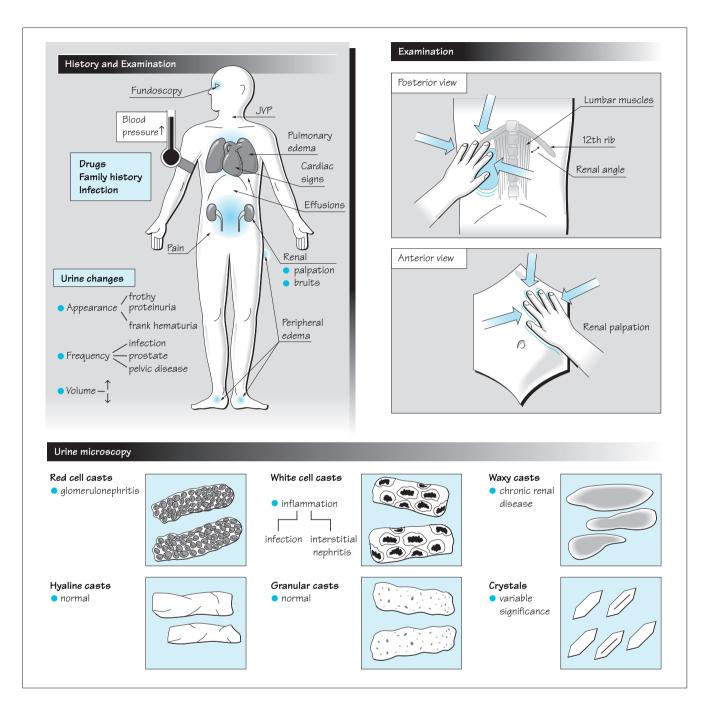




Clinical features of kidney disease



As there are more nephrons in each kidney than are needed to sustain life, significant renal damage can occur without obvious clinical effects. Kidney disease may not become clinically apparent until there is substantial loss of renal function. For this reason, slowly progressive renal diseases can be asymptomatic in the early stages.

History

Pain

Pain is uncommon with renal disease, but can occur if there is

urinary obstruction, especially from renal stones. Infection or distention of the renal capsule or of renal cysts, especially in polycystic kidney disease, can also cause pain. Inflammation of the bladder or urethra, usually as a result of infection, can cause dysuria (discomfort on micturition). Rarely, glomerular disease can cause a dull lumbar ache.

Urine appearance and volume

Proteinuria can produce frothy urine and frank hematuria is obvious as red or pink urine. Dark urine can also occur with

18 Introduction Clinical features of kidney disease



the myoglobinuria of rhabdomyolysis or the hemoglobinuria of hemolysis. Recurrent intermittent frank hematuria suggests immunoglobulin A (IgA) glomerulonephritis in young people or renal tract cancer in elderly people. Glomerular bleeding is present throughout the stream, whereas hematuria only at the beginning of the stream suggests urethral bleeding and hematuria only late in the stream suggests bladder or prostate bleeding.

Increased urinary frequency is an increase in the frequency of micturition. Polyuria is an increase in total urine volume. Increased *urinary frequency*, especially at night, can suggest prostatic enlargement in men or urinary tract infection. *Polyuria* suggests a defect of renal urine-concentrating mechanisms or excess water ingestion. Prostatic enlargement can also cause hesitancy and terminal dribbling as well as obstruction and urinary retention. Total *anuria* is rare and usually suggests urethral or bilateral ureteric obstruction, a severe rapidly progressive glomerulonephritis, or aortic or bilateral renal arterial occlusion.

General history

Always take a full history. Establish whether the patient has a previous history of hypertension, diabetes mellitus, malignancy, or other systemic diseases. Any recent infection, but typically a streptococcal throat infection, can trigger a post-infective glomerulonephritis. The drug history may indicate use of nephrotoxic drugs, especially analgesics or non-steroidal anti-inflammatory drugs. A family history of renal disease can suggest a hereditary disorder, especially polycystic kidney disease. Symptoms of itching, muscle cramps, anorexia, nausea, and even confusion are consistent with chronic renal impairment. Hemoptysis suggests a vasculitic disease, particularly Goodpasture's syndrome.

Examination

Carry out a full examination including blood pressure measurement, fundoscopy, examination for edema, and rectal and vaginal examinations where appropriate. Check for a distended bladder. Look for signs of systemic disease in all systems, especially neurological and rheumatological signs. Cardiac valve lesions raise the possibility of glomerulonephritis associated with infective endocarditis. Peripheral bruits or absent peripheral pulses indicate vascular disease and such patients are at risk of renal artery stenosis, which may result in renal artery bruits.

Kidneys

Enlarged kidneys may be palpable. The right kidney, which lies lower than the left because of the liver, is sometimes palpable when normal. To palpate the kidneys, place the right hand over the upper abdomen on the relevant side. On the same side, place the left hand with the fingers in the renal angle

formed by the lateral margin of the lumbar muscles and the twelfth rib. As the patient inspires, push the fingers of the left hand anteriorly several times. You will feel an enlarged kidney with the right hand as it moves down the abdominal cavity during inspiration and is pushed anteriorly by the fingers of your left hand.

Fluid status

It is important to determine whether the patient has an excess or a deficiency of body water. Useful physical signs to look for include peripheral pitting edema, detectable especially at the ankles and sacrum, signs of pulmonary edema, effusions, the jugular venous pulse pressure, and skin turgor. A cardiac gallop rhythm may suggest hypervolemia. A low blood pressure, especially with a postural drop (orthostatic hypertension), indicates hypovolemia.

Bedside investigation of urine

Dipstick test urine for hematuria, proteinuria, and glucosuria. Use a microscope, ideally with phase contrast, to examine fresh urine. If possible, centrifuge the urine and discard most of the supernatant to concentrate cells or casts.

Red cells. These can arise from anywhere in the urinary tract, but deformed (dysmorphic) red cells indicate glomerular bleeding.

White cells. These suggest inflammation, resulting from bacterial infection if they are polymorphonuclear cells or interstitial nephritis if they are eosinophils or lymphocytes.

Casts. These are cylindrical aggregates formed in the distal tubule or collecting ducts. Red cell casts indicate glomerular bleeding, usually due to glomerulonephritis. White cell casts suggest acute infection, usually bacterial. Hyaline casts and fine granular casts are normal findings. Hyaline casts are mainly protein and may be increased in proteinuria. Granular casts are also mainly protein. Fatty casts can occur in the nephrotic syndrome. Waxy casts are large and occur in dilated tubules in chronic renal failure.

Crystals. These may indicate a stone-forming tendency, but are not always of pathological significance because they can form after urine collection. Ideally, examine urine for crystals when it is fresh and at $37\,^{\circ}$ C.

Infectious agents. Nitrites and leukocyte esterases on dipstick analysis suggest infection. Take a midstream urine sample for microscopy and culture.

Proteinuria. Quantify any proteinuria with a spot urine protein: creatinine ratio or a 24-h urine collection.

Clinical features of kidney disease Introduction 19