KIDNEY AND URETER

MUSEUM CATALOGUE

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KIDNEY AND URETER

INTRODUCTION

Pathology is all about understanding disease – how it arises, its patterns, complications and how it causes symptoms and signs. That understanding is aided by having a visual appreciation of the morphological changes in tissues.

Powers of observation and description are not just of use in pathology. These are important when examining patients also. As soon as a patient walks into a room you should be observing them (are they fat, thin, pale, yellow, short of breath etc). Specific site, size, colour, texture, appropriate terminology etc are also important for describing lumps and skin lesions on a patient, and knowledge of pathological features is important in radiological diagnosis, so the observational and descriptive skills which you learn in pathology have a broader application.

The kidneys are situated in the retroperitoneum. Each normally weighs around 150gm and is about 11cm long. They consist of an outer cortex and inner medulla, the medulla containing a number of pyramids, the apices of which are the papillae. Urine drains from the collecting ducts into first the minor then the major calyces that join to form the renal pelvis. It is important to remember that the fat within the renal hilus is normal.

The pathology pots within the museum display a variety of diffuse and focal abnormalities of the kidney. Diseases of the kidney tend to affect one of its 4 main components: blood vessels, glomeruli, tubules and interstitium. There is not much to see macroscopically in the glomerulonephritic diseases, so few of these are represented. Scattered simple cysts in the kidney are not uncommon.

Any comments on this catalogue are welcome. Please contact a member of the department.

HOW TO USE THIS CATALOGUE

This catalogue can be used as a tool to develop your knowledge, as well as provide an opportunity for revision.

It is divided into:

- Introduction and approach to specimens of kidney and ureter (pages 2-5).
- Index (pages 6-9). Examples of specific diagnoses can be found via the index.
- Core and classic disease processes of the kidney and ureter (pages 10-26). This gives examples and discussion of core and/or classic diseases of the kidney and ureters. These are the specimens that students should focus on being able to identify initially. However, it depends to some extent on what you have covered in lectures and practical classes or resource sessions as to what you should know. Some of the specimens and discussion are directed more towards clinical medical students.
- Main catalogue (pages 27- 48). This section covers the specimens in numerical order. Questions and/or comments accompany some of the specimens to help you expand your knowledge. In order to fit more specimens in the museum, not all of the pots are in numerical order on the shelves, and large specimens are often found on the bottom shelves.

You might find it useful to work quietly with a few friends and to have a few textbooks handy (e.g. pathology, medical, anatomy). You will also find that you can learn some anatomy and clinicopathological correlation from the specimens and information given.

You do not have to examine every single specimen in the museum. However, just as in clinical practice, you will not become proficient in diagnosing something if you have only seen one case. Exposure to a variety of cases (specific diagnoses can be found via the index) to experience the variability in morphology will help your learning greatly. In general red and blue dots indicate basic and straightforward cases, whereas yellow dots indicate a more complex case. This is not a hard and fast rule, and you will find yellow dot specimens turning up in resource sessions/practical classes and even exams, if they represent classic pathology.

In general

- read the clinical information given
- look at the entire specimen, not just the front
- identify and orientate the organ or tissue.
- from your knowledge of pathology (which will come with time) look for relevant features to help you make the diagnosis. Of course to appreciate the abnormal you first need to have an appreciation of normal anatomy to be able to recognize and orientate the organ/tissue and the abnormalities
- make a diagnosis or differential diagnosis using any clinical information given to you – it is often relevant – sometimes the diagnosis is only made with knowledge of the clinical features. Even when you know the diagnosis, attempt to identify relevant features in the specimen and understand why this is the diagnosis.
- attempt to correlate the pathological features with the clinical features (clinico-pathological correlation) i.e. explain how the pathological features have caused the patients symptoms and signs (when relevant)
- try to answer any questions presented yourself before reading the answers.

You may prefer to look at the specimen 'blind', without reading the clinical information given first.

Remember that some of these specimens are very old, and some of the investigations and treatments mentioned may be out of date.

Limits to diagnosis on macroscopic examination

In all cases a diagnosis is given in the catalogue, sometimes it was made based on the stated clinical history and histopathological findings. In some cases the macroscopic appearance is classic and even without the clinical information and histopathological findings you should be able to make the diagnosis from the appearance, in others, it might only be possible for you to give a list of differential diagnoses or a more general diagnosis.

In relation to pathology pot specimens in examinations, you may be asked

- for a diagnosis
- for a description
- about the pathogenesis of the disease
- about the predisposing factors and/or causes of the disease
- about the potential complications of the disease and how they arise

- to explain a patient's clinical symptoms and signs or investigation results in light of the pathological abnormalities present
- to describe the expected histological abnormalities in the abnormal areas

or other searching questions that we can concoct.

BASIC APPROACH TO INTERPRETATION AND DESCRIPTIONOF OF KIDNEY AND URETER PATHOLOGY SPECIMENS

Students are expected to be able to give a brief succinct description of relevant macroscopic features of a specimen using appropriate terminology, as well as to arrive at a diagnosis or differential diagnosis. Even if not asked for a description, identification of relevant features is helpful in the diagnostic process. Your descriptive skills will improve with practice.

In any aspect of medicine, one needs to approach things in a systematic manner; otherwise important points may be omitted.

- Read the clinical history, it will often provide relevant information
- Look at the front of the pot first (i.e. the one with the number and the dot), but always make sure to look at the back and sides as well.
- Identification of and description of the abnormality.
- Decide and state whether the organ is of normal size, too small (is it atrophic?) or too large (oedematous, other)
- Is the abnormality focal, diffuse (involving the entire organ, region or tissue) or multifocal? The lesion itself should then be described.

Focal lesion

The description of a discrete or focal macroscopic lesion can incorporate a number of features.

Size: Give an approximate measurement

Shape

Colour: What colour is it? Is it all one colour or is it many colours (variegated)? Does it look homogenous (all the same the whole way through)?

Consistency: This is of course difficult when the specimen is in a pot and you are unable to touch it. But even just by looking you can get some idea: Does it look solid or firm? Does it look friable (as if it is falling to pieces) or are there bits missing or greyish areas (altered blood) to suggest necrosis?

Margins: Are they well defined/demarcated, or irregular or diffuse? Renal cell carcinomas, although malignant, typically have well-circumscribed margins and may even appear encapsulated. Their cut surface is typically very heterogenous with areas of necrosis, haemorrhage, fibrosis and even cystic degeneration.

Diffuse

There are a number of pots that show small kidneys. For those with a finely granular surface,

consider hypertension, diabetes, chronic glomerulonephritic conditions or generalised chronic ischaemia

(from renal artery stenosis). In these cases there is also often variable thinning of the cortex.

Small chronic pyelonephritic kidneys classically show larger broader depressed surface scars.

There are a number of pots that show a slightly enlarged kidney. For these consider:

- an infiltrative process such as amyloid (extracellular protein deposition) or leukaemia (cell infiltration) where the organ is often pale and there is reduced demarcation between the cortex and medulla
- an acute inflammatory process (acute pyelonephritis) causing oedema, where there may also be hyperaemia and microabscess formation
- acute severe glomerular injury such as with malignant nephrosclerosis or a rapidly progressive glomerulonephritis where there are often petechial haemorrhagic spots.

Use the clinical information given to help in your diagnosis. Definite diagnosis of course generally requires histological confirmation.

Adult polycystic kidney disease is another diffuse process where there are numerous large cystic spaces replacing much of the renal parenchyma. Hydronephrosis may also result in a large kidney.

- Identification of the major pathological process. In some cases it may be helpful to identify the general pathological process that the abnormality represents e.g. inflammatory or neoplastic (benign or malignant, primary or metastatic). This will be especially useful if you don't immediately know what the diagnosis is, at least you will be able to 'ball park' it. To do this it may be helpful to go through the surgical/pathological sieve.
- Identification of related lesions. By now you should have some idea of what you think the diagnosis, or at least the differential diagnosis, is. You should now think about what you know of this condition and look for, and describe, other relevant features that may confirm or refute this diagnosis. It may be useful to include relevant negatives.
- Other pathologies. Have a look at the rest of the specimen to see if there are any other abnormalities. If they are present, describe them. A few scattered small or even sometimes large simple cysts are common in the kidney.
- Diagnosis. State your diagnosis or differential diagnosis. Be as precise and specific as possible. Use any relevant clinical information given to help you. Sometimes a precise diagnosis is not possible but a presumptive diagnosis based on the macroscopic and/or clinical findings is. If you can't decide on one diagnosis, give a list of reasonable differential diagnoses, in order of decreasing likelihood, give a more general diagnosis (e.g. malignant tumour), or at least attempt to identify the pathological process.

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CORE AND CLASSIC DISEASE PROCESSES

KIDNEY: POLYCYSTIC KIDNEY

CASE 50287/83

Clinical information
The patient was a woman aged 62 who developed chronic renal failure in her fifties.

Describe the specimen
The specimen is an enlarged kidney in which the parenchyma has been destroyed by numerous cysts measuring up to 4.5cm in diameter. The cysts are thin-walled and filled with clear, turbid or brown fluid.

What is the diagnosis?
Polycystic kidney: probably adult polycystic kidney disease

What is adult polycystic kidney disease?
This is a disease arising from a genetic abnormality. Most cases are acquired as an autosomal dominant inherited disease, however, a small proportion of cases arise from spontaneous mutations. The cysts develop from dilated tubules that subsequently cause pressure atrophy of adjacent parenchyma.

What clinical problems does this disease cause?
Patients typically present in the third or fourth decade of life. They may develop hypertension, chronic renal failure, loin pain, haematuria, renal infections and/or stones.

What are the extra-renal manifestations of adult polycystic kidney disease?
Patients may also have intracranial berry/saccular aneurysms, colonic diverticula, mitral valve prolapse and (asymptomatic) cysts in the liver, spleen and pancreas.

Comment
There are a number of different cystic diseases of the kidney, including congenital ones and a number of other inherited ones that have different appearances. This is the commonest, apart from scattered small sporadic simple cysts that are very common.

KIDNEY: ACUTE PYELONEPHRITIS

CASE 50348/83

Clinical information
The patient was a man aged 72 with lymphoma. He developed septicaemia secondary to bronchopneumonia.

Describe the specimen
The specimen consists of the kidney that is slightly enlarged and demonstrates diffuse mottling from congestion. The renal pelvis is also congested.

What is the diagnosis?
Acute pyelonephritis

What is the normal route of spread of infection to the kidney and what organisms are normally responsible?
Most cases of acute pyelonephritis arise as a result of bacteria ascending from the lower urinary tract. They are therefore usually caused by G-ve bacteria from the gut.

What are the predisposing factors for acute pyelonephritis?
Predisposing factors for ascending infection from the bladder include

- urinary obstruction
- instrumentation
- vesicoureteric reflux (due to congenital anatomic anomaly usually in males)
- pregnancy
- immunocompromisation
- neurogenic bladder e.g. spinal cord injury, diabetes, multiple sclerosis
- renal stones

Predisposing factors for blood-borne infection

- bacteraemia (non-enteric organisms) or fungaemia, spreading from distant foci of infection (e.g. in the bone, skin, heart) usually in the immunocompromised patient

What clinical symptoms and signs may this patient have demonstrated?
Fever, malaise, loin pain and tenderness, haematuria, dysuria, cloudy urine, tachycardia

What are the potential complications of acute pyelonephritis?

- septicaemia and septic shock
- renal or perinephric abscess
- pyonephrosis
- acute papillary necrosis
- recurrent infections -> chronic pyelonephritis +/- chronic renal failure

Comment
Histology showed oedema and congestion with extensive but patchy infiltration of the parenchyma by neutrophils and numerous colonies of bacteria. Staphylococcus aureus was cultured from the kidney at post-mortem. The renal infection in this case will have been blood borne.

KIDNEY: ACTIVE CHRONIC PYELONEPHRITIS WITH PYONEPHROSIS AND HYDROURETERS

CASE 20508

Clinical information
The patient was a man aged 28 who died at the end of a 7-year illness resulting from an ependymoma of the cervical spinal cord, with paralysis progressing to quadriplegia. Recurrent urinary and chest infections complicated the quadriplegia and there were bedsores and osteoporosis.

Describe the specimen
The specimen consists of both kidneys, the ureters and the bladder. The bladder is dilated. Both ureters are dilated and tortuous. The kidneys measure 12 and 13cm in length and show dilatation of the pelvis and calyces that contain patchy purulent exudate. The surviving renal substance is congested and thinned and there are large depressed scars on the renal surfaces, more obvious in the larger kidney.

What is the diagnosis?
Active chronic pyelonephritis with pyonephrosis and hydroureters

Why has this patient developed these pathologies?
Interference with the nerve supply to the bladder in spinal cord injury leads to neurogenic bladder and chronic urinary retention. This predisposes to bacterial infection and reflux, an additional predisposing factor being the repeated urinary tract instrumentation that these patients often need. Reflux leads to dilatation of the ureters and renal pelvis. Urinary stasis and reflux also promote acute and chronic or recurrent renal infection, the former causing congestion and pus within the kidney, the latter causing chronic inflammation and scarring, typically causing large depressed cortical scars.

KIDNEY: RENAL TUBERCULOSIS WITH HYDRONEPHROSIS

CASE 21388

Clinical information

The patient was a man aged 33 who gave a history of "cystitis" 5 years previously. For the last 2 days there had been haematuria, frequency and dysuria. On examination there was an enlarged seminal vesicle and an irregular prostate. The urine contained many leucocytes and acid-fast bacilli. IVP showed calyceal dilatation and delay in excretion of the right kidney and a normal left kidney. He was treated for 2 years with streptomycin, PAS and INH, and the urine became sterile. However there was progressive deterioration in the function of the right kidney from pelvi-ureteric and uretero-vesical obstruction. The kidney and ureter were therefore resected. Recovery was uneventful.

Describe the specimen

The specimen of kidney measures 10cm in length. A portion of the upper ureter is attached. There is marked intrarenal hydronephrosis and there is thick caseous exudate in the dilated lower calyces. The renal substance has been totally destroyed in the mid and lower parts of the organ but a comparatively broad rim of renal tissue remains at the upper pole. The ureteric wall is focally thickened and the lumen dilated.

What is the diagnosis?

Renal tuberculosis with hydronephrosis

Comment

It is not possible to tell macroscopically that this is tuberculosis, as the exudate could also represent pus, but tuberculosis should be in the differential.

How did the tubercle bacilli gain access to the kidney in this case?

This patient may well have had a tuberculous prostatitis with involvement of a seminal vesicle. The organisms probably gained access to these organs via the blood from a pulmonary infection. The organisms could then have got into the bladder and ascended the ureters. Alternatively, they could have gained access to the kidneys by direct blood spread.

KIDNEY: OLD RENAL TUBERCULOSIS

CASE 18218

Clinical information

The patient was a hypertensive man aged 52 who was admitted with pulmonary oedema. He was known to have a non-functioning right kidney and on cystoscopy the right ureteric orifice could not be found. The pulmonary oedema responded to treatment and he was discharged, but he was readmitted 4 months later with a left hemiplegia involving face, arm and leg and the left 7th and 12th nerves. Carotid angiogram showed internal carotid occlusion. He slowly improved but two months later had a further episode involving a similar distribution. He died of bronchopneumonia 3 months later. At post-mortem there was an old myocardial infarct, extensive confluent bronchopneumonia and an acute abscess in the left upper lobe. A large cystic softening involving almost the whole of the right middle cerebral territory was seen in the brain, and the right internal carotid artery was occluded by thrombus. The left kidney was larger than normal. The right kidney was small.

Describe the specimen

The specimen is a slice of this kidney with the upper 10cm of the ureter. The kidney measures 7cm in length and shows old inspissated caseous material filling the calyces. The surrounding renal parenchyma has mostly been destroyed. The ureter is filled with old caseous material.

What is the diagnosis?

Renal tuberculosis (old)

Comment

The changes here are more readily diagnosed macroscopically as tuberculosis than those in the previous specimen.

KIDNEY: MULTIPLE RECENT INFARCTS

CASE 25281

Clinical information
The patient was a man aged 76 who died of congestive cardiac failure. He also had ulcerating atherosclerosis of the aorta.

Describe the specimen
The specimen consists of sections of both kidneys. The kidney on the left shows simple cortical infarctions that have pale centers and congested borders. A small rim of cortical tissue survives over the infarct beneath the capsule. There are also similar small infarcts in the kidney on the right. Single occluded small vessels are seen in the hilum on each side.

What is the diagnosis?
Multiple recent renal infarcts

Why is there congestion around the infarcts?
Congestion arises from dilatation of blood vessels that happens as part of the acute inflammatory response to dead tissue.

What type of necrosis occurs with renal infarction?
Coagulative

What is the most likely pathogenesis of these infarcts?
Multiple infarcts generally arise from embolic occlusion of arteries. In this case, it was probably athero-emboli from aortic atherosclerosis.

KIDNEY: BENIGN NEPHROSCLEROSIS

CASE 25232

Clinical information
The patient was a woman aged 88 who died following an intracerebral haemorrhage. It is not known whether she was previously hypertensive, however, on the final admission her BP was 180/100.

Describe the specimen
The specimen consists of a coronal slice of kidney that is slightly smaller than normal. The surface demonstrates fine granular scarring. The cut surface shows narrowing of the cortex. The pelvis and calyces appear normal.

What is the diagnosis?
Benign nephrosclerosis

What is benign nephrosclerosis and how does it arise?
Benign nephrosclerosis is a term given to kidneys that demonstrate a finely granular surface macroscopically. They are also variably smaller than normal. The granularity of the surface is due to small cortical scars that arise as a result of chronic ischaemia due to hyaline arteriolosclerosis related narrowing of renal arterioles. This causes tiny foci of ischaemic atrophy of tubules and glomeruli with chronic inflammation and scarring of the interstitium, with resultant macroscopic foci of depression in the cortex. Hyaline arteriolosclerosis and benign nephrosclerosis happen to some extent with age but are more marked in patients with systemic hypertension and diabetes.

What are sclerosed glomeruli?
Sclerosed (or obsolescent or hyalinized) glomeruli are small, shrunken, fibrotic non-functioning glomeruli. They are seen histologically as small rounded eosinophilic masses. They develop from chronic damage to the glomeruli of any cause (e.g. immunological, ischaemic, metabolic in diabetes)

KIDNEY: SMALL SCARRED KIDNEYS

CASE 11889

Clinical information
The patient was a man aged 65 who had exertional dyspnoea for 18 months accompanied by frequency of micturition during the day. The BP was 190/100, the haemoglobin low and creatinine elevated. He suffered a severe epistaxis and died of uraemia.

Describe the specimen
The specimen consists of coronal slices of both kidneys with attached arteries and aorta. Each kidney is grossly shrunken and measures only 7cm in length. Their surfaces are granular and the cut surface shows gross diminution of cortical thickness with loss of demarcation between cortex and medulla.

What is the diagnosis?
Small, scarred kidneys

What is its pathogenesis?
Chronic injury to any component of the kidney (e.g. immunological, metabolic or ischaemic damage to glomeruli, chronic inflammatory damage to tubules) ultimately results in damage to with fibrosis of other components also (e.g. glomerular damage will impair blood flow through the peritubular vascular system resulting in tubular atrophy and interstitial scarring, primary tubulointerstitial disease can ultimately result in glomerular scarring). Extensive atrophy and fibrosis results in the kidney becoming small and scarred.

What diseases may cause such changes and how do they do so?
- Immunological
  - Certain glomerulonephritides (e.g. membraneous nephritis) which primarily damage glomeruli via deposition of immune complexes
- Vascular
  - Systemic hypertension which exacerbates hyaline arteriolosclerosis causing chronic ischaemic damage
  - Renal artery stenosis causing chronic generalized renal ischaemia
  - Diabetes which exacerbates hyaline arteriolosclerosis (advanced glycosylation end product mechanism) causing chronic ischaemic damage
- Metabolic
  - Diabetes which damages glomeruli and tubules via thickening of basement membranes and mesangium and exacerbates hyaline arteriolosclerosis, causing chronic ischaemic damage to glomeruli
- Chronic tubulointerstitial nephritis e.g.
  - Chronic pyelonephritis from chronic/recurrent infection related inflammation and scarring in tubules and interstitium. Kidneys affected by chronic pyelonephritis typically have broad depressed cortical scars.
  - Certain drugs
  - Urate nephropathy

How will such changes affect renal function?

Such changes will cause progressive deterioration in renal function and ultimately chronic renal failure, as in this patient.

KIDNEY: RENAL CALCULUS AND HYDRONEPHROSIS

CASE 22621

Clinical information
The patient was a man aged 89 who was known to have renal calculi and chronic pyelonephritis for 10 years. He died 5 days after an overdose of Carbrital.

Describe the specimen
The specimen consists of a kidney measuring 15cm in length. There is great intrarenal dilatation of the pelvis and calyces. The renal substance is reduced to a thin rind, which is thickest on the upper and lateral aspect. A large staghorn calculus is wedged in the pelvis and upper ureter.

What is the diagnosis?
Renal calculus and hydronephrosis

What type of renal calculus is this and how does it develop?
This is a large staghorn calculus, typically composed of magnesium ammonium phosphate. These usually develop following infection by urea-splitting bacteria, e.g. Proteus, which convert urea to ammonia and thus make the urine alkaline, causing the precipitation of magnesium ammonium phosphate.

How has the hydronephrosis developed and why has the overlying parenchyma become thinned and atrophied?
The hydronephrosis has developed as a result of obstruction to urinary outflow at the renal pelvis. Backup of urine has caused dilatation of the calyces and pressure atrophy of the parenchyma. Stones and obstruction also predispose to infection, so scarring from chronic pyelonephritis may have contributed to the cortical thinning.

What are the main types of renal calculus and how does each form?
4 main types

- calcium: most common, usually a mix of Ca^{++} oxalate and Ca^{++} phosphate
    - most related to hypercalciuria without hypercalcaemia
    - others related to underlying disease causing hypercalcaemia (e.g. hyperparathyroidism, diffuse bone disease) or to hyperuricosuria, acting as a nucleus for the deposition of calcium, or to hyperoxaluria
- magnesium ammonium phosphate
    - largely follow infection by urea-splitting bacteria e.g. Proteus, which convert urea to ammonia and thus make the urine alkaline, causing the precipitation of magnesium ammonium phosphate
    - form some of the largest stones
- uric acid stones
    - common in patients with hyperuricaemia e.g. gout
    - others may have excessively acidic urine
- cysteine stones
    - from genetic defect in the renal reabsorption of amino acids including cysteine

Most stones are caused by the presence of an increased concentration of the stone's constituents within the urine to a level of super saturation. However, not all patients with

hypercalciuria, hyperoxaluria or hyperuricosuria form stones and there may be other precipitating factors or a deficiency of inhibitory factors.

What are the potential clinical effects and complications of renal calculi?

- may not cause any problems
- smaller stones may pass into the ureter and cause spasm → renal colic (severe pain) and ureteric obstruction
- gross or microscopic haematuria
- predispose to infection -> acute +/- chronic pyelonephritis
- hydronephrosis
- if bilateral, chronic pyelonephritis etc can -> chronic renal failure

KIDNEY: AMYLOIDOSIS

CASE 9934

Clinical information

The patient was a 41-year old woman with pulmonary tuberculosis for at least 3 years, treated at various sanitaria. She was transferred to the RAH because of the development of generalized oedema which did not respond to treatment. The BP was 90/65 and there was marked proteinuria. The serum creatinine was normal but albumin reduced. The liver function tests were normal except for slightly raised serum alkaline phosphatase. The sputum contained acid-fast bacilli and the chest x-ray showed cavitation affecting both lungs. She deteriorated gradually and died.

Describe the specimen

The specimen is of the kidneys and spleen that are slightly enlarged and have a brownish tinge to their cut surfaces. The spleen shows 'sago' change on the cut surface.

What is the diagnosis?

Amyloidosis

What is this condition and how does it arise?

- amyloid is a pathologic extracellular protein with a characteristic fibrillary ultrastructural appearance
- there are multiple biochemically distinct forms
    - AL/primary amyloid
        - associated with 'plasma cell dyscrasias' especially multiple myeloma
        - composed of immunoglobulin light chains, usually lambda type
        - usually deposits in multiple organs
    - AA/secondary amyloid
        - occurs in occasional cases of various systemic chronic inflammatory conditions and tumours e.g. inflammatory bowel disease, TB, rheumatoid arthritis, Hodgkin's disease, renal cell carcinoma
        - synthesized from serum amyloid associated protein (SAA) produced by the liver
        - usually deposits in multiple organs
    - CNS amyloid
        - A-beta protein derived from amyloid precursor protein (APP)
        - seen in senile plaques in Alzheimer's Disease and in vessel walls in Alzheimer's Disease and congophilic angiopathy
    - Other types include
        - senile cardiac amyloid (composed of transthyretin, or atrial natriuretic peptide)
        - endocrine amyloid: seen in medullary carcinoma of thyroid (calcitonin), other neuroendocrine tumours, also in islets of Langerhan's in type 2 diabetes
        - patients on long term haemodialysis (composed of beta-2-microglobulin)
- the protein differs depending on the cause/associated disease but its formation appears to be related to immunological mechanisms

- it deposits in basement membranes, vessel walls and connective tissue and is seen as amorphous eosinophilic material on light microscopy. Characteristically shows 'apple green birefringence' on a Congo red stain
- organs/sites of deposition depend on the underlying disease association, may be multiple organs or single organ. Function of organ will deteriorate with progressive amyloid deposition
- affected organs usually mildly enlarged, firm
- prognosis in patients with systemic amyloidosis is poor

What type of amyloid is this patient likely to have and why?
This patient probably has AA/secondary amyloid that has developed secondary to tuberculosis.

Why has this patient developed proteinuria and generalised oedema?
As well as causing chronic renal failure (which this patient reportedly did not have), renal amyloidosis can cause proteinuria and the nephrotic syndrome (which it appears this patient did have). Deposition of amyloid in the glomeruli can result in massive proteinuria with resultant hypoalbuminaemia and subsequent oedema.

What are the other main causes of the nephrotic syndrome?
- Diabetes mellitus
- Minimal change nephropathy (mainly in children)
- Membraneous nephropathy
- Several other glomerulonephritides

KIDNEY: RENAL CELL CARCINOMA

CASE 18074

Clinical information
The patient was a man aged 50 who had pain and a palpable mass in the flank.

Describe the specimen
The specimen consists of a slice of kidney. In the lower pole is a well-circumscribed, apparently encapsulated, oval tumour measuring 90mm in maximum dimension. It demonstrates a variegated cut surface with fibrous, yellow, haemorrhagic and cystic areas. The remaining renal substance appears essentially normal.

What is the diagnosis?
Renal cell carcinoma

Comment
This has the typical appearance of a renal cell carcinoma, the commonest renal malignancy. They are usually well circumscribed though not necessarily encapsulated and show a variegated cut surface, often with cystic change.
These tumours not uncommonly macroscopically invade the renal vein and tumour emboli end up in the lung, a common site for metastases. Bone is another common site of metastases.
These tumours are reknowned for causing a variety of paraneoplastic syndromes including polycythaemia (from the production of erythropoietin), hypertension (via the production of renin), hypercalcaemia (from the production of a PTH-like substance) and Cushing's syndrome (from the production of an ACTH-like substance).

KIDNEY: WILM'S TUMOUR

CASE 12732

Clinical information
The patient was a male child who at 3.5 years of age presented with haematuria, loss of appetite and a mass palpable in the left flank. He underwent left nephrectomy and died one year later despite post-operative radiotherapy.

Describe the specimen
The specimen consists of a bisected kidney that has been mounted inverted in the pot. The kidney has an enlarged lower pole where the architecture is distorted and replaced by a focally necrotic and haemorrhagic homogeneous tan coloured tumour measuring 6cm in maximum diameter. Tumour invades the renal pelvis and the upper calyces are dilated.

What is the diagnosis?
Wilm's tumour

Comment
This diagnosis cannot be made macroscopically but is the favoured diagnosis for a renal tumour in a young child.

KIDNEY: LEUKAEMIC INFILTRATION

CASE 14977

Clinical information

A boy of 16 developed acute leukaemia 4 months before death. At the onset he was hypertensive and uraemic, considered to be due to nephritis. The uraemia responded to treatment but the hypertension persisted. He was discharged on steroids and antimitotic drugs and remained reasonably well under outpatient supervision. Finally he developed persistent epistaxis and purpura and was admitted in coma. He died less than 24 hours later. At postmortem widespread purpuric haemorrhages were found and there was massive leukaemic infiltration of lymph nodes, liver, spleen and kidneys, and acute cerebellar haemorrhage.

Describe the specimen

The specimen consists of a coronal slice of a greatly enlarged kidney that measures 14cm in length and 9cm across. The surface is uniformly pale with widening of the cortex and medulla and reduced demarction between cortex and medulla. There is a patch of recent haemorrhage beneath the mucosa of the pelvis.

What is the diagnosis?

Leukaemic infiltration of the kidney

Comment

Leukaemic cells in the blood may be filtered out in various organs, the reticuloendothelial organs (spleen, liver, bone marrow, lymph nodes and other lymphoid tissues) in particular, but other organs such as kidney may also be involved.

KIDNEY: CARCINOMA OF THE RENAL PELVIS

CASE 13785

Clinical information
The patient was a woman who had intermittent haematuria for 12 months. Pyelogram showed a filling defect in the renal pelvis. The kidney was removed.

Describe the specimen
The specimen shows a coronal slice of kidney that measures 9cm in length. The renal substance appears essentially normal but there is a 2.5cm diameter papillary tumour consisting of delicate pale fronds filling the pelvis.

What is the diagnosis?
Carcinoma of the renal pelvis

What type of epithelium lines the renal pelvis and ureters?
Transitional

What histological type of tumour would you expect this to be?
Transitional cell carcinoma

Comment
Tumours of the renal pelvis and ureters are typically transitional cell carcinomas. Some are papillary, some are more solid. Haematuria is a typical presenting symptom.