Session Learning Objectives

This session aims to

- Comprehend the causes, pathogenesis, clinical features and management of renal vascular diseases
- Describe the aetiology, pathophysiology, signs and symptoms, investigations and management of various types of glomerular diseases:
  • Glomerulonephritis
  • Nephrotic syndrome
  • Acute nephritic syndrome
  • Minimal change nephropathy
RENNAL VASCULAR DISEASE AND GLOMERULAR DISEASE

- Renal vascular disease
  - Renal artery stenosis

- Glomerular disease
  - Glomerulonephritis
RENAL ARTERY STENOSIS

- **Presentations**
  - Hypertension
  - Deterioration of renal function on ACE inhibitors
  - Flash pulmonary oedema
  - Acute renal infarction

- **Aetiology**
  - Reduction of renal blood flow, >70% narrowing of the artery
  - Most common cause – atherosclerosis
  - Stenosis → global renal ischaemia → ischaemic nephropathy (shrinkage) or renal failure
RENAL ARTERY STENOSIS

Arrow indicates area of stenosis – Left Kidney

RENAL ARTERY STENOSIS

○ Investigations
  • Ultrasound
  • Renal isotope scanning
  • Renal angiography

○ Management
  • Medical
    – Anti-hypertensive, low-dose aspirin, lipid-lowering drugs
  • Angioplasty
  • Surgical resection and re-anastomosis
### DISEASES OF SMALL INTRA-RENAL VESSELS

A number of conditions can lead to acute damage and occlusion of small blood vessels in the kidneys

- Thrombotic microangiopathy
- Disseminated intravascular coagulation
- Accelerated phase hypertension
- Systemic sclerosis
- Atheroembolic renal disease
GLOMERULAR DISEASES

Glomerulonephritis

Glomerulopathies are third common cause of end-stage renal disease

- **Pathogenesis**
  - Many types of GN are immunologically mediated and respond to immunosuppressive drugs
  - Immune response is directed against antigens (sometimes unknown)
  - Evidence of immune reactions are presence of circulating antibodies, glomerular deposition of antibodies, immune complexes, complement and fibrin
GLOMERULONEPHRITIS (GN)

Classification (histo-pathological)

- Minimal change nephropathy
- Focal segmental glomerulosclerosis
- Membranous nephropathy
- IgA nephropathy
- Post infection
# GLOMERULONEPHRITIS (GN)

## Investigations

- Urine microscopy – RBCs
- Urinary protein
- Blood tests
  - Serum urea, creatinine
  - Anti-Streptolysin-O (ASO) titer
  - C3 and C4 level
  - Anti-Glomerular Basement Membrane (GBM)
- Renal imaging
- Renal biopsy
GLOMERULONEPHRITIS

A

B


http://web2.airmail.net/uthman/specimens/images/dpgn.jpg
MINIMAL CHANGE NEPHROPATHY

- Conditions which cause idiopathic nephrotic syndrome
- Minimal change disease accounts for nephrotic syndrome in most children and one-quarter of adults

**Features of nephrotic syndrome**
- Proteinuria
- Hypoalbuminaemia
- Oedema and generalised fluid retention

**Management**
- Minimal change disease usually responds to corticosteroid therapy and does not progress to CRF
PRIMARY FOCAL SEGMENTAL GLOMERULOSCLEROSIS (FSGS)

- FSGS presents as idiopathic nephrotic syndrome but less responsive to treatment than minimal change and may progress to renal impairment.
# PRIMARY FOCAL SEGMENTAL GLOMERULOSCLEROSIS (FSGS)

## Epidemiology

Common cause of nephrotic syndrome in Hispanic and African Americans

## Pathology

- Characterised by sclerosis
- Not all glomeruli are affected
- May be associated with reduced oxygen in the blood/ HIV/ other forms of glomerulonephritis
<table>
<thead>
<tr>
<th>PRIMARY FOCAL SEGMENTAL GLOMERULOSCLEROSIS (FSGS)</th>
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<tbody>
<tr>
<td><strong>Clinical features</strong></td>
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<tr>
<td>• Hypertension</td>
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<td>• Focal sclerosis</td>
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<td><strong>Treatment</strong></td>
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<td>• Corticosteroids</td>
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<td><strong>Complications</strong></td>
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<td>Most people progress to kidney failure with 5-10 years</td>
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MEMBRANOUS NEPHROPATHY

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<tr>
<th>Common cause of nephrotic syndrome in adults</th>
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**Causes**
- Drugs
- Heavy metals
- Hepatitis B virus
- Malignancy

**Treatment**
- Corticosteroids and immunosuppressive drugs

**Prognosis**
- 1/3 remit spontaneously,
- 1/3 remain in nephrotic state,
- 1/3 show progressive loss of renal function
# IGA NEPHROPATHY AND HENOCH-SCHONLEIN PURPURA

**Aetiology** – usually idiopathic

- Presents in many ways – haematuria, proteinuria, hypertension
- Common cause of end-stage renal failure (ESRF)
- Henoch-Schonlein syndrome comprises skin rash, abdominal pain, joint pain and GN due to systemic vasculitis
- Response to immunosuppressive therapy is poor
HENOCH-SCHONLEIN RASH

http://www.riversideonline.com/source/images/image_popup/ans7_hspurpura.jpg
GN ASSOCIATED WITH INFECTION

- Bacterial infections usually subacute (e.g. subacute bacterial endocarditis) cause GN
- GN associated with malaria, hepatitis B & C, and other chronic infections is common
- Now rare in developed countries

Presentation

- Presents with severe salt and fluid retention, hypertension, haematuria and oliguria

Management

- Usually resolves spontaneously
ACUTE POST-INFECTIONOUS GN

- GN following infection with certain strains of streptococcus and often called post-streptococcal nephritis
- More common in children
- GN develops >10 days after throat infection or skin infection

**Presentation**
- Sodium retention, hypertension and oedema

**Management**
- Fluid and sodium restriction, diuretics, hypotensive agents

ACUTE POST-INFECTIONOUS GN

Infiltration of mononuclear Lymphocytes in glomerulus

http://eyepathologist.com/images/Ki0210.jpg
RAPIDLY PROGRESSIVE GN

- Extreme inflammatory nephritis which causes rapid loss of renal function over days to weeks

This syndrome occurs when kidney disease damages the filters in the kidney, causing leakage of protein from the blood into urine and a build up of water (oedema) in the kidney.
NEPHROTIC SYNDROME

Clinical features:
Heavy urinary protein loss, hypoalbuminemia, oedema and hypercholesterolemia.

Pathophysiology:
- **Proteinuria** – due to structural damage and reduction in the fixed negatively charged elements in the capillary wall.
- **Oedema** – due to reduced concentration of osmotically active albumin in the blood.
NEPHROTIC SYNDROME

NEPHROTIC SYNDROME

Aetiologies

- Membranous GN in adults
- Minimal change GN in children
- And the GN’s caused by:
  - Secondary to infections such as malaria
  - Drugs such as penicillin and captopril
  - Heavy metals such as mercury, cadmium and gold
- Reactions to allergens such as poison ivy, pollens, bee stings, cow’s milk
# NEPHROTIC SYNDROME

**Clinical presentation**

- Frothy urine
- History of exposure to drug or allergen
- Heavy protein loss in urine
- Low protein in the blood
- High cholesterol in the blood
- Oedema of lower legs, sacral, peri-orbital, arms and genitalia
- Ascites
- Butterfly rash of SLE, retinopathy, or neuropathy for Diabetes Mellitus
NEPHROTIC SYNDROME

Investigations

- 2 hour urinary protein (>3-5 gms in adults) and serum albumin levels (<30 gms/L)
- Renal function tests
- Renal biopsy for histological changes
- Lipid profile for high cholesterols
SLE BUTTERFLY RASH

RETINOPATHY – RETINAL LESIONS

http://imaging.ubmmedica.com/consultantlive/images/articles/2003/07012003/0307ConPERheum1A.jpg

http://www.tedmontgomery.com/the_eye/eyephotos/pics/HypertensiveRetinopathy-2.jpg
Readings and Resources

Resources:

- **Set Textbooks:**

- **Additional textbooks:**
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