Acute Poststreptococcal Glomerulonephritis:

Group A β-hemolytic streptococcal infections are common in children and can lead to the post infectious complication of acute glomerulonephritis (GN). Acute poststreptococcal glomerulonephritis (APSGN) is a classic example of the acute nephritic syndrome characterized by the sudden onset of:

- Gross hematuria
- Edema
- Hypertension
- Renal insufficiency.

ETIOLOGY AND EPIDEMIOLOGY

APSGN follows infection of the throat or skin by certain "nephritogenic" strains of group A β-hemolytic streptococci.

Following streptococcal pharyngitis	Following skin infections
Cold-weather months	Warm-weather months
Early school age	Preschool age
Serotype M1, M4, M25, and M12	Serotype M49
Boys: girls 2:1	Girls=boys

PATHOLOGY

Glomeruli appear enlarged and relatively bloodless and show diffuse mesangial cell proliferation, with an increase in mesangial matrix. Polymorphonuclear leukocyte infiltration is common in glomeruli during the early stage of the disease.

CLINICAL MANIFESTATIONS

- ✓ PSGN is most common in children ages 5-12 yr. and uncommon before the age of 3 yr.
- ✓ The typical patient develops an acute nephritic syndrome 1-2 wk. after an antecedent streptococcal pharyngitis or 3-6 wk. after a streptococcal pyoderma.
- ✓ The onset is usually abrupt. The earliest symptoms are dark color urine, mild periorbital edema, and decreased urine output, nonspecific symptoms such as malaise, lethargy, abdominal pain, or flank pain.
- ✓ The severity of kidney involvement varies from asymptomatic microscopic hematuria with normal renal function to gross hematuria with acute renal failure.
- ✓ Patients are at risk for developing encephalopathy and/or heart failure secondary to hypertension or hypervolemia.
- ✓ Hypertensive encephalopathy must be considered in patients with blurred vision, severe headaches, altered mental status, or new seizures.
- ✓ Respiratory distress, orthopnea, and cough may be symptoms of pulmonary edema and heart failure.
- ✓ The acute phase generally resolves within 6-8 wk. Although urinary protein excretion and hypertension usually normalize by 4-6 wk. after onset, persistent microscopic hematuria can persist for 1-2 yr. after the initial presentation, an increase hematuria may occur with exercise or with intercurrent illness during this period.

DIAGNOSIS

- Urine color is usually reddish brown(cola ,rusty).
- Urinalysis demonstrates red blood cells, often in association with red blood cell casts, proteinuria, and polymorphonuclear leukocytes and granular cast.
  Protein loss in urine <u>less than 1 gm/24hr.</u>

- Urine volume is reduced during the first 3-5 days occasionally the patient is anuric.
- A mild normochromic anemia may be present from hemodilution and low-grade hemolysis
- Elevated blood urea and serum creatinine levels ,patients may develop hyperkalemia and metabolic acidosis
- The serum C3 level is significantly reduced in >90% of patients.
  C4 is most often normal in APSGN
- Confirmation of the diagnosis requires clear evidence of a prior streptococcal infection. A rising antibody titer to streptococcal antigen(s) confirms a recent streptococcal infection. The ASOT titer is commonly elevated after a pharyngeal infection but rarely increases after skin infections.
  The best single antibody titer to document cutaneous streptococcal infection is the antideoxyribonuclease B level.

- Magnetic resonance imaging of the brain is indicated in patients with severe neurologic symptoms
- Chest x-ray is indicated in those with signs of heart failure or respiratory distress,

PREVENTION

Early systemic antibiotic therapy for streptococcal throat and skin infections will reduce but not eliminate the risk of GN

TREATMENT

Any child with acute PSGN should be hospitalized.

Systemic antibiotic therapy with 10 days course of penicillin is recommended to limit the spread of the nephritogenic organisms.

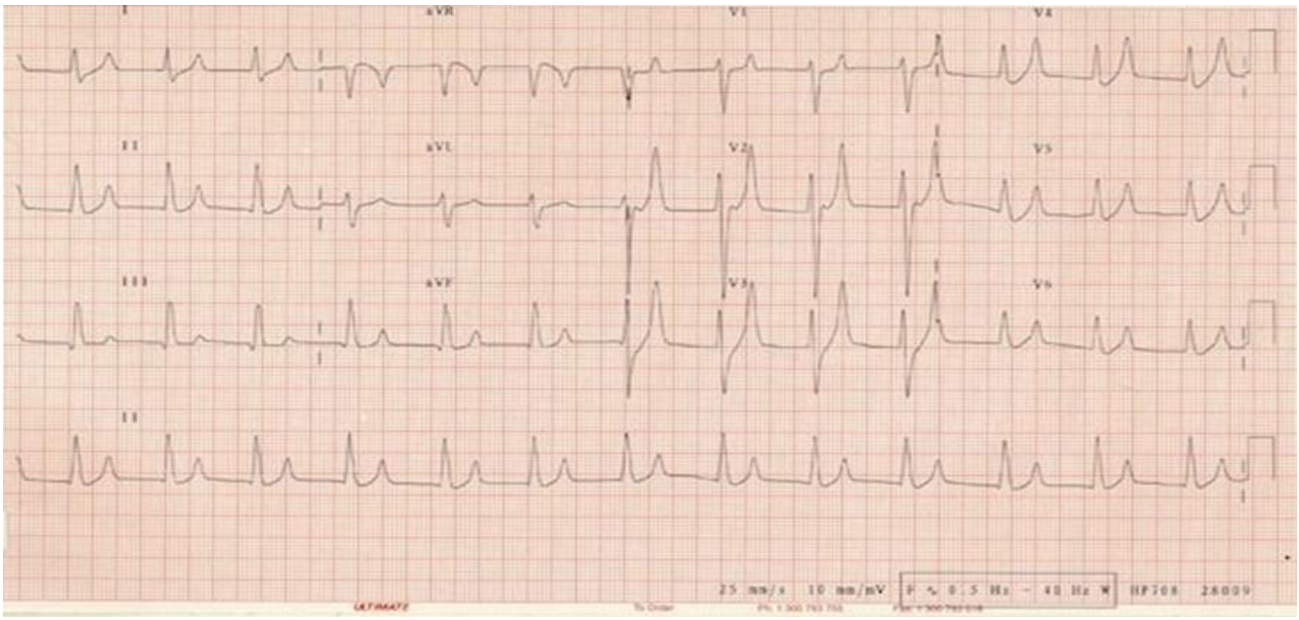
The major life threating complications during initial 1-2 weeks are acute renal insufficiency and acute hypertension.

Treatment of acute renal insufficiency

- Fluid restriction to 400 ml/m2/24 hr. which is the insensible loss + UOP
- If still anuria with evidence of volume overload, restrict fluids further.
- If still anuria, do renal dialysis.
- Management of other sequelae of ARF: hyperkalemia, hypertension and metabolic acidosis.

❖ Hyperkalemia; (serum potassium level > 6 mEq/L) can lead to cardiac arrhythmia, cardiac arrest, and death.

1. The earliest electrocardiographic change seen in patients with hyperkalemia is the appearance of peaked T waves.
2. This may be followed by widening of the QRS intervals
3. ST segment depression,
4. ventricular arrhythmias, and cardiac arrest



Early ECG changes showing Peaked T waves

Procedures to deplete body potassium stores should be initiated when the serum potassium value rises to >6.0 mEq/L

1) Exogenous sources of potassium (dietary, intravenous fluids) should be eliminated.
2) Sodium polystyrene resin (Kayexalate), 1 g/kg, should be given orally or by retention enema. This resin exchanges sodium for potassium and can take several hr. to take effect. A single dose of 1 g/kg can be expected to lower the serum potassium level by about 1 mEq/L.
3) More severe elevations in serum potassium (>7 mEq/L) require emergency measures in addition to Kayexalate. The following agents should be administered:
4) Calcium gluconate 10% solution, 1.0 mL/kg IV, over 3-5 min
5) Sodium bicarbonate, 1-2 mEq/kg IV, over 5-10 min
6) Regular insulin, 0.1 units/kg, with glucose 50% solution, 1 mL/kg, over 1 hr.

   Calcium gluconate counteracts the potassium-induced increase in myocardial irritability but does not lower the serum potassium level. Administration of sodium bicarbonate, insulin, or glucose lowers the serum potassium level by shifting potassium from the extracellular to the intracellular compartment.

7) A similar effect has been reported with the acute administration of β-adrenergic agonists
8) persistent hyperkalemia should be managed by dialysis.

Treatment of hypertension:

Bp should be checked at interval of 4-6 hours, with evidence of hypertensive encephalopathy or signs of pulmonary edema or diastolic Bp>95mmmercury treatment is indicated.

Salt and water restriction is critical, and diuretic administration may be useful

Isradipine (0.05-0.15 mg/kg/dose, maximum dose 5 mg qid) may be administered for relatively rapid reduction in blood pressure.

Longer-acting agents such as calcium channel blockers (amlodipine) or β blockers (propranolol, labetalol) may be helpful in maintaining control of blood pressure.

Children with severe symptomatic hypertension should be treated with continuous infusions of nicardipine, sodium nitroprusside, labetalol, or esmolol and converted to intermittently dosed antihypertensive when more stable.

PROGNOSIS

Complete recovery occurs in >95% of children. Recurrences are extremely rare. Mortality in the acute stage can be avoided by appropriate management of ARF, HFand HT