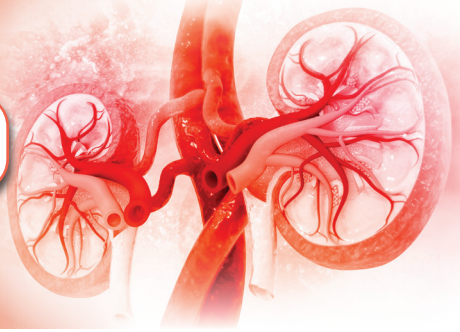
FACT FILE

CHRONIC KIDNEY DISEASE



This fact file has been written and compiled by Katy Stuart, RD. Katy has 18 years of dietetic experience in the NHS, working mainly in critical care, renal and complex nutrition support. She is currently working as a part-time Renal Dietitian with United Lincolnshire Hospitals NHS Trust. Katy is also a supplementary prescriber.

There are around 1.8 million people in the UK diagnosed with chronic kidney disease (CKD), and there are thought to be a million people worldwide who have the condition but are undiagnosed, resulting in 1.8 million deaths per year.[1,2] Prompt diagnosis and management of CKD is vital to improve a sufferer's quality of life and reduce the risk of requiring renal replacement therapy. Dietary and lifestyle factors play an important role in treatment.

WHAT ARE THE STAGES OF CKD?

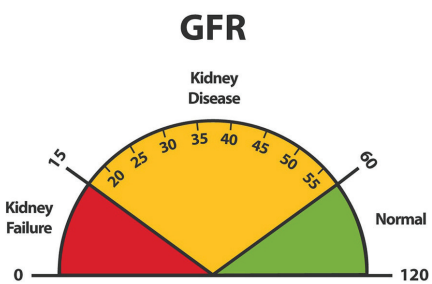
Five stages classify CKD depending on the level of kidney damage and function and the ability of the kidneys to filter blood.[3] The estimated glomerular filtration rate (eGFR) is a lab measurement of the amount of blood that passes through the glomeruli (filters) in the kidneys every minute, and the eGFR falls as the disease progresses.[3]

Table 1: The five stages of CKD

Stage of CKD	Description	eGFR level
1	Kidney function remains normal but urine findings suggest kidney disease	90ml/min or more
2	Slightly reduced kidney function with urine findings suggesting kidney disease	60-89ml/min
3	Moderately reduced kidney function	30-59ml/min
4	Severely reduced kidney function	15-29ml/min
5	Very severe or end-stage kidney failure	Less than 15ml/min on dialysis

Source: Kidney Research UK[22]

HOW IS CKD DIAGNOSED?

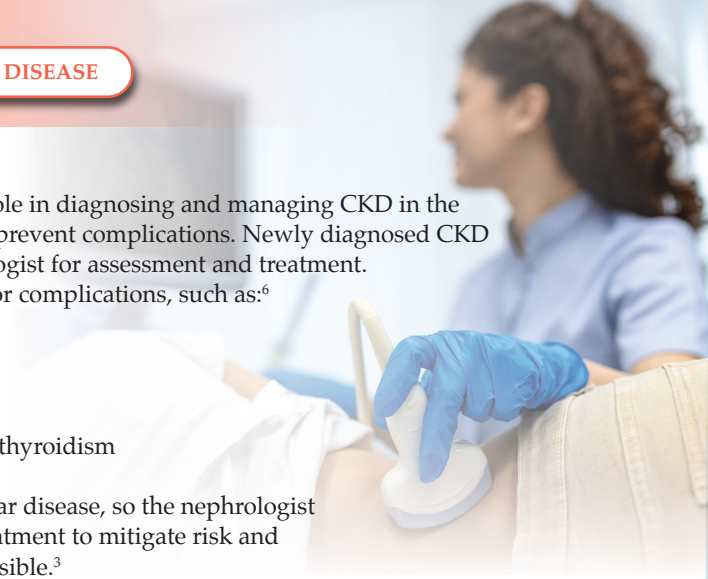


CKD is often asymptomatic, particularly in the early stages. Patients will present to their GP or hospital with advanced kidney impairment symptoms of the following:

- Nausea
- Vomiting
- Fluid weight gain
- Fatigue

Blood tests are performed to diagnose suspected CKD and biochemical indicators (as well as eGFR), including uraemia, raised creatinine, hyperkalemia, hyperphosphatemia, anaemia and metabolic acidosis.[3,4] Urinalysis is also necessary, as protein in the urine indicates kidney disease.[5]

For article references please visit www.NHDmag.co.uk/article-references.html



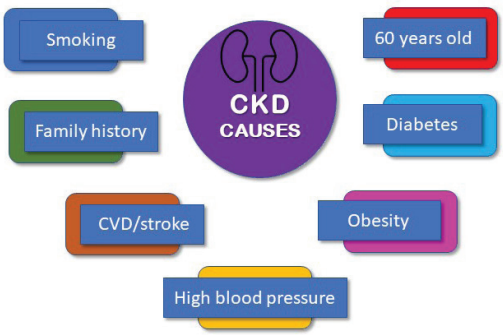
ONGOING MONITORING

Primary care physicians play a crucial role in diagnosing and managing CKD in the community to slow its progression and prevent complications. Newly diagnosed CKD patients should be referred to a nephrologist for assessment and treatment. They will require ongoing monitoring for complications, such as:[6]

- Raised electrolytes
- Acid-base balance
- Fluid management
- Blood pressure control
- Vitamin D deficiency and hyperparathyroidism
- Bone disease

CKD is a risk factor for cardiovascular disease, so the nephrologist will also make recommendations on treatment to mitigate risk and prolong kidney function as much as possible.[3]

WHAT ARE THE CAUSES AND RISK FACTORS?



There are many causes of CKD; however, it is often a combination that leads to kidney failure. Risk factors include diabetes, hypertension, obesity, certain medications and ageing.[4,7] The risk increases if more than one of these conditions is present and more significant stress is placed on the cardiovascular system and renal blood flow. Other conditions that can lead to kidney failure include:[7]

- Genetic disorders such as polycystic kidney disease (PKD) and Alport syndrome
- Renal artery stenosis
- Ischaemia
- Infection
- Trauma
- Drugs that are toxic to the kidneys, such as NSAIDs
- Poisoning such as lead poisoning
- IgA glomerulonephritis and vasculitis
- Autoimmune disorders such as Anti-GBM (Goodpasture's) disease and lupus
- Haemolytic uremic syndrome in children

Sometimes chronic kidney disease can be idiopathic and the cause might not be identified.

WHAT IS THE TREATMENT?

Preservation of kidney function is the most critical aspect of treatment and, in turn, symptom control and improving quality of life, weight loss and blood pressure control, eg, renin-angiotensin system blockade with ACE inhibitors and blood glucose.[4,8]

When eGFR <10mls/min and symptoms become severe, the decision will be made between the patient and the nephrologist regarding what renal replacement therapy treatment would be most appropriate, eg, dialysis or transplant.

THE ROLE OF DIET

Diet has a vital role in CKD treatment. Advice on a healthy diet and lifestyle is usually the first line, and renal dietitians will advise specifically on aspects such as weight management and renal dietary restrictions if indicated.

Low salt

Low salt intake reduces urinary albumin excretion by 32%.[9] Low salt diets will reduce blood pressure and volume expansion and aid blood pressure control.[9] The Dietary Approaches to Stop Hypertension (DASH) study showed that a diet high in fruits and vegetables and low in animal protein is associated with lower endogenous acid load and thus lower workload for each kidney nephron.[9] Patients with CKD should be encouraged to prepare meals without added salt and avoid processed foods where possible.[9]

Low protein

Low-to-moderate protein diets will mitigate glomerular hyperfiltration and preserve kidney function by lowering intraglomerular hypertension, mitigating renal interstitial fibrosis, and lowering urea and phosphate levels.[4] However, the benefits of protein restriction need evaluation. Current guidelines recommend 0.6-0.8g/kg day when eGFR is <50ml/min to ensure a safe and adequate intake.[4,8] There is a risk of protein-energy wasting and muscle mass and strength loss, particularly in the elderly.

Plant-based diets

The type of protein intake is also as important as the quantity.[8] There is now a focus on plant-based lower-protein diets to help with weight loss and preserve kidney function.[23] Those replacing animal-based proteins with plant-based proteins have shown reductions in the severity of hypertension, hyperphosphatemia and metabolic acidosis.[10] Plant-based proteins contain a form of phosphate called phytic acid, which requires the enzyme phytase to absorb. This enzyme has low activity in humans. Therefore, plant protein sources are suitable for renal dietary phosphate restrictions.[9] Plant-based proteins are not only adequate but have been deemed safe for patients with CKD, including those with proteinuria, so a plant-based diet is more feasible for CKD patients than was previously thought.[10,11]

Vegetarianism

Strict vegetarians can exceed their minimum requirement for protein and consume, on average, 72.3g of protein per day.[10] CKD patients can consume 0.7-0.9g/kg/day of primarily plant-based protein without any adverse effects.[10] The 'biological value' of a protein is considered an antiquated method of measuring protein quality.[10] More sophisticated methods are currently used based on ileal and faecal digestibility.[12] These methods include protein energy efficiency ratio (PER), net protein ratio (NPR), protein digestibility corrected amino acid score (PDCAAS), and digestible indispensable amino acid score (DIAAS).[12]

Omega-3s

There is some evidence that omega-3 fats may slow the progression of kidney disease, but these studies have been small and heterogeneous, so there is not enough evidence yet for agreed guidelines.

Dietary fibre

Dietary fibre plays a role in treatment. High-fibre diets help the growth of commensal bacteria, such as *Bifidobacterium*, and strengthen the gut barrier.[9] In addition, a high-fibre diet facilitates regular stool elimination and excretion of urea and potassium.[9] Urea disrupts the gut protective barrier increasing intestinal permeability and the risk of bacterial translocation.

MAINTAINING NUTRITIONAL STATUS

Patients with CKD are at high risk of developing nutritional disorders, which reduce the quality of life and lead to poor outcomes.[13] Protein-energy wasting (PEW) prevalence increases from <2% in CKD stages 1-2 to 54% in stages 3-5.[14,15] Kidney failure patients can develop a form of cachexia, the pathophysiology of which is similar to PEW and affects the same metabolic pathways as in other chronic diseases such as cancer.[14] The systemic nature of CKD involves stimulation of skeletal muscle protein degradation pathways combined with activation of mechanisms that impair muscle protein synthesis caused by metabolic acidosis, insulin, IGF-1 resistance, cytokines, and inflammation.[16] This persistent imbalance between protein degradation and muscle synthesis results in wasting, which is unlikely to be reversible.[15,17]

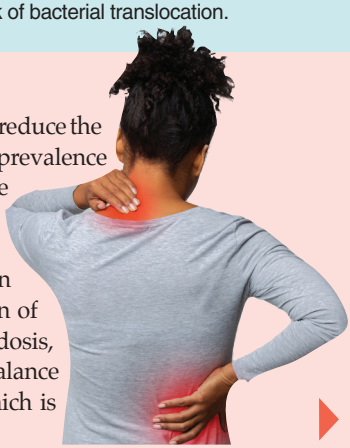


Table 2: Criteria for the clinical diagnosis of protein-energy wasting (PEW) and cachexia in adults with CKD[18]

Criteria	PEW	Cachexia
Serum chemistry	- Serum albumin <38g/L - Serum pre-albumin <300mg per 100mL (for maintenance dialysis patients only) - Serum cholesterol <100mg per 100mL	- Serum albumin <32g/L - Anaemia <12g/dL - Increased inflammatory markers CRP (>5.0mg/L), IL-6 (>4.0pg/mL)
Body mass	- BMI <23kg/m2 - Unintentional weight loss over time at least 5% over three months or 10% over six months - Total body fat percentage <10%	- BMI <20kg/m2 - Unintentional weight loss of at least 5% in 12 months
Muscle mass	- Muscle wasting: reduced muscle mass of 5% over three months or 10% over six months - Reduced mid-arm muscle circumference area (reduction >10% in relation to 50th percentile of reference population)	- Reduction of appendicle skeletal muscle index by DEXA (kg/m2) <5.45 in women and <7.25 in men. - Reduced mid-arm muscle circumference area (<10th percentile for age and gender)

Muscle wasting, known as sarcopenia, is an essential feature in the pathophysiology of cachexia and increases the risk of comorbid complications.[14] Appetite loss has the most robust prognostic power for predicting the risk of PEW.[13] Monitoring of mid-arm muscle circumference and body mass index is required for CKD patients and regular assessment of nutritional status.

ORAL NUTRITIONAL SUPPLEMENTS (ONS)

Treatment with ONS is associated with improved survival rates.[13] Enteral nutrition (EN) is indicated if oral intake (with or without ONS) is not sufficient to meet at least 70% of daily requirements.[13]

ENTERAL AND PARENTERAL NUTRITION

EN and PN are given to critically ill hospitalised patients with acute kidney injury (AKI) or AKI on CKD when they cannot achieve at least 70% of macronutrient requirements with oral nutrition.[13] The European Society of Parenteral and Enteral Nutrition (ESPEN) recommends 30-35kcal/kg/day for hospitalised patients with AKI or AKI on CKD without acute/critical illness and 0.8-1.0g/kg BW/day protein.[13] Using predictive equations and weight-based formulae is problematic in CKD with/without critical illness. The frequent fluid overload in these patients and the inability to distinguish fat from muscle leads to bias and imprecision. No disease-specific EN and PN products are required for AKI and CKD patients. However, the presence of hyperkalaemia, hyperphosphatemia and fluid overload will require a lower electrolyte and energy-dense low-volume feed product.[13] Similarly, common electrolyte ONS products may be more appropriate, but this should not be routinely recommended for all patients with CKD.

DIETARY RESOURCES FOR CKD

A renal dietitian should assess all CKD stage 3-5 patients, and dietary advice should be individualised.[19,20] Those newly embarking on a plant-based diet should consult a renal dietitian to ensure nutritional adequacy.[19] There are many online resources that patients can be directed to for helping them to eat as healthily as possible within the dietary restrictions, including those from the National Kidney Foundation[20] and the Kidney Kitchen.[21]

END NOTE

CKD will likely become more prevalent due to the rise in associated diseases such as diabetes, obesity, hypertension and the growing elderly population. Healthcare resources need to be re-evaluated to ensure adequate treatment modalities are available for those with end-stage renal disease. Continued emphasis must be placed on diet and lifestyle management and prevention of CKD and the importance of the renal dietitian and other healthcare professionals in renal care. They are crucial in helping CKD patients maintain their kidney function for as long as possible.