below are used to identify *Micrococci.*

Gram stain	Gram positive cocci in tetrads
MacConkey agar	Lactose fermenting colonies
catalase	positive
Modified oxidase test	positive

IDENTIFICATION OF *Streptococcus pyogenes* [14]

The following bacteriological techniques given below are used to identify *Streptococcus pyogenes.*

Gram stain	Gram positive cocci in pairs and short chains
blood agar	beta hemolysis
catalase test	negative
bacitracin 0 .04 units sensitivity	sensitive
Bile Esculin test	negative

IDENTIFICATION OF *Enterococcus faecalis* [14]

The following bacteriological techniques given below are used to identify *Enterococcus faecalis.*

Gram stain	Gram positive cocci in pairs and short chains
blood agar	Non- haemolytic tiny colonies
catalase test	negative
Bile Esculin test	positive
Arginine dihydrolase test	positive
Mannitol	fermented
Arabinose	Non-fermented
Heat tolerance test (surviving at 60˚for 30 minutes)	positive

IDENTIFICATION OF *Diphtheroids* [15]

The following bacteriological techniques given below are used to identify *Diphtheroids.*

Gram stain	Gram positive bacilli
Blood agar	White opaque colonies
hanging drop method	Motile bacilli
catalase test	positive
oxidase test	negative
nitrate reduction test	negative
urease test	negative
glucose	fermented
Bile Esculin test	negative
Arginine dihydrolase test	negative
Voges proskauer test	negative

IDENTIFICATION OF *Escherichia coli* [16]

The following bacteriological techniques given below are used to identify *Escherichia coli* .

Gram stain	Gram negative bacilli
MacConkey agar	lactose fermenting colonies
hanging drop method	motile bacilli
catalase test	positive
oxidase test	negative
nitrate reduction test	positive
indole test	positive
Methyl red	positive
Voges proskauer	negative
citrate utilization test	negative
Triple sugar iron agar	acid butt and acid slant with gas
urease test	negative
Sugars (glucose,lactose,sucrose)	fermentation of sugars with acid and gas.

IDENTIFICATION OF *Proteus vulgaris* [16]

The following bacteriological techniques given below are used to identify *Proteus vulgaris* .

Gram stain	Gram negative bacilli
MacConkey agar	Non lactose fermenting colonies
Blood agar	Grey white colonies with swarming
hanging drop method	motile bacilli
catalase test	positive
oxidase test	negative
nitrate reduction test	positive
indole test	positive
Methyl red	positive
Voges proskauer	negative
citrate utilization test	positive
Triple sugar iron agar	acid butt and alkaline slant with H_2S production
urease test	positive
phenylalanine deaminase test	positive

IDENTIFICATION OF *Proteus mirabilis* [16]

The following bacteriological techniques given below are used to identify *Proteus mirabilis.*

Gram stain	Gram negative bacilli
MacConkey agar	Non lactose fermenting colonies
Blood agar	Grey white colonies with swarming
hanging drop method	motile bacilli
catalase test	positive
oxidase test	negative
nitrate reduction test	positive
indole test	negative
Methyl red	positive
Voges proskauer	negative
citrate utilization test	positive
Triple sugar iron agar	acid butt and alkaline slant with H_2S production
urease test	positive
phenylalanine deaminase test	positive
Ornithine decarboxylation	positive

IDENTIFICATION OF *Klebsiella oxytoca* [16]

The following bacteriological techniques given below are used to identify *Klebsiella oxytoca.*

Gram stain	Short Gram negative bacilli
MacConkey agar	lactose fermenting mucoid colonies
Blood agar	Grey white colonies
hanging drop method	Non-motile bacilli
catalase test	positive
oxidase test	negative
nitrate reduction test	positive
indole test	positive
Methyl red	negative
Voges proskauer	positive
citrate utilization test	positive
Triple sugar iron agar	acid butt and acid slant with gas
urease test	positive
Sugars (glucose,lactose,sucrose)	fermentation of sugars with acid and gas

IDENTIFICATION OF *Klebsiella pneumoniae* [16]

The following bacteriological techniques given below are used to identify *Klebsiella pneumoniae* .

Gram stain	Short Gram negative bacilli
MacConkey agar	lactose fermenting mucoid colonies
Blood agar	Grey white colonies
hanging drop method	Non-motile bacilli
catalase test	positive
oxidase test	negative
nitrate reduction test	positive
indole test	negative
Methyl red	negative
Voges proskauer	positive
citrate utilization test	positive
Triple sugar iron agar	acid butt and acid slant with gas
urease test	positive
Sugars (glucose,lactose,sucrose)	fermentation of sugars with acid and gas

IDENTIFICATION OF *Pseudomonas aeruginosa* [17]

The following bacteriological techniques given below are used to identify *Pseudomonas aeruginosa*.

Gram stain	Slender Gram negative bacilli
MacConkey agar	Non-lactose fermenting colonies
Nutrient agar	production of bluish green pigment colonies
hanging drop method	motile bacilli
catalase test	positive
oxidase test	positive
nitrate reduction test	positive
indole test	negative
Methyl red	negative
Voges proskauer	negative
Hugh&Leifson O/F medium	oxidative reaction
Triple sugar iron agar	alkaline butt and alkaline slant
arginine dihydrolase test	positive
lysine decarboxylation test	negative

IDENTIFICATION OF *Acinetobacter baumannii* [18]

The following bacteriological techniques given below are used to identify *Acinetobacter baumannii* .

Gram stain	Gram negative coccobacilli
MacConkey agar	Non-lactose fermenting colonies
Growth at 42˚	present
hanging drop method	Non-motile bacilli
catalase test	positive
oxidase test	positive
nitrate reduction test	negative
Methyl red	negative
Voges proskauer	negative
Hugh&Leifson O/F medium	oxidative reaction
Triple sugar iron agar	alkaline butt and alkaline slant
Citrate test	positive
Urease test	negative

ANTI MICROBIAL SUSCEPTIBILITY TESTING [19]

All Bacterial isolates grown in various samples were tested for antimicrobial susceptibility pattern using Kirby –Bauer Disc diffusion Method.

ANTIMICROBIAL SUSCEPTIBILITY PATTERN TESTING BY KIRBY-BAUER DISC DIFFUSION METHOD

Inoculum Preparation and procedure

3-5 similar colonies from 24 hour culture was transferred to a sterile test tube containing 3 ml of peptone water with the help of sterile bacteriological loop.

The colony was emulsified in the peptone water and turbidity matched with 0.5 McFarlands standards.

1. A Sterile cotton swab was dipped in the suspension and was evenly streaked over cation adjusted Mueller Hilton agar in three directions approximately at an angle of 60° .
2. After allowing the plates to dry for 3-5 minutes antibiotic disks (HiMedia)were placed on the agar plate with the help of a sterile forceps.

After keeping the drugs the petridishes were incubated at37°C aerobically for 24 hours.The diameter of Zone of inhibition were read with the ruled template.

Interpretation of the zone of inhibition was done according to the CLSI guidelines.

Quality control tests were done every week with following ATCC strains to test the efficacy of media and drugs.

The following **ATCC** control strains were used

- *Staphylococcus aureus*–ATCC 25923
- *Escherichia coli*-ATCC 25922
- *Pseudomonas aeruginosa*-ATCC 27853
- *Klebsiella pneumoniae*(ESBL)-ATCC 700603

Identification of Methicillin resistant Staphylococcus aureus [20]

Screening test

3-5 colonies from overnight culture was transferred to 2 ml peptone water and emulsified .The turbidity was matched with 0.5 McFarlands standard.

Lawn culture was made with the same on Muller Hilton agar plate and Incubated overnight at 33-35°C.Cefoxitin 30µg disk was placed on the agar plates. The Zone of Inhibition was interpreted according to CLSI guidelines.

A similar lawn culture of ATCC *Staphylococcus aureus* 25923 was put up as Quality control strain

Interpretation of Zone of Inhibition

Organism	**Methicillin Sensitive**	**Methicillin Resistant**
Staphylococcus aureus	≥22 mm	≤21mm

Determination of Extended Spectrum Beta Lactamase (ESBL) production [21]

Screening test

All Gram negative isolates were screened with Two disk Cefotaxime 30µg and Ceftazidime 30µg and considered to be ESBL producers if Zone of inhibition for

Cefotaxime 30µg-≤ 27 mm

Ceftazidime 30µg - ≤ 22 mm

These isolates were subjected to phenotypic confirmatory test.

Lawn culture of the isolates were made on Mueller Hilton agar plate. Ceftazidime 30µg, Ceftazidime- Clavulanate 30µg/10µg disks and Cefotaxime30µg, Cefotaxime-Clavulanate 30µg/10µg disks were placed and incubated at 37ºC for 18 hours.

INTERPRETATION

An increase in Zone of inhibition by ≥ 5 mm diameter for either antimicrobial agent tested in combination with β Lactamase inhibitor was confirmed as ESBL producer.

MICROBIOLOGICAL ANALYSIS

Infections cause relapses of Nephrotic syndrome in children who were in remission phase. [2,22] The most Common infections seen in nephrotic syndrome are urinary tract infection, acute respiratory tract infection,skin and soft tissue infection and peritonitis.[1,2,22,23].

Other infections were respiratory tract infection, cellulitis, etc.[10,22].

URINE ANALYSIS

According to Gulati S et al study UTI is the most common infection accounting about 40.26 % of the infections .[1,24]

Osmolality of urine plays an important role in enhancing the bacterial growth [25]

Accurate diagnosis of UTI can be made only when urine is collected free of contamination and processed within 2 hours of collection.

Gram staining is done by keeping a drop of well mixed urine on a clean glass slide and stained and examined under oil immersion as outlined by Washington et al.[26].

Culture of urine is done by semiquantitative technique since quantitative techniques are time consuming.The most commonly used semiquantitative techniques are Standard loop as outlined by Hoprich [27]. Other methods include Filter paper strip outlined by Leigh and Williams [28] and Dip slide by Guttmann and Naylor [29]

In culture a colony count of more than 100000 (or) 10^5 CFU/ ml is considered as significant bacteriuria which was first described by Kass [30]..

In UTI the common organisms causing infection were *Escherichia coli* followed by *Klebsiella* . [1,31].. these pathogens were sensitive to cefotaxime and amikacin.[1]

Due to the production of Extended spectrum beta lactamases there is beta lactum resistance in Enterobacteriaceae which causes therapeutic problems.[32]

THROAT SWAB ANALYSIS

According to P.Senguttuvan et al study the third common infection in NS is Acute respiratory infection.[1]

Approximately 15 to 30% of paediatric throat infections are caused by *Streptococcus pyogenes* (Group A *streptococcus* [GAS]). [33]

PERITONEAL FLUID ANALYSIS

According to P.Senguttuvan et al study the second common infection was peritonitis which was caused by *Escherichia coli* followed by *Klebsiella* .[1] But according to Se Jin Park,Jae Il Shin study *Streptococcus pneumoniae* causes primary peritonitis followed by *beta-hemolytic Streptococci, Haemophilus* and other Gram-negative bacteria.[10]

WOUND SWAB ANALYSIS

Wound swab taken for skin infections are usually polymicrobial. According to P.Senguttuvan et al study, skin infections due to bacteria accounted for 4.2% cases and most of the pathogens causing skin infections were sensitive to cefotaxime and amikacin.[1]

Results

RESULTS

This study was done in 100 hospitalized children with Nephrotic syndrome

TABLE 1: AGE WISE DISTRIBUTION OF CASES (n=100)

AGE	MALE	FEMALE	TOTAL
1 to ≤ 3	12	12	24
>3 to ≤ 5	19	5	24
>5to≤ 12	25	27	52
Total	56 (56%)	44 (44%)	100

Out of 100 cases, 52 cases were found in the age group of > 5 to ≤ 12 years,24 cases were found in the age group of > 3 to ≤ 5 years, 24 cases were found in the age group of 1 to ≤ 3 years.

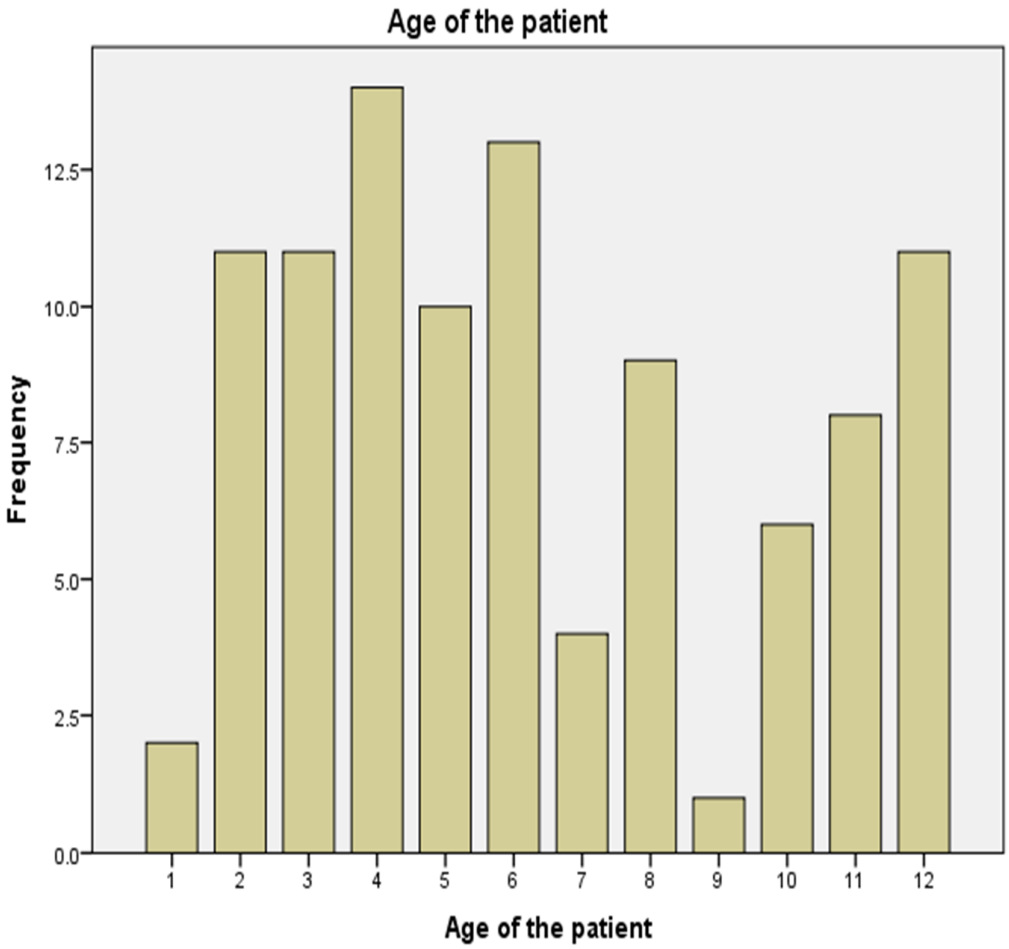

Age of the patient
Frequency
12.5
10.0
7.5
5.0
2.5
0.0
1
2
3
4
5
6
7
8
9
10
11
12
Age of the patient

TABLE 2: SEX WISE DISTRIBUTION OF CASES (n=100)

MALE CHILD	FEMALE CHILD	TOTAL
56	**44**	**100**

Out of 100 patients, Fifty six were male children and Forty four were female children.

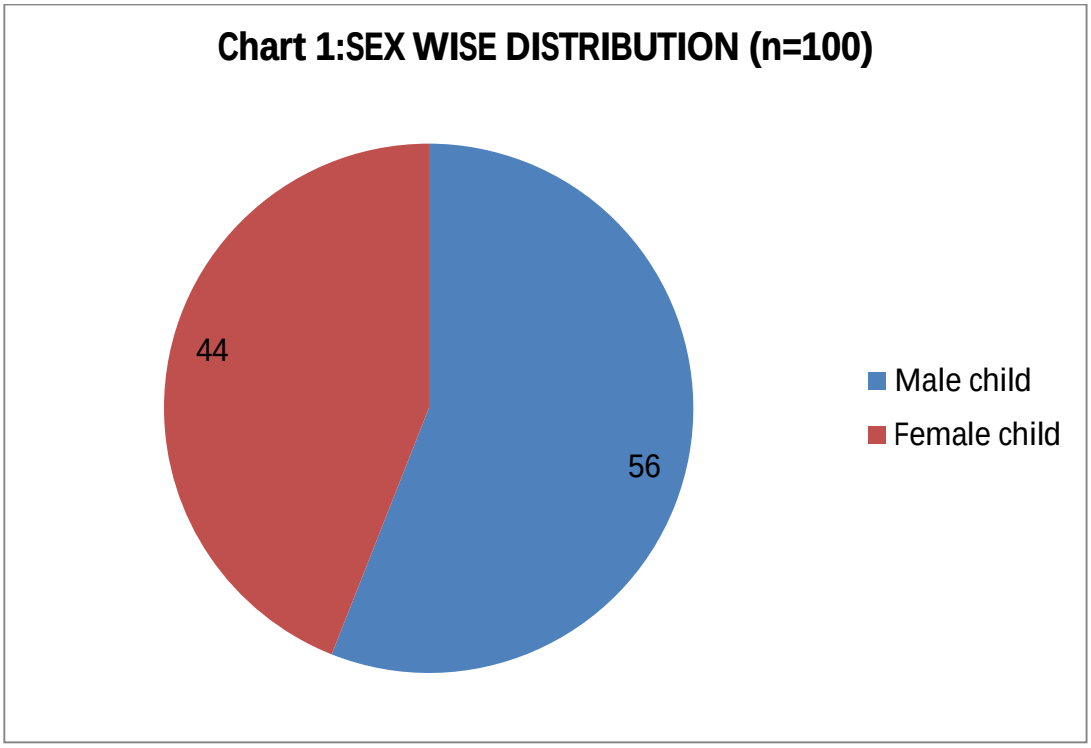


TABLE 3: SAMPLE WISE DISTRIBUTION (n=122)

Total number of patients	Total number of samples	Total number of urine samples	Total number of Throat swab samples	Total number of Blood samples
100	**122**	**100**	**15**	**7**

In total number of 100 patients 122 samples were taken.Out of 122 samples 100 were urine samples, 15 were Throat swab samples and 7 were Blood samples.

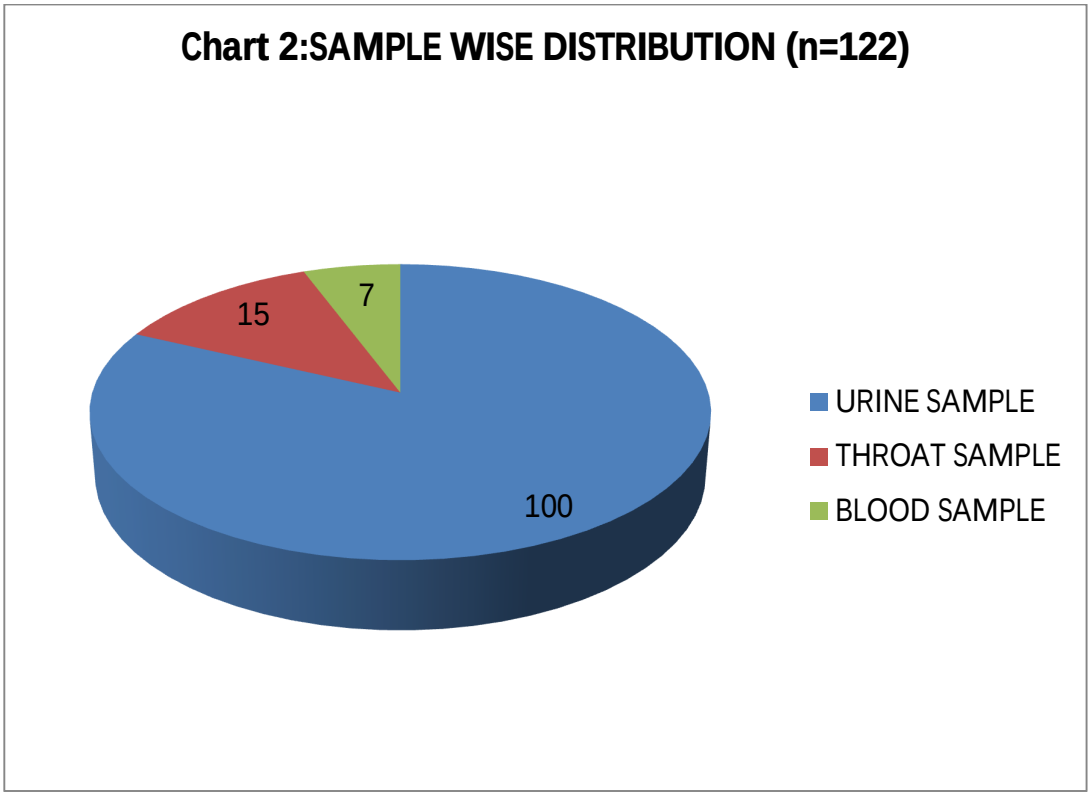


TABLE 4 : MULTIPLE SAMPLE DISTRIBUTION

Total number of urine and throat swab samples	Total number of urine,Blood and throat swab samples	Total number of urine and Blood samples
12	**3**	**4**

TABLE 5 : PERCENTAGE OF INFECTIONS IN TYPES OF NEPHROTIC SYNDROME

TYPES OF NEPHROTIC SYNDROME	PERCENTAGE
FIRST EPISODE	**14%**
STEROID DEPENDENT NS	**49%**
STEROID RESISTANT NS	**37%**

Infections were more common in the Steroid Dependent NS followed by Steroid Resistant NS. Infections were less in the First episode of NS.

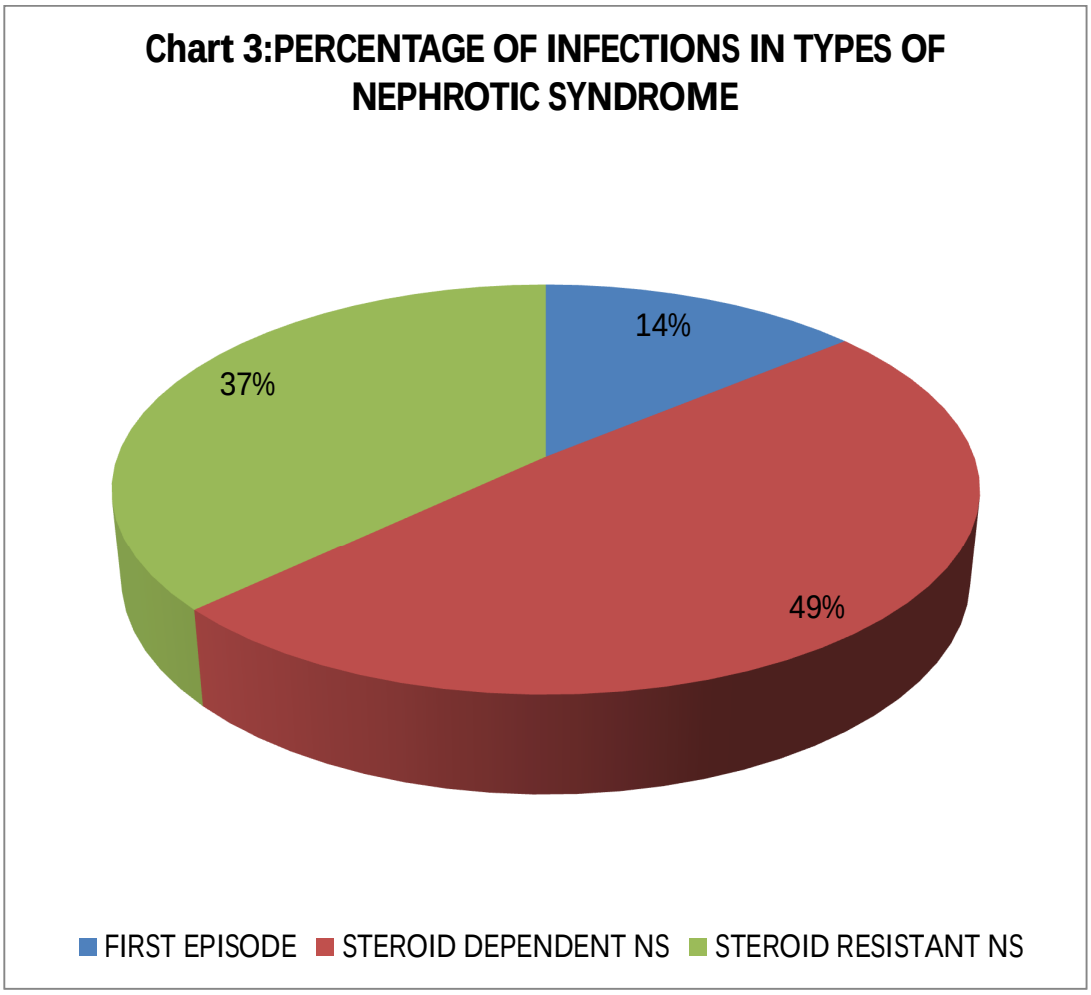


Chart 3:PERCENTAGE OF INFECTIONS IN TYPES OF NEPHROTIC SYNDROME
14%
37%
49%
FIRST EPISODE
STEROID DEPENDENT NS
STEROID RESISTANT NS

TABLE 6 : SPECTRUM OF INFECTIONS

INFECTION	MALE CHILD	FEMALE CHILD	TOTAL
Urinary tract infection	19	16	35 (35%)
Acute Upper respiratory tract infection	5	4	9(9%)
Both (UTI and AURI)	1	-	1(1%)

UTI was present in 35% of the children and was found to be more in male children than female children. Acute upper respiratory tract infection was present in 9% of the children and was more common in male children. 1% of male children had both UTI and acute upper respiratory tract infection

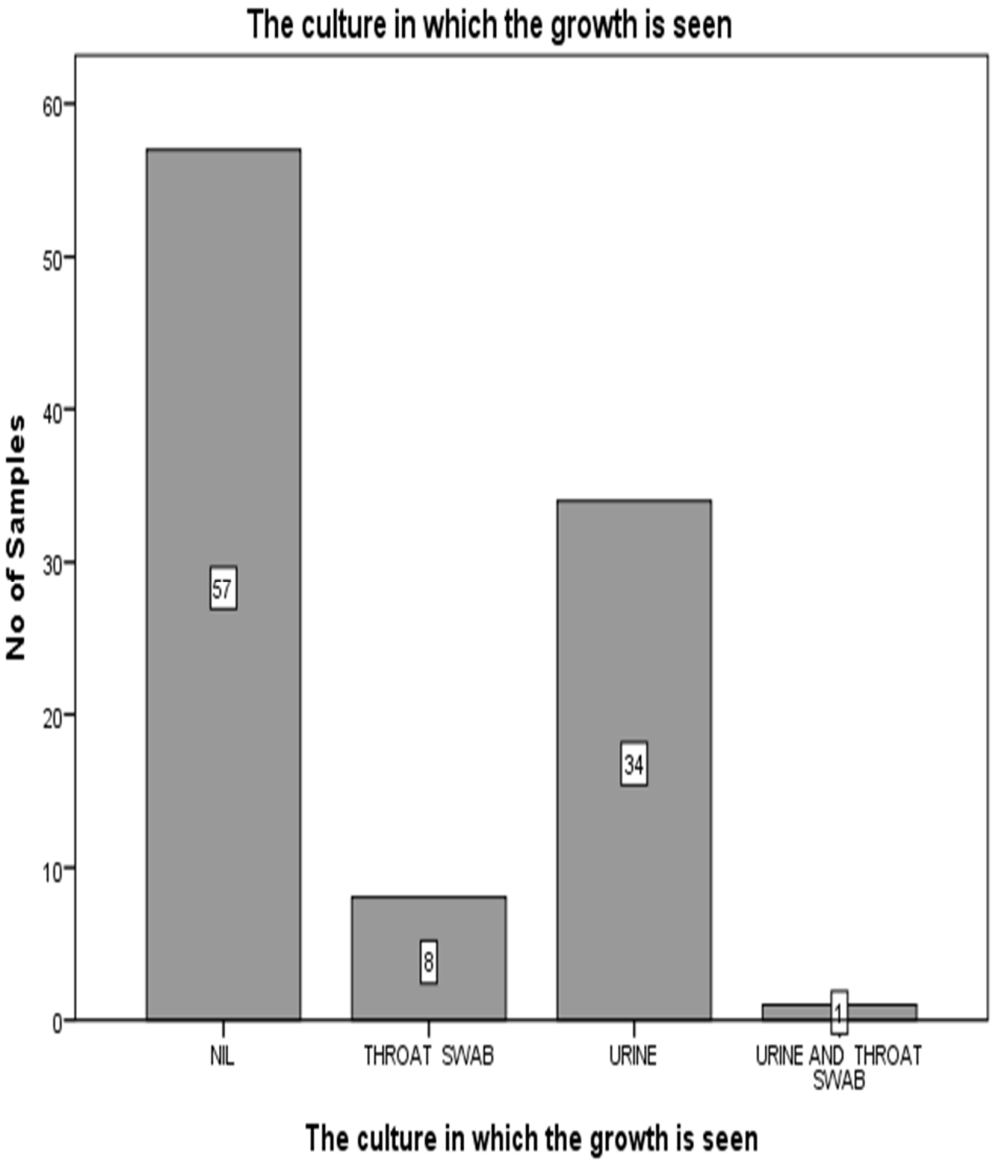

The culture in which the growth is seen
No of Samples
60
50
40
30
20
10
0
57
8
34
1
NIL
THROAT SWAB
URINE
URINE AND THROAT SWAB
The culture in which the growth is seen

TABLE 7 : PATHOGENS ISOLATED IN URINE SAMPLE –(n=35) (35%)

Name of the organism	No of isolates	percentage
Escherichia Coli	**19**	**54%**
Escherichi Coli (ESBL)	**2**	**6%**
Staphylococcus aureus (MSSA)	**4**	**11%**
Klebsiella pneumoniae	**3**	**9%**
Klebsiella pneumoniae (ESBL)	***2***	***6%***
Klebsiella oxytoca (ESBL)	**2**	**6%**
Pseudomonas spp	**2**	**6%**
Acinetobacter baumannii	**1**	**3%**
TOTAL	**35**	

Out of 35 pathogens in urine 54% were *Escherichia coli* ,11% were *Staphylococcus aureus (MSSA)* ,9% were *Klebsiella pneumoniae* , 6% were *Escherichia coli (ESBL)* 6% were *Klebsiella pneumoniae (ESBL)* , 6% were *Klebsiella oytoca (ESBL)* , 6% were *Pseudomonas spp* and 3% were *Acinetobacter baumannii*

Chart 4: ISOLATES IN URINE SAMPLE

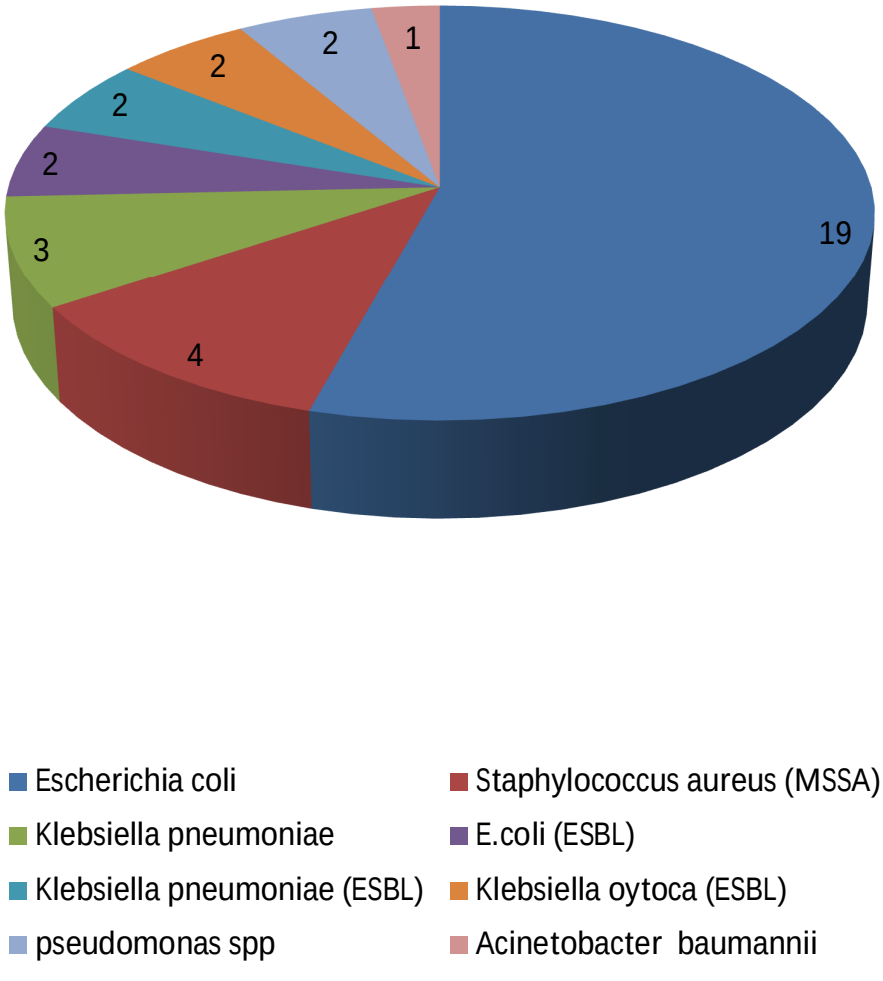
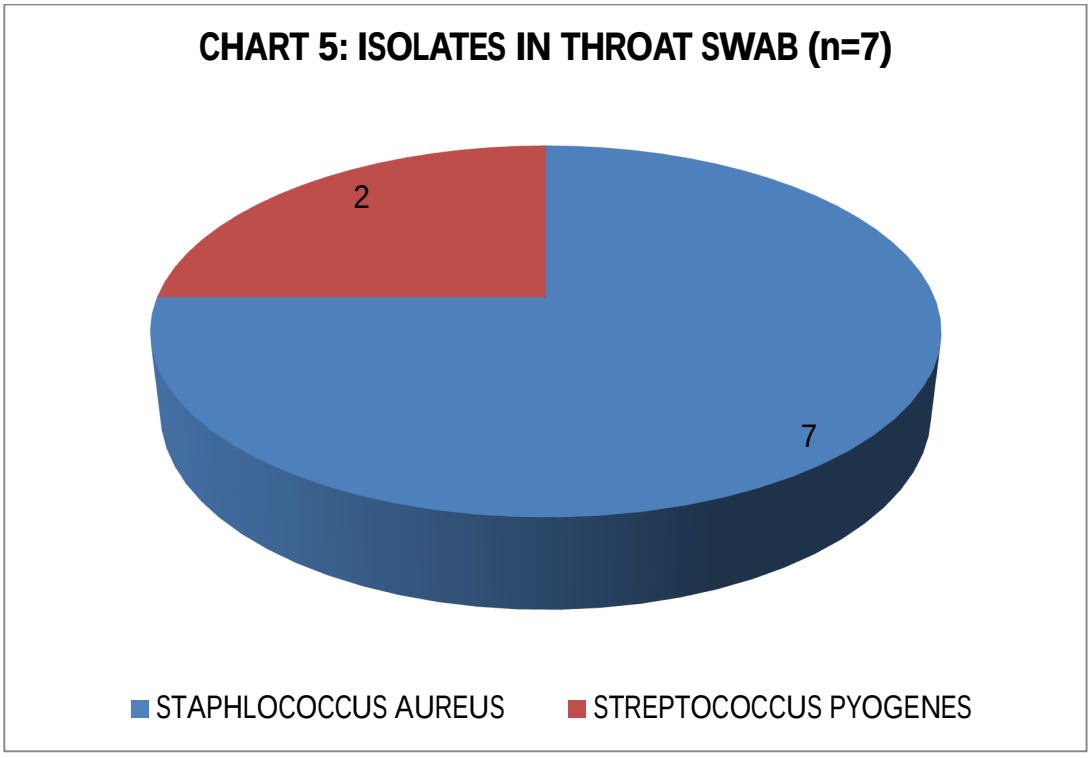


TABLE 8 : PATHOGENS ISOLATED IN THROAT SWAB SAMPLES –(n= 9) (60%)

Name of the organism	No of isolates	percentage
Staphylococcus aureus (MSSA)	**7**	**78%**
Streptococcus pyogenes	**2**	**22%**
TOTAL	**9**	

Out of 9 pathogens in Throat swab 78% were *Staphylococcus aureus (MSSA)* followed by *Streptococcus pyogenes* which was 22%.



ANTIMICROBIAL SUSCEPTIBILITY PATTERN OF THE ISOLATES.

Antimicrobial susceptibility pattern of the isolates were determined by disk diffusion method and MIC determination for Vancomycin and interpreted according to the CLSI guidelines.

TABLE 9 : ANTIBIOTIC SENSITIVITY OF PATHOGENS IN URINE IN PERCENTAGE (%) –GNB

Name of the organism	Total no of isolates	AK 30µg	COTRI 1.25/23.75 µg	PT 100/10 µg	CIP 5µg	CAZ 30 µg	CTX 30 µg	TETRA 10µg	IMP 10 µg	NOR 10 µg	NITRO 300 µg
Escherichia Coli	19	95%	100%	100%	NA	NA	84%	100%	100%	74%	68%
Klebsiella pneumoniae	3	67%	67%	100%	NA	NA	67%	100%	100%	100 %	100%
Pseudomonas spp	2	100 %	NA	100%	NA	100 %	NA	NA	100%	50%	NA
Acinetobacter baumannii	1	100 %	100%	100%	100 %	R	NA	100%	100%	NA	NA

KEY TO THE TABLE

AK-Amikacin , CIP-Ciprofloxacin, COTRI-Cotrimoxazole, TETRA-Tetracycline, PT-Piperacillin- Tazobactam, CAZ-Ceftazidime, CTX-Cefotaxime, IMP- Imipenem, NOR- Norfloxacin, NITRO – Nitrofurantoin.NA-Not Applicable

TABLE10: ANTIBIOTIC SENSITIVITY OF ESBL PATHOGENS IN URINE IN PERCENTAGE (%) –GNB

Name of the organism	Total no of isolates	AK 30µg	COTRI 1.25/23.75 µg	PT 100/10 µg	CTX 30 µg	TETRA 10µg	IMP 10 µg	NOR 10 µg	NITRO 300 µg
Escherichia Coli (ESBL)	2	50%	50%	100%	R	100%	100%	R	100%
Klebsiella pneumoniae (ESBL)	2	100%	100%	100%	R	50%	100%	100%	50%
Klebsiella oxytoca (ESBL)	2	100%	100%	100%	R	100%	100%	R	100%

KEY TO THE TABLE

AK-Amikacin, COTRI-Cotrimoxazole, TETRA-Tetracycline, PT-Piperacillin-Tazobactam, CTX-Cefotaxime, IMP- Imipenem, NOR- Norfloxacin, NITRO – Nitrofurantoin.

Among the 21 Isolates of *Escherichia coli* 2 were ESBL Producers, 5 Isolates of *Klebsiella pneumoniae* 2 were ESBL Producers and 2 Isolates of *Klebsiella oxytoca* both were ESBL Producers by Screening Test with CTX disk. They were confirmed by Phenotypic confirmatory test

There was no Amp C beta lactamase and Metallo beta lactamase (MBL) producers in this study

TABLE 11 : ANTIBIOTIC SENSITIVITY OF PATHOGENS IN URINE IN PERCENTAGE (%)-GPC

Name of the organism	Total no of isolates	PEN 10µg	GM 10µg	NOR 10 µg	COTRI 1.25/23.75 µg	TETRA 10µg	VAN	NITRO 300 µg
Staphylococcus aureus(MSSA)	**4**	**100%**	**100%**	**100%**	**100%**	**100%**	**100%**	**100%**

KEY TO THE TABLE

PEN- Penicillin, COTRI-Cotrimoxazole, GM-Gentamicin,TETRA-Tetracycline, VAN-Vancomycin, NITRO – Nitrofurantoin.

TABLE 12: ANTIBIOTIC SENSITIVITY OF PATHOGENS IN THROAT SWAB IN PERCENTAGE (%)-GPC

Name of the organism	Total no of isolates	PEN 10µg	ERY 15µg	GM 10 µg	CIP 5µg	COTRI 1.25/23.75 µg	TETRA 10µg	CK 30µg	VAN	CTX 30 µg	OF 5µg
Staphylococcus aureus (MSSA)	7	100%	100%	100 %	100 %	100%	100%	100 %	100 %	NA	NA
Streptococcus pyogenes	2	100%	50%	NA	NA	NA	100%	100 %	100 %	100 %	50%

KEY TO THE TABLE

PEN- Penicillin, ERY- Erythromycin, COTRI-Cotrimoxazole, CIP-Ciprofloxacin GM-Gentamicin,TETRA-Tetracycline,VAN-Vancomycin,CK-chloramphenicol,OF-Ofloxacin,CTX – Cefotaxime,NA-Not Applicable

Discussion

DISCUSSION

This cross sectional study was conducted at the Institute of Microbiology, Madras Medical College, Rajiv Gandhi Government General Hospital in association with the Department of Nephrology, Institute Of Child Health, Madras Medical college, Chennai.

A total number of 100 children with Nephrotic syndrome hospitalised who have satisfied the inclusion criteria were included in the study.

Out of 100 patients with Nephrotic syndrome 52% of children belonged to age group > 5 to ≤ 12 followed by age groups 1-≤ 3 (24%) and > 3 to -≤ 5 (24%) (Table : 1).In the article " Infections encountered in childhood nephrotics in a pediatric renal unit"by P. Senguttuvan et al quotes that the mean age for the onset of Nephrotic syndrome is 5.95 years .(3,1,)

In this study the prevalence of Nephrotic syndrome is higher (56%) in male children than in female children (44%) . (Table : 2).This correlates with study by O.T. Adedoyin et al study which showed a male to female ratio of 2.6:1.(34)

Totally 122 samples were collected from 100 patients.Urine sample was collected from all the patients.Throat swabs were collected from 15 children who presented with clinical symptoms of acute upper respiratory tract infection.Blood sample was collected from 7 patients who had febrile illness. (Table : 3)

Multiple samples were collected from 19 patients. Out of this urine and throat swab samples were collected from 12 patients , Urine and Blood samples were collected from 4 patients , urine blood and throat swab samples collected from 3 patients. (Table : 4)

In this study , percentage of infection varies in different types of Nephrotic syndrome . Percentage of infection is higher in the Steroid Dependent Nephrotic Syndrome (49%) followed by Steroid Resistant Nephrotic syndrome (37%) and First Episode of Nephrotic syndrome (14%) (Table : 5) . This study shows that infections are more common in children who are on immunosuppresants for treatment and also it indicates that steroid intake causes more immunosuppression compared to other drugs. This is in contrast to the study by P. Senguttuvan et al which showed that there was no difference in infections between children who received steroid therapy alone and who received both steroid and cyclophosphamide. [(1)]

The percentage of infection in children with Nephrotic syndrome in this study is higher in male children (24%) when compared to female children (22%) (Table : 6) This study co-rrelates with the study done by Moorani KN et al which showed72.58% were male children and 27.42% were female children in a ratio of 2.5 : 1. [(35)]

The spectrum of bacterial infection this study shows that Urinary tract infection is the most common infection (35%) followed by acute upper respiratory tract infection (9%) (Table :6). This co-rrelates with the study conducted by

Gulati S et al which showed urinary tract infection being the commonest 13.7% and upper respiratory infections 5.2%. [24]

Among UTI male children were more infected (19%) than female children (16%) (Table :6).

Acute upper respiratory tract infection is more common in males (5%) than female children (4%) (Table :6).

Both UTI and Acute Upper Respiratory tract Infection were present in one male child.

Among the Pathogens isolated from urine the predominant one was gram – negative bacilli .Among the Gram- negative bacilli the most common pathogen was *Escherichia coli* (60%) followed by *Klebsiella pneumonia* (15%) ,*Klebsiella oxytoca* (6%),*Pseudomonas spp* (6%) and *Acinetobacter baumannii* (3%)) (Table :7). This co-rrelates with O.T. Adedoyin et al study which showed that coliforms commonly produces UTI in children with NS ,*Klebsiella* 8.6% and *Pseudomonas* was 5.7%. [34]

The only Gram – positive pathogen isolated in the urine was *Staphylococcus aureus* .(11%).This was in contrast with the study done by Ibadin MO which showed 54.3% growth of *Staphylococcus aureus*. [36]

The predominant pathogen that was isolated in Throat swab was Gram-positive cocci *Staphylococcus aureus(MSSA)* (78%) followed by *Streptococcus pyogenes* (22%)) (Table :8).

Among the antibiotic sensitivity pattern of pathogens in urine the common pathogen is *Escherichia coli* which was 100% sensitive to Trimethoprim-sulphamethoxazole ,Piperacillin-Tazobactam,Tetracycline and Imipenem. 95% sensitive to Amikacin.84% sensitive to Cefotaxime. 74% sensitive to Norfloxacin. 68% sensitive to Nitrofurantoin .This was in contrast with the study done by Ibadin MO which showed 100% sensitive to cefotaxime and Amikacin.[36]

Klebsiella pneumoniae was 100% sensitive to ,Piperacillin-Tazobactam, Tetracycline, Imipenem, Norfloxacin and Nitrofurantoin. 67% sensitive to Amikacin, Trimethoprim-sulphamethoxazole and cefotaxime. This was in contrast with the study done by Ibadin MO which showed 100% sensitive to Amikacin.[36]

Pseudomonas spp was 100% sensitive to Amikacin, Piperacillin-Tazobactam ,ceftazidime and Imipenem.50% sensitive to Norfloxacin. This correlates with the study done by Ibadin MO which showed 100% sensitive to Amikacin and ceftazidime. [36]

Acinetobacter baumannii was 100% sensitive to Amikacin, Trimethoprim-sulphamethoxazole, Piperacillin - Tazobactam, Ciprofloxacin, Tetracycline and Imipenem. It was resistant to ceftazidime (Table :9).

Among the isolates of Enterobactericeae in urine 17% were ESBL producers by phenotypic confirmatory method.

Two isolates of *Escherichia coli* were ESBL producers. They were 100% sensitive to Piperacillin-Tazobactam ,Tetracycline,Imipenem, and Nitrofurantoin. 50% sensitive to Amikacin, Trimethoprim- sulphamethoxazole. They were resistant to Cefotaxime and Norfloxacin

Among the two *Klebsiella oxytoca* isolates both were ESBL producers.They were 100% sensitive to Amikacin Trimethoprim-sulphamethoxazole,Piperacillin-Tazobactam, Imipenem and Norfloxacin.50% sensitive to Tetracycline and Nitrofurantoin..All were resistant to cefotaxime.

Among the five *Klebsiella pneumoniae* isolates two were ESBL producers. They were 100% sensitive to Amikacin, Trimethoprim-sulphamethoxazole, Piperacillin-Tazobactam, Tetracycline, Imipenem and Nitrofurantoin. They were resistant to cefotaxime and Norfloxacin(Table :10)..

The Gram-positive pathogen *Staphylococus aureus (MSSA)* in urine was 100% sensitive to Penicillin, Gentamycin, Norfloxacin, Trimethoprim-sulphamethoxazole, Tetracycline , Vancomycin and Nitrofurantoin(Table :11)..

In Throat swab *Staphylococus aureus (MSSA)* was 100% sensitive to Penicillin, Erythromycin, Gentamycin, ciprofloxacin, Trimethoprim-sulphamethoxazole, Tetracycline , chloramphenicol and Vancomycin (Table :12)..

Streptococcus pyogenes was 100% sensitive to Penicillin, ,Tetracycline , chloramphenicol, Vancomycin and cefotaxime. 50% sensitive to Erythromycin and Ofloxacin(Table :12).

Out of 7 Blood samples there was no Growth in none of the samples.

Summary

SUMMARY

- 100 children with Nephrotic syndrome hospitalised in the Department of Nephrology, Institute Of Child Health were included in the study.
- Out of 100 patients with Nephrotic syndrome 52% of children belongs to age group > 5 to ≤ 12.
- 56% were Male children and 44% were female children .
- From 100 patients total number of various samples collected was 122.Urine sample was collected from 100 patients,Throat swab sample from 15 patients ,Blood sample from 7 patients. Multiple samples were collected from 19 patients.
- Percentage of infection was 49% in the children with Steroid Dependent Nephrotic Syndrome, 37% in Steroid Resistant Nephrotic syndrome and 14% in First Episode of Nephrotic syndrome .
- 24% of male children with Nephrotic syndrome had infections and 22% of female children had infections.
- 35% of the children had Urinary tract infection which is the most common infection and 9% of the children had acute upper respiratory tract infection.No growth was found in Blood samples.
- Gram- negative bacilli were the predominant pathogen in urine. *Escherichia coli* was the commonest organism to be isolated in 60% of the children

followed by *Klebsiella pneumonia* in 15% ,*Klebsiella oxytoca* in 6% ,*Pseudomonas spp* in 6% and *Acinetobacter baumanii* in 3% of the children.

- Gram – positive cocci present in the urine was *Staphylococcus aureus* in 11% of the children.

- Among the pathogens in urine the common pathogen *Escherichia coli* was 100 % sensitive to Trimethoprim- sulphamethoxazole ,Piperacillin-Tazobactam,Tetracycline and Imipenem. *Klebsiella pneumoniae* was 100 % sensitive to ,Piperacillin-Tazobactam,Tetracycline, Imipenem ,Norfloxacin and Nitrofurantoin. *Pseudomonas spp* was 100% sensitive to Amikacin, Piperacillin-Tazobactam ,ceftazidime and Imipenem. *Acinetobacter baumanii* was 100% sensitive to Amikacin, Trimethoprim- sulphamethoxazole, Piperacillin-Tazobactam,Ciprofloxacin,Tetracycline and Imipenem.

- Among the isolates of Enterobactericeae in urine 17% were ESBL producers by phenotypic confirmatory method.

- Two isolates of *Escherichia coli* were ESBL producers and were 100% sensitive to Piperacillin-Tazobactam ,Tetracycline,Imipenem, and Nitrofurantoin . The two *Klebsiella pneumoniae* ESBL producers were 100% sensitive to Amikacin ,Trimethoprim sulphamethoxazole, Piperacillin-Tazobactam, Imipenem and Norfloxacin. The five *Klebsiella oxytoca* ESBL producers were 100% sensitive to Amikacin, Trimethoprim-sulphamethoxazole Piperacillin-Tazobactam, Tetracycline, Imipenem and Nitrofurantoin.

- Gram-positive *Staphylococus aureus (MSSA)* in urine was 100% sensitive to Penicillin, Gentamycin,Norfloxacin, Trimethoprim- sulphamethoxazole ,Tetracycline , chloramphenicol, Vancomycin and Nitrofurantoin
- Gram- positive cocci *Staphylococcus aureus(MSSA)* was the predominant pathogen seen in 78% of the Throat swab followed by *Streptococcus pyogenes* seen in 22%.
- *Staphylococus aureus (MSSA)* grown in Throat swab was 100% sensitivetoPenicillin,Erythromycin,Gentamycin,ciprofloxacin,Trimethoprim-sulphamethoxazole ,Tetracycline , chloramphenicol and Vancomycin.
- *Streptococcus pyogenes* grown in Throat swab was 100% sensitive to Penicillin, ,Tetracycline , chloramphenicol, Vancomycin and cefotaxime.

Conclusion

CONCLUSION

- Nephrotic syndrome is predominant in the age group > 5 to ≤ 12.
- One of the major complication of Nephrotic syndrome is Infections.
- The incidence of infection was higher in children with Steroid Dependent Nephrotic Syndrome than Steroid Resistant Nephrotic syndrome and First Episode of Nephrotic syndrome .
- The incidence of infection in male children was 24% and it was 22% in female children.
- Urinary tract infection is the most common infection in children with Nephrotic syndrome followed by acute upper respiratory tract infection.
- The most common organism in UTI was *Escherichia coli* which was sensitive to Trimethoprim- sulphamethoxazole ,Piperacillin-Tazobactam,Tetracycline and Imipenem.
- Among the Enterobacteriaceae family ,ESBL production is a cause of antimicrobial resistance and AmpC beta lactamase and Metallo beta lactamase producers were not detected in this study.

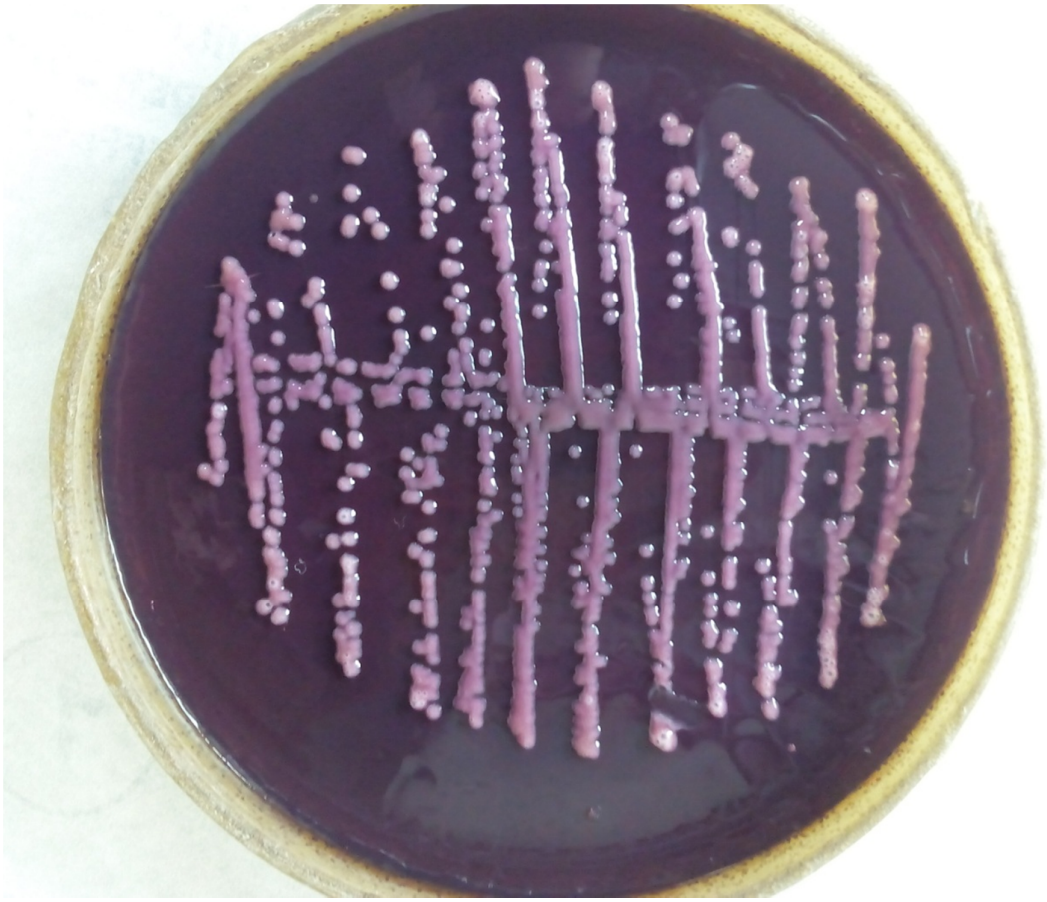
- The most common organism in acute upper respiratory tract infection was *Staphylococus aureus (MSSA*) that was 100% sensitive to Penicillin, Erythromycin, Gentamycin, Ciprofloxacin, Trimethoprim- sulphamethoxazole, Tetracycline, Chloramphenicol and Vancomycin.

Infections in Nephrotic syndrome should be diagnosed earlier and treated with appropriate antibiotics so that prolonged Remission and Relapse can be prevented. Also earlier treatment reduces the Morbidity and Mortality.

COLOUR PLATES

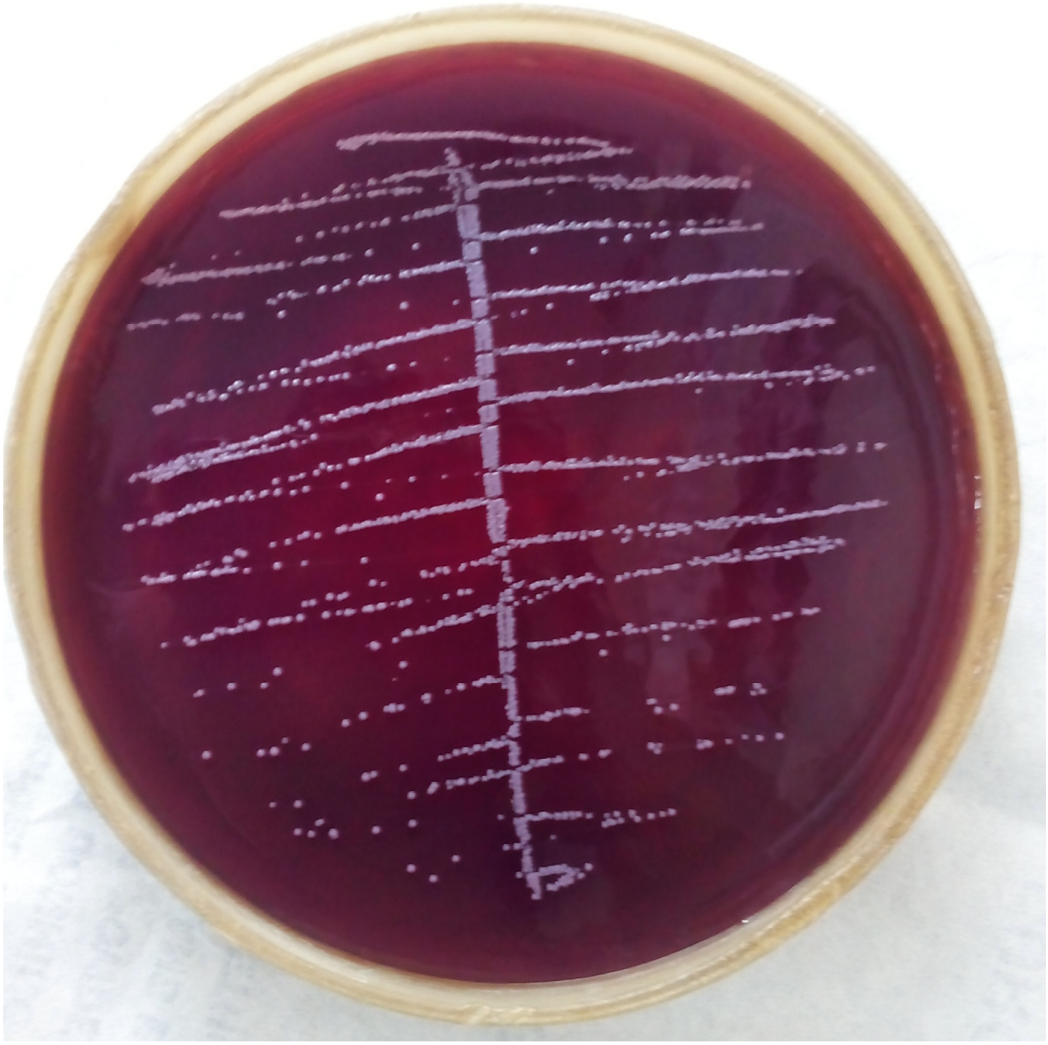
Beta-Hemolytic Streptococcus pyogenes

Lactose fermenting colonies of *Escherichia coli* in urine sample



Mucoid and Lactose Fermenting colonies of *Klebsiella pneumoniae*

in urine sample



Opaque colonies of *Staphylococcus aureus* in urine sample

Appendix

APPENDIX I

ABBREVATIONS

UTI	URINARY TRACT INFECTION
MCNS	MINIMAL CHANGE NEPHROTIC SYNDROME
I.V	INTRAVENOUS
NSAID	NON-STEROIDAL ANTI INFLAMMATORY DRUGS
ACE	ANGIOTENSIN CONVERTING ENZYME
ATN	ACUTE TUBULAR NECROSIS
BP	BLOOD PRESSURE
OPV	ORAL POLIO VACCINE
IPV	INJECTABLE POLIO VACCINE
DPT	DIPTHERIA,PRTUSSIS,TETANUS
H.INFLUENZA B	HEMOPHILUS INFLUENZA B
HEP-B	HEPATITIS B
MMR	MEASLES,MUMPS,RUBELLA
BAP	BLOOD AGAR PLATE
CLSI	CLINICAL AND LABORATORY STANDARD INSTITUTE
ATCC	AMERICAN TYPE CULTURE COLLECTION
ESBL	EXTENDED BROAD SPECTRUM BETA LACTAMASE
CFU	COLONY FORMING UNIT
NS	NEPHROTIC SYNDROME
AURI	ACUTE UPPER RESPIRATORY TRACT INFECTION
MSSA	METHICILLIN SENSITIVE STAPHYLOCOCCUS AUREUS
GNB	GRAM NEGATIVE BACILLI
MIC	MINIMUM INHIBITORY CONCENTRATION
MBL	METALLOBETA LACTAMASE
GPC	GRAM POSITIVE COCCI

APPENDIX II

A. STAINS AND REAGENTS

1. Gram staining

Methyl violet (2%)	l0g Methyl violet in 100ml absolutealcohol in 1 litre of distilled water(primary stain)
Grams Iodine	l0g Iodine in 20g KI (fixative)
Acetone	Decolourising agent
Carbolfuchsin 1%	Secondary stain.

B. MEDIA USED

1.Mac Conkey agar

Peptone	20g
Sodium taurocholate	5g
Distilled Water	1 ltr
Agar	20 g
2% neutral red in 50% ethanol	3.5ml
10% lactose solution	l00ml

Dissolve peptone and taurocholate in water by heating. Add agar and dissolveit in steamer. Adjust pH to 7.5. Add lactose and neutral red shake well and mix.

Heatin free steam (100°C) for 1 hour, then autoclave at 115℃ for 15 minutes.

2. Blood agar (5% sheep blood agar)

Peptone l0g

NaCl 5g

Distilled water 1 Ltr

Agar l0g

Dissolve ingredients in distilled water by boiling, and add 5% sheepblood(sterile) at 55℃ adjust pH to 7.4.

3. Cation adjusted Mueller- Hinton Agar

Beef infusion	300ml
Caeseinhydrolysate	17.5g
Starch	1.5g
Agar	l0g

Distilled water 1 ltr

pH = 7.4

Steriliseby autoclaving at 121°C for 20 mins

C. MEDIA REQUIRED FOR BIOCHEMICAL IDENTIFICATION

1. Oxidase Reagent

Tetra methyl p-phenylenediaminedihyrochloride- 1% aqueous solution.

2. Catalase

3% hydrogen peroxide

3.Coagulase test

Tube coagulase test

1. Prepare a 1 in 6 dilution of the plasma in saline and place 1 ml volume of the diluted plasma in small tube.
2. Emulsify a colony of the Staphylococcus under test in the tube of the diluted plasma
3. Appropriate controls were put up
4. Incubate the tubes at 37°Cfor 4 hours
5. Examine the tubes at 1,2,3 4 hours for Clot formation
6. Leave the tubes at room temperature overnight and reexamine
7. Read as positive any degree of clot formation

Slide Coagulase test

Emulsify a staphylococcus colony in a drop of water on a microscopeslide with a minimum of spreading .Make a similar suspensions of control positive and negative strains to confirm the proper reactivity of the plasma. Stir the adhering plasma into Staphylococcal suspension on the slide.Formation of Clumps are read as Positive.

4.Indole test

Kovac's reagent

Amyl or isoamyl alcohol 150ml Para dimethyl amino benzaldehyde – l0gConcentrated hydrochloric acid - 50ml

Dissolve the aldehyde in the alcohol and slowly add the acid. Prepare in small quantities and store in the refrigerator. Shake gently before use.

5.Christensen's Urease test medium

Peptone lg

Sodium chloride 5g

Dipotassium hydrogen phosphate 2g

Phenol red 6ml

Agar 20g

Distilled water 1 ltr

10% sterile solution of glucose 10ml

Sterile 20% urea solution 100ml

Sterilize the glucose and urea solutions by filtration. Prepare the basal medium without glucose and urea, adjust to pH 6.8-6.9 and sterilize by autoclaving in a flask at 121°C for 30min. Cool to about 50°C, add the glucose & urea, and tube the medium as slopes.

6. Simmon's Citrate Medium

Koser's medium 1 ltr

Agar 20 g

Bromothymol blue 0.2% 40ml

Dispense, autoclave at 121°C for 15 min and allow to set as slopes

7. Triple Sugar Iron medium

Beef extract	3g
Yeast extract	3g
Peptone	20g
Glucose	lg
Lactose	10 g
Sucrose	l0g
Ferric citrate	0.3g
Sodium chloride	5g

Sodumthiosulphate	0.3g
Agar	12g
Phenol red 0.2% solution	12ml
Distilled water	1 ltr

Heat to dissolve the solids, add the indicator solution, mix and tube. Sterilize at 121℃ for 15 min and cool to form slopes with deep butts.

8. Glucose phosphate broth

Peptone	5g
Dipotassium hydrogen phosphate	5g
Water	1 ltr
Glucose 10% solution	50ml

Dissolve the peptone and phosphate and adjust the pH to 7.6. Filter dispense in 5mlamounts and sterilize at 121℃ for 15min. Sterilize the glucose solution by filtration and add 0.25ml to each tube.

Methyl Red Reagent

Methyl Red	l0mg
Ethyl alcohol	30ml
Distilled water	20ml

VogesProskauer Reagent

Reagent A: Alpha naphthol	5g
Ethyl alcohol	100ml
Reagent B: Potassium hydroxide	40g
Distilled water	100ml

9. Peptone water fermentation test medium

To the basal medium of peptone water, add sterilised sugars of 1% indicator bromothymol blue with Durham's tube. Basal medium peptone water Sugar solutions:

Sugar	1ml

Dislilled water	100ml	pH = 7.6.

10.Mannitol motility medium

Agar	5g
Peptone	lg
Potassium nitrate	1g
Mannitol	2g
Phenol red indicator	
Distilled water	1000ml
pH	7.2

12. Potassium nitrate broth

Potassium nitrate (KNO3)	0.2gm
Peptone	5.0gm
Distilled water	100ml

The above ingredients were mixed and transferred into tubes in 5 mlamount and autoclaved.

13. Phenyl alanine deaminase test

Yeast Extract	3g
Dl-Phenylalamine	2 g
Disodium hydrogen phosphate	l g
Sodium Chloride	5 g
Agar	12g
Distilled water	1 lr
PH	7.4

Distributed in tubes and sterilized by autoclaving at 121° C for 1 5 minutes,allowed to solidify as long slopes.

14. Sugar fermentation medium

Peptone	15g
Andrade's indicator	10 ml

Sugar to be tested	20g
Water	1 litre

Andrade's indicator is prepared from 0.5% aqueous acid fuchsin to which sufficient 1M sodium hydroxide has been added to turn the colour of the solution yellow.

Dissolve the peptone and Andrade's indicator in 1 litre of water and add 20g of the sugar; sugars to be tested generally include glucose, sucrose, lactose and maltose. Distribute 3ml amounts in standard test tubes containing an inverted Durham tube. Sterilize by steaming at 100 degree C for 30 min on 3 consecutive days.

APPENDIX III

Panel of antibiotic Disk used for*Staphylococcus aureus* (Hi Media)

ANTIBIOTIC DISK	CONENT	SENSITIVE (mm)	INTERMEDIATE (mm)	RESISTANT (mm)
Penicillin	10 units	≥29	-	≤28
Cefoxitin	30µg	≥22	-	≤21
Gentamicin	10µg	≥15	13-14	≤12
Tetracycline	30µg	≥19	15-18	≤14
Ciprofloxacin	5µg	≥21	16-20	≤15
Erythromycin	15µg	≥18	14-17	≤13
Trimethoprim-Sulfamethoxazole	1.25/23.75µg	≥16	11-15	≤10
Norfloxacin	10 µg	≥ 17	13-16	≤12
Nitrofurantoin	300 µg	≥ 17	15-16	≤14

Panel of drugs used for *β Hemolytic streptococcus spp*

Antibiotic	Content	Sensitive (mm)	Intermediate (mm)	Resistant (mm)
Pencillin	10 units	≥24	-	-
Cefotaxime	30µg	≥24	-	-
Vancomycin	30µg	≥17	-	-
Erythromycin	15 µg	≥21	16-20	≤15
Tetracycline	30 µg	≥23	19-22	≤18
Ofloxacin	5 µg	≥16	13-15	≤12
Chloramphenicol	30µg	≥21	18-20	≤17

Panel of drugs used forEnterobacteriaceae

Antibiotic	Disk Content	Sensitive(mm)	Intermediate(mm)	resistant(mm)
Cefotaxime	30 µg	≥26	23-25	≤22
Amikacin	30 µg	≥17	15-16	≤14
Tetracycline	30 µg	≥15	12-14	≤11
Norfloxacin	10 µg	≥ 17	13-16	≤12
Trimethoprim-Sulfamethoxazole	1.25/23.75 µg	≥16	11-15	≤10
Imipenem	10 µg	≥23	20-22	≤19
Piperacillin-tazobactam	100/10 µg	≥21	18-20	≤17
Nitrofurantoin	300 µg	≥ 17	15-16	≤14

Panel of drugs used for *Pseudomonas aeruginosa*

Antibiotic	Disk Content	Sensitive(mm)	Intermediate(mm)	Resistant(mm)
Piperacillin-tazobactam	100/10 µg	≥21	15-20	≤14
Ceftazidime	30µg	≥18	15-17	≤14
Imipenam	10µg	≥19	16-18	≤15
Amikacin	30µg	≥17	15-16	≤14
Norfloxacin	10 µg	≥ 17	13-16	≤12

Panel of drugs used for *Acinetobacter baumannii*

Antibiotic	Disk Content	Sensitive (mm)	Intermediate (mm)	Resistant (mm)
Piperacillin-tazobactam	100/10 µg	≥21	15-20	≤14
Ceftazidime	30µg	≥18	15-17	≤14
Imipenem	10µg	≥22	19-21	≤18
Amikacin	30µg	≥17	15-16	≤14
Tetracycline	30µg	≥15	12-14	≤11
Norfloxacin	10 µg	≥ 17	13-16	≤12
Trimethoprim-Sulfamethoxazole	1.25/23.75 µg	≥16	11-15	≤10

Annexure

ANNEXURE - I

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No. 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. Meenakshi S
Post Graduate in MD (Microbiology),
Institute of Microbiology,
Madras Medical College
Chennai 600 003

Dear Dr. Meenakshi S

The Institutional Ethics Committee has considered your request and approved your study titled **"A Study on Bacteriological Profile and Antimicrobial susceptibility pattern of infections in hospitalised children with Nephrotic Syndrome.". No. 04092015**

The following members of Ethics Committee were present in the meeting held on 08.09.2015 conducted at Madras Medical College, Chennai-3.

1.	Prof.C.Rajendran, M.D.,	: Chairperson
2.	Prof.R.Vimala, M.D., Dean, MMC, Ch-3	: Deputy Chairperson
3.	Prof.Sudha Seshayyan, M.D., Vice-Principal, MMC, Ch-3	: Member Secretary
4.	Prof.B.Vasanthi, M.D., Professor Pharmacology, MMC	: Member
5.	Prof.P.Ragumani, M.S., Professor, Inst.of Surgery, MMC	: Member
6.	Prof.Baby Vasumathi, Director, Inst.of O&G, Ch-8	: Member
7.	Prof.V. Amudavalli, Professor, Inst.of Biochemistry, MMC	: Member
8.	Prof.Srinivasagalu, Director, Inst.of Inter Med. MMC	: Member
9.	Thiru S.Rameshkumar, B.Com., MBA	: Lay Person
10.	Thiru S.Govindasamy, B.A., B.L.,	: Lawyer
11.	Tmt.Arnold Saulina, M.A., MSW.,	: Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee
MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

ANNEXURE – II

PROFORMA

NAME: DATE:

AGE: IP NO:

SEX:

WARD NO:

ADDRESS:

PRESENTING COMPLAINTS :

PAST HISTORY :

TREATMENT HISTORY:

LABORATORY EVALUATION:

BIOCHEMICAL PARAMETERS

CBC	:
Blood urea	:
Serum creatinine	:
Serum protein	:
Serum Albumin	:
Serum cholesterol	:
Urine routine	:

MICROBIOLOGICAL INVESTIGATIONS

SPECIMEN:

GRAM'STAIN:

CULTURE:

MAC :

BAP :

ISOLATION IDENTIFIED:

ANTIMICROBIAL SENSITIVITY:

ANNEXURE – III

INFORMATION SHEET

STUDY TITLE: "A STUDY ON BACTERIOLOGICAL PROFILE AND ANTIMICROBIAL SUSCEPTIBILITY PATTERN OF INFECTIONS IN HOSPITALISED CHILDREN WITH NEPHROTIC SYNDROME".

INVESTIGATOR : **Dr.S.MEENAKSHI,**
II yearM.D Microbiology Post Graduate,
Institute of Microbiology,
Madras Medical College,
Chennai - 600003.

GUIDE : **Dr.R.VANAJA MD.,**
Professor of Microbiology,
Institute of Microbiology,
Madras Medical College,
Chennai 600 003.

Children with nephrotic syndrome are at increased risk of infection. The following factors may contribute to this problem.

1. Reduced serum concentrations of immunoglobulin G
2. Impaired ability to make specific antibodies
3. Decreased levels of the alternative complement pathway factors B and D
4. Immunosuppresive therapy.

Infections still remain major cause of death and also be responsible for a poor response to therapy or induce relapse.

I am going to evaluate the organism infecting the children with nephrotic syndrome and determine the antibiotic susceptibility pattern. I am going to collect urine,blood,throat swab,peritoneal fluid and wound swab samples from patient and process them accordingly.100 patients are included in this study after getting informed consent only. This study is entirely voluntary and patient can withdraw any time from this study. Extra cost will not be incurred to the patients in this study. Any doubt regarding this study will be willingly clarified. Results of the study will be published. In case of any doubt, please contact Dr.S.Meenakshi, Cell: 965553272.

Signature of investigator Signature of participant

Date:

PATIENT CONSENT FORM

TITLE OF THE STUDY :"
"A STUDY ON BACTERIOLOGICAL PROFILE AND ANTIMICROBIAL SUSCEPTIBILITY PATTERN OF INFECTIONS IN HOSPITALISED CHILDREN WITH NEPHROTIC SYNDROME".

Name :
Age :
Sex :
Date :
IP No :
Project Patient No :

Documentation of the informed consent from parents/ guardian
I _____ have read the information in this form (or it has been read to me). I was free to ask any questions and they have been answered. I hereby give my consent for my child to be included as a participant in " **A STUDY ON BACTERIOLOGICAL PROFILE AND ANTIMICROBIAL SUSCEPTIBILITY PATTERN OF INFECTIONS IN HOSPITALISED CHILDREN WITH NEPHROTIC SYNDROME".**

I have read and understood this consent form and the information provided to me.

1. I have had the consent document explained to me.
2. I have been explained about the nature of the study.
3. I have been explained about my rights and responsibilities by the investigator.
4. I have been informed the investigator of all the treatments that my child is taking or have taken in the past _____ months including any native (alternative) treatment.
5. I have been advised about the risks associated with my child's participation in this study.
6. I agree to cooperate with the investigator and I will inform him/her immediately if my child suffer unusual symptoms.
7. My child have not participated in any research study within the past _____ month(s).
8. I am aware of the fact that my child can opt out of the study at any time without having to give reason and this will not affect my child's future treatment in this hospital.
9. I am also aware that the investigator may terminate my child's participation in the study at any time, for any reason, without any consent.
10. I hereby give permission to the investigators to release the information obtained from my child as result of participation in this study to the sponsors, regulatory authorities, Govt. agencies, and IEC. I understand that they are publicly presented.
11. I have understand that my child's identity will be kept confidential if my data are publicly presented.
12. I have had my questions answered to my satisfaction.
13. I have decided my child to be in the research study.

I am aware that if I have any question during this study, I should contact the investigator. By signing this consent form I attest that the information given in this document has been clearly explained to me and understood by me, I will be given a copy of this consent document.

For participants:
Name& signature of the parent/guardian.)

Name _____ Signature_____Date_____

Name and Signature of the investigator or his representative obtaining consent:

Name _____ Signature_____Date_____

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B´Äö\´¯¨£k®uø»¨¦:

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இச்சுய ஒப்புதல் படிவத்தில்கையெழுத்திடுவதன் மூலம் இதிலுள்ள AøÚzxÂå¯[PÐ® GÚUS öuÎÁõP ÂÍUP¨£mhxGßÖ® öu¶ÂUQ÷Óß. Ca_¯ J¨¦uÀ£iÁzvß J¸{PÀGÚUS öPõkUP¨£k®GßÖ® öu¶¢x öPõs÷hß.

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B´ÁõÍºøPö¯õ¨£® ÷uv:

Master Chart

MASTER CHART

S. No	age	sex	IP NO	chief complaints	Duration of presenting complaints	Diagnosis	Total count	Blood urea	Serum creatinine	Serum Total protein	Serum albumin	Serum choles terol	Urine Routine	urine sample	Blood sample	Throat swab sample	DGS
1	11	M	883192	fever,edema and abdominal distension	2 days	SRNS	12000	88	0.7	2.7	1.7	420	3+	Y	Y	N	1.NO PUS CELLS ,NO ORGANISM
2	5	M	883205	facial puffiness,abdominal distension pedal edema	1 week	SDNS	8600	20	0.5	3	1.6	324	2+	Y	N	N	NO PUS CELLS ,NO ORGANISM
3	12	F	883352	fever,edema and oliguria	2 days	SRNS	7700	22	1	2.9	1.5	352	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
4	2	M	880956	edema, oliguria ,abdominal distension	6 weeks	First Episode	10,100	37	0.8	5	3	215	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
5	11	M	884025	facial puffiness,oliguria	2 months	SRNS	10,800	20	0.6	3.4	2.1	356	4+	Y	N	N	NO PUS CELLS ,NO ORGANISM
6	5	M	885831	oliguria,edema	2 days	SDNS	8200	30	1	3	1	350	3+	Y	N	N	FEW PUS CELLS ,NO ORGANISM
7	3	F	885317	facial puffiness,oliguria	2 months	SRNS	8,000	22	0.8	3	1.5	360	2+	Y	N	N	NO PUS CELLS ,NO ORGANISM
8	11	M	886917	edema,cold,cough	2 weeks	SDNS	13,500	18	0.6	4.5	2	320	3+	Y	N	Y	1.MANY PUS CELLS ,NO ORGANISM 2.FEW PUS CELLS.MANY GPC IN CLUSTERS SEEN
9	12	F	886961	facial puffiness, edema,oliguria,cough	1 week	SDNS	30,200	28	1.1	4.3	2.9	420	3+	Y	N	Y	1.NO PUS CELLS ,NO ORGANISM 2.FEW PUS CELLS.MANY GPC IN CLUSTERS SEEN
10	4	M	886976	edema,oliguria	1 week	SDNS	6,900	25	0.6	5.2	3.5	324	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
11	8	M	886394	edema,oliguria,cold,cough	2 days	SRNS	16,800	16	0.7	4.5	2.5	320	3+	Y	Y	Y	1.MANY PUS CELLS ,NO ORGANISM 2. NO PUS CELLS.FEW GPC IN PAIRS AND SHORT CHAINS SEEN
12	6	F	885991	facial puffiness,abdominal distension pedal edema	4 days	SRNS	12,900	40	0.8	3	1.5	350	3+	Y	N	N	.MANY PUS CELLS ,NO ORGANISM
13	12	M	888159	facial puffiness,oliguria	2 months	SRNS	10,000	32	0.8	3	1.5	320	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM

14	5	M	887308	fever,facial puffines,abdominal distension	3 days	SDNS	12,300	23	0.6	3.5	1.4	456	3+	Y	Y	Y	1.NO PUS CELLS ,NO ORGANISM 3.FEW PUS CELLS.MANY GPC IN CLUSTERS SEEN
15	3	F	888088	facial puffiness,cold,cough	1 week	First Episode	11,000	15	0.4	3	1.5	360	2+	Y	N	Y	1.NO PUS CELLS ,NO ORGANISM 2NO PUS CELLS ,NO ORGANISM.
16	2	F	887416	abdominal distension,facial puffiness, pedal edema,oliguria	3 days	SRNS	12,000	28	0.4	3.6	1.7	360	3+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
17	2	M	887588	facial puffiness,oliguria,	4 days	First Episode	14,320	47	1	3	1.8	418	3+	Y	N	Y	1.NO PUS CELLS ,NO ORGANISM 2NO PUS CELLS ,NO ORGANISM.
18	11	F	888307	abdominal pain,vomiting	2 days	SRNS	17,100	24	0.6	3	1.5	360	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
19	12	M	888173	facial puffiness,abdominal distension pedal edema	4 days	SRNS	8,500	42	1.2	3.7	1.8	450	2+	Y	N	N	NO PUS CELLS ,NO ORGANISM
20	12	M	888623	abdominal distension,facial puffiness,	5 days	SRNS	16,700	40	1.5	3.5	1.5	300	3+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
21	12	F	889225	oliguria,edema,facial puffiness	3 days	SRNS	7,800	23	0.6	2.6	1.3	400	4+	Y	N	Y	1.NO PUS CELLS ,NO ORGANISM 2.FEW PUS CELLS.MANY GPC IN CLUSTERS SEEN
22	8	M	889071	facial puffiness,abdominal distension pedal edema	1 week	First Episode	11,600	14	0.4	2.6	1.6	485	4+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
23	12	F	889509	oliguria,edema,facial puffiness	1 month	SRNS	12,700	43	1.1	5.7	3.9	402	1+	Y	N	N	NO PUS CELLS ,NO ORGANISM
24	7	F	889523	facial puffiness,abdominal distension pedal edema,oliguria	3 days	SRNS	18,000	44	0.5	4	2.5	350	1+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
25	7	F	889560	abdominal pain,vomiting,oliguria	1 week	SDNS	12,300	34	0.6	3.5	2.4	360	2+	Y	N	N	MANY PUS CELLS FEW GNB SEEN
26	7	M	890363	oliguria,edema,facial puffiness	5 days	SDNS	11,000	20	0.8	3	1.5	300	3+	Y	N	N	FEW PUS CELLS ,NO ORGANISM
27	10	M	889705	abdominal distension,facial puffiness, pedal edema,oliguria	10 days	SDNS	11,300	26	0.5	3.5	2	380	3+	Y	N	N	FEW PUS CELLS ,NO ORGANISM

28	6	F	889977	oliguria,edema,facial puffiness	5 days	First Episode	16,700	22	0.6	4	2.5	280	3+	Y	Y	N	1.NO PUS CELLS ,NO ORGANISM
29	5	M	890529	abdominal distension,facial puffiness, pedal edema,oliguria	3 days	SRNS	14,000	24	0.8	3	1.5	370	4+	Y	N	Y	1.NO PUS CELLS ,NO ORGANISM 2.FEW PUS CELLS.MANY GPC IN CLUSTERS SEEN
30	4	M	892013	oliguria,pedal edema,facial puffiness	5 days	SDNS	9,100	29	0.6	4	2.5	420	1+	Y	N	N	NO PUS CELLS ,NO ORGANISM
31	4	M	891615	abdominal distension,facial puffiness, pedal edema,oliguria	3 days	SDNS	22,700	48	0.8	4	2	390	4+	Y	N	N	NO PUS CELLS ,NO ORGANISM
32	5	M	892123	oliguria,edema,facial puffiness	5 days	SRNS	12,000	30	0.6	3.5	1.5	400	1+	Y	N	N	NO PUS CELLS ,NO ORGANISM
33	3	M	891643	abdominal distension,facial puffiness,	5 days	SRNS	23,100	17	0.5	3.4	2.3	305	4+	Y	N	Y	1.NO PUS CELLS ,NO ORGANISM 2.FEW PUS CELLS.MANY GPC IN CLUSTERS SEEN
34	3	M	892017	facial puffiness,cold,cough	1 week	SDNS	15,400	14	0.5	4	2	360	4+	Y	N	Y	MANY PUS CELLS ,NO ORGANISM
35	1	m	890185	abdominal distension,facial puffiness, pedal edema,oliguria	3 days	SRNS	16,700	46	0.5	4	2.5	350	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
36	2	F	893724	abdominal distension,facial puffiness, pedal edema,oliguria,cold , cough	3 days	First Episode	15,000	20	0.5	4.4	2.1	568	3+	Y	N	Y	1.NO PUS CELLS ,NO ORGANISM 2.NO PUS CELLS. NO ORGANISMS
37	4	M	893212	abdominal distension,facial puffiness, pedal edema,oliguria	2 days	SDNS	7,400	27	0.4	4	2.5	400	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
38	10	F	893572	oliguria,pedal edema,facial puffiness	5 days	SDNS	12,500	28	0.7	4	2	390	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
39	2	F	893790	facial puffiness, pedal edema,oliguria	2 weeks	SRNS	18,000	15	0.8	3.9	1.8	656	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
40	1	F	893786	abdominal distension,facial puffiness, oliguria	4 days	SDNS	14,200	19	0.5	4	2	385	3+	Y	N	N	FEW PUS CELLS ,NO ORGANISM
41	10	F	894240	abdominal distension,facial puffiness, pedal edema,oliguria,cold , cough	2 days	SDNS	11,600	29	0.6	4	1.8	360	4+	Y	N	Y	1.NO PUS CELLS ,NO ORGANISM 2.FEW PUS CELLS.MANY GPC IN PAIRS AND SHORT CHAINS SEEN

42	8	F	894370	oliguria,pedal edema,facial puffiness	5 days	SRNS	27,700	18	0.6	4	2.5	425	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
43	4	M	894287	abdominal distension,facial puffiness, pedal edema,oliguria,cold , cough	2 days	SDNS	10,200	12	0.5	3.5	1.5	400	3+	Y	Y	Y	1.NO PUS CELLS ,NO ORGANISM 2.FEW PUS CELLS.MANY GPC IN PAIRS AND SHORT CHAINS SEEN
44	10	F	894583	oliguria,edema	2 days	SDNS	9,100	35	0.8	4	2	360	4+	Y	N	N	NO PUS CELLS ,NO ORGANISM
45	2	F	894968	abdominal distension,facial puffiness, pedal edema,oliguria	1 week	SDNS	18,700	57	0.9	4.8	1.3	656	3+	Y	N	N	MANY PUS CELLS FEW GNB SEEN
46	2	M	894320	abdominal distension,facial puffiness, pedal edema,oliguria	2 days	SDNS	13,400	16	0.8	3.4	1.8	448	4+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
47	2	F	895883	oliguria,pedal edema	2 days	SDNS	11,000	19	0.4	4	2	400	4+	Y	N	N	NO PUS CELLS ,NO ORGANISM
48	4	M	895940	abdominal distension,facial puffiness, pedal edema,oliguria,cold , cough	5 days	SDNS	12,100	16	0.7	3.8	2.5	360	4+	Y	N	Y	1.NO PUS CELLS ,NO ORGANISM 2.FEW PUS CELLS.MANY GPC IN CLUSTERS SEEN
49	12	M	895886	abdominal distension,facial puffiness, pedal edema,oliguria	3 days	SRNS	10,500	18	0.6	4	2	400	4+	Y	N	N	NO PUS CELLS ,NO ORGANISM
50	6	M	895890	abdominal distension,facial puffiness, pedal edema,oliguria,	5 days	SDNS	13,600	30	0.7	4	2	360	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
51	6	M	895922	facial puffiness , oliguria	10 days	First Episode	17,300	35	0.7	4	1.5	420	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
52	5	M	896066	facial puffiness , oliguria	2 days	SRNS	11,000	28	0.8	4	2	360	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
53	6	M	896289	facial puffiness , oliguria	10 days	First Episode	10,700	20	0.5	3	1.8	356	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
54	4	M	896830	facial puffiness , oliguria	2 days	SDNS	10,000	27	0.5	4	2.6	370	3+	Y	N	N	FEW PUS CELLS ,NO ORGANISM
55	12	M	896749	facial puffiness,scrotal edema	4 days	SDNS	17,600	37	1.3	4	1.3	560	1+	Y	N	N	FEW PUS CELLS ,NO ORGANISM
56	6	F	897433	facial puffiness , oliguria	2 days	SRNS	9,800	40	1.2	3.8	1.5	520	4+	Y	N	N	NO PUS CELLS ,NO ORGANISM
57	8	M	898496	oliguria ,abdominal pain	4 days	SDNS	24,900	38	1	3.4	1.3	520	4+	Y	Y	N	1.NO PUS CELLS ,NO ORGANISM
58	2	M	898750	abdominal pain	2 days	SDNS	9,400	30	1.2	4	1.8	460	2+	Y	N	N	NO PUS CELLS ,NO ORGANISM
59	10	F	898296	facial puffiness,abdominal distension	5 months	First Episode	16,200	20	0.7	5.8	3.6	420	3+	Y	N	N	MANY PUS CELLS ,NO ORGANISM

60	8	F	898638	pedal edema	2 days	SRNS	7,400	30	1	3.3	1.3	480	1+	Y	N	N	NO PUS CELLS ,NO ORGANISM
61	6	M	904353	oliguria,pedal edema,facial puffiness	3 days	SRNS	17,900	19	0.6	3.6	2.1	420	3+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
62	8	F	903388	abdominal pain , vomiting	2 days	SRNS	10,600	45	0.7	4.1	1.9	842	1+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
63	3	F	904268	facial puffiness	2 days	SDNS	10,600	16	0.5	4.7	3.1	480	1+	Y	N	N	NO PUS CELLS ,NO ORGANISM
64	11	M	904014	abdominal pain,facial puffiness	1 month	First Episode	5,000	30	0.6	3.6	1.7	376	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
65	5	M	904367	facial puffiness,abdominal distension,oliguria	2 days	SDNS	17,900	29	0.4	4.4	1.4	602	3+	Y	Y	N	MANY PUS CELLS ,NO ORGANISM
66	3	M	904372	oliguria	3 days	SDNS	11,000	38	0.8	4	1.8	600	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
67	4	M	903656	facial puffiness , oliguria	1 week	First Episode	12,000	28	0.4	4.2	2	480	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
68	8	F	905361	facial puffiness,abdominal distension,oliguria	1 week	SDNS	15,400	77	0.5	4	1.8	520	3+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
69	3	F	905449	facial puffiness,abdominal distension	3 days	SDNS	8,600	38	0.7	4.1	2.5	450	3+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
70	8	M	905342	facial puffiness,pedel edema	3 days	SDNS	13,530	52	1	3	1.6	687	2+	Y	N	N	NO PUS CELLS ,NO ORGANISM
71	4	M	906058	facial puffiness,abdominal distension	6 days	SDNS	13,800	15	0.8	4.1	2.6	340	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
72	12	F	906225	fever,facial puffines	2 days	SDNS	8,700	38	0.7	4	2.1	420	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
73	5	M	906198	facial puffiness,abdominal distension,pedal edema	20 days	SDNS	20,000	24	0.9	3.8	1.6	520	4+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
74	4	F	906287	,facial puffines	2 days	SDNS	11,000	30	0.9	4.1	2.2	520	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
75	5	M	906351	,facial puffines,oliguria	1 week	SRNS	12,500	30	0.9	4.5	2	420	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
76	3	M	906431	facial puffiness,abdominal distension,pedal edema	2 days	SDNS	7,700	16	0.6	4.7	2.6	227	3+	Y	N	N	FEW PUS CELLS ,NO ORGANISM
77	4	F	905579	facial puffiness,abdominal distension,pedal edema	15 days	SRNS	14,600	28	0.6	4	1.7	522	3+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
78	5	F	906889	,facial puffines	2 days	SDNS	12,600	30	0.8	3.8	2	616	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
79	6	F	906673	,facial puffines	2 days	SRNS	14,300	30	0.7	4	2.5	560	nil	Y	N	N	NO PUS CELLS ,NO ORGANISM
80	6	M	907117	,facial puffines	2 days	SRNS	10,000	30	0.9	4.2	3.1	460	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
81	12	F	907114	,facial puffines	2 days	SDNS	10,600	40	1	5	3.1	462	3+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
82	6	M	907113	,facial puffines	2 days	SDNS	13,000	25	1	4.2	3.1	600	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM

83	4	F	908211	,abdominal distension,oliguria	2 days	SRNS	8,800	30	0.8	4	2.5	480	4+	Y	N	N	NO PUS CELLS ,NO ORGANISM
84	10	M	908728	,facial puffines,pedal edema	1 week	SDNS	9,400	77	0.8	4.2	3	520	4+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
85	11	F	908902	,facial puffines	3 days	First Episode	9,900	39	1.2	3.8	1.6	600	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
86	8	M	908641	facial puffiness,abdominal distension pedal edema,oliguria,fever,vomiting	3 days	SDNS	8,160	27	0.7	4.1	2.6	450	1+	Y	N	Y	1.MANY PUS CELLS ,NO ORGANISM2.No pus cells, No organisms
87	6	F	909013	,facial puffines,pedal edema	1 week	SDNS	10,200	20	0.6	4	1.5	560	2+	Y	N	N	NO PUS CELLS ,NO ORGANISM
88	6	M	909018	,facial puffines,pedal edema	4 days	SDNS	7,970	20	0.8	3.6	1.8	520	4+	Y	N	N	NO PUS CELLS ,NO ORGANISM
89	6	F	910671	facial puffiness,abdominal distension,pedal edema	2 days	SRNS	18,000	21	0.5	4	1.7	623	3+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
90	9	F	910731	,facial puffines,pedal edema	1 week	SDNS	9,100	19	0.7	4.2	1.9	582	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
91	4	F	910855	,facial puffines,pedal edema	2 days	SDNS	17,500	31	0.5	4.3	1.8	460	4+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
92	11	F	910905	,facial puffines,pedal edema	1 week	SRNS	16,700	29	0.6	3.8	1.5	560	2+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
93	3	F	911307	,facial puffines,oliguria	4 days	First Episode	18,000	24	0.4	3.5	1.8	460	3+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
94	2	F	911705	,facial puffines,pedal edema	1 week	SRNS	13,800	48	1.8	4	1.8	560	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
95	11	F	910765	abdominal distension,facial puffiness, pedal edema,oliguria	10 days	First Episode	9,800	18	0.5	3.9	1.7	320	4+	Y	N	N	NO PUS CELLS ,NO ORGANISM
96	2	M	911738	facial puffiness,abdominal distension,pedal edema	2 days	SRNS	10,800	24	0.8	3.5	1.5	450	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM
97	3	M	912615	,facial puffines,oliguria	4 days	SRNS	12,800	40	0.9	4	2	520	4+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
98	3	M	912516	,facial puffines,pedal edema	1 week	SDNS	10,600	30	0.8	4	1.5	480	4+	Y	N	N	NO PUS CELLS ,NO ORGANISM
99	7	M	912884	,facial puffines,pedal edema	1 week	SRNS	8,500	40	0.7	3.5	1.5	520	3+	Y	N	N	MANY PUS CELLS ,NO ORGANISM
100	4	M	912963	,facial puffines,oliguria	4 days	SDNS	7,400	20	0.8	3.8	1.2	620	3+	Y	N	N	NO PUS CELLS ,NO ORGANISM

S. No	ISOLATE IDENTIFIED IN URINE	ISOLATE IDENTIFIED IN BLOOD	ISOLATE IDENTIFIED IN THROAT SWAB	colony count in Urine C/S	Infection present in	sensitivity PATTERN	PEN	ERY	AK	CIP	COTRI	PT	GM	CAZ	CTX	TETRA	IMP	VANCO	NORFLOX	NITRO	CHLORAMPHENICOL	OFLOX
1	No growth	No growth	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
2	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
3	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
4	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
5	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
6	Staphylococcus aureus (MSSA)	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	S	S	NT	NT	S	NT	S	NT	NT	S	NT	S	S	S	S	NT
7	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
8	E.coli(ESBL)	NA	Staphylococcus aureus (MSSA)	**> 10^5 CFU/ml**	**URINE AND THROAT SWAB**	PRESENT	2.S	2.S	1.R	2.S	1.R 2.S	1.S	2.S	NT	1.R	1.S 2.S	1.S	2.S	1.R	1.S 2.S	NT	NT
9	No growth	NA	Staphylococcus aureus (MSSA)	0	**THROAT SWAB**	PRESENT	S	S	NT	S	S	NT	S	NT	NT	S	NT	S	NT	NT	S	NT
10	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
11	E.coli	No growth	Normal throat commensals grown in culture	**1. > 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	S	S	S	NT	S	S	NT	NT
12	.E.coli	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	S	S	S	NT	S	S	NT	NT
13	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

14	No growth	No Growth	Staphylococcus aureus (MSSA)	0	**THROAT SWAB**	PRESENT	NT	NT	S	S	NT	S	NT	NT	S	NT	NT	S	NT	S	S	S
15	No growth	NA	Normal throat commensals grown in culture	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
16	E.coli	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	S	S	S	NT	R	R	NT	NT
17	No growth	NA	Normal throat commensals grown in culture	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
18	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
19	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
20	E.coli	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	S	S	S	NT	S	S	NT	NT
21	No growth	NA	Staphylococcus aureus (MSSA)	0	**THROAT SWAB**	PRESENT	S	S	NT	S	S	NT	S	NT	NT	S	NT	S	NT	NT	S	NT
22	E.coli	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	S	S	S	NT	S	S	NT	NT
23	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
24	E.coli	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	S	S	S	NT	S	S	NT	NT
25	Klebsiella pneumoniae (ESBL)	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	R	R	S	NT	S	S	NT	NT
26	Staphylococcus aureus (MSSA)	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	S	S	NT	NT	S	NT	S	NT	NT	S	NT	S	S	S	S	NT
27	Staphylococcus aureus (MSSA)	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	S	S	NT	NT	S	NT	S	NT	NT	S	NT	S	S	S	S	NT

28	No growth	No growth	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
29	No growth	NA	Staphylococcus aureus (MSSA)	0	**THROAT SWAB**	PRESENT	S	S	NT	S	S	NT	S	NT	NT	S	NT	S	NT	NT	S	NT
30	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
31	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
32	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
33	No growth	NA	Normal throat commensals grown in culture	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
34	E.coli	NA	Normal throat commensals grown in culture	**1. > 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	R	NT	S	S	NT	NT	S	S	S	NT	S	R	NT	NT
35	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
36	No growth	NA	Staphylococcus aureus (MSSA)	0	**THROAT SWAB**	PRESENT	S	S	NT	S	S	NT	S	NT	NT	S	NT	S	NT	NT	S	NT
37	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
38	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
39	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
40	E.coli (ESBL)	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	R	S	S	NT	R	S	NT	NT
41	No growth	NA	streptococcus pyogenes	0	**THROAT SWAB**	PRESENT	S	S	NT	NT	NT	NT	NT	NT	S	S	NT	S	NT	NT	S	S

42	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
43	No growth	No Growth	streptococcus pyogenes	0	**THROAT SWAB**	PRESENT	S	R	NT	NT	NT	NT	NT	NT	S	S	NT	S	NT	NT	S	R
44	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
45	Klebsiella. Pneumoniae	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	R	NT	R	S	NT	NT	R	S	S	NT	S	S	NT	NT
46	E.coli	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	S	S	S	NT	S	S	NT	NT
47	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
48	No growth	NA	Staphylococcus aureus (MSSA)	0	**THROAT SWAB**	PRESENT	S	S	NT	S	S	NT	S	NT	NT	S	NT	S	NT	NT	S	NT
49	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
50	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
51	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
52	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
53	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
54	Pseudomonas species	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	NT	S	NT	S	NT	NT	S	NT	R	NT	NT	NT
55	Pseudomonas species	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	NT	S	NT	S	NT	NT	S	NT	S	NT	NT	NT
56	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
57	No growth	No growth	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
58	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
59	E.coli	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	R	S	S	NT	R	S	NT	NT

60	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
61	Klebsiellapneumoniae	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	S	S	S	NT	S	S	NT	NT
62	Klebsiellapneumoniae	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	S	S	S	NT	S	S	NT	NT
63	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
64	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
65	Klebsiella oxytoca (ESBL)	No Growth	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	R	S	S	NT	R	S	NT	NT
66	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
67	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
68	Acinetobacter baumanii	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	S	S	S	NT	R	NT	S	S	NT	NT	NT	NT	NT
69	Klebsiella pneumoniae (ESBL)	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	R	S	S	NT	S	R	NT	NT
70	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
71	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
72	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
73	Klebsiella oxtoca (ESBL)	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	R	S	S	NT	R	S	NT	NT
74	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
75	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
76	Staphylococcus aureus (MSSA)	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	S	S	NT	NT	S	NT	S	NT	NT	S	NT	S	S	S	S	NT
77	E.coli	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	S	S	S	NT	S	S	NT	NT
78	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
79	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
80	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
81	E.coli	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	R	S	S	NT	R	S	NT	NT
82	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

83	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
84	E.coli	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	S	S	S	NT	S	S	NT	NT
85	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
86	E.coli	NA	Normal throat commensals grown in culture	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	R	S	S	NT	S	S	NT	NT
87	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
88	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
89	E.coli	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	S	S	S	NT	S	S	NT	NT
90	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
91	E.coli	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	S	S	S	NT	S	S	NT	NT
92	E.coli	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	S	S	S	NT	R	S	NT	NT
93	E.coli	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	S	S	S	NT	S	S	NT	NT
94	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
95	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
96	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
97	E.coli	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	S	S	S	NT	S	S	NT	NT
98	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
99	E.coli	NA	NA	**> 10^5 CFU/ml**	**URINE**	PRESENT	NT	NT	S	NT	S	S	NT	NT	S	S	S	NT	S	S	NT	NT
100	No growth	NA	NA	0	NIL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

KEY TO MASTER CHART

S.NO	SERIAL NUMBER
M	MALE
F	FEMALE
IP NO	INPATIENT NUMBER
SRNS	STEROID RESISTANT NEPHROTIC SYNDROME
SDNS	STEROID DEPENDENT NEPHROTIC SYNDROME
DGS	DIRECT GRAM STAINING
MSSA	METHICILLIN SENSITIVE STAPHYLOCOCCUS AUREUS
E.COLI	ESCHERICHIA COLI
Y	YES
N	NO
NA	NOT APPLICABLE
S	SENSITIVE
R	RESISTANT
NT	NOT TESTED
CFU	COLONY FORMING UNIT
PEN	PENICILLIN
ERY	ERYTHROMYCIN
AK	AMIKACIN
CIP	CIPROFLOXACIN
COTRI	CO-TRIMOXAZOLE
PT	PIPERACILLIN-TAZOBACTAM
GM	GENTAMYCIN
CAZ	CEFTAZIDIME

CTX	CEFOTAXIME
TETRA	TETRACYCLINE
IMI	IMIPENEM
VAN	VANCOMYCIN
NOR	NORFLOXACIN
NITRO	NITROFURANTOIN
OFLOX	OFLOXACIN

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