Assessment of the Renal Patient

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Nephrologists encounter a spectrum of renal disease, from asymptomatic incidental findings to newly diagnosed renal impairment to life-threatening metabolic disturbance in a critically ill patient. Many of these patients will have significant comorbidity, often alongside previously diagnosed chronic renal disease. These factors mean the assessment of the 'renal patient' can be a challenging proposition. There are numerous approaches to the assessment of patients with kidney disease, and practitioners will develop their own style with time. The 'practical' strategy we outline here is only one of many but one that we find successful in day-to-day practice.

In this chapter we approach the assessment of the renal patient with a series of such questions. This strategy is outlined in Fig. 1.1. Unsurprisingly, as with any area of acute medicine, the first priority will be to ask if the patient is safe. This question provides the starting point of this chapter.

After addressing any life-threatening emergency, the usual next question in any renal patient will be to ask: What is this patient's intravascular volume status? An accurate assessment of this is absolutely central to both the diagnosis and management of patients with kidney disease. Assessment of fluid balance is usually based on multiple sources of clinical information, and we discuss the utility of these in the second section of this chapter. However assessing fluid balance is primarily a practical skill, so although tips and guidance can be provided in a textbook, this can be no substitute for repeated practice.

Alongside the assessment of intravascular volume status, the other critical step will be to clarify is this patient already being managed for an underlying kidney condition. The approach to the patient with established kidney disease, for example, a patient receiving dialysis or being treated for an inflammatory glomerulonephritis, will be completely different from the approach to the patient who presents with a renal disorder for the first time. In these cases where the patient is known to a renal service, the assessment will be focused on optimising the management of the underlying condition and addressing associated complications. This aspect of the assessment of the kidney patient is addressed in the last section of the chapter.

If the current presentation is the patient's first consultation with a renal specialist, the primary purpose of the assessment will be to establish the underlying cause of the renal disorder. This is a clinical scenario in which there is often diagnostic uncertainty, representing some of the most interesting and sometimes challenging areas of the nephrology practice. This aspect of the clinical assessment comprises the majority of this chapter. In this section the approach to different renal syndromes is outlined.

Urgent Assessment for Renal Emergencies

As with any acutely unwell patient, the first priority must be to assess and correct potentially life-threatening physiological dysfunction. Alongside the maintenance of an adequate airway, ventilation and circulation, urgent renal-specific issues are likely to relate to assessment and treatment of either metabolic disturbance, most often hyperkalaemia, acidaemia, or fluid overload. Any of these scenarios may be and indication for urgent renal replacement therapy (RRT) if

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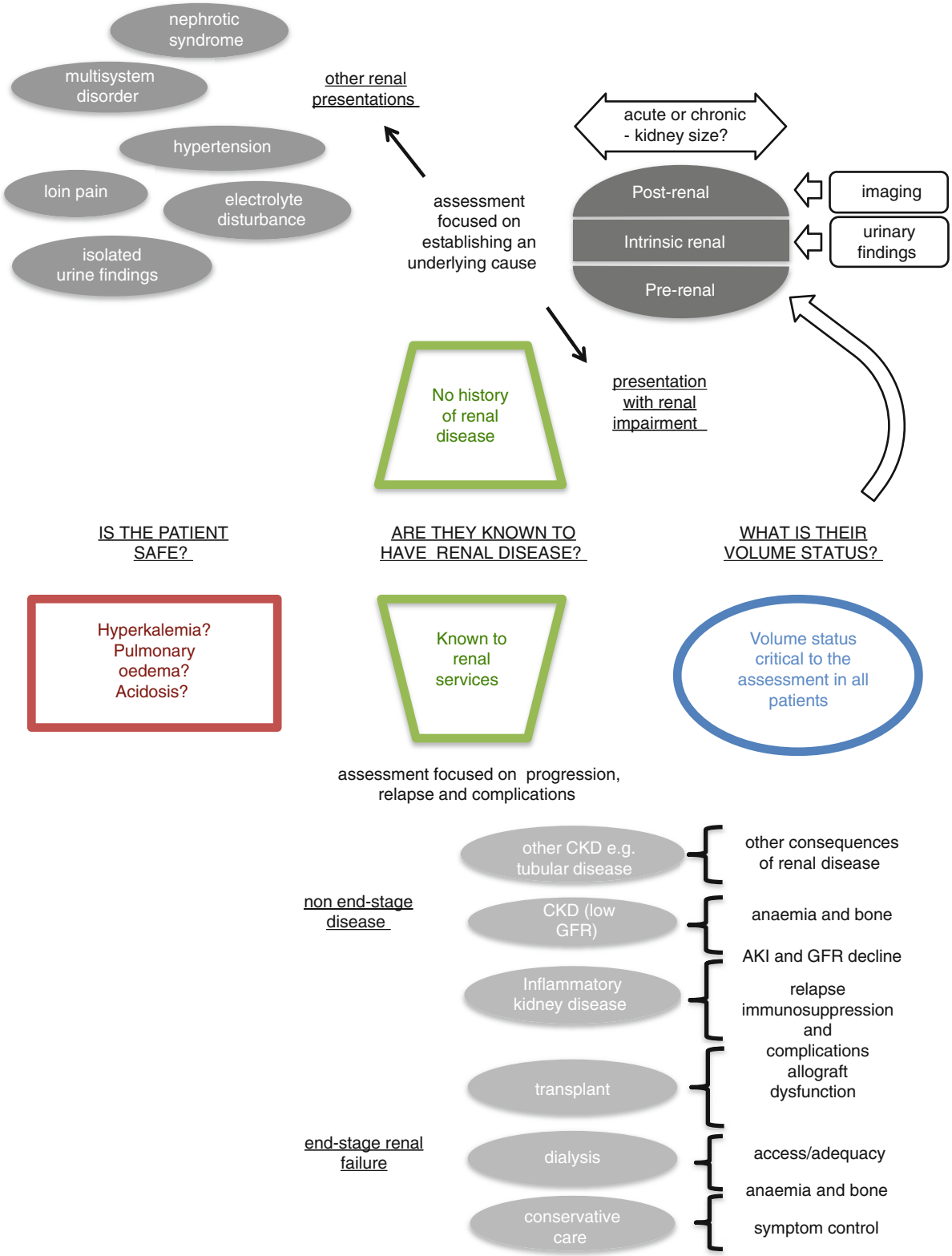

Fig. 1.1 An approach to the assessment of the 'renal patient'. Our approach is initially based on asking three questions. The first priority is always to ensure any life-threatening clinical situation is addressed. Establishing whether a patient is known to renal services is always important. The assessment of a patient with a known renal problem will be focused on progression and complications of their condition. Conversely the work-up will be will be concentrated on making a diagnosis if the patient has no known renal history. Classification of a new presentation into one of the many 'renal syndromes' e.g. acute pre-renal impairment or nephrotic syndrome, will mean further assessment can be appropriately targeted. In parallel, the patient's intravascular volume status will likely be relevant to almost every 'renal patient' whether in the evaluation of potential pre-renal acute kidney injury, management of oedema and hypertension in a patient with nephrotic syndrome or to establish the cause of intradialytic hypotension in a patient receiving outpatient haemodialysis

not responsive to medical manoeuvres. Furthermore uraemic encepholapathy, pericarditis are likely to be absolute indications for instituting dialysis (see Chaps. 7, 9 and 10 on acute renal replacement, acid-base and electrolyte disturbances, respectively).

When there is an indication for urgent renal replacement therapy (RRT) attention must be given to the most appropriate form of dialysis or haemofiltration. Requirements will depend on the need for other organ support, but the use of an extracorporeal circuit, even with the low blood flows used in continuous therapies such as haemofiltration, risks haemodynamic instability. Therefore adequate, usually invasive, monitoring and access to vasopressors or inotropes are mandatory in cardiovascularly unstable patients receiving RRT. Critically, immediate management often depends on whether the acidaemic or hyperkalaemic patient has a remediable medical condition; a patient with urinary retention and potassium of 7.2 mmol/L may respond rapidly to medical therapy following relief of obstruction, whereas a septic oliguric hypotensive patient with a potassium of 5.8 mmol/L is much more likely to need renal replacement in the coming hours.

Assessment of Intravascular Volume Status

The evaluation of fluid status is critical in the assessment and management of all renal patients.

Table 1.1 Clinical assessment of fluid status

History	Paroxysmal nocturnal dyspnoea, orthopnoea, increased weight and worsening oedema are relatively sensitive and fairly specific symptoms of fluid overload. Thirst is a relatively sensitive marker of dehydration or salt overload. A history of significant fluid loss or reduced fluid intake may contribute to the overall assessment of fluid balance
Examination	
Pulse	Tachycardia is a non-specific marker of intravascular volume depletion and may be associated with excessive intravascular volume in the context of heart failure
Blood pressure	Relative hypotension (in comparison with historical blood pressure) and episodes of documented hypotension (intraoperative, hospital or community based) are always significant findings in patients with renal dysfunction. Although trends may be informative, hypotension alone is a non-specific marker of intravascular volume depletion
Orthostatic changes	Changes in blood pressure with changes in posture are useful findings. Changes of 20 mmHg in systolic and 10 mmHg in diastolic blood pressure upon change in posture are widely used as significant thresholds. Reflex tachycardia (increase in 30 beats per minute or more) is non-specific and sensitive for acute large blood loss but insensitive for smaller bleeds or other causes of hypovolaemia
Peripheral temperature	Cool nose, hands or feet at room temperature imply either decreased intravascular volume (low JVP) or cardiac failure (raised JVP). These are not sensitive but easy and reproducible. Unhelpful in patients with peripheral vascular disease or vasodilated patients (sepsis, cirrhosis, thyrotoxicosis)
Jugular venous pressure (JVP)	Operator dependent and sensitivity highly influenced by body habitus. Raised JVP may represent either increased intravascular volume or high right ventricular filling pressure (cardiac failure, pulmonary hypertension, tricuspid regurgitation or stenosis, restrictive defects, tamponade). Common for ESRD patients with previous central venous catheters to have internal jugular stenosis or occlusion (including secondary to current line) or superior vena cava obstruction
Oedema	Diurnal variation (feet swollen in the evening, face in the morning) usually rules out any anatomical cause. A bed-bound patient may have normal ankles but retain substantial salt and water in the sacral or flank oedema. Oedema will also be influenced by drugs and plasma oncotic pressure
Third heart sound (S3 gallop rhythm)	Insensitive indicator of ventricular failure/overload
Ascites and pleural effusions	Non-specific and poorly sensitive
Weight	Extremely useful serial measurement for general nephrology patients as well as those on dialysis with a ‘dry weight’. Serial measurements for inpatients are extremely valuable measures of total body water (but not necessarily intravascular volume)
Urine output	Non-specific but important part of AKI classification and relatively a sensitive marker of intravascular volume. Unhelpful as a marker of intravascular volume if coexistent AKI or concentrating defect from an alternative cause, e.g. post-obstructive diuresis
Documentation of inputs and outputs	Anaesthetic charts, ward charts including drain losses and stool charts can be invaluable if accurate
Urine specific gravity	Can be useful but acutely or chronically injured kidneys lose the ability to concentrate urine normally and therefore has limited value in patients with renal disease
Urinary sodium	A low spot urinary sodium or fractional excretion of sodium is indicative of reduced renal perfusion and can be helpful in identifying intravascular volume depletion or hepatorenal syndrome but is invalid in the face of acute tubular injury, diuretics or dopamine

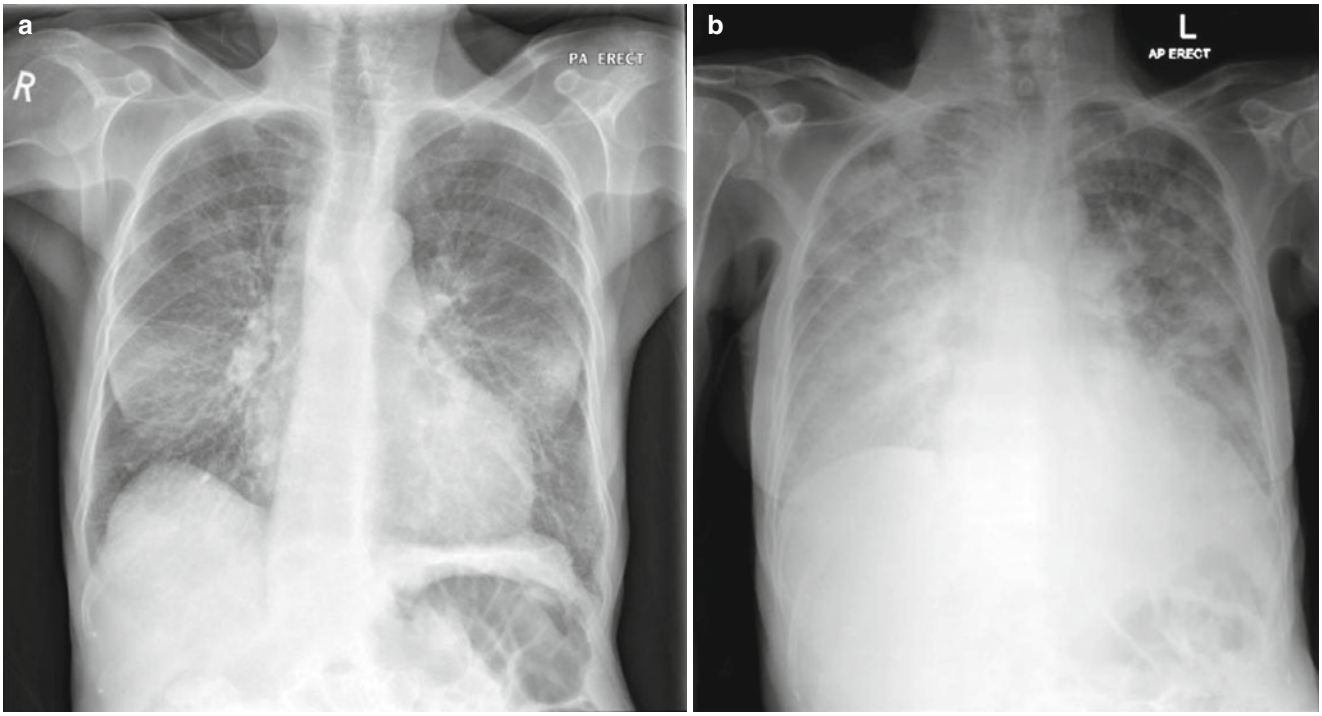

Fig. 1.2 (**a**) CXR of a patient with raised left ventricular end-diastolic pressure (LVEDP) showing upper lobe blood diversion, fluid in the fissure and Kerley B lines. This approximately equates to an LVEDP of 18 mmHg or above. (**b**) Frank pulmonary oedema in the same patient within 24 h in the absence of any filling or cardiac event but in the presence of sepsis. This demonstrates that pulmonary oedema on a CXR is not necessarily indicative of excessive filling pressures per se

Table 1.1 shows some of the clinical parameters that have merit in assessing fluid balance. Some such as serial measurement of weight and orthostatic blood pressure should be incorporated into routine ward observations even on non-renal wards. Where appropriate patients such as those discharged home with recovering AKI or nephrotic syndrome may also benefit from monitoring their own weight and postural blood pressure to help inform changes in fluid status. Other signs such as peripheral temperature and observation of central veins are often useful, whereas skin turgor and dryness of mucous membranes are on the whole poor, insensitive and non-specific clinical tools.

Investigations may also be helpful in assessment of intravascular volume status. The chest radiograph can confirm fluid overload, but is not particularly sensitive for mild/moderate overload, and acute lung injury can occur without an increase of right sided filling pressure (see Fig. 1.2a, b). In one study using pulmonary artery measurements, where fluid overload was defined as a pulmonary artery occlusion pressure ≥18 mmHg, radiologists achieved a diagnostic sensitivity of 55–65 %. This improved to 70 % by including cardiothoracic ratio (CTR) >0.55 and a vascular pedicle width (VPW) (the horizontal distance between the point where the superior vena cava crosses the right main bronchus and the origin of the left subclavian artery) measurement of >70 mm [1]. Therefore in practical terms a single chest-x-ray is unlikely to be useful, it may be a valuable tool when serial measurements are used.

In addition, there are also near-patient tests that may become more commonly utilised. Bioimpedance is one such technique and is an inexpensive and non-invasive technique to estimate body water. Given the difficulties with the clinical assessment of fluid status, the possibility of an easily usable objective measure is attractive. Unfortunately as with many of the clinical parameters guiding assessment of fluid balance, a single measurement in an individual patient does appear to add significant value to clinical assessment. Serial measures may again be more useful, so this technique may be most appropriate in patients regularly attending dialysis or the outpatient department rather than in the acute situation [2].

Biochemical measures that have been advocated as indices of fluid status such as a disproportionately high urea:creatinine ratio, raised serum urate or elevated serum lactate also lack sensitivity and specificity but may be helpful as part of a constellation of features associated with intravascular volume depletion.

A number of methods using continuous monitoring and aimed at detecting changes in cardiac output in response to

altered venous return (e.g. using respiratory cycle variation, passive leg raising or a fluid challenge) are useful in experienced hands. Importantly however, measures of fluid-responsiveness are likely to reflect a number of variables rather than just intravascular volume, so should always be considered as part of a more comprehensive assessment. Finally a variety of invasive cardiovascular measurements can be used to more accurately assess fluid status where sophisticated monitoring is available and are reviewed comprehensively elsewhere [3]. There remains significant debate as to the advantages and disadvantages of the various methods of invasive monitoring, and the knowledge base in this area of critical care is continuously evolving.

Assessment of the Patient When the Renal Disorder Has Yet to Be Identified and/or Characterised

This next section of this chapter discusses the approach to the patient when they present for the first time with a renal disorder to the reviewing nephrologist. In these cases a diagnosis needs to be established. Although renal physicians are faced with an enviable variety of clinical scenarios, these can usually be grouped into a number of renal 'syndromes'. These renal syndromes although not diagnoses in themselves are useful descriptions to help direct further investigation and to facilitate communication between physicians. An initial approach to these renal syndromes is outlined below. Following this the more general aspects of the history and the clinical examination, which might be helpful in establishing the cause of a renal disorder in patients presenting with any one of these renal syndromes is presented.

Presentation with Renal Impairment

An impaired kidney function, as identified by an increase in the serum creatinine, is the commonest reason for referral to a nephrologist. Patients may first present with symptoms directly related to renal dysfunction (Table 1.2). Often however, the presence of renal impairment will be identified by blood tests performed following a clinical presentation unrelated to the kidney.

Does the Patient Have Acute or Chronic Renal Injury?

The urgency of further investigation in a patient with renal impairment is critically dependent on whether the identified kidney dysfunction is acute (occurring over days to weeks) or chronic (occurring over months to years). Differentiation of these clinical scenarios is straightforward if the patient has been historically diagnosed with CKD, if previous blood tests are available or if the patient is aware of previous episodes of AKI or CKD. The distinction can be all the more difficult in cases of acute on chronic renal dysfunction where only knowledge of baseline creatinine figures will uncover whether there is an element of acute deterioration in GFR. Suggestive evidence may come from a likely acute precipitating event, the long-standing presence of a risk factor or a childhood history raising the possibility of previously undiagnosed renal dysplasia. Often, however, these clues are not immediately to hand, so other evidence may needed to judge the likely time course of the presenting renal dysfunction.

The duration of symptoms can be a useful indicator of chronicity; however, the high prevalence of subclinical advanced renal impairment means that in many cases the underlying disorder may have been present for some time before the patient reports the onset of illness. Furthermore the presence of risk factors or a systemic disorder known to put the patient at risk of chronic kidney disease (see below) does not provide definite evidence for pre-existing renal dysfunction. The single best markers of long-standing (and consequently irreversible) renal impairment are radiological measurements. Bipolar kidney length <10 cm in combination with an increase in cortical echogenicity (defined as a higher pixel density in the renal cortex than the liver) are the best predictors of advanced chronic kidney disease [4]. Exceptions to this rule occur where the size of chronically diseased kidneys is preserved, most typically in diabetes but also cystic diseases such as APCKD, protein deposition diseases such as amyloid, chronic obstruction, xanthomatous pyelonephritis and HIV nephropathy.

Table 1.2 Symptoms attributable to renal dysfunction

Uraemia	Anorexia, nausea, pruritus, malaise and sleep disturbance are all common symptoms in ESRD. Patients may also complain of headaches, reduced mental agility, chest pain (pericarditis), prolonged bleeding or bruising
Anaemia	Lethargy, palpitations, shortness of breath
Electrolyte disturbance	Palpitations, musculoskeletal pain, cramps, restless limbs, seizures, confusion
Fluid maldistribution or excess	Facial or peripheral swelling, shortness of breath or reduced exercise tolerance, orthopnoea, paroxysmal nocturnal dyspnoea
Urinary symptoms	Oliguria, nocturia (a common symptom in CKD)

Biochemical markers such as potassium, calcium, phosphate, parathyroid hormone and haemoglobin have been described in textbooks as indicators of chronicity of renal dysfunction. However, these parameters have limited utility in practice since all can also change rapidly in the context of AKI.

Diagnostic Breakdown of Acute or Chronic Renal Dysfunction

Assessment of a patient with AKI is described in detail in Chap. 5; however, the classical subdivision into prerenal, intrinsic and postrenal causes of impairment is a useful approach in any patient presenting with either acute or chronic renal impairment, so it will also be outlined here.

Prerenal

A 'prerenal' aetiology suggests hypoperfusion of the kidney. This is a critical component of the assessment of patients with AKI as this form of insult is usually associated with an acute presentation. However, renal artery stenosis and chronic hypovolaemia in patients with high output gastrointestinal stomas may develop more insidiously and present as CKD. Therefore, potential precipitants of prerenal injury should be sought in all patients with renal dysfunction.

Circulatory shock, whether vasodilatory, cardiogenic or hypovolaemic, will invariably lead to an acute prerenal injury (even if it is subclinical) and should be straightforward to identify. Other more subtle forms of prerenal kidney injury (such as those due to a less clinically apparent degree of intravascular volume depletion) may be less obvious initially but remain important considerations. The potential precipitants of prerenal injury are outlined in Table 1.3.

Postrenal Causes

Postrenal causes of renal impairment are particularly important to detect since the associated renal injury often reversible with relief of the obstruction. Furthermore, obstruction is a common cause for unexpected deterioration in patients with established kidney disease.

Although the clinical history or examination (Table 1.4) may suggest an obstructive cause of kidney injury, the exclusion of a postrenal cause of renal impairment cannot be made without radiological investigation. A renal ultrasound is often the first-line investigation. There are important limitations which should be considered when interpreting evidence of obstruction on renal ultrasound:

- 'False positives' can occur in cases of chronic dilation of the pelvi-calyceal system. Functional imaging should be pursued in attempting to establish the significance of morphological abnormality where kidney function is adequate for these tests to be useful. Renograms with the addition of diuretics using nuclear medicine tracers can be helpful in this context (discussed in Chap. 38).
- 'False negatives' can occur in anuric patients who may not have pelvi-calyceal dilatation. If the history is suggestive, more detailed anatomical imaging such as MRI or CT may be helpful. Occasionally in these cases a diagnostic trial of relief of obstruction should be considered.

Symptomatic changes in urine volume are not usually diagnostically helpful. Patients with significant obstruction usually notice no change in their urine volume. Clinical presentations and past history that might suggest a postrenal cause of kidney dysfunction are shown in Table 1.4.

Table 1.3 Assessment of potential precipitants of prerenal kidney injury

Relative hypotension, i.e. drop in BP for whatever cause in normally hypertensive patient	
Cardiogenic shock	
Intravascular volume depletion	
Blood loss	
Poor oral intake	Consider the confused patient or the patient without access to fluids
Fever, sweating	
Vomiting, diarrhoea	Patients with short bowel syndrome or high output stomas are at particular risk of volume depletion
Polyuria	Prescribed or non-prescribed diuretics, mannitol
	Central polyuria – salt-losing nephropathy, diabetes insipidus
	Tubular dysfunction – Addison's disease, nephrogenic DI, lithium toxicity, salt-losing nephropathies, hyperglycaemia, diuresis associated with recovery of kidney function
Fluid redistribution with reduced arterial perfusion	
Ascites	Liver disease
Fluid accumulation in the GI tract	Bowel obstruction, post-op ileus
Oedema secondary to nephrotic syndrome	
Septic/anaphylactic shock	
Local hypoperfusion	
Arterial occlusion	Emboli, most commonly following endovascular intervention; arterial dissection; malignant infiltration
Renal artery stenosis	May be suggested by 'crash' pulmonary oedema, profound reduction in GFR post-renin-angiotensin blockade
Venous obstruction	Renal vein thrombosis, consider in association with a procoagulant state (nephrotic syndrome); Page kidney following kidney biopsy or trauma

NB Patient notes (including anaesthetic records) are invaluable for identifying episodes of relative hypotension, poor IV or oral intake, excessive losses or weight changes. The better the documentation, the more accurate the interpretation is likely to be

Table 1.4 Symptoms, findings and clinical history suggestive of urinary tract obstruction or conditions associated with obstruction

Anuria	Suggests bilateral ureteric or bladder outlet obstruction. Beware of nonobstructive causes
Symptoms of bladder outlet obstruction	Urinary frequency, dysuria, poor flow, nocturia, urgency, double micturition, hesitancy, post-micturition dribbling, incontinence (overflow obstruction), sensation of incomplete emptying
NB Acute urinary retention can be painless with a neuropathic bladder	
Spraying on micturition	Urethral stricture, phimosis or paraphimosis
Loin pain	Any cause of obstruction
	On micturition (suggestive of vesicoureteric reflux)
	On excessive drinking (suggestive of PUJ obstruction)
Visible haematuria	Nephrolithiasis, malignancy, papillary necrosis
Medications	Anticholinergics, withdrawal of alpha blockers
Disseminated or pelvic malignancy	
Iatrogenic obstruction	Pelvic surgery or radiation therapy
Nephrolithiasis	
Pregnancy	Collecting systems can be dilated without functional obstruction
Childhood UTI or enuresis	May suggest congenital abnormalities of the urinary tract
Schistosomiasis infection	

Table 1.5 Simple classification and diagnostic clues to intrinsic renal causes of renal dysfunction

Toxins: endogenous	Any history to suggest rhabdomyolysis, intravascular haemolysis, tumour lysis syndrome, multiple myeloma, enteric hyperoxalosis, cholesterol emboli, hypercalcaemia
Toxins: exogenous	Drugs prescribed or illicit, herbal remedies, poisons
	Sepsis, snake bite, IV contrast
Glomerular injury	Features to suggest nephritic (or nephrotic) syndrome, haematuria or cola-coloured urine, recent or current infections, constitutional symptoms consistent with systemic inflammatory or autoimmune disease or malignancy, e.g. fevers, weight loss, ENT symptoms, red eyes, alopecia, rashes, haemoptysis, pleurisy, arthralgia, oedema. Bruising or bleeding in TMA such as HUS
Primary tubulointerstitial disease	Autoimmune, infiltrative, e.g. lymphoma, or inflammatory, e.g. TB or sarcoid
Any cause of unresolved prerenal injury	

Intrinsic Renal

Intrinsic renal causes of AKI and CKD are numerous but can be grouped into broad categories for the purpose of initial assessment (Table 1.5). Other chapters in the rest of this textbook are devoted to the assessment and management of each of these conditions.

Examination of the urine is absolutely critical in establishing the diagnosis of intrinsic renal injury, and the role of urinalysis and urine microscopy is discussed in detail in Chap. 2.

Presentations Other than with Biochemical Evidence of Change in Renal Function

Although the majority of patients referred for the first time to nephrologists will have a degree of kidney dysfunction, not all patients will present with renal failure. Patients with isolated urinary abnormalities are often sent for expert review, and the assessment of isolated haematuria and proteinuria is discussed in detail in the following chapter. In addition to urinary abnormalities, nephrologists will find themselves faced with a diverse range of clinical syndromes secondary to disorders of the kidney. Similarly these syndromes and the logical diagnostic approaches are discussed in detail in the chapters devoted to the presenting syndrome or the underlying diagnoses. However an introduction to these presentations alongside important diagnostic clues is discussed briefly below.

Haematuria

Haematuria can originate from anywhere along the urinary tract, so it is important to identify coexistent lower urinary tract symptoms in any patient with visible blood in the urine. Intermittent visible haematuria can be temporarily associated with an upper respiratory tract infection (suggestive of IgA nephropathy) or related to exercise. Periodic haematuria can be seen in patients with endometriosis. Finally myoglobinuria will often lead to a false-positive dipstick urinalysis for haematuria.

Proteinuria

Proteinuria is identified either on dipstick testing, during routine medical, as part of a surveillance programme (e.g. for

diabetes), or for the investigation of associated oedema, but patients with nephrotic range proteinuria can on occasion present having noticed frothy urine. History of past episodes of dipstick-positive proteinuria is helpful but is rarely recalled by patients.

Nephrotic Syndrome

The presentation of nephrotic syndrome is usually unambiguous. Diagnostic difficulties can occur in the presence of anuria, advanced renal failure or in the context of other causes for hypoalbuminaemia. For patients with nephrotic syndrome, swelling of the face or 'puffy eyes' in the morning can be an early symptom, pedal oedema and frothy urine may be noted, and fatigue or tiredness are also common complaints. For some individuals nephrotic syndrome may be precipitated by vaccinations, insect bites or non-specific infections. The definition, investigation and initial management of nephrotic syndrome are covered in Chap. 12.

Hypertension

Uncontrolled or unexpected hypertension is a fairly common referral to the nephrologist. Beyond emergency care, and determining duration, severity and compliance, the main consideration is the degree to which an underlying cause should be sought (Table 1.6). A secondary cause is more likely if hypertension occurs before the age of 40 years (the younger the patient, the greater the likelihood of a secondary cause), there is a history of severe end-organ damage, it presents as accelerated hypertension or there is a family history of early hypertension/stroke. It is important to recognise that the commonest cause of secondary hypertension relates to underlying CKD.

Loin Pain

Although kidney stones are a common and potentially serious cause of loin pain, pain arising from the kidneys can occur in numerous conditions (Table 1.7).

Nephrolithiasis is usually accompanied by severe symptoms although it may only be on direct questioning that patients mention passing 'gravel', i.e. small sand-like material. Working or living in hot, dehydrating environments, multiple long flights and salt intake as well as any dietary precipitants such as betel nut are all associated with stones in epidemiological studies. Any history of obstruction, lithotripsy or stone removal is also clearly important. Identifying if and where any stones have been analysed is very helpful and can expedite appropriate preventative therapy if reports can be obtained.

Electrolyte Disorders

The renal physician is often asked to provide assistance in the diagnosis and management of patients with electrolyte abnormalities. Renal tubular syndromes are discussed in detail later in this book, although differentiation of renal tubular abnormalities from endocrine, metabolic or gastroenterological aetiologies can be difficult.

Table 1.6 Evaluation of the patient with hypertension

Age of onset	Early onset <40 years more suggestive of secondary cause
Adherence	Agents tried, evidence of concordance
Severity/end-organ damage	Number of agents, retinopathy, left ventricular hypertrophy/failure, cerebrovascular disease
History of renal disease	
Family history	Examples include Liddle's or Gordon's syndrome but more usually a family history without diagnosis
Abnormalities of great vessels	Congenital heart disease, murmurs or abnormal pulses
Renal artery stenosis	Flash pulmonary oedema is rare. A history of macrovascular disease, deterioration in renal function with ACEI/ARB, absent peripheral pulses are more common
Phaeochromocytoma	*Episodic* headache, palpitations (64 %), sweating (70 %), pallor, hypotension, tremor, flushing, dyspnoea and epigastric pain
Obstructive sleep apnoea	Usually typical history and body habitus
Other endocrine causes	Cushingoid features, signs of acromegaly, etc.

Table 1.7 Pain associated with renal disease

Pyelonephritis	Acute and chronic such as xanthogranulomatous pyelonephritis
Renal stones	Typically severe, sudden onset and radiating (loin to groin)
Acute obstruction	Stone, sloughed papillae, blood clot, intermittent PUJ obstruction (particularly after fluid challenge)
Reflux	Occasionally patients describe loin pain on micturition
Wunderlich syndrome	Spontaneous renal haemorrhage from renal carcinoma, angiomyolipoma (renal AML) or arteriovenous malformation
Abdominal pain	Polyarteritis nodosa, infiltration with tumour
Infarction	Arterial or venous occlusion
Loin pain haematuria	Nutcracker *syndrome*

Where an abnormal finding is unexpected or sudden, the possibility of an aberrant value should be considered. For example, the recognition of the abnormal lab finding as being the result of blood sampling from a drip arm or patient misidentification can save significant anxiety. Symptomatology can be non-specific, such as muscle weakness with hyperkalaemia, or classic such as perioral paraesthesia and latent tetany in hypocalcaemia. The urgency of investigation and treatment will typically depend on both the severity and the chronicity of any abnormality.

Changes in Urinary Volume

Urinary volumes in the absence of disease can vary at least tenfold, so aside from anuria patients may find it difficult to recognise clinically important changes in urinary volume. Oliguria is a manifestation of advanced renal impairment of any cause; however, acute absolute anuria is rare and the causes are listed in Table 1.8.

Conversely, patients may find polyuria difficult to distinguish from urinary frequency, but this is important to differentiate. One approach to assessment is shown in Table 1.9.

Renal Manifestation of Multisystem Disorders

Abnormalities of the kidney occur in a large number and diverse range of systemic disorders with vascular, inflammatory, malignant or infective aetiologies (Table 1.10). Indeed, where the kidney represents the first clinical manifestation of these disorders, the renal physician may be best placed to establish diagnosis with predominantly extrarenal involvement. In these cases it is often attention to a comprehensive clinical history and examination that will reveal an underlying disorder.

General Aspects of the Clinical History Relevant to Establishing a Renal Diagnosis

In patients first presenting to a nephrologist once the presenting renal syndrome (e.g. new renal impairment nephrotic syndrome or an electrolyte disorder) has been determined, a further clinical history and examination can be pursued. A clear understanding of what brought the patient to seek medical attention at this time alongside a past medical and surgical history is likely to provide further valuable information.

Table 1.8 Causes of acute anuria

Vascular 1. Arterial catastrophe	Aortic dissection or thromboembolic event to single functioning kidney
Vascular 2. Venous thrombosis	Bilateral venous thrombosis (e.g. nephrotic syndrome or IVC occlusion)
Urinary leak	Usually traumatic rupture, occasionally after instrumentation or surgery
Anti-GBM disease	Probably the only intrinsic renal disease that can result in abrupt anuria
Profound shock	In patient with underlying CKD
Obstruction	Bilateral obstruction or obstruction of single functioning kidney, bladder outlet obstruction, surgical obstruction
Page kidney	Single functioning kidney
Urinary leak	For example, bladder rupture, leaking transplant ureter

Table 1.9 Assessment of polyuria

Polyuria associated with thirst	
Due to increased urine output:	
Renal tubular disorders, congenital	
Nephrogenic diabetes insipidus	Patients may give a childhood history of drinking from puddles or any available water
Bartter syndrome	
Medullary cystic kidney disease	Nephronophthisis, childhood thirst and polyuria
Renal tubular disorders acquired	
Recovery from AKI	
Medication	Lithium, diuretics, etc.
Acquired medullary pathology	Pyelonephritis, obstructive uropathy, HbSS, analgesic nephropathy, light chains
Hypercalcaemia, hypokalaemia	
Osmotic diuresis	Glucose, mannitol, contrast
Endocrine causes	Cranial diabetes insipidus, Addison's disease, hyporeninemic hypoaldosteronism
Due to increased intake:	Xerostoma (Sicca syndrome) anticholinergic medication
Polyuria without thirst	
Psychogenic polydipsia	
Following fluid loading	Beer drinking, IV fluids

Table 1.10 Common multisystem disorders with renal involvement

Diabetes	Diabetic nephropathy or atherosclerosis
Atherosclerosis	Large vessel or small vessel renal involvement
Connective tissue disorders	Scleroderma renal crisis
	Interstitial disease: sarcoidosis, treatment related (calcineurin inhibitors)
	Glomerulonephritis: systemic lupus erythematosus, systemic vasculitis, treatment related (gold penicillamine)
	AA amyloidosis: rheumatoid arthritis
Malignancy (primary or metastatic disease)	Obstruction, hypercalcaemia, tumour lysis syndrome, AL amyloid (paraproteinaemia)
	Direct infiltration
	Thrombotic microangiopathy
	Glomerulonephritis: membranous (breast, lung, GI), minimal change (lymphoma)
	Treatment related
Infections	
Tuberculosis	Sterile pyuria, haematuria, cystitis, nephrolithiasis
	Interstitial disease
	Glomerular – MCGN type 2, focal proliferative, amyloid
	Treatment-associated nephropathy
Enterohaemorrhagic bacteria	Thrombotic microangiopathy
Other bacterial infections	Postinfectious glomerulonephritis
Schistosomiasis	Chronic cystitis, bladder fibrosis, malignancy, ureteric obstruction and vesicoureteric reflux
	Interstitial fibrosis
	Glomerulopathy
Blood-borne viruses, e.g. hepatitis B and C, HIV	Disease associated. Glomerular, thrombotic microangiopathy, cryoglobulin
	Treatment-related nephropathy
Chronic suppurative infection	AA amyloidosis
Gout	Urate nephropathy, uromodulin disorder
Chronic pain	Analgesia use – nephropathy/TIN
Chronic neurological disorder	Bladder dysfunction
Inflammatory bowel disease	Short bowel syndrome/ileostomy losses, treatment associated, oxalate nephropathy, AA amyloidosis
Hepatic failure	Hepatorenal syndrome
Ear, nose and throat disorders	Deafness – Alport's syndrome, Anderson-Fabry's disease
	Epistaxis – cocaine or systemic vasculitis
Pulmonary renal syndromes	Haemoptysis: systemic vasculitis, lupus, anti-GBM syndrome
	Asthmatic: eosinophilic granulomatosis with polyangiitis

Age

The age at presentation can be a clue to diagnosis in a patient with renal impairment. A patient presenting with ESRD in his or her 20s may well have an inherited or congenital cause, and it is important to determine if symptoms started in childhood or adolescence. Age may make some diagnoses much less likely. For example, it is very unusual for lupus nephritis to present late in life, whereas primary vasculitides may well present in a patient's eighth or ninth decade. In addition, the significant age-associated decline in GFR and increase in the prevalence of CKD mean that the elderly are more vulnerable to acute kidney injury (AKI).

Gender

Some renal diseases show a significant gender bias and patterns of inheritance may give a significant clue to the diagnosis when a clear family history is available. For example, X-linked conditions like Alport's syndrome and Anderson-Fabry's disease have a male bias, whereas conditions such as Takayasu's arteritis, fibromuscular dysplasia and systemic lupus erythematosus have a very strong female preponderance.

Ethnicity and Country of Origin

Ethnicity and country of origin may be pointers to increased exposure to risk factors for some renal diseases and comorbid conditions that may affect the kidneys. There are many examples of this and some examples are given in Table 1.11.

Family History

In a new patient it is important to ask about a family history of any kidney problems such as dialysis, renal transplantation or familial renal disease (Table 1.12). If there is evidence of a family history and the diagnosis is not obvious, then age of onset, severity and phenotypic characteristics are helpful. Efforts should be made to obtain (with consent) medical diagnoses of the relatives; information from another renal

Table 1.11 Ethnicities and countries of origin associated with increased risk of kidney disease

Examples of populations at increased risk of CKD in the West	
UK	South Asian and Black populations have higher rates of diabetic nephropathy and hypertension
	Sickle cell nephropathy, systemic lupus erythematosus (SLE), focal segmental glomerulosclerosis in the Black population. Possibly chronic TIN in Asian population
United States	Increased CKD in the Black and Native American population
Australasia	Aborigines, Maoris and Pacific Islanders at increased risk of diabetic nephropathy and hypertension
Examples of geographically prevalent nephropathy	
Danube river	Balkan nephropathy is chronic tubulointerstitial nephropathy. A familial predisposition also exists. There is also a higher prevalence of renal tract tumours in this population
Tunisia and France	Ochratoxin associated with chronic interstitial nephritis
China and Indian subcontinent	Aristolochic acid causing 'Chinese herb nephropathy'
	Takayasu's arteritis
	Heavy metal poisoning
Italy	Hepatitis C-associated kidney disease
Cyprus	CFHR5 nephropathy
Africa and Indian subcontinent	Genitourinary tuberculosis in distribution of TB prevalence
Sub-Saharan Africa	Schistosoma haematobium – lower urinary tract disease and glomerulopathy
	HIV-associated nephropathy
Africa and South/Central Asia	Schistosoma mansoni – glomerulopathy
Africa, Australasia and Indian subcontinent	Post-streptococcal glomerulonephritis
East Asia	IgA nephropathy, SLE, hepatitis B-associated glomerulonephritis
Middle East	Nephrolithiasis
	Familial Mediterranean fever
Central American Pacific Coast	Tubulointerstitial disease

Table 1.12 Family history in renal disease

CKD or ESRD (dialysis or transplantation)	Cause as identified by renal unit, age of onset, pattern of inheritance
Hypertension and diabetes	Polygenic influence, RCAD syndrome
Stones	Calcium nephrolithiasis
Reflux nephropathy	Dysplastic kidneys, PUV or any other congenital abnormality of the urogenital tract
Renal tumours	Age of onset, numbers, other non-renal malignancies (including phaeochromocytoma), epilepsy, learning disabilities, pneumothorax, fibroids, skin lesions – may indicate VHL, BHD, HLRCC or TSC
Subarachnoid haemorrhage	May indicate PKD or the need for screening if known PKD; in haematuria may suggest hereditary angiopathy with nephropathy, aneurysms and cramps (HANAC) syndrome
Deafness	Alport's syndrome, female family members may have history of isolated haematuria suggesting X-linked Alport's syndrome, Fabry's disease, autosomal dominant history suggestive of branchio-oto-renal syndromes, MYH9 mutations, mitochondrial disorders, ciliopathies
Microscopic haematuria	X-linked or autosomal recessive Alport's syndrome; thin basement membrane nephropathy; CFHR5 nephropathy; HANAC syndrome; MYH9-associated nephropathies (Epstein/Fechtner's syndromes)
Retinitis pigmentosa	Bardet-Biedl syndrome and other ciliopathies; mitochondrial disorders
Liver fibrosis and cysts	Autosomal recessive polycystic kidney disease
Heart disease	Anderson-Fabry's disease; hereditary amyloidosis
Gout	Uromodulin-associated disease (MCKD2)
Electrolyte disorders	Hyper- or hyponatraemia, kalaemia, calcaemia, magnesaemia or phosphataemia or a history of polyuria, periodic paralysis

unit may expedite a diagnosis and prevent an unnecessary further investigation. The pattern of inheritance is crucial: affected siblings with unaffected parents suggest recessive disease, especially if there is endogamy or consanguinity. Affected individuals in consecutive generations are consistent with autosomal dominant, mitochondrial and X-linked inheritance; male-to-male transmission of a trait excludes X-linked disease or mitochondrially encoded disorder.

Obstetric History

An abnormal obstetric history can be a pointer to chronic underlying renal disease. Moreover, dipstick urinalysis and blood pressure measurement are recorded regularly from booking until delivery as part of routine maternity care. These findings may unveil both pregnancy-associated and pre-existing renal disease. If the patient is not clear about the details, it is often worthwhile pursuing via her family practitioner or obstetric unit.

The number of pregnancies, including miscarriages, stage of pregnancy reached and any reason for early delivery can be highly relevant as outlined in Table 1.13.

Occupation

Occupation may be a factor in the risk of developing some renal conditions (Table 1.14). Sometimes the exposure is not immediately apparent, and a detailed history of both occupation and hobbies is important to avoid continued risk of deterioration.

Travel and Hobbies

In addition to considering the patient's geographical origins, a travel history should be sought. This is particularly important in the context of AKI. Relevant questions are shown in Table 1.15. Relevant hobbies include water sports (leptospirosis), pets (hantavirus) and endurance sports (haematuria, rhabdomyolysis).

Psychosocial History

There are several aspects of the psychosocial history that are germane to the care of the renal patient. There remains a significant bias to CKD in lower socio-economic groups. Lower educational attainment, mental health problems, substance misuse and unstable home circumstances can pose barriers to engagement with medical professionals, delay recognition of ill health and diagnosis, limit adherence and influence suitability for home therapies. Getting a clear understanding of a patient's psychosocial history is therefore fundamental to the delivery of full and effective care. In addition, there are certain abused substances that have associations with renal disease shown in Table 1.16.

Medication and Allergy

The kidneys are susceptible to a wide range of adverse effects from medications and their active metabolites. Different medications may cause toxicity in a variety of sites within the kidney. It is essential to obtain a history of (1) prescribed and (2) non-prescription drugs and (3) recreational or illicit drugs. Ward prescription charts and inpatient procedural records such as anaesthetic charts, as well as redundant

Table 1.13 Obstetric history and renal disease

Multiple miscarriages	May suggest anti-cardiolipin antibody syndrome. Occurs in advanced CKD of any cause
Hypertension	Stage of pregnancy, severity and number of agents required to treat. Hypertension early in pregnancy, e.g. at booking, is highly suggestive of a non-pregnancy-related cause
Proteinuria	Proteinuria (or haematuria) at booking or heavy proteinuria early in pregnancy is very suggestive of underlying renal disease. Prolonged proteinuria post-partum may also suggest that pre-eclampsia was not the only cause for proteinuria
Pre-eclampsia	Early (<20/40) PET is suggestive of underlying renal disease (20 % have underlying CKD). An underlying renal cause is more likely if hypertension worsens in second pregnancy (with same partner)
UTIs (lower tract or pyelonephritis)	Pyelonephritis is more common in pregnancy and may result in renal scarring
Obstetric sepsis/severe haemorrhage	AKI following either may rarely result in cortical necrosis

Table 1.14 Occupations predisposing or exacerbating renal disease

Occupation/exposure	Pathology
Solvents	Glomerular and tubular pathology
Aniline dye	Urothelial tumours
Sewage workers	Leptospirosis
Outdoor workers in endemic areas	Hantavirus, leptospirosis
Old paint work/plumbing	Lead nephropathy
Cooks	Renal stones
Heavy metal workers/exposure/soldering	Heavy metal nephropathy

Table 1.15 Key travel questions

Details of travel, particularly in the previous 12 months
Pretravel vaccinations and malaria prophylaxis
Rural vs. urban destinations
Unwell contacts
Time period between return and onset of symptoms
Accommodation and food/drink exposures
Fresh water swimming
Animal contacts or bites – tick, animal, bird or bat bites and scratches
Occupation and hobbies – e.g. water sports or agricultural employment
Recent dental work, surgical procedures
Sexual history

Table 1.16 Substance misuse

Smoking	Hastens the progression of renal disease associated with both diabetes and hypertension
	Increased microalbuminuria
	Increased likelihood of pulmonary haemorrhage in anti-GBM disease
Alcohol	IgA nephropathy in alcoholic liver cirrhosis
	Cirrhosis and hepatorenal syndrome
Solvent	Toluene in 'glue sniffing' has been associated with numerous tubular and glomerular lesions
Cocaine	Renal ischaemia, vasculitis or rhabdomyolysis
Heroin	FSGS
Intravenous drug injection	Blood-borne viruses
	Infective endocarditis
	AA amyloid
Ketamine	Inflammatory cystitis and obstructive uropathy
Ecstasy/MDMA	Increased risk of rhabdomyolysis and acute hyponatraemia

Table 1.17 Key elements of medication history

Prescribed medication
Start and stop dates
Route of administration
Previous exposure and timeline
Non-prescribed, over-the-counter or commercially purchased medication
Particularly analgesic and NSAID use which may not be volunteered
Include use of creams and gels with consideration to systemic absorption
Drug interactions
Anticipated/dose adjustment/drug level monitoring
Drug reactions
Fever, rash, arthralgia may occur in suspected AIN
Reversibility of effect
Concomitant administration of prophylaxis for side effects
IV radiocontrast
Dietary supplements
Creatine-based sports supplements
Dieting supplements
Nutritional additives
Laxatives and diuretics
Herbal preparations
Whenever possible ascertain the origin and obtain a sample for analysis since it may contain heavy metals or NSAIDs
Consider interactions with prescription medication, for example, via cytochrome P450
Illicit drug abuse
Modality – frequency of needle use and sharing between individuals or participation in needle exchange
Route – intravenous or subcutaneous (increased risk of amyloid) associated with thrombophlebitis or cellulitis
Risk factors for infective endocarditis

completed drug charts, should be reviewed to identify potential toxins in use at the onset of renal dysfunction. Accurately identifying a complete drug history may be time-consuming, but confirming prior use of aminoglycosides, exposure to a common cause of tubulointerstitial nephritis or that a patient is taking a product containing aristolochic acid may be critically important to establishing the cause of renal dysfunction. Key elements of the drug history are shown in Table 1.17.

Clues on Clinical Examination to Help Establish the Cause of a Renal Disorder

A thorough clinical examination is an essential part of the approach to any patient presenting either acutely or to the outpatient department. Accurate fluid assessment will be critical to most presentations to the renal physician and is discussed in detail above. Alongside intravascular volume assessment, other findings on clinical examination can provide important clues to the aetiology of a renal presentation. These examination findings are summarised in Table 1.18.

Assessment of the Patient with Known Renal Disease

Many renal conditions are chronic, and it is common to encounter patients who have previously been investigated or treated either locally or in another centre. When a patient is known to suffer from a renal disorder, the aim of the assessment will usually be focused on the management of the underlying condition or associated complications rather than the underlying cause of the renal disease (which may or may not have already been diagnosed). Those in contact with renal services frequently have complex and chronic histories, and the importance of handling the transfer of care cannot be overstated (see Chap. 49). Fortunately patients are increasingly involved in their own care, have a good understanding of their disease and have access to their clinical records. When they do not, it is important to make the effort to trace historical imaging, blood and urine results. In all patients known to have renal disease, the nature and duration of the condition, as well as histological details if available, are an essential starting point of any assessment. Patients with known renal disease will broadly fall into the following groups allowing the clinician to focus their clinical assessment on relevant issues.

Table 1.18 Examination tips and renal disease

Habitus	Obesity (OSA), Bardet-Biedl syndrome (renal cysts) (short stature), Noonan syndrome (short and webbed neck – renal dysplasia), Turner's syndrome (short stature, webbed neck – horseshoe kidney), Down's syndrome (renal dysplasia), Jeune's syndrome (short limbs, narrow rib cage). Lipodystrophy (MPGN). Limb abnormalities VACTERL association. *Any form of CKD in childhood can result in short stature*
Hair	Scaring alopecia (SLE), diffuse alopecia (heavy metal poisoning, tacrolimus, steroids), hirsutism (cyclosporine)
Ears	Otitis, inflammation of the pinna with granulomatosis with polyangiitis (GPA), preauricular pit, sensorineural deafness (branchio-oto-renal syndrome (BOR), Alport's syndrome, Anderson-Fabry's disease (AFD), CHARGE syndrome (ear abnormalities))
Nose	Crusting, nasal bridge collapse, GPA; cocaine
Mouth	Dentition (infective endocarditis (IE)), mouth ulcers (vasculitis SLE, herpes viral infections), fungal infections, macroglossia (useful sign of amyloid)
Polydactyly	Jeune's, Bardet-Biedl, Meckel-Gruber syndromes
Nails	Periungual fibromas (tuberous-sclerosis complex (TSC), dysplastic nails (nail patella syndrome), splinter haemorrhages (infective endocarditis), Muehrcke's bands (episodes of nephrotic syndrome))
Skin	*Signs of renal disease*: vasculitic rashes, palpable purpura (Henoch-Schönlein purpura (HSP)), palpable subcutaneous nodules/ulcers (polyarteritis nodosa), malar flush (systemic lupus erythematosus (SLE)), cutaneous lupus erythematosus, alopecia (SLE, tacrolimus), neurofibroma, viral exanthem, erythema nodosum, tracheostomy scar (previous ICU admission), xanthelasma, nicotine stains (atherosclerotic disease), Janeway lesions (IE), bruising (amyloid), livedo reticularis (cholesterol emboli, SLE, anti-cardiolipin syndrome), angiokeratoma (AFD), Raynaud's disease (SLE, scleroderma, anti-cardiolipin syndrome), facial angiofibromas, ash-leaf macule and shagreen patch (TSC) *Signs of immunosuppression*: purpura, thin skin, gum hypertrophy (cyclosporine), sebaceous gland hyperplasia, actinic keratosis, Kaposi's sarcoma, squamous cell carcinoma, basal cell carcinoma, hypertrichosis (cyclosporine), cushingoid features and striae (steroids) *Signs of advanced CKD*: xerosis, acquired perforating dermatosis, porphyria cutanea tarda, calciphylaxis
Eyes	Retinopathy (hypertensive, diabetic); retinitis pigmentosa/dysplasia (Bardet-Biedl; Senior-Loken syndrome, nephronophthisis; Jeune's syndrome; Kearns-Sayre mitochondrial cytopathy); uveitis (tubulointerstitial nephritis with uveitis); band keratopathy, sicca (Sjögren's syndrome); corneal clouding (cystinosis); lenticonus (Anderson-Fabry's disease); proptosis (GPA, IgG-4-related disease); angiomatosis retinae (VHL); coloboma (renal coloboma syndrome CHARGE and COACH syndromes), periorbital bruising (amyloid), iritis, scleritis, retinal vasculitis (vasculitis), drusen (dense deposit disease)
Lymphoproliferative	Lymphadenopathy (tuberculosis, lymphoma); splenomegaly (IE, sarcoid, lymphoproliferative disorder)
CVS	Atrial fibrillation (emboli), pericardial rub (SLE, infections, uraemic pericarditis), murmur/pacing wire (endocarditis), radiofemoral delay/missing pulses (aortic coarctation/mid-aortic syndrome, Takayasu's arteritis), bruits (renovascular disease, fibromuscular dysplasia), ventricular failure (right or left sided)
Chest	Pneumothorax (tuberosclerosis); pleural rub (SLE, vasculitis, infection); asthma (eosinophilic granulomatosis with polyangiitis); pulmonary fibrosis (systemic vasculitis, scleroderma, SLE, Sjogren's syndrome, drugs); signs of bronchiectasis (amyloid)
Abdominal	Signs of chronic liver disease (hepatorenal syndrome, viral hepatitis), stoma (high output), absent abdominal musculature (prune-belly syndrome)
Neurological	Asterixis/tremor (uraemic encephalopathy, calcineurin inhibitor toxicity), hemiparesis (bladder dysfunction, infection-associated amyloid), spina bifida (occulta)
Musculoskeletal	Polyarthropathy (rheumatoid arthritis, SLE, ankylosing spondylitis), monoarthritis (hyperuricaemia), infection including IE, Charcot joint, absent patellae (nail patella syndrome)

Patients with ESRD Treated with Dialysis

Patients with ESRD may have been receiving renal replacement therapy for many years and have been treated with several treatment modalities. Renal physicians will often be integral to the holistic care of patients on dialysis programmes. Therefore, alongside dialysis-related issues the renal specialist increasingly needs at least a basic understanding of a broad range of medical, surgical and psychiatric problems so that appropriate further expertise can be sought when necessary. When thinking about dialysis-related problems, presentations will commonly be related to dialysis access (including infection) and intradialytic issues (instability, adequacy or complications). Important considerations for the assessment of the patient on dialysis are outlined in Table 1.19.

Table 1.19 Clinical assessment specific to the dialysis patient

Underlying cause of ESRF	
Duration of ESRF and different treatment modalities	
Most recent dialysis session and intradialytic problems	Date/duration of last session, problems with treatment, loss of circuit
Dialysis access PD catheter Temporary vascular access for HD AVF or AV grafts	Date of formation/insertion, signs of infection, adequate function, position (temporary HD catheters or PD catheters)
Fluid status	
Dialysis adequacy	URR or KT/V, serum potassium, residual native kidney function, ultrafiltration volumes, intradialytic weight gains (HD), constipation (PD)
Blood pressure control	Antihypertensive medication, sodium intake, intradialytic hypotension
Traditional cardiovascular risk factors	Smoking, dyslipidaemia and treatment
Complications of CKD and treatment	Anaemia, bone-mineral disorder
Transplant listing status	May impact on decisions regarding transfusion, imminent recipient of live donor transplant, etc.
Nutritional status	Changes in 'dry weight' may indicate occult chronic infection or malignancy

Table 1.20 Clinical assessment specific to the transplant patient

Underlying cause of ESRF	
Duration of ESRF and different treatment modalities	
Previous transplantation	Date, duration, reason for graft loss
Current transplant	
Donor details	Age, donor type, donor comorbidity/COD
Immunological details	Overall sensitisation (% CRF) (donor-specific and non-specific antibodies), mismatch, cross-match details, posttransplant donor-specific antibodies
Surgical details	Cold and warm ischaemic times, arterial and venous anatomy, ureteric anastomosis
Post-operative course	Delayed graft function, infection, rejection, thrombosis, obstruction (stent removal)
Allograft biopsies	Tubular injury, rejection, recurrence of primary disease, degree of fibrosis
Immunosuppressive treatment	Induction, maintenance, treatment for rejection, steroid withdrawal, drug levels
Infection risk and prophylaxis	Donor and recipient viral immunity, prophylaxis, infection history, BK virus
Recent allograft imaging	Ultrasound, MRA/angiography, nuclear medicine
Baseline function	Look out for slow declines over many months
Non-allograft issues	
Fluid status	
Evidence of infection	Urine, chest, GI, neurological, atypical organisms
Evidence of malignancy	Weight loss, breast/cervical screening
Blood pressure control	Antihypertensive medication, sodium intake
CV risk	Smoking, dyslipidaemia (posttransplant), diabetes
Osteoporosis	Fractures, prophylaxis
Complications of CKD and treatment	Anaemia, bone-mineral disorder

Patients with ESRD with Functioning Kidney Transplants

The management of graft dysfunction will represent a substantial workload for any transplant unit, so the nephrologist needs to be fluent in the further management of a transplant patient with a rise in the serum creatinine. Once again assessment of intravascular volume status is a critical element. In addition, the management of immunosuppression-related complications such as infection and malignancy will need specific attention in transplant patients. Finally, patients with kidney transplants will often present to their 'home' transplant unit with transplant-related issues and problems not immediately associated with the allograft or associated complications. Table 1.20 outlines aspects of clinical assessment important in the patient with a functioning renal allograft.

Table 1.21 Clinical assessment specific to the patient with inflammatory renal disease

Underlying diagnosis	
Duration of disease, relapses, multisystem involvement	
Baseline kidney function	
Renal biopsies	Active disease, chronic fibrosis
Immunosuppressive treatment	Induction, maintenance, steroid withdrawal, drug levels
Evidence of relapse	Including extrarenal symptoms and signs
Fluid status	
Infection risk and prophylaxis	Infection history, viral immunity, prophylaxis
Evidence of infection	Urine, chest, GI, neurological, atypical organisms
Evidence of malignancy	Weight loss, breast/cervical screening
Blood pressure control	Antihypertensive medication, sodium intake
CV risk	Smoking, statins, diabetes
Osteoporosis	Fractures, prophylaxis
Complications of CKD	Anaemia, bone-mineral disorder

Patients with ESRD on Conservative Care Programmes

Increasingly renal units have large and successful conservative care programmes. One of the reasons patients may have decided that they do not wish to receive active care for ESRF is to reduce time spent in a medical environment. Although these patients may have decided not to undergo dialysis or transplantation, the assessment and prompt management of fluid status, anaemia, bone-mineral disorders, nausea and pain can have a significant impact on quality of life and should be pursued as a priority.

When patients are clearly close to death the renal physician may need to play an active part in ensuring that the appropriate end-of-life care can be effectively delivered and in the most appropriate environment.

Patients with CKD and a Reduced GFR

Some form of chronic kidney disease is thought to affect between 5 and 10 % of the population in Western countries. Nephrologists will often be asked to help manage this group of patients who may often suffer with multiple comorbidities. The increasing recognition of the increased risk of AKI in those with CKD as well as the contribution of AKI to the future progression of CKD means that a key focus of the assessment of this group of patients surrounds the management, and prevention, of a further decline in kidney function. The approach to this clinical situation should be similar to that set out in the section on establishing the cause of previously undiagnosed renal impairment set-out in detail above.

Patients with CKD also commonly present with fluid overload, and the nephrologist will often be asked for advice, again underlying the importance of thorough assessment of intravascular volume status.

In this group of patients particular attention should also be focused on the prescription of medication at the dose appropriate for the patients GFR. Failure to dose-reduce medications can lead to acute-on-chronic kidney injury or alternatively significant adverse effects due to drug or metabolite accumulation (see Chap. 56).

The management of patients with stable CKD also needs to address the complications of decreased renal function, specifically anaemia, bone-mineral disorder and cardiovascular risk. These are addressed in the chapters on chronic kidney disease management.

Patients with Inflammatory Renal Diseases and/or Requiring Immunosuppression

Patients suffering from systemic vasculitis, SLE, nephrotic syndrome or other inflammatory renal conditions will often be taking immunosuppressive drugs. In addition to the issues related to CKD or dialysis, the possibility of disease relapse and the consequences of immunosuppression needs to be considered when these patients present to renal services. An approach to this group of patients is outlined in Table 1.21.

Summary

Patients attending renal specialists present with an enormous diversity of clinical problems. An approach to the review of patients using a system similar to the one outlined in this chapter allows for comprehensive and organised recognition of both the background and current problems. In this group of often extremely complex patients, it is by focusing the assessment on answering the relevant questions at hand that clinical problems will be appropriately prioritised and timely and safe treatment instigated. The critical importance of accurate assessment of intravascular volume status in almost all areas of renal medicine cannot be overemphasised, and this is a skill that can only be learned with repeated practice. Although as experience is gained each clinician will develop their own unique approach to clinical assessment, it is only with a systematic approach that nephrologists can be confident of providing safe, efficient and high-quality care to their patients.

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