Kidney infections

Acute pyelonephritis
Inflammation of renal parenchyma and renal pelvis
Typically fever, chills and costovertebral angle tenderness +/- LUTS
Gram negative bacteria

- E. coli
- Proteus
- Klebsiella
- Pseudomonas
- Enterobacter
- Citrobacter
- Serratia
- Enterococcus, S aureus and S epidermidis occasional GP orgs.

*E. coli expressing Type II p-fibriae responsible for 80% of cases

Diagnosis
Urinalysis typically positive for nitrities and leucocytes
MSU > 10⁴ cfu/ml with pyuria suggestive (?arbitrary definition) – 20%
have counts less than 10⁵
WBC casts
Elevated CRP and ESR

Imaging
Not indicated in acute uncomplicated pyelonephritis
Reserved for complicated cases or those not responding after 72 hrs

IVU
Renal enlargement in 20% if done acutely – may be focal
(lobar nephronia) appearing as a mass lesion
Reduced nephrogram and delay secondary to cortical vasoconstriction

USS
Excludes obstruction, identifies stones and gross focal changes

CT
Excellent for focal changes and identification of gas and stones. Generally reserved for those failing to improve.
Upper urinary tract infections

Management

Mild, outpatient

PO TMP-SMX 14 days

*PO Ciprofloxacin 500mg bd 7 days

7 day course of cipro more effective and fewer side effects than 14 day course of TMP-SMX (Talan 2000).

Third generation cephalosporin cefpodoxime proxetil 200mg bid equivalent efficacy cf. ciprofloxacin 500mg bid.

No adequately powered studies of penicillin/BLI vs. fluoroquinolone or TMP-SMX

Table 2.4: Oral treatment options of acute uncomplicated pyelonephritis in adult pre-menopausal non-pregnant women according to level of evidence and grade of recommendation. (For oral therapy, see text.)

<table>
<thead>
<tr>
<th>Substance</th>
<th>Dosage</th>
<th>Duration</th>
<th>LE</th>
<th>OR</th>
<th>Author, year</th>
<th>Ref</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ciprofloxacin</td>
<td>500 mg bid</td>
<td>7 days</td>
<td>Ic</td>
<td>A</td>
<td>Slen 2000</td>
<td>09</td>
<td>a. Ciprofloxacin significantly more effective than ceftriaxone/IMP/AMPS and with trend towards less AE.</td>
</tr>
<tr>
<td>Ciproflax</td>
<td>1000 mg od</td>
<td>7-10 days</td>
<td>Ic</td>
<td>A</td>
<td>Tanen 2004</td>
<td>10</td>
<td>b. Efficacy and tolerance of extended release ciprofloxacin (ciproflax) 1000 mg od equivalent with 10-day conventional ciproflax</td>
</tr>
<tr>
<td>Cefpodoxime</td>
<td>200 mg bid</td>
<td>10 days</td>
<td>Ic</td>
<td>U</td>
<td>Miller 2011</td>
<td>74</td>
<td>c. Clinically equivalent with ciprofloxacin 500 mg bid</td>
</tr>
<tr>
<td>Cefuroxime</td>
<td>400 mg od</td>
<td>10 days</td>
<td>Ic</td>
<td>A</td>
<td>Miller 2006</td>
<td>74</td>
<td>d. Equivalent with cefuroxime 600 mg bid, not available in Europe</td>
</tr>
<tr>
<td>Levofloxacin</td>
<td>250 mg od</td>
<td>10 days</td>
<td>Ic</td>
<td>A</td>
<td>Richard 1999</td>
<td>12</td>
<td>e. Equivalent with ciproflaxacin 500 mg bid</td>
</tr>
<tr>
<td>Cefuroxime</td>
<td>400 mg od</td>
<td>10 days</td>
<td>Ic</td>
<td>U</td>
<td>Richard 1999</td>
<td>74</td>
<td>f. Only statistically undersusceptible</td>
</tr>
<tr>
<td>TMP-SMX</td>
<td>1600/600 mg bid</td>
<td>14 days</td>
<td>Ic</td>
<td>U</td>
<td>Stamm 1987</td>
<td>68</td>
<td>g. Only if uropathogen is known to be susceptible to TMP</td>
</tr>
</tbody>
</table>

*Cefuroxime profile

LE = level of evidence; OR = grade of recommendation; TMP = trimethoprim; SMX = trimethoprim/sulfamethoxazole; bid = three times daily; od = twice daily; od = once daily; AE = adverse events.

Mod/severe, I/P

IVI, IVABx, antiemetics, painkillers 14-21 days

IV Gent and ampicillin (7mg/kg; 1g qds)

IV Ceftriaxone 1-2g qds

IV Ciprofloxacin 400mg bd

Pregnant

IV Ceftriaxone 1-2g qds

IV Gent and ampicillin

IV Tazocin 4.5mg tds

IV Imipenem 500mg qds

Imaging for complicating factors

10-30% relapse rate after 14 days appropriate Rx – repeat MSU 4 days on and 10 days off Rx. Usually cured after further 2 week course.

Acute focal bacterial nephritis

Uncommon form of acute pyelonephritis

Similar presentation but more unwell

Often in diabetics and immunocompromised

Mass on USS

Wedge shaped area of poor perfusion on contrast enhanced scan

Management as for acute pyelonephritis

Emphysematous pyelonephritis

Rare severe necrotising infection of kidney; a/w mortality rates of 20-43%

Usually in diabetics (70-90%), always in adults, often with a history of UUT obstruction

Classic triad of fever, loin pain and vomiting always seen; 50% palpable mass, occasionally with crepitation
E Coli is usually responsible – produces carbon dioxide from metabolism of sugars

**Diagnosis**

AXR shows gas in renal parenchyma in 85% cases (mottled vs. crescent), but CT investigation of choice

**Classification**

Divided into type 1 and type 2 based on CT findings:

- **Type 1**
  - Generalised renal infection
  - Gross parenchymal destruction
  - Small gas locules throughout kidney
  - Minimal fluid collection
  - ~60% mortality

- **Type 2**
  - Localised renal infection
  - Mild/moderate focal destruction
  - Coalescence of gas focally
  - Air-fluid level
  - ~20% mortality

**Management**

- Resuscitation
- Broad spectrum antibiotics
- IV insulin sliding scale
- Emergent nephrectomy (medical therapy alone a/w 60-80% mortality)

**Renal abscess**

Pus in the renal parenchyma

**Organisms**

- (i) Gram-negative
  - Most common
  - Almost universally from ascending route
  - Two-thirds in association with stones or renal damage;
  - occasionally a/w reflux disease
  - Experimental and clinical data suggest haematogenous seeding
  - by GNB rare, except in instances of complete collecting system obstruction

- (ii) Gram-positive
  - Uncommon
Historically majority of renal abscesses were secondary to haematogenous seeding by staphylococci. Now rare, except for immunocompromised and IVDU

(iii) Mycobacteria

Very rare even in patients with genitourinary TB

Usually parenchymal scarring, wall thickening and calcification, hydronephrosis and hydrocalycosis

Isolated hydrocalycosis may be mistaken for renal abscess however

Clinical features:
Fever, chills, rigor
Malaise and lethargy
Abdominal/flank pain
Leucocytosis
Positive UTI (GNB only)

Diagnosis:
USS
Initially indistinct echopoor parenchymal SOL with varying degree of internal echoes
maturation into discreet lesion of variable echotexture with hyperechoic margin
Presence of air casts dense echo/shadow

CT
Renal enlargement
Discreet low attenuation mass with surrounding rim of higher attenuation (ring-sign)

Management:
Dependent on size
Small abscesses <= 3cm a/w resolution with prompt administration of appropriate ABx
Abscesses > 3cm suitable for percutaneous drainage.
Failure to respond to above should prompt a search for perinephric abscess
Surgery may be considered as primary surgery for abscesses > 5cm

Infected hydronephrosis/pyonephrosis
Definitions below from Campbells

Infected hydronephrosis – bacterial infection in hydronephrotic kidney
Pyonephrosis – infected hydronephrosis with suppurative destruction of renal parenchyma a/w total or near total loss of renal function

Clinical presentation: severe sepsis, with fever, chills, rigors, abdominal pain, shock, leucocytosis. Dipstick may be negative in complete obstruction

USS diagnosis of pyonephrosis dependent upon identification of internal echoes in dependent portion of kidney. Focal echopoor areas within parenchyma suggest destruction highly suggestive of pyonephrosis vs. infected hydronephrosis.

Management:
Antimicrobials and judicious drainage
Percutaneous vs. endoscopic drainage
Perinephric abscess
Pus within Gerota’s fascia [cf. paranephric abscess = outside Gerota’s fascia]
Routes of infection:
  - Kidney (60-80%)
    - ruptured cortical abscess (Staphylococcal seeding)
    - ruptured corticomedullary abscess (GNB)
    - ruptured calyx
      - pyonephrosis
      - calyceal diverticulum
      - staghorn
  - Haematogenous (10-30%)
    - Skin, mouth, lung infections
    - Infected perinephric haematoma
  - Paraneophric space (10%)
    - Bowel (Crohns etc.)
    - Pancreas
    - Subphrenic/subhepatic abscess
    - Spine (Pott’s etc.)
Clinical presentation
  - Insidious development of symptoms – 60% present > 14 days
  - Unexplained fever (absent in ~30%)
  - Nightsweats
  - Weight loss
  - Antalgic gait, flexion and external rotation with psoas irritation
  - Leucocytosis and pyuria in >75% cases
  - Multiple organisms = MSU and blood culture frequently miss organisms
    - Edelstein 1988 (therefore broad spectrum irrespective of results)
Diagnosis
  - AXR  Normal in 40%
    - Loss of renal outline and/or psoas shadow
    - Scoliosis in up to 50%
    - Rarely gas or air/fluid levels
  - USS  Generally hypoechoic with debris (occasionally air)
    - Hyperechoic thick, irregular wall
  - CT  Investigation of choice
Management
  (i)  Appropriate antibiotics [NB. a number of reviews have shown that virtually all patients with acute pyelonephritis are rendered afebrile with 4 days of appropriate IV antibiotics (Thorley 1974, Fowler 1994) – thus if patient continues to spike after 4 days needs CT to exclude parenchymal or perinephric abscess]
  (ii) Drainage (percutaneous or surgical)
Xanthogranulomatous pyelonephritis
Destruction of renal parenchyma with granulomatous infiltrates containing lipid-laden macrophages
Rare
Women > men
Adults > children
Peak incidence 40-60 yrs
Aetiology
Unknown
Nephrolithiasis in ~80%
Upper tract obstruction +/- stones in others (PUJO, VUR)
Increased risk in diabetes and immunocompromised

Presentation
Fever
Flank pain
Weight loss
Palpable mass
Occasionally fistula

Pathology
Macro
Hugely enlarged kidney with normal contour typical – occasionally focal
Lesion starts in PC system and invades into renal parenchyma

Micro
Lipid-laden (foamy) macrophages (difficult to distinguish from CCRCC on microscopy, and especially difficult on frozen section)
Visible bacteria

Diagnosis
Blood investigations as for pyelonephritis
Positive urine cultures in 50-75%
Proteus > E. Coli
Positive renal tissue culture in >90%

Radiology
Classic triad in >50%
Unilateral renal enlargement
Non-function
Calculus

USS
Hypoechoic centre and hyperechoic rim

CT
Investigation of choice
Enlarged kidney with normal contour
Water-density central lesion (often with stone at centre) surrounded by enhancing rim (differs from RCC)
Management
Nephrectomy Rx of choice
Estabishes diagnosis
Kidney often non-functioning
Removes infective focus
Antibiotics should be commenced ASAP and continued peri-operatively
Partial nephrectomy may be an option for localised disease
Conservative Mx reported but excluding malignancy difficult on Bx alone

Malakoplakia
= ‘Soft plaque’
Originally described by Michaelis and Guttman in 1902
Chronic inflammatory condition affecting urinary tract believed to represent abnormal response to infection
Characterised by presence of lipid laden (foamy) macrophages containing pathognomonic Michaelis-Guttman bodies
Urinary tract involved in ~60% of cases (GIT, lung, skin and LNs also may be affected)
Rare
Female > males 4:1
Age > 50 yrs
Aetiology
Unknown
Coliform UTI (usually E. Coli) a consistent finding on MSU
~ 50% immunosuppressed or serious sytemic disease
Believed to represent a failure of macrophages to adequately phagocytose bacteria
Macrophages have reduced cGMP and increased alpha-1 antitrypsin

Presentation
Usually patient has a history of recurrent coliform UTIs
Bladder irritative LUTS and haematuria
Ureter obstruction and haematuria
Kidney
flank pain fever and mass
Abnormal mass on imaging
Rarely a/w renal vein thrombosis

Pathology
Soft yellow-brown plaques
Grow to form polypoid lesions
Micro Large histiocytes (vonHansellmann cells) containing basophilic inclusions (Michaelis-Guttman bodies – arrowed.

Diagnosis
Biopsy confirms diagnosis
Occasionally early disease a/w absence of MG bodies
IHC of histiocytes highly positive for alpha-1 AT which suggests diagnosis

Management
Chronic antibacterial prophylaxis
Rifampicin, doxycycline and TMP especially good as have intracellular activity
Nephrectomy for symptomatic unilateral lesions
Overall mortality 15% (~20% for renal disease)

Chronic pyelonephritis
Common
25% of ESRF population
Aetiology controversial. Some believe that untreated chronic infection – vasoconstriction (TXA2) – ischaemia – atrophy, but most believe that chronic infection alone is not sufficient to damage renal parenchyma.
Damage may have occurred in childhood with reflux of infected urine into kidney
Histology shows non-specific infiltrate of lymphocytes, plasma cells and occasionally PMNs.
Management – renal support and treatment of UTIs

Renal echinococcus
Extremely rare
Larva from dog tapeworm – human gut – duodenum penetration – liver – lungs. 3% pass through lungs to systemic circulation to kidneys
Typical renal hydatid cysts
Single in 95% of cases; bilateral in 5%
Slow growth – 1cm/yr
Triple epithelium – outer fibroblasts, middle capsule layer, inner germinal layer (producing more larva known as scoleces in daughter cysts) Daughter cysts detach from germinal layer to float freely within main cyst
Vague back pain, mass and haematuria
Predilection for seeding in renal poles

Diagnosis
<50% eosinophilia
Immunology variously reliable
Daughter cysts in urine diagnostic but usually not present

Management
Surgical
Avoid rupture – risk of anaphylaxis
Pretreat with mebendazole/albendazole
? inject scolicidal agent prior to removal
Mebendazole/albendazole unreliable as medical Rx alone