

Chronic kidney disease is irreversible, but management and treatment options are available to help patients self-manage the condition and maintain their quality of life

# Managing and treating chronic kidney disease

## In this article...

- › The classifications of chronic kidney disease
- › Treatment options available for different stages of CKD
- › Complications that can occur as a result of CKD

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Chronic kidney disease is a long-term condition that commonly exists with other conditions such as diabetes, hypertension and heart failure. The majority of people with CKD are managed in the community by a GP and community nursing team rather than a nephrologist. This article provides an overview of its stages, management and treatment.

The 2010 health survey for England found 6% of men and 7% of women had stage 3-5 chronic kidney disease. It showed a large variation by age: fewer than 1% of men and women aged 16-24 years had stage 3-5 CKD, compared with 29% of men and 35% of women aged 75 years and over (Roth et al, 2011).

Patients with CKD have irreversible kidney damage; as kidneys have several functions, this can lead to multiple health problems. CKD has several causes including hereditary diseases such as polycystic kidney disease, structural deformities, infection or long-term conditions such as diabetes and hypertension. Patients can develop complications such as high blood pressure, poor nutritional health, low haemoglobin levels and heart disease.

Late management of people with CKD increases morbidity, mortality and associated healthcare costs. Early detection and treatment can improve the outcome (Renal Association, 2013a).

## Kidney function

The gut absorbs nutrients from the gastrointestinal tract into the bloodstream. Waste from metabolism is deposited back in the bloodstream; the blood is filtered by the kidneys and what is not required by the body (toxins and water), is filtered out in the form of urine to be excreted.

If the kidneys are not working properly, toxins and water are retained in the body. When kidney function deteriorates, toxin levels and water retention both increase, causing CKD symptoms (Box 1). Toxins and water retention can rise to dangerous levels and, if not adequately treated, lead to death.

The most practical way of assessing the level of kidney function is to calculate the glomerular filtration rate. GFR is a measurement of how much waste fluid the kidneys filter from the blood per minute (mL/min); in normal kidney function, this should be >90mL/min. As it is difficult to measure the GFR directly, an estimated GFR (eGFR) is calculated using a formula using creatinine levels and taking into account age, gender and ethnic group.

## Classifying CKD

Identifying the CKD stage helps with care planning. The National Institute for Health and Care Excellence (2014) recommends classifying CKD using the albumin/creatinine ratio (ACR), which measures the amount of albumin in urine and takes into account the level of creatinine in the blood.

EGFR and ACR are important because the lower the eGFR or the higher the ACR, the more likely are complications of CKD. These complications require monitoring and intervention depending on the CKD stage. NICE (2014) and the National Kidney Foundation (2002) have developed charts

## 5 key points

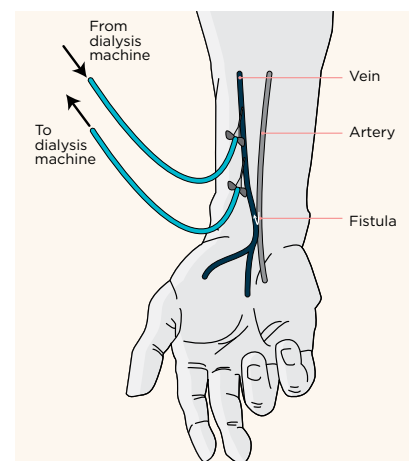
**1** Chronic kidney disease is an irreversible, long-term condition

**2** CKD should be monitored and treated in the early stages to optimise outcome and slow deterioration

**3** Dialysis and transplants are treatments, not cures, and are not suitable for all patients

**4** Patients require close management of diet, fluid and medicines to compensate for reduced kidney function

**5** Specialist guidelines are available to help with management



Arterio-venous fistula: this is formed for most patients requiring haemodialysis

and tools to help with classification and, once the stage has been identified, guidance on monitoring and intervention (Table 1).

### Managing and treating CKD

Caring for patients with CKD means managing treatments that carry out the kidney's functions. Most patients can be managed by their GP but some may need investigations to find the cause of CKD and any reversible factors. The Renal Association (2013b) provides clear guidelines on when to refer a patient to a nephrologist.

In the early stages of CKD, toxin and water control can be managed by dietary restrictions, which should be monitored by a renal dietitian. Depending on CKD severity, dietary advice may include potassium, phosphate, calorie and fluid intake.

As too much potassium in the blood can cause heart dysfunction, the first dietary change is usually to follow a low-potassium diet. This involves limiting the intake of fruit, potatoes, mushrooms and chocolate, as well as learning to cook certain foods differently; for example, boiling vegetables before adding them to recipes.

Special diets should be provided by professionals to ensure patients are properly assessed and that the education and management provided is appropriate to them.

A low-protein diet should not be offered as this can lead to malnourishment if sustained over a long period. Renal teams must monitor and manage kidney function to ensure symptoms are kept to a minimum; once they are unmanageable using diet and medicines, renal replacement therapy of the patient's choice should be started.

When patients have later-stage CKD (stages 4 and 5), they must be given robust information on treatment options. These comprise dialysis, transplantation and conservative management. Not every option will be appropriate for every patient; suitability will be determined by the patient's physical and mental condition.

### Dialysis

Patients at stage 5 CKD or those who have an eGFR below 10 are offered dialysis. There are two types of dialysis: peritoneal dialysis; and haemodialysis.

#### Peritoneal dialysis

This uses the peritoneal membrane, which lines the abdomen. A dialysis catheter is inserted into the membrane and a special dialysis fluid drained through the catheter into the peritoneum. Toxins, waste products and excess fluid are pulled from the membrane's blood supply into the fluid by osmosis and diffusion (Fig 1). The fluid

**TABLE 1. CHRONIC KIDNEY DISEASE CLASSIFICATION**

GFR categories (mL/min/1.73m <sup>2</sup> ), description and range		ACR categories (mg/mmol), description and range			Increasing risk
		<3 Normal to mildly increased	3-30 Moderately increased	>30 Severely increased	
		A1	A2	A3	
≥90 Normal and high	G1	No CKD in the absence of markers of kidney damage			Increasing risk ↓
60-89 Mild reduction related to normal range for a young adult	G2				
45-59 Mild-moderate reduction	G3a <sup>1</sup>				
30-44 Moderate-severe reduction	G3b				
15-29 Severe reduction	G4				
<15 Kidney failure	G5				

ACR = albumin:creatinine ratio. CKD = chronic kidney disease. GFR = glomerular filtration rate  
Source: Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group (2013). Adapted with permission

must be changed regularly; the frequency depends on the type of PD carried out.

Continuous ambulatory peritoneal dialysis requires the patient to drain the fluid out and replace it with more fluid (do an exchange), which takes around 30 minutes, four times a day (roughly every six hours). The fluid is drained into the peritoneum and left there for four to eight hours. The patient can carry out everyday activities, such as working and sleeping, during this time but it may not suit some occupations. This form of dialysis gives patients control

of their treatment. The volume of fluid inserted into the abdomen can lead to hernias and infection (peritonitis) which, if spotted quickly, can be treated effectively.

Automated PD requires the patient to be connected to a dialysis machine. Small amounts of fluid are put in and removed from the peritoneum as the patient sleeps so exchanges are not required during the day. This appeals to people who want to control their treatment but have commitments that mean they cannot carry out four exchanges a day.

### BOX 1. CKD SYMPTOMS

Most people do not have CKD symptoms until the later stages. These include:

- Fatigue, lack of energy
- Poor appetite
- Nausea, vomiting, taste changes
- Need to pass urine more often, especially at night
- Swollen feet and/or ankles
- Shortness of breath
- Dry, itchy skin
- Difficulty concentrating

#### Haemodialysis

This type of dialysis removes toxins and waste products using a haemodialysis machine. The machine creates a continual cycle of blood being taken from the patient, cleaned using an artificial kidney (filter), then returned to the patient. It can also remove excess fluid. Most patients attend a dialysis unit three times a week and have a four-hour dialysis session; a smaller proportion dialyse more frequently and/or at home.

Access is needed to the blood supply and, for most patients, this is by an arteriovenous fistula. This is formed by joining

an artery to a vein in the arm, which requires a small operation. The blood in the artery goes directly into the vein causing the vein to enlarge, thereby making venous access easier. Fistula needles can then be inserted into the fistula at the start of each dialysis session.

A smaller proportion of patients dialyse using a:

- » Graft – this is similar to a fistula but an artificial tube is used to connect the artery and vein;
- » Permcath – a flexible plastic catheter inserted into the internal jugular vein and tunnelled over the chest wall.

### Transplantation

Kidney transplantation is not appropriate for all patients who progress to stage 5, especially those with comorbidities. Patients can receive a transplant through:

- » Cadaver donation – from someone who has died;
- » Living donation – from a living person the patient knows (family/friend), an anonymous donor (altruistic), or as part of a chain in which the relative donates to another kidney patient and the original patient receives a kidney from the family of the other kidney patient (paired exchange).

The type of transplant offered depends on the transplant unit the patient attends, personal circumstances, organ availability and preferences.

### Conservative management

Some patients, especially if they are very old, very frail or have other medical problems, may find dialysis too much of a burden. In these cases, medications can be taken to control symptoms. These medications do not replace dialysis or transplantation and discussing end-of-life care is part of management. These patients are usually managed in the community by a community team and/or a specialist renal nurse.

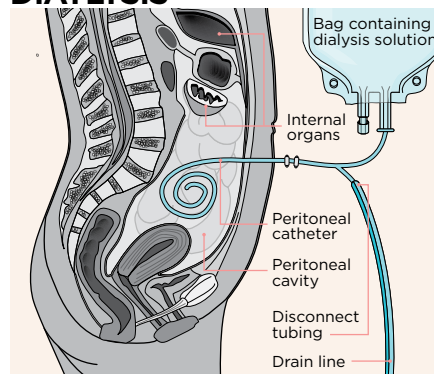
### Anaemia in CKD

Anaemia is a common complication of CKD and has many causes, the most common of which is reduced erythropoietin production. Erythropoietin, a hormone produced mainly by the kidneys, controls the production of red blood cells in the bone marrow. Reduced kidney function leads to less circulating erythropoietin so fewer red blood cells are produced; this leads to anaemia.

Another factor contributing to renal anaemia is iron deficiency. This can be:

- » Functional – iron stores exist but are not available for the body to use;
- » Absolute – iron stores are so low the

FIG 1. PERITONEAL DIAALYSIS



bone marrow does not have enough to support red blood cell manufacture.

Anaemia in CKD can occur as early as stage 3. In general, the lower the level of kidney function, the greater the severity of the anaemia. There are many guidelines to help practitioners treat renal anaemia, including NICE's (2011) guidance. This advises that anaemia be considered, investigated and managed in people with CKD if:

- » Haemoglobin levels fall to 11g/dL or less (or 10.5g/dL or less if under two years old);
- » They develop symptoms attributable to anaemia (such as tiredness, shortness of breath, lethargy and palpitations).

Measuring the eGFR is an important part of investigations – if it is >60mL/min, anaemia is unlikely to be due to CKD and other causes should be investigated.

### Treatment

The first-line treatment of anaemia in CKD is correction of any iron deficiency. Iron supplements can be administered:

- » Orally – sometimes tried in patients not receiving dialysis, but treatment may be ineffective or cause side-effects including nausea, constipation, diarrhoea and gastrointestinal discomfort;
- » Intramuscularly – rarely used in CKD as it often requires painful injections;
- » Intravenously – commonly used, especially in patients receiving dialysis, and can be administered as a bolus injection, an infusion or directly into the lines of a haemodialysis machine.

Blood transfusions are avoided because of potential complications and as alternative therapies are available. This is especially the case for potential transplant recipients as blood transfusion can increase the percentage of cytotoxic antibodies, which can cause rejection of a donor organ. This makes it difficult to find a cross-match negative donor and may mean a longer wait for a transplant.

Erythropoiesis stimulating agent (ESA) – a genetically engineered copy of erythropoietin – should be considered if the patient remains anaemic once the iron deficiency has been corrected. Once patients start treatment for anaemia, it is usually indefinite (unless they receive a kidney transplant), so frequent monitoring of haemoglobin and iron levels are required. It can be harmful to correct anaemia to a normal range when patients are treated with ESA because of an increased risk of stroke, hypertension and vascular access (fistula) thrombosis; NICE (2011) recommends treating to keep the haemoglobin at 100-120g/L.

### Blood pressure

Controlling blood pressure can slow the progression of CKD in some people. Unfortunately, hypertension is a complication of CKD so its management can become difficult. Recommendations suggest people with CKD should aim to keep their systolic blood pressure <140 mmHg (target 120-139mmHg) and their diastolic blood pressure <90mmHg (NICE, 2014). For those with CKD and diabetes, systolic blood pressure should be <130mmHg (target 120-129mmHg) and diastolic blood pressure <80mmHg (NICE, 2014).

### CKD-mineral and bone disorder

CKD-mineral and bone disorder (CKD-MBD) is a common complication of CKD that develops due to reduced kidney function. Bone changes from CKD-MBD can occur years before symptoms appear; if left untreated, bones gradually become thin and weak, leading to an increased risk of fractures and symptoms such as bone and joint pain. In healthy adults, bone tissue is continually remodelled and rebuilt. The kidneys are key to maintaining healthy bones; they do this by controlling calcium and phosphate levels in the blood and activating vitamin D received from sunlight and food.

Calcium builds and strengthens bones. If calcium levels in the blood become too low, the parathyroid glands produce parathyroid hormone PTH, which causes the bones to release calcium so levels in the blood rise. If this occurs for a sustained period, the parathyroid glands enlarge and produce more PTH, even when calcium levels are normal or elevated. This results in too much calcium being removed from the bones and weakens them over time.

Phosphate also helps to regulate bone calcium levels. Healthy kidneys remove excess phosphate from the blood but, when they are not working properly, levels in the

blood rise. This leads to lower levels of calcium in the blood and higher PTH levels, and the loss of calcium from the bones.

Healthy kidneys convert vitamin D into an active form called calcitriol. This helps the body absorb dietary calcium and phosphorus into the blood and bones. Calcitriol and PTH work together to keep calcium balance normal and bones healthy. As kidney function decreases, less vitamin D is converted to calcitriol and less calcium is absorbed; as a result, PTH levels increase, removing calcium from the bones. The combination of decreased calcium absorption from food and PTH drawing calcium from the bones makes the bones weak and brittle.

Another complication of CKD-MBD is vascular or soft tissue calcification. The reason for this is not completely understood, but is thought to be due to high calcium and phosphate levels in the blood. Calcium is deposited outside the skeleton, in tissues such as the blood vessels, lungs, kidneys, heart and gastric mucosa. This can cause problems with tissue or blood supply function, including tissue necrosis.

The underlying principle of treatment for CKD-MBD is to keep the levels of calcium and phosphate in the blood as close to normal as possible to prevent the parathyroid glands from becoming overactive. This is achieved through diet and medication.

### Diet

Reducing dietary intake of phosphate is one of the most important steps in preventing bone disease. A dietitian can advise on foods that contain high levels of phosphate, such as milk, cheese and nuts, which should be avoided.

### Medication

If diet alone is not enough to keep phosphate levels normal, a phosphate binder can be prescribed. This is taken with meals; it binds with phosphate and passes through the gut, preventing absorption.

Various forms of vitamin D can be prescribed. These increase calcium absorption from the diet and suppress the parathyroid gland. Calcimimetic drugs “trick” the body into thinking calcium levels are raised, which causes PTH levels to decrease and calcium levels to fall to normal.

### Regulation of acid-base balance

Acids are produced primarily from the metabolism of ingested protein. To maintain acid-base balance, acid production must balance with neutralisation or excretion. The lungs and kidneys control this balance.

The lungs eliminate carbon dioxide. This is mildly acidic so exhaling decreases the blood’s acidity. The kidneys maintain the body’s pH balance by excreting either hydrogen ions (acid) or bicarbonate ions (alkaline) from body fluids. If the bicarbonate concentration is greater than normal, the kidneys excrete more bicarbonate ions and the urine will become more alkaline. If the hydrogen ion concentration is greater than normal, the kidneys will excrete more hydrogen ions and urine becomes more acidic (White, 2005). Control is also maintained by the kidneys reabsorbing bicarbonate, which can act as a buffer to take up the excess hydrogen ions.

In CKD, acidosis is often seen when the GFR decreases to <20-25% of normal. The degree of acidosis correlates approximately with the severity of kidney failure (Cibulka and Racek, 2007). As the number of functioning nephrons (the unit of the kidney where blood is filtered) declines in CKD, each nephron increases activity to ensure acid excretion is maintained. As kidney function decreases further, there are not enough nephrons to manage the level of acid excretion required, so acid levels start to build up. Hydrogen ion regulation in the body is managed through buffer systems (bicarbonate, phosphate and protein buffer systems). Initially, buffers in the blood combine with the excess hydrogen ions so there are no symptoms. As the number of hydrogen ions increases, fewer buffers are available to bind with the ions. This is reflected in blood tests showing a reduction in serum bicarbonate level.

With further reduction in kidney function and the kidneys’ reduced ability to remove acid from the blood, the pH of the blood becomes lower (acidic) and the body tries to get rid of the excess hydrogen ions in other ways (Kallenbach et al, 2005). The reduction in pH stimulates respiration, and breathing may become deeper or faster as the body tries to expel carbon dioxide. Patients with mild metabolic acidosis may experience nausea, vomiting and fatigue. As the acidosis worsens, they will begin to feel extremely weak and drowsy and may become confused.

Acidosis in CKD is corrected by administering sodium bicarbonate. Patients not receiving dialysis take this orally. Those receiving PD and haemodialysis receive bicarbonate during the procedure. Bicarbonate is transferred from the dialysate to the blood; the diffusion of the bicarbonate helps the patient achieve acid-base balance by buffering the hydrogen ions (Kallenbach et al, 2005).

### Self-management

Self-management is key to optimising outcomes. Patients should be offered information and education on blood pressure management, smoking cessation, exercise, diet and medications, and encouraged to have a healthy lifestyle and diet.

Managing a long-term condition can be stressful. If patients need to attend clinic appointments, have frequent dialysis or be admitted to hospital due to complications, holding down a job or studies can be difficult. This can cause financial problems and changes in family dynamics. Patients may need time or professional support to help them develop coping strategies. Depression in people with CKD is reported to be 20-40% (Zalai and Novak, 2008).

### Conclusion

CKD is an irreversible, complex, long-term condition. Management involves a dietary and fluid restrictions, medicines and possibly dialysis or transplantation. Patients should be involved in treatment choices and self-management. They may require psychological or social support as CKD can affect mood and wellbeing, as well as having social and financial implications. **NT**

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