Glomerulonephritis

Department of Paediatrics

Definition

- Inflammation and multiplication of cells within the glomerulus.

- Injury → Immunologically mediated
  → Uncertain
- GN → Primary
  → Secondary

Primary GN

1. Immune complex GN
    A) Acute post streptococcal
    B) Mesangial IgA (Berger disease)
    C) MPGN
    D) Membranous
2. Anti GBM mediated GN
   eg:- Goodspasture syndrome
3. Uncertain aetiology – MCGN, FSGS

Secondary GN

1. **Immunologically mediated**

   SLE, HSP

   Infections such as SABE, shunt nephritis, syphilis, malaria, hepatitis B

2. **Hereditary disorders**

   sickle cell anaemia

Rapidly progressive (crescentic) GN

APSGN, MPGN, SLE, HSP, Idiopathic

Histological Classification

- **Minimal change**

  L/m & I/m normal

  E/m – fusion of foot processes

  eg:- SSNS

- **Focal segmental glomerulosclerosis**

  Normal glomeruli + sclerosed glomeruli

- **Membranous nephropathy**

  Diffuse thickening of the wall

Classification contd.

- **Proliferative GN**
  - Diffuse proliferative – exudate GN
    e.g. PSGN
  - Pure mesangial proliferation e.g. HSP
  - GN with crescents e.g. SLE, HSP
  - Focal proliferative GN e.g. HSP, Berger's
  - Membranoproliferative GN

Manifestations of GN

- Asymptomatic haematuria / protienuria
- Acute nephritic syndrome
- Nephrotic syndrome

Acute post streptococcal GN (Acute Nephritic Syndrome / Acute Nephritis)

- Haematuria
- Oedema
- Hypertension
- Oliguria
- Renal insufficiency

Aetiology

- Lancefield group A beta haemolytic streptococci (only nephritogenic strains 12,4,25,49)

Pathogenesis & Pathology

- Immune complex disease
- A diffuse proliferative, exudate GN

Macroscopy : symmetrical enlarged kidneys

L/M : all glomeruli are enlarged and diffuse multiplication of mesangial cells

I/M : lumpy bumpy deposits of Ig & complements on GBM.

Clinical Presentations

- gross haematuria / oedema
- acute HT / hypertensive encephalopathy
- acute left heart failure
- anuria.

Epidemiology :

Age group 5-10 years , uncommon <3 years. male > female

Clinical features

- H/O throat / skin infection prior to 1-2 weeks
- **Gross haematuria** - typical smoky brown colour, lasts 1-14/ d , microscopic haematuria lasts longer, rarely no haematuria
- **Oedema -** usually periorbital, occasionally generalized
- **Hypertension** - not always present, diastole of 100mmHg alarming, BP >/110mmHg ⟶ imminent HT encephalopathy, HT persists up to one week

Clinical features contd.

- **Oliguria** < 300ml/m^s/24 hrs (<10ml/kg/24hrs)
- **Proteinuria** – varying degree
- If gross protienuria + ⟶ nephrotic on nephritic syndrome
- **Non specific symptoms** – malaise, lethargy, abdominal pain, fever, headache

The disease has two distinct phases

1. **Initial oliguric phase with hypervolaemia**

   oliguria, oedema, hypertension, pulmonary oedema

2. **Phase of diuresis and natriuresis**

   1-2 weeks after the hypervolaemic phase

Investigations

Urine macroscopy

smoky brown colour, less volume

Full Report

proteinuria varying degree; trace or +

microscopy – RBC , RBC & granular casts

(Urine culture and ABST when the diagnosis is not obvious)

Investigations contd.

Renal function test when indicated

- blood urea, creatinine & electrolytes

Evidence of strptococcal infection

- ASOT / streptozyme test (anti DNAase B antigen), Throat swab – not helpful
- Definitive diagnosis – ASOT + decreased C3

Others when indicated E.g. CXR -

Differential Diagnosis

Other causes of GN including HSP

Other causes of haematuria

Other causes of periorbital oedema

Management

- **Monitoring**

  Daily - weight, input output(volume & colour), BP thrice daily

  - Renal function test if in renal failure

Management contd.

- **Restriction of sodium and water**

  - until diuresis occurs

Daily intake = insensible loss

400ml/m^2/d + UOP in last 24 hours

To be reduced further if oliguria is severe

Protein is restricted to 0.5g/kg/d if in renal failure

Management contd.

Bed rest - only during hypertensive period

Antibiotics - oral penicillin for 10 days

Treatment of hypertension

- fluid restriction
- hydralazine, propranolol
- frusemide
- diazoxide
- nifedipine

Management of complications

- **HT encephalopathy**

  Hydrallazine 0.2 mg/kg/dose

  Frusemide 2 mg/kg/dose

  Diazepam / paraldehyde for fits

- **Pulmonary oedema**

  Fluid restriction, frusemide,

  peritoneal dialysis

- **Acute renal failure**

Prognosis

- Recovery rate >95%

  Microscopic haematuria clears in several months to years.

  Few develop progressive GN
- Indications for renal biopsy:
  - ARF>14 days
  - Deterioration after acute stage
  - Persistent haematuria and protienuria >2 years

Haematuria

Macroscopic (gross)

Microscopic >5 RBC/hpf in 10ml

or >10 RBC/mm3 of urine

Gross Haematuria

Origin → kidney - brown or coke colour + casts

→ lower tract - red and +/- clots

Causes of red/brown colour urine

Blood

Haemoglobin

Myoglobin

Beetroot, food colourings, urate

Therefore prior microscopy is essential

Microscopic haematuria

- **Transient**

  heavy exercise, febrile illness, NS, UTI, contamination from outside.

Persistent

haematuria + in 3 consecutive samples

taken at monthly intervals

Causes

Vary with age and place of living

In children

PSGN UTI

NS HUS

Neoplasms Hydronephrosis

Stones Trauma

Bleeding and clotting conditions

Recurrent gross haematuria syndrome

Drugs, parasitic diseases, other GN

In infancy

- Infections
- HUS
- Renal vein thrombosis
- Tumours

In newborn

- vascular injuries
- polycystic kidneys
- renal vein thrombosis
- extra renal & GN

Evaluation

- A thorough history and physical examination

e.g. * H/O sore throat/ skin infection/

HT + oliguria → **PSGN**

* dysuria, frequency, fever → **UTI**

* A flank mass → **Tumour/**

R.V.thrombosis/ polycystic kidney

* Recurrent gross haematuria →

Berger disease/ Alport syndrome

* Oedema & gross protienuria → **NS**

Investigations

* UFR
* Urine culture and ABST

* Other investigations appropriate to the individual case

THE END