Renal Medicine

Summary

Kidneys may
- Become inflamed (nephritic)
- Leak protein (nephrotic)
- Stop functioning (renal failure)
- Bleed (haematuria)

Acute Renal failure
- Pre-renal
- Renal  - progression of pre-renal with acute tubular necrosis
  - intrinsic renal disease eg rapidly progressive glomerulonephritis
- Post renal

Acute nephritis (acute nephritic syndrome)
- Smoky brown haematuria with red cell casts
- Hypertension
- Oliguria
- Proteinuria

Acute Nephritic syndrome: aetiology
- Post streptococcal
- IgA nephropathy (commonest glomerulonephritis)
- Vasculitis eg systemic lupus

Investigation
- Throat swab and ASO titre
- 24 hour urinary protein

Treatment
- Penicillin if streptococci present
- Salt restriction
- Anti-hypertensive drugs

Nephrotic syndrome
- Proteinuria (>5g per 24hours) makes urine frothy
- Hypoalbuminaemia (<30g/dl)
- Oedema (loss of protein onctic pressure allows salt and water into extracellular space; increased salt and water retention by kidney)

(and: Hypercholesterolaemia as liver makes more cholesterol- linked to compensatory increase in albumin synthesis)
Causes in children
Minimal change GN in children
(idiopathic and nearly always steroid-responsive; does not progress to renal failure)

Causes in adults
*Glomerulosclerosis* especially due to
- diabetes

*Membranous GN*, usually idiopathic but sometimes associated with:
- Malignancy
- Malaria, hepatitis B
- Systemic lupus
- Drugs: Gold and penicillamine, high-dose captopril

Amyloid

Treatment
- Oedema- diuretics and salt restriction
- Minimal change glomerulonephritis- steroids

Acute renal failure
- Urine output less than 400ml per 24 hours
- Pre-renal- hypovolaemia, shock (septicaemic, cardiogenic)
- Renal- rapidly progressive GN and many other causes
- Post renal- compression or obstruction
Clinical features
- Oliguria / anuria
- Unwell
- Hyperkalaemia (arrhythmias)
- Acidosis

Acute renal failure
- A Arterial blood gases to assess degree of acidosis
- B Bloods: FBC, U+E, creatinine, blood cultures
- C Catheter to assess fluid balance
- D Dietary protein restriction
- E Electrolytes and ECG
- U Ultrasound to exclude obstruction and assess renal size
  (small indicates previous renal disease, discrepancy between sides ?renal artery stenosis; if normal size and no known reason, ?biopsy)

Treatment
- Catheterise
- Correct fluid balance
- Correct metabolic abnormalities (eg hyperkalaemia)
- Treat underlying cause if identified
- Renal support- dialysis

Chronic renal failure
- Progressive irreversible deterioration in renal function
- Causes:
  - Hypertension, vascular disease
  - Diabetes
  - Drugs
  - Infection (chronic pyelonephritis)
  - Chronic obstruction

Clinical features
- Symptoms and signs of underlying cause eg diabetes
- Fatigue, lethargy, anorexia, weight loss
- Pruritis, gout
- Hypertension
- Skin pigmentation
- Peripheral neuropathy
- Dialysis- peritoneal catheter or a-v fistula

Chronic renal failure: the “P’s”
- Pigmentation
- Pruritis
- Pulmonary oedema
- Peripheral neuropathy
- Parathyroid over-activity (phosphate retention reduces ionised calcium; PTH stimulates osteoclasts; bone resorption from tips of terminal phalanges and skull – “pepperpot”)
Glomerulonephritis

- Confusing as only weak correlation between:
  - Histology
  - Cause
  - Type of presentation
  - Limited number of glomerular responses to injury

**Presentations of Glomerulonephritis**
(Grossly Simplified)

<table>
<thead>
<tr>
<th>Non proliferative</th>
<th>Proliferative</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Minimal change</td>
<td>• Mesangio</td>
</tr>
<tr>
<td>• Membranous</td>
<td>capillary</td>
</tr>
<tr>
<td>• Focal segmental</td>
<td>• Diffuse</td>
</tr>
<tr>
<td>sclerosis</td>
<td>proliferative</td>
</tr>
</tbody>
</table>

Nephrotic syndrome

Acute nephritis

Recurrent haematuria

Acute renal failure

**Summary**

**Kidneys may**

- Become inflamed (nephritic)
- Leak protein (nephrotic)
- Stop functioning (renal failure)
- Bleed (haematuria)

**Acute Renal failure**

- Pre-renal

- Renal  - progression of pre-renal with acute tubular necrosis
  - intrinsic renal disease eg rapidly progressive glomerulonephritis

- Post renal
Notes on Chronic Renal Failure:

Would you ask this patient with chronic renal failure some questions? 
Think "The B's"
Bothersome symptoms, blood pressure, breathing, blood, bones, bags

Bothersome symptoms
Tiredness (?anaemia of chronic disease)
Anorexia
Nausea and vomiting, diarrhoea
Gout (urate retention)
Pruritis
Neuropathy- stocking distribution sensory loss
Pericarditis

Blood pressure
? long history hypertension preceeding renal problems
? other vascular problems (angina, MI, claudication, stroke)
? on treatment
Most with end stage renal failure are hypertensive whatever the aetiology

Breathing
Breathlessness may be due to pulmonary oedema, anaemia or acidosis.

Pulmonary oedema
Previous vascular problems (eg previous MI with dilated damaged ventricle), Hypertensive heart disease (with ventricular hypertrophy)
Fluid retention)
? swollen ankles ?PND
? is patient on diuretics
Weighing the patient sequentially is helpful

Blood
Normochromic normocytic anaemia of chronic disease (normal ferritin)
Is the patient receiving erythropoietin (subcutaneous injection)?
? on NSAIDs- may cause GI blood loss and iron deficiency picture
Many patients with CRF have a raised ESR (probably related to high fibrinogen)
If very high ESR, important to check for myeloma
Many patients have mild thrombocytopenia and easy bruising

Bones
? bone pain- may suggest renal bone disease
? on calcium and vitamin D treatment
? on phosphate binders
? previous parathyroidectomy

Bags
? having chronic ambulatory peritoneal dialysis (CAPD)
Typically 4 exchanges per day with 2 litres each time
Proportion of hypertonic and isotonic bags (hypertonic has higher osmolality; used in greater proportions if patient is fluid overloaded)
? any cloudy bags (suggests infection- peritonitis)
Renal impairment: factors favouring chronic disease
Normochromic normocytic (NCNC) anaemia
Peripheral neuropathy
Small kidneys on U/S or IVU
Bone disease (low calcium, high phosphate, raised alkaline phosphatase and raised parathyroid hormone levels).
Band keratopathy (rare- calcification in cornea)

Causes of chronic renal failure: "idiot chap"
I idiopathic
D diabetes
I infection: chronic pyelonephritis (assoc. ureteric reflux)
O obstruction eg chronic prostatic hypertrophy
T tuberculosis
C chronic glomerulonephritis (especially IgA nephropathy)
H hypertension
A analgesic nephropathy
P polycystic kidneys

Chronic glomerulonephritis, hypertension and diabetes are the commonest and most important causes. Idiopathic cases are those presenting late with small shrunken kidneys where histology is unhelpful. Some of these are likely to be due to undiagnosed chronic glomerulonephritis and some to undiagnosed hypertension.

Would you examine this patient with chronic renal failure?
? pallor
? sallow complexion with lemon yellow tinge or brown pigmentation of skin
? brown discolouration over distal nails ("half and half nails")
? level of consciousness ?metabolic tremor ?hiccoughs
? arteriovenous shunt for haemodialysis
? fluid retention (pulse, JVP, sacral and ankle oedema, weight)
? peritoneal catheter (Tenchkoff catheter) for CAPD; transplant scar
? evidence gout or neuropathy affecting feet
? pericardial rub
Check blood pressure in all patients

Urinalysis- checking for glycosuria, proteinuria and infection (nitrites and leucocytes)

Investigation of CRF: to assess severity and general features
Urine microscopy- for evidence of infection and for "casts" which may suggest glomerulonephritis
FBC- normochromic normocytic anaemia
Urea and creatinine- some guide to renal impairment but poor correlation with glomerular filtration rate. Better to assess:
Creatinine clearance- 24hour urine collection together with blood sample
24 hour urinary protein usually done on same sample
Serum urate and phosphate- both retained in CRF
Calcium, alkaline phosphatase- low calcium and high ALP suggests renal bone disease
Parathyroid hormone levels- may become high before phosphate retention occurs and indicate need for phosphate restriction and binders.
Investigation of chronic renal failure to establish cause:
- Urine: test for glucose; MSU for infection; Bence Jones protein for myeloma
- Blood tests: hepatitis B serology, antinuclear factor, serum electrophoresis, immunology (including complexes, complement etc)
- X-ray abdomen (calcification may suggest stones or, if extensive, tuberculosis)
- Renal ultrasound (to exclude obstruction; picks up polycystics)
- IVU- looking for
  - discrepancy in renal size (renal artery stenosis)
  - dilated pelvic calyces and cortical scarring (chronic pyelonephritis)
  - loss of papillae (analgesic nephropathy)
- Renal biopsy if there is real diagnostic doubt and not already in end stage renal disease (ESRD). Biopsy looks for evidence and type of chronic glomerulonephritis.

Indications for dialysis
- Symptomatic uraemia despite conservative treatment
- Pericarditis or bone disease
- Progressive decline in renal function (to prevent symptoms)
- Volume overload despite fluid restriction and diuretics
- Hyperkalaemia despite treatment

Dietary management
- Healthy balanced diet encouraged
- Very low protein diets no longer in favour
- Avoid high protein intake
- Care with potassium (instant coffee, chocolate, fruit and fruit juice)
- Care with salt intake
- Avoid salt substitutes (contain potassium)
- Low phosphate-reduced milk and fish intake

Medication
- Calcium acetate (phosphate binder-preventing absorption, taken with meals)
- Erythropoietin- subcutaneous injection
- Iron supplements- if evidence iron deficiency
- Calcium - taken between meals
- Activated vitamin D (alfacalcidol; "One-alpha")
- Anti-hypertensives
- Statins if hyperlipidaemia as high risk of vascular events
- Allopurinol if hyperuricaemia

ECG changes of hyperkalaemia
- Low flat P waves
- Broad bizarre QRS
- Slurring into the ST segment
- Tall tented T waves
- Long PR interval

Hyperkalaemia is more common in acute renal failure. It may occur in chronic renal failure too eg after a GI bleed, excess dietary intake or missing a dialysis session.

Treatment of life threatening hyperkalaemia
- First arrange ECG monitor and access to resuscitation equipment
- Intravenous calcium gluconate (stabilises myocardium)
- Glucose and insulin (drives potassium into cells)
- Dialysis if patient unwell