Clinical assessment of renal disease

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Abstract

Kidney disease presents in a number of different ways and to a variety of practitioners. The presentation may be non-specific and a high index of suspicion is required to allow early detection and intervention. A systematic clinical assessment is vital to facilitate timely referral and appropriate management of renal disease. The history and examination should be tailored to the type of presentation, and will be dictated by the degree of chronicity of the disease process. In all cases the urine dipstick is a crucial part of the clinical examination.

This article presents an overview of the approach to evaluation and diagnosis in the patient with kidney disease. It is intended to be read in conjunction with the other articles in these chapters.

Keywords acute kidney injury; chronic kidney disease; intrinsic renal disease; post-renal; pre-renal; urinalysis

Introduction

Patients with kidney disease may present with hypertension, with impaired kidney function, with symptoms and signs of proteinuria or haematuria, or with features of a systemic disease that is known to involve the kidneys. Asymptomatic patients may also come to the attention of a clinician after the discovery of abnormalities in routine blood or urine tests, or on imaging of the renal tract. In this chapter we outline a practical approach to the assessment and diagnosis of kidney disease in both the acute and chronic settings.

History

A detailed history is crucial in order to identify the aetiology of kidney disease, as well as to determine the chronicity of the process. The focus of the history will depend on the initial mode of presentation but should always be targeted at forming a differential diagnosis, which in turn will inform initial investigations and management. It is useful to consider the three main clinical presentations that are encountered: acute kidney injury (AKI), sub-acute or intrinsic renal disease, and chronic kidney disease (CKD). For convenience we will discuss the history in these three scenarios separately, but it must be borne in mind that certain intrinsic renal diseases can present as AKI, and the presence of underlying CKD increases the risk of developing AKI, so in practice all aspects of the history are relevant to all cases.

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Acute kidney injury

AKI is identified by a rise in serum creatinine or oliguria. History in this context is directed at identifying recent precipitating factors, which may be categorized as pre-renal, renal or post-renal. The ***renal*** causes of AKI will be discussed in the intrinsic renal disease section below.

Pre-renal insults include sepsis, hypovolaemia, low cardiac output or a combination of these, leading to (relative) hypotension and subsequent renal hypoperfusion. A history of preceding infection, cardiac failure or diarrhoea and vomiting should be sought.

The drug history may reveal medications such as angiotensin-converting enzyme inhibitors and non-steroidal anti-inflammatory drugs (NSAIDs), which impair the ability of the kidneys to autoregulate and maintain glomerular filtration rate (GFR). Diuretic use will prevent the kidneys from concentrating urine appropriately, thereby exacerbating hypovolaemia. It is important to recognize that in patients with a history of undertreated hypertension, renal hypoperfusion can occur in the face of normotension.

Aminoglycosides are direct tubular toxins and can cause or exacerbate AKI, and penicillins can cause an acute interstitial nephritis (AIN). Proton pump inhibitors can also cause AIN, which has become a growing problem in recent years during which they have been prescribed with increasing frequency. Over-the-counter medications are often not recognized by patients as being relevant to their drug history, so specific questions should be asked about purchased NSAIDs and herbal remedies.

A history of recent intervention, or imaging with the use of iodinated contrast, should also be directly sought. Instrumentation of the aorta for procedures such as percutaneous coronary intervention (PCI) can lead to the showering of cholesterol emboli into the renal vasculature, causing AKI, which is often irreversible; by comparison, there is a good chance of recovery of function following contrast-induced nephropathy (CIN).

A history of crush injury, or prolonged immobility, for example after a drug overdose, raises the possibility of rhabdomyolysis. HIV, hepatitis B and hepatitis C can all cause kidney disease and therefore risk factors for acquisition of blood-borne viruses should be elicited. A history of bone pain may be due to undiagnosed myeloma. This disease can lead to AKI via a number of mechanisms, including cast nephropathy and hypercalcaemia.

Post-renal — absolute anuria is suggestive of obstruction and a history of preceding prostatic symptoms in men (hesitancy, poor stream, nocturia) is important in this situation. A history of gynaecological malignancy, radiotherapy or recent pelvic surgery in women raises the possibility of bilateral ureteric obstruction. In anuric patients, imaging of the renal tract should be a priority.

Subacute or intrinsic renal disease

Even in rapidly progressive glomerulonephritides, symptoms can be mild and non-specific. Fatigue, weight loss, nausea and reduced appetite are all relevant, and the time of onset of these symptoms may give some idea about the duration of the disease. Patients should be asked specifically about sinus problems, epistaxis, haemoptysis, rashes, and joint pains that may be associated with systemic vasculitides. Anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis, anti-GBM disease (Goodpasture's syndrome), lupus nephritis (class IV)

and the crescentic form of immunoglobulin A (IgA) nephropathy can all cause an acute necrotizing glomerulonephritis, which leads to a rapid deterioration in renal function and requires urgent nephrological assessment and treatment to prevent irreversible damage. The history is key in identifying these conditions. Upper and lower respiratory tract involvement is seen in ANCA-associated vasculitis, which may be manifest as epistaxis or haemoptysis secondary to pulmonary haemorrhage. Anti-GBM disease affects the kidneys and lower respiratory tract only (Goodpasture's syndrome). Lupus nephritis occurs in up to 50% of patients with systemic lupus erythematosus (SLE)[1] and can be the presenting feature of the disease, but in many cases other systemic manifestations such as arthralgia, rash, hair loss, mouth ulcers, previous miscarriages or venous thromboses will predate overt nephritis.

Fevers and night sweats can occur in vasculitis, but also raise the possibility of an infectious aetiology such as TB or bacterial endocarditis. A history of sore throat or skin infection 2–3 weeks before developing renal impairment is suggestive of post-streptococcal GN, whereas visible haematuria associated with IgA nephropathy classically occurs within a few days of a URTI (synpharyngitic). The presence of a purpuric rash on extensor surfaces of the legs and buttocks, along with arthralgia, abdominal pain and dipstick haematoproteinuria, is highly suggestive of Henoch–Schönlein purpura (HSP).

Oedema may be the only presenting feature of nephrotic syndrome, though some patients notice that their urine has become 'frothy' due to heavy proteinuria. Occasionally venous thromboembolism may be the presenting feature of this syndrome, so it should be considered in patients presenting with unexplained pulmonary embolism or deep vein thrombosis.

Chronic kidney disease

Diabetes mellitus and hypertension are the most common causes of CKD in the UK. However, because these conditions are common, they may co-exist with other causes of CKD. It is essential to ascertain the underlying burden of disease, which is a function of the duration and adequacy of control of these conditions. Both hypertension and diabetes can be present for many years before diagnosis and it is crucial to ask about complications of disease in other organs (e.g. diabetic retinopathy, including the need for laser therapy, symptoms of peripheral or autonomic neuropathy, vascular disease) in order to assess the likelihood of associated nephropathy. Whatever the primary cause of renal disease, optimal control of blood glucose and blood pressure is a key factor in reducing the rate of progression.

A history of vascular disease, in particular peripheral vascular disease, raises the possibility of associated renovascular disease. Frequent childhood urinary tract infection (UTI) or prolonged nocturnal enuresis suggests reflux nephropathy.

The presence of a systemic disease such as SLE, myeloma, sarcoidosis or scleroderma in a patient with CKD naturally raises the possibility of secondary renal involvement. However, it is also important to consider whether any medications used to treat the systemic disease could be nephrotoxic.

A family history of hypertension, diabetes, or inherited primary renal diseases must be elicited. In patients with a family history of autosomal dominant polycystic kidney disease (ADPKD), one should ask about the progression of disease in their relatives, and any family history of sudden death or intracerebral bleed. In patients with a family history of renal disease who do not know the diagnosis, the pattern of inheritance may be informative (e.g. X-linked inheritance of Alport's disease).

Symptoms attributable to CKD itself – 'uraemic symptoms', such as fatigue, nausea, vomiting, itching, weight loss, hiccups and altered taste – occur late and do not tend to manifest until GFR has fallen below 15 ml/minute (stage 5).

Examination

A detailed history will have provided clues as to the underlying cause of renal disease, and a thorough examination will allow the clinician to prioritize the investigations and management appropriately.

Acute kidney injury

In the context of AKI, adequate assessment of the circulation is key, in order to identify whether or not there has been a significant pre-renal insult. Volume status should be examined by assessing the jugular venous pressure (JVP), systemic blood pressure (including postural blood pressure) and peripheral circulation. Skin turgor and mucous membrane appearance are notoriously unreliable, especially in the older patient. Blood pressure should be considered in the context of the patient's usual blood pressure, if known, and the JVP should always be interpreted with caution in patients with a history of lung disease, who may have impaired right ventricular function. If the patient has postural hypotension, or is in frank circulatory shock, assessment of the peripheral circulation, including pulse volume, temperature of the peripheries and capillary refill will indicate the underlying pathology. Warm, dilated peripheries with a high-volume pulse and tachycardia suggest the high cardiac output state of sepsis. Cold, shut-down peripheries with a low-volume pulse indicate reduced cardiac output. This may be due to hypovolaemia (in which case the JVP will be low, and there will be no pulmonary oedema), or to impaired cardiac function (in which the JVP may be elevated, the apex beat may be displaced, and there may be a third heart sound, peripheral oedema, and basal crackles on auscultation of the lungs).

In the absence of any signs of pre-renal injury, other findings that could indicate the cause of AKI must be sought. Hypertension along with salt and water overload may be secondary to an acute nephritis. Accelerated-phase hypertension can itself be a cause of AKI, and ophthalmoscopy may reveal hypertensive retinopathy with flame haemorrhages (Grade 3) or even papilloedema (Grade 4).

The skin should be carefully examined for maculopapular erythematous rashes associated with drug reactions, or the cutaneous manifestation of a systemic vasculitis. Patients should be examined for systemic signs of bacterial endocarditis such as splinter haemorrhages, Roth spots and a new or evolving heart murmur. Evidence of cholesterol embolization in the feet, or 'trash feet', suggests that the same process may be occurring in the kidneys.

Extensive bruising or a tense and tender muscle compartment may be found in rhabdomyolysis. The presence of compartment syndrome should prompt urgent referral to an orthopaedic

