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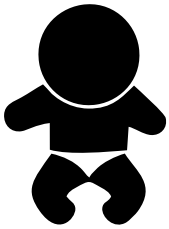

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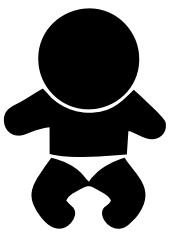

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CONTENTS

LIST OF ABBREVATIONS

1. AGN - Acute glomerulo nephritis
2. APSGN - Acute Post streptococcal glomerulonephritis
3. AKI - Acute kidney injury
4. ASLO - Anti streptolysin O titre
5. ARF - Acute renal Failure.
6. BUN- Blood Urea Nitrogen
7. BP - Blood Pressure
8. CKD - Chronic Kidney Disease
9. C3 - Complement C3
10. CCF - Congestive cardiac failure
11. HSP - Henoch Schonlein Purpura
12. HTN- Hypertension
13. PIGN - Post infectious glomerulonephritis
14. PSGN - Post streptococcal glomerulonephritis
15. UTI - Urinary tract infection

1

INTRODUCTION

Acute glomerulonephritis is one of the most common kidney disorder in children accounting for 70% of all nephrology cases. Commonest cause for AGN in children is post infectious etiology. The prototype of PIGN is PSGN.[1] PSGN results from antecedent infection of skin or throat caused by nephritogenic strains of group A beta hemolytic streptococci.[2,3,4] The world wide burden of PSGN is 4,72,000 per year with approximately 4,04,000 cases in children and 4,56,000 occurring in underdeveloped countries.[5] AGN has wide range of presentation and natural history has been changing now-a-days. Although mortality is less, many morbidities are associated with AGN. Only few studies on clinic-etiological profile, prognosis and outcome of PIGN have been reported in India in recent years.

HISTORY

In 1812, Scientist Well's described clinical features of acute nephritis which developed after scarlet fever i.e. development of edema and urine that contains both RBC's (red substance) and protein (coagulable substance).[6] After a decade later Richard bright combined the clinical features like urine abnormalities and dropsy and autopsy evidence of kidneys derangement.[7]

The form of kidney disorders developing after scarlet fever came to be known as acute hemorrhagic Bright's disease.[8] During last decade of 19-20th century several descriptions of post scarlitinal glomerulonephritis appeared and were termed acute glomerulonephritis.[9,10,11]

Dick and Dick [12] and Dochez and Sharman [13] demonstrated that beta hemolytic streptococcus was etiologic agent of scarlet fever led to use the term post streptococcal glomerulonephritis. By 1940, anti-streptococcal antibodies[14] and hypocomplementemia were noted in post streptococcal glomerulonephritis [15] and itbecame clear that glomerulonephritis followed both upper respiratory and cutaneous infection with beta hemolytic streptococci.[16, 17]

In 1941, Seegal and Earle described the concept of nephritogenic strains of streptococci that were different from those that caused rheumatic fever.[18] Ellis, the famous nephrologist defined two types of nephritis:-

- Type 1 nephritis: - It was associated with hematuria, uremia and fluid retention and it occurred following infections in young children who usually recovered. He also recognized a subset of patients who did not completely recover and who had persistent hematuria and proteinuria. Probably many of them had IgA nephropathy or other types of glomerulonephritis. Arthur Ellis's article on Bright's disease published in 1942 summarized an impressive 20 year systemic recording of 600 patients with kidney disease at London hospital.

- Type 2 nephritis: - It was a condition with proteinuria but not hematuria and edema and had a more insidious onset. Complications like uremia was common. Microscopic examination revealed no inflammation and glomerular signs were often subtle.

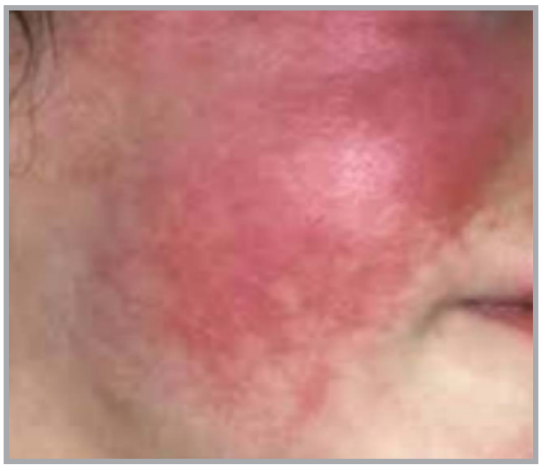

Fig 1: Image of a scarlet fever

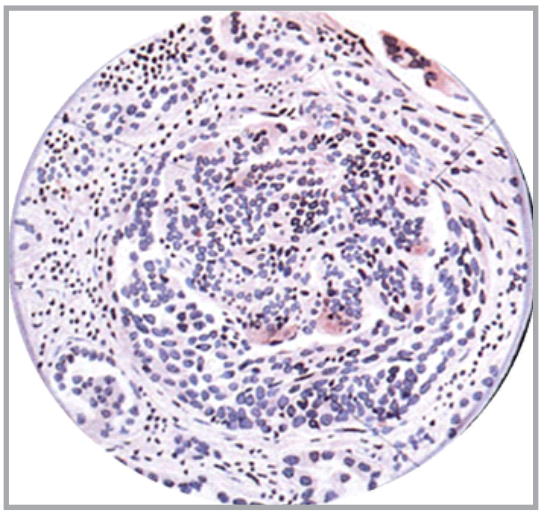

Fig 2: Acute glomerulonephritis with crescent formation

DEFINITIONS

- Hematuria is defined as presence of red blood cells in the urine.

- *Gross hematuria* is visible discoloration of the urine due to the presence of red blood.

- *Microscopic hematuria* is defined as the presence of more than 5 RBCs/high power field on a centrifuged urine specimen (or >5 RBCs/mm3 on an uncentrifuged specimen).

Common causes of hematuria :

- Glomerulonephritis.
- Idiopathic hypercalciuria.
- Stones.
- Hemorrhagic cystitis.
- Pyelonephritis.
- Urethritis.

Presence of abnormal type and amount of protein in urine. Physiological proteinuria occurs commonly and is the result of

- Filtration of small amounts of albumin through the glomerular basement membrane.

- Non reabsorption of low molecular weight proteins from tubular epithelial cells.

- The secretion of endogenous renal proteins synthesized by the tubules (Tamm Horsfall Protein or THP) into the urine.

Causes of Proteinuria :

- Glomerular proteinuria.

- Tubular proteinuria.

ACUTE GLOMERULAR NEPHRITIS

Acute glomerulonephritis is characterized by a relatively abrupt onset of variable degrees of hematuria, edema, hypertension, oliguria along with diminished glomerular filtration rate , salt and fluid retention and resulting congestion . AGN may follow infection with a variety of microorganisms, when it is called as *post infectious.*

Table 1: Etiology of acute glomerulonephritis

Postinfectious
- Strept04:(ieci, staphylococci, pneumococci, meningococci, Treponema pallidum. Salmonella typhi. loptospira
- Plasmodium malanae, P. falciparum, toxoplasma, filaria
- Hepatitis B and C. cytomogalovin is, parvovirus, Ebstein-Barr virus. coxsackievirus. echovir us. vaticella
- Associated with severe infections; infection of shunts, prostheses, bacterial endocarditis

Systemic vasculitis
- Henoch-SchOnlein purpura
- Microscopic polyaderitis. Wegener's granulomatosis

Others
- Membranoproliferative glomerulonephritis
- IgA nephropathy
- Hereditary nephropathy
- Systemic lupus etythernalosus
- Acute interstitial nephritis

POST-STREPTOCOCCAL GLOMERULONEPHRITIS
is defined clinically in the presence of:

a) Hematuria and proteinuria

b) Clinico-serological evidence of recent streptococcal infection(pyoderma or pharyngitis; positive antistreptolysin-O and

c) Low serum C3 levels, with normalization on 8 wk follow up

NEPHROTIC SYNDROME
Clinical manifestation of glomerular disease associated with nephrotic range proteinuria,hypoalbuminemia(<2.5g/dl),edema and hyperlipidemia (cholesterol>200mg/dl).

2

REVIEW OF LITERATURE

Acute glomerulonephritis is one of the most common kidney disorder in children accounting for 70% of all nephrology cases. Commonest cause for AGN in children is post infectious etiology. The prototype of PIGN is PSGN.[1] PSGN results from antecedent infection of skin or throat caused by nephritogenic strains of Group A beta hemolytic streptococci.[2, 3, 4] The world wide burden of PSGN is 4,72,000 per year with approximately 404000 cases in children and 4,56,000 occurring in underdeveloped countries.[5] AGN has wide range of presentation and natural history has been changing now-a-days. Although mortality is less, many morbidities are associated with AGN.

HISTORICAL BACKGROUND

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In 1941, Seegal and Earle described the concept of nephritogenic strains of streptococci that were different from those that caused rheumatic fever.[18]

EPIDEMIOLOGY:

PSGN is one of the important non suppurative complication of streptococcal infection worldwide. Nearly 492000 cases occur worldwide, among them 404000 cases occur in children.[19] In tropical countries pyoderma associated PSGN is common[20] whereas in temperate countries pharyngitis associated PSGN predominates.[21, 22]

The serotypes most frequently associated with pyoderma associated glomerulonephritis is M49, the red-lake strain,[23,24] other associated strains are M2,M42,M56,M57 and M60.[25] Pharyngitis associated glomerulonephritis strains are M1,M4,M2 and some of M12 strains.[26,27]

In geographic areas having distinct seasons pyoderma associated PSGN tend to occur in late summer or early rainy season.[28.29.30.31] whereas in regions with constant tropical climate cases occur in all season.[32]

The overall decline in the incidence of both developed and underdeveloped countries in the world is likely due to the early use of antibiotics for skin infections leading to decreased transmission of virulent strains. Also earlier access to health care and fluoridation of water might play a role.

ETIOLOGY OF PSGN

Table 2: Etiological agents of PIGN

BACTERIA	VIRUSES	FUNGI	PARASITES
Pharyngitis or skin infections	Hepatitis B[a]	*Coccidioides immitis*	Malaria
Group A *Streptococcus (pyogenes)*	Hepatitis C[a]	Histoplasmosis	*Plasmodium malariae*[a]
Group C and G *Streptococcus*	Human immunodeficiency virus[a]		*Plasmodium falciparum*
Endocarditis	Cytomegalovirus[b]		*Plasmodium vivax*
Streptococcus viridans	Varicella		Leishmaniasis (*Leishmania donovani*)
Staphylococcus aureus	Epstein-Barr[b]		Toxoplasmosis[a]
Staphylococcus epidermidis	Parvovirus B19		Schistosomiasis (*Schistosoma mansoni*)
Abscess	Enteroviruses		Filariasis (*Wuchereria bancrofti*)
Streptococcus viridans	Echovirus		
Staphylococcus aureus	Coxsackievirus		
Gram-negative bacilli	Paramyxoviruses		
Intraventricular shunt infections	Measles		
Staph epidermidis	Mumps		
Staph aureus			
Diphtheroids			
Pneumonia			
Streptococcus pneumoniae			
Mycoplasma			
Legionella			
Enterocolitis			
Yersinia enterocolitica			
Salmonella Typhi			
Campylobacter jejuni			
Rickettsial disease			
Rocky Mountain spotted fever			
Q fever (*Coxiella*)			
Ehrlichiosis			
Others			
Neisseria meningitides			
Syphilis (*Treponema pallidum*)[a]			

AGE AND SEX PREDILECTION

A nationwide study in New Zealand was conducted to define epidemiology and clinical features of PSGN in children hospitalized with the illness. The study found higher incidence of PSGN in socioeconomically deprived children as well as with Pacific and Maori children of New Zealand.[33]The disease is more frequent in children aged 2-12yrs with high prevalence in 5-6 years[34].

No racial predilection is seen in PSGN. The condition is reported in all ethnic and cultural groups in urban populations. A predilection toward minority populations is observed; however this may be related more to the socioeconomic factors of overcrowding than to any racial predilection.

In developing countries PSGN occurs predominantly in males and often as epidemics. PSGN usually seen as sporadic cases but epidemics are seen in densely populated communities with poor hygienic conditions with high incidence of malnutrition, anemia and intestinal parasites.

Although a male predominance is noted in symptomatic cases for unknown reasons when subclinical and clinical disease is taken into account, the rates are the same in both the sexes.

The disease was previously known as Bright's disease after Richard Bright. Historically disease gains the name after Richard Bright who was the founder of nephrology and later rewarded as father of nephrology and hence the disease is popularly called as Bright's disease. The word Bright's disease was used for more than100 years for any type of kidney diseases and later particularly for glomerular diseases.

PATHOLOGY

Glomerular changes in AGN include proliferation of mesangial and epithelial cells and endothelial cells and C3 deposition with inflammatory exudate followed by IgG deposition. This immune deposition has been classified into 3 types[35] and those are as follows:-

a) Starry sky pattern: -In this type irregular and finely granular deposits of IgG and C3 along glomerular capillary walls and in mesangium.

b) Mesangial pattern: - In this type mainly C3 and some amount of IgG is deposited in the mesangium.

c) Garland pattern: - Here dense deposits are seen along the capillary walls of glomerulus and it is associated with severe proteinuria and poor prognosis.

PATHOGENESIS

The pathognomonic feature of PSGN is immune complex deposition in glomerular basement membrane. Streptococcal antigen lead to the activation of

complement pathway and/or activation of plasmin or production of circulating immune complexes. Permeability of glomerular basement membrane increases leading to the immune complex deposition and protein and RBC leakage. The streptococcal nephritogenic antigen is responsible for recruitment of immune cells, tissue destruction, IgG deposition and complement (C3) deposition and complement activation which leads to release of cytokines that in turn attract phagocytes. Proliferation of intrinsic cells and formation of membrane attack complex also aggravates the process.

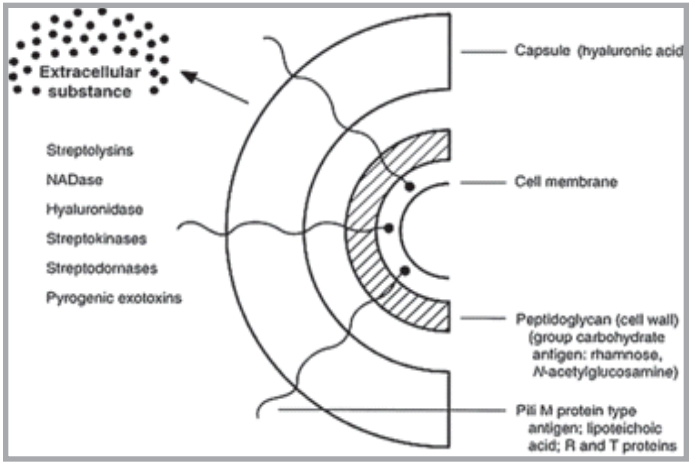


Figure 3: Structure of cell wall of streptococcus

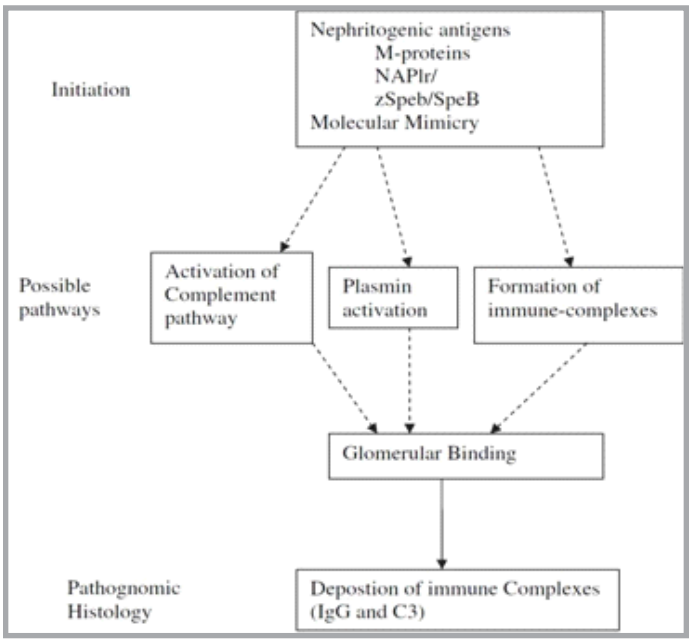


Figure 4: Pathogenesis (mechanism) of PSGN

Numerous streptococcal antigenic factors have been proposed as triggers of glomerulonephritis. These are M proteins, M like proteins and later GADPH (Glyceraldehyde phosphate dehydrogenase) or endostreptosin {nephritis associated plasmin receptor (NAPlr/Plr)} and streptococcal pyrogenic exotoxin B (zymogen, SPEB), streptokinase.[36, 37, 38]

Nephritogenic M proteins are type [1,2,3,4,25,49,12] following skin infections and type [47,49,55,2,60,57] following throat infections. M proteins antigenically cross react with glomerular basement membrane.[36.39] The kidney has a limited number of ways of responding to any type of injury. Similar pathological signs may be the product of many different processes, produced by different initiating factors and different cytokines.

Association with HLA antigens, HLA DRW4[40] and HLADRB1*03011 [41] and with polymorphisms of endothelial nitric oxide synthase gene intron 4 a/b has been described [42] but frequency of alleles does not correlate with the severity of disease.

Pathognomonic features of AGN are hematuria, proteinuria and glomerular hypercellularity with C3 and IgG deposition. Variations may occur with all these findings not being present at the same time. Severe diffuse glomerulonephritis can occur without C3 or IgG depositon or without severe hematuria or proteinuria. On the other hand, macroscopic hematuria can occur with minimal histological findings. Same type of histological findings can be found with other infectious diseases[39.43] and conditions like alcoholism, IgA nephropathy and diabetic nephropathy.[44]

CLINICAL FEATURES

AGN is more frequent in the age group of 2-12 years with high prevalence in 5-6years.[34] AGN rarely occurs before 2 years.[45,46,47] This may be because of low rate of streptococcal pharyngitis in this age group and immature immune(antibody) response.

The classical diagnostic triad of AGN is edema, hematuria and hypertension. Children with AGN often seek medical attention for edema or gross hematuria. Rarely signs and symptoms of essential hypertension will be the presenting feature leading to diagnosis.There are 3 phases of the disease: latent phase, acute and recovery phase. History of preceding throat pain and pyoderma is identified in majority of the cases. The latent period between the streptococcal infection and the onset of signs and symptoms of AGN is 3-33days[48] but on an average it is 7-14 days.

Hypertension occurs in 80-90% of cases. [49, 50] Complications of hypertension like seizures, mental status changes, headache and visual abnormalities occurs in 30- 35% of patients according to one case series. Hypertension may be because of fluid and sodium retention with expansion of extracellular space.[51]

FeNa (fractional excretion of sodium) is less than 1% similar to prerenal failure[52,53] and renin levels are low at presentation.[54,55] Hematuria is present in almost all patients. Tea or cola colored urine is seen in 25-60% of cases. Proteinuria is also common but nephrotic range proteinuria is rare. Patients may present with clinical and radiological signs of pulmonary edema.

ATYPICAL PRESENTATIONS OF AGN

1. Acute pulmonary edema: - child presents with dyspnea and restlessness.

Pathophysiology:-In AGN, expansion of extracellular volume occurs which further leads to congestive heart failure and pulmonary edema. Clinical features being sudden onset breathlessness, respiratory distress, cough and crepitation. Blood pressure is high in the initial phases but later stages when heart failure and shock supervenes there may be hypotension. Chest x ray reveals mild cardiomegaly and features of pulmonary edema.

Treatment is monitoring of vital signs, Oxygen therapy, diuretics (loop diuretics) play a vital role.

2. Hypertensive encephalopathy:-child presents with nausea, vomiting, headache, convulsions and altered sensorium. Children develop encephalopathy at comparatively lower levels of blood pressure. MRI brain shows signs of posterior reversible leukoencephalopathy(PRESS) syndrome.

3. Acute renal failure:-In rare cases, oligo-anuria and azotemia are the chief presenting features and gross hematuria and edema is absent. Rapidly progressive disease is seen in minority of cases.

4. Nephrotic syndrome:-Mixed picture of nephritic and nephrotic syndrome may be present in some of the cases. The child presents with gross hematuria, hypertension, generalized edema and heavy proteinuria. Glomerular lesions are invariably severe in these cases and extensive crescent formation may be seen.

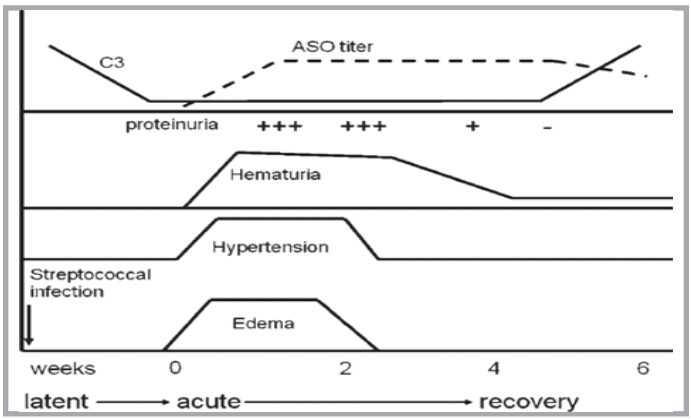


Figure 5: typical clinical course of PSGN

Subclinical infections are also very common. One of the study suggested that PSGN occurs in about 20% of asymptomatic family members of patients with the AGN.[56]

Meherban singh et al conducted study among 100 patients admitted with AGN in institute of child health, Kabul. Their study revealed antecedent history of sore throat 1-3 week before the onset of the disease in 57% of cases. Males were affected twice as frequently as female. Disease was ushered as acute onset of edema (93%), cola colored urine (56%), HTN(40%) and fever(41%). Urine abnormalities were detected in 96% of the patients and azotemia in 85% of cases. The main complications of the disease were CHF 8%, hypertensive encephalopathy 5% and acute renal failure 5%.[57]

Atypical presentations of AGN include those presenting with acute illness, usually related to HTN or edema in the absence of obvious urine abnormalities.[58]Serial examination of the urine may confirm the diagnosis of AGN later. Another atypical feature is presence of typical HSP rash[59, 60, 61]. The diagnosis of PSGN is confirmed by renal biopsy in these cases.

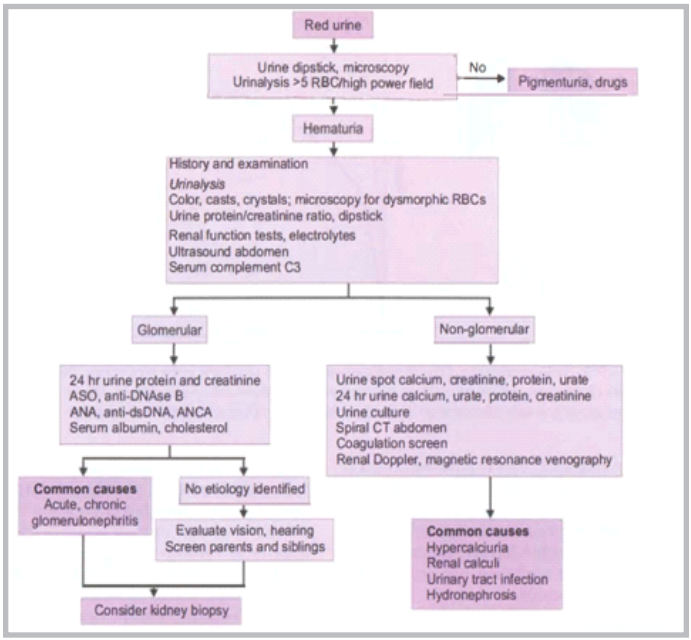
Table 3: Distinguishing clinical features of other glomerulonephritis

TABLE 2. **Distinguishing Clinical Features of Other Glomerulonephritides and Glomerular Diseases**

DIAGNOSIS	HISTORY	SIGNS, SYMPTOMS, AND PHYSICAL EXAMINATION FINDINGS	LABORATORY FINDINGS
ANCA-associated vasculitis (WG, MPA)	Female-male ratio of 4:1 in WG Middle-aged adults Flulike symptoms presenting for 2 months	Fever, weight loss, arthralgia Purpuric rash (MPA) Mononeuritis (MPA) Chronic sinusitis, epistaxis (WG) Dyspnea, cough (WG) Saddle nose deformity (WG)	Pulmonary infiltrates on radiograph Positive ANCA (c-ANCA with WG and p-ANCA with MPA)
Goodpasture disease	Typically young men aged 20–30 years	Hemoptysis Chest pain	Anti–glomerular basement membrane antibodies
Hereditary nephritis (Alport syndrome)	Family history in males of hearing loss and renal dysfunction	High-frequency hearing loss Anterior lenticonus of the eyes	
HSP		Palpable purpura (buttocks, extensor surfaces of legs) Colicky abdominal pain Joint pain or swelling Orchitis in boys	
HUS	Exposure to undercooked meat, animal feces (petting zoo, farm)	Bloody diarrhea Vomiting Pallor	Hemolytic anemia (schistocytes on blood smear, LDH level elevated, low haptoglobin level) Thrombocytopenia Stool culture positive for *Escherichia coli* O157:H7
IgA nephropathy	Typically manifests in preadolescents to young adults (second or third decade of life)	Sympharyngitic simultaneous with URI (or GI) symptoms	
MPGN	Associated with chronic hepatitis B infection	Asymptomatic 50% of cases	Persistent decrease in serum C3
SLE	Female-male ratio of 4:1 in children Family history of lupus or autoimmune disorders Preponderance in African American and Hispanic females	Discoid or malar rash Photosensitivity Arthralgias Oral or nasal ulcers Serositis	Cytopenias (anemia, leukopenia, and/or thrombocytopenia) Antinuclear antibody titer Anti–double-stranded DNA titer

ANCA=antineutrophil cytoplasmic antibody; GI=gastrointestinal; c-ANCA=cytoplasmic antineutrophil cytoplasmic antibody; HUS=hemolytic uremic syndrome; LDH=lactate dehydrogenase; MPA=microscopic polyangiitis; MPGN=membranoproliferative glomerulonephritis; SLE=systemic lupus erythematosus; p-ANCA=perinuclear antineutrophil cytoplasmic antibody; URI=upper respiratory tract infection; WG=Wegener granulomatosis.

APPROACH TO A CHILD WITH HEMATURIA



Approach to any child with complaints of red urine should be systematic as shown in the flow chart.

APPROACH TO PROTEINURIA

PROTEINURIA

Presence of abnormal type and amount of protein in urine. Physiological proteinuria occurs commonly and is the result of

- Filtration of small amounts of albumin through the glomerular basement membrane.
- Non reabsorption of low molecular weight proteins from tubular epithelial cells,
- The secretion of endogenous renal proteins synthesized by the tubules (Tamm Horsfall protein or THP) into the urine.

Causes of proteinuria

- Glomerular proteinuria.
- Tubular proteinuria.

Glomerular proteinuria

Nephrotic syndrome (minimal change disease, focal segmental glomerulosclerosis, congenital nephrotic syndrome)
Membranoproliferative glomerulonephritis, membranous nephropathy
Hepatitis B and C nephropathy, HIV nephropathy
Reflux nephropathy
Amyloidosis
Associated hematuria: Postinfectious glomerulonephritis, IgA nephropathy, Henoch-Schönlein nephritis, lupus nephritis, Alport syndrome

Tubular proteinuria

Drug induced nephropathy (analgesics)
Heavy metal nephropathy (e.g. gold, lead, cadmium)
Renal tubular acidosis
Interstitial nephritis, pyelonephritis
Intermittent or transient proteinuria
Postural (orthostatic)
Fever
Exercise

INVESTIGATIONS

Urine analysis is very important and useful investigation. Presence of dysmorphic RBCs, leukocytes, RBC and WBC casts are identified in urine examination. In the early part of acute phase, leukocytes may predominate over RBCs. Mild anemia may be seen which is due to hemodilution and low grade hemolysis. One large series showed that only 10% of 155 patients had hemoglobin level of >12gm/dl and over 50% were below 10gm/dl.[62] Increased BUN was noted in 60-65% of patients and may be because of decreased glomerular filtration rate.

Serum complement levels C3 is decreased in acute phase and returns to normal levels within 6 weeks of onset. Normalization of C3 levels within 6 weeks is of diagnostic importance when renal biopsy is not performed. Low serum complement level may indicate the following systemic diseases:

- Systemic lupus erythematosus (SLE) (focal, diffuse).
- Subacute bacterial endocarditis.
- Visceral abscess.
- "Shunt" nephritis.
- Cryoglobulinemia.

Low serum complement level may indicate the following renal diseases:

- Acute post infectious glomerulonephritis.
- MPGN: Type I , type 2.

A normal serum complement level may indicate the following systemic diseases:

- Polyarteritis nodosa group.
- Hypersensitivity vasculitis.
- Wegener granulomatosis.
- Henoch-Schönlein purpura.
- Good pasture syndrome.

A normal serum complement level may indicate the following renal diseases:

- Immunoglobulin (Ig) A (or Ig G-IgA) nephropathy.
- Idiopathic rapidly progressive glomerulonephritis (RPGN).
- Anti-glomerular basement membrane (GBM) disease.
- Negative immunofluorescence findings.
- Immune complex disease.

ASLO titers are increased in patients with pharyngitis associated PSGN and anti DNAse B, antihyaluronidase are elevated in pyoderma cases. In a study, the sensitivity of elevated ASLO titers was very high (97%) but the specificity was 80%,may be because of 20% of unaffected normal controls show evidence of streptococcal exposure with raised ASLO titre.[63] In Roy's series of biopsy proven cases, 60% had elevated ASLO titre >333 Todd units.[64]

Study conducted by Longcope and Dodge et al showed ASLO titre continued to increase over 4 weeks after presentation and mean titre peaked at 3 weeks. Chest x-ray is indicated in patients with signs of pulmonary edema and respiratory distress. Renal biopsy in considered in following conditions

- Fever, rash, heart disease and joint pain.
- Absence of serological evidence of streptococcal infection.

- Normal C3 levels.
- Combined features of AGN and nephritic syndrome.
- RPGN.
- Nephritic range proteinuria beyond 2 weeks.
- Low C3 levels beyond 12 weeks.
- Persistent proteinuria beyond 6 months.
- Gross hematuria beyond 3-4 weeks.
- Persistent microscopic hematuria beyond 12-18 months.
- Oliguria, hypertension, azotemia beyond 4-10 days.

Treatment is symptomatic.

- Edema and circulatory overload are treated with diuretics and restriction of salt and water.
- Daily monitoring of weight, urine output and blood levels of urea and creatinine.
- Hypertension is treated with calcium channel blockers like nifedipine
- In hypertensive emergencies IV labetalol, sodium nitroprruside and nicardipine are used.
- Systemic antibiotic therapy with penicillin for 10 days is recommended to limit the spread of nephritogenic organisms. However antibiotic therapy does not alter the natural history of the disease.
- Patients with acute renal shut down require correction of electrolyte abnormalities and sometimes renal replacement therapy.

Acute complications in AGN are because of hypertension and acute renal dysfunction. The complications being acute pulmonary edema, CCF, hypertensive encephalopathy, nephrotic on nephritic syndrome, ARF, prolonged HTN leading to IC bleed, electrolytes imbalances like hyperkalemia, hypophosphatemia, hypocalcemia, metabolic acidosis and uremia.

Kuralavanan Gunasekaran, et al conducted a prospective study on PIGN in Pondicherry observed that PIGN was significant contributor to morbidity in

children with AGN. PSGN was the most common etiology among PIGN cases and complications were AKI in 20.8% of cases, hypertensive emergency in 89.4% of cases, CCF in 11.1% cases, encephalopathy in 4.2% and retinopathy in 1.4% cases.

One more prospective study conducted by Sarkissian A et al found that PSGN was more common after streptococcal pharyngitis (30%) than streptococcal pyoderma (13%) and hypo complementemia was noted in 95% of cases, hypertension in 72%, encephalopathy in 3% and CCF in 10% of patients.

The prognosis of PSGN is favourable in children as compared to adults. Epidemic PSGN carry a better prognosis than sporadic cases.[50] It may be because of high index of suspicion in epidemics leading to identification of higher number of cases.

Complete recovery occurs in more than 95% of cases of PSGN. Loss of edema and decrease in blood pressure occurs in first week. Gross hematuria resolves early whereas microscopic hematuria may persist for 6-12 months. Serum levels of C3 normalises within 8 weeks. Proteinuria resolves early but orthostatic or intermittent proteinuria may persist longer. Recurrences are very rare. Prognosis is usually very good unless associated with severe renal failure and crescentic glomerulonephritis.

Prospective study conducted by M. Vijay Kumar et al on AGN revealed that PSGN was most common cause of AGN in children and it was one of the commonest cause of renal edema in children. Early recognition, prompt treatment and aggressive therapy and adequate follow up was made mandatory. Prognosis was usually good unless associated with renal failure and crescentic glomerulonephritis.

3

CONCLUSION

- AGN is one of the most common kidney disorder in childhood.
- AGN is most common in the age group of 6-8 years and it is uncommon below 3years. This may be because of low rate of streptococcal pharyngitis in this age group and immature immune response (antibody response).
- The prototype of PIGN is PSGN, which continues to be a non suppurative complication of Group A streptococcal infection.
- In tropical countries pyoderma associated PSGN is common whereas in temperate countries pharyngitis associated PSGN predominates.
- The classical diagnostic triad of AGN is edema, hematuria and hypertension.
- Latent period between streptococcal infection and AGN is 7-14 days.
- Hypertension occurs in 80-90% of cases and it is one of the common cause of hypertensive emergency in paediatric age group.
- Hematuria is present in almost all patients and microscopic hematuria may persist 6- 12 months or longer. Proteinuria is common but nephrotic range proteinuria is rare. Anuria is rare and if it is persistent suggests rapidly progressive glomerulonephritis.
- Mild anemia may be seen due to hemodilution and low grade hemolysis. Serum complement levels C3 levels are decreased in acute phase and returned to normal levels within 6-8 weeks. Normalization of C3 levels within 6-8 weeks is of diagnostic importance when renal biopsy is not performed. ASLO titres are increased in patients with pharyngitis associated PSGN and antiDNAse B ,anti hyaluronidase are elevated in pyoderma associated PSGN.
- Acute complications in AGN are because of hypertension and acute renal dysfunction. Acute complications being acute pulmonary edema, CCF, hyper

tensiveencephalopathy, ARF, electrolyte imbalance, metabolic acidosis, seizures, prolonged hypertension leading to intracranial bleed.

- The prognosis of PSGN is favourable in children as compared to adults. Complete recovery occurs in more than 95% cases of PSGN.

- Prevention of PSGN in developing countries is based on public health measures such as improved hygiene and better housing condition with better sanitary practices, therefore decreasing the streptococcal infections.

- Availability of vaccine for Group A streptococci is highly desirable and anticipated, both in terms of preventing invasive disease and non suppurative complications.

- A vaccine that eradicates all Group A streptococci would eliminate PSGN in near future.

4

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