

## A Few Words About Kidney Damage

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### Abstract

*Kidney damage can be detected early and simply by urine and blood tests and blood pressure measurements. About one in ten adults has some form of kidney disease. It can develop at any age, and chronic kidney disease is becoming more common with increasing age. The kidneys are an extremely important organ for the health of the whole organism. About 200 liters of blood are purified daily. The kidneys remove waste material that is excreted in the urine. Regardless of age, simple and timely treatment can slow the progression of kidney disease, prevent complications and improve quality of life.*

**Keywords:** Kidney, Damage, CKD, Markers

### Introduction

The kidneys are symmetrical organs in humans and are roughly the size and weight of the pig kidneys one might see in a butcher's shop [1]. They lie outside the abdominal peritoneal cavity under the diaphragm (the structure separating the thorax from the abdomen). The right kidney lies lower than the left. Each has an outer portion termed the 'cortex' and an inner one termed the 'medulla'. Within the cortex each kidney contains approximately one million filtering units (glomeruli) comprising a leash of small blood vessels (capillaries) through which blood passes. A large volume of blood – a quarter of cardiac output or approximately 1-1.5 litres-passes through the two million glomeruli each minute. The pressure gradient between the blood within the glomerular capillaries and the space outside them provides the driving force for ultrafiltration of fluid across the glomerular capillary walls. This fluid is virtually free of red blood cells and other formed elements of the blood and protein, but does contain waste products of body metabolism, water and electrolytes. Many drugs (in particular those that are soluble in water) are removed from the body by this process of glomerular filtration. Fine tubules lead from the extracapillary space and dip down into the medulla of the kidney. During the process of passage of fluid through the renal tubules, modification of the contents of the tubular fluid are made by reabsorption into the blood stream or secretion from the blood stream into the tubular fluid. Immediately after the extraglomerular capillary space, the tubule is termed a 'proximal tubule' and further down the system there is a 'distal tubule'. Distal tubules lead into collecting ducts which ultimately open into the main collecting system of the kidneys at the renal papillae. Tubular fluid enters the calyces of the kidney and then passes on into the renal pelvis, down the ureter and into the bladder from where urine is voided. The rate of filtration across glomerular capillaries in the average human being is approximately 180 litres per day. The vast majority of this ultrafiltrate is reabsorbed into the circulation.

Eventual daily urine volume is usually of the order 1-1.5 litres and varies according to fluid intake and losses in sweat, from the gut and by other routes. From this it can be seen that even modest reductions in renal reabsorptive capacity may have a major impact upon the volume of urine produced. It is not surprising that an early feature of kidney damage is the production of an excess volume of urine (polyuria) and the need to get up at night to pass urine (nocturia), although other causes for these symptoms exist as well.

### Functions

The four main functions are:

- Elimination of waste products;
- Maintenance of water and electrolyte balance, in particular the balance of sodium and potassium in the body;
- Maintenance of acid base balance, ie the balance between the concentration of hydrogen ions taken in and eliminated; and
- Production of hormones and enzymes.

### Fluid

Changes in fluid volume and movement have a huge impact in cell size, cardiac output, and even oxygen delivery [2]. Changes in circulating volume and total body water can be acutely compensated in a number of ways including regulating resistance (vasoconstriction, vasodilation), flow (cardiac function), or generating intra-extracellular fluid shifts. These changes, however, will provide a temporary solution to a much bigger problem: what is causing these fluid shifts, is there too much volume, not enough? The brain can tell the body is thirsty by recognizing changes in body fluids, it can help you find water and physically control the process of drinking. Your small intestine can help you absorb water into the bloodstream. But none of this would matter without the kidney. The kidney is the only organ that can regulate the total amount of water and electrolytes in the body.

The kidney is the body's filter. It filters blood to produce urine, which contains the "waste products" found in blood. It achieves this

through three basic functions: filtration, reabsorption, and secretion. Arterial blood will arrive at the kidney through the renal arteries and a series of high-pressure conduits that will aid in filtration. After the blood is filtered (which is a massive and basically unregulated process), a mode of “purification” takes place, that is, reabsorption and secretion. These actions are highly regulated and specific, in that even the most minute ionic concentrations can be altered.

### Cardiorenal Syndrome

Renal dysfunction is a common comorbidity complicating the natural course of heart failure (HF), and similarly patients with kidney disease often present accompanying heart disease [3]. This has led to the concept of cardiorenal syndrome (CRS), which is defined as ‘pathophysiologic disorder of the heart and kidneys whereby acute or chronic dysfunction in one organ induces acute or chronic dysfunction in the other’. Although, guidelines for the management of patients with HF and kidney disease are well developed and regularly updated by the cardiology and nephrology societies, there are no agreed guidelines/recommendations for the management of patients with cardiorenal and/or renocardiac syndromes, as these patients have typically been excluded from clinical trials. In practice, however, such patients are commonly seen by either cardiologists or nephrologists and often become a real clinical challenge. The question which often arises is whether management of patients with HF requires modification for those with impaired renal function and vice versa.

Coronary artery disease and hypertension are the commonest causes of chronic HF, followed by valvular heart disease and cardiomyopathies. Renal dysfunction is a common finding in patients with HF, strongly associated with increased morbidity and mortality. It is estimated that more than 50% of HF patients may have at least moderately impaired renal function and its prevalence increases with HF severity, age, a history of hypertension or diabetes mellitus. Conversely, even mild renal dysfunction constitutes a risk factor for HF development and this association becomes stronger with deterioration in renal function.

The underlying cause of renal dysfunction should always be sought in order to detect potentially reversible causes (such as hypotension and/or dehydration due to drug overdosing, deterioration in renal function due to ACE inhibitors, angiotensin receptor blockers [ARBs] or other concomitant medications, e.g. non-steroidal anti-inflammatory drugs). When coincidental renal disease is suspected, further renal investigations are indicated. Renal dysfunction is not only a consequence of HF, but itself may play a key role in the pathophysiology of the HF syndrome. Thus, in all HF patients renal function should be regularly monitored by measurements of blood urea nitrogen, serum creatinine levels and/or estimation of glomerular filtration rate. Therapy in HF patients with concomitant renal dysfunction is not evidence-based, as these patients are not satisfactorily represented in randomized clinical trials of HF.

HF patients with renal dysfunction often have excessive salt and water retention, which requires more intensive diuretic treatment. In patients with a creatinine clearance of <30 ml/min, thiazide diuretics are less effective and loop diuretics are preferred. Patients may be at a higher risk of further deterioration in renal function when high doses of diuretics are used. More potent options may be diuretic infusions instead of intravenous boluses and a combination of loop diuretic and thiazide/metolazone.

### Chronic Kidney Disease

Chronic kidney disease (CKD), defined as reduced excretory kidney function (glomerular filtration rate (GFR) <60 mL/min/1.73m<sup>2</sup>) or evidence of kidney damage (such as proteinuria) for a period of at least 3 months, is considered a major global public health problem [4]. The prevalence of CKD has been estimated at between 10-15% in industrialised countries and is increasing, likely as a result of population ageing and the increasing incidence of diabetes, vascular disease and obesity.

More recent staging classification systems have attempted to improve CKD risk stratification by incorporating proteinuria. Within the continuum of patients with CKD, there is a wide range of disease severities, from patients with an excellent long-term renal prognosis through to patients with end-stage kidney disease (ESKD) who require renal replacement therapy.

Many patients with CKD follow a predictable clinical course following disease initiation, with progressive renal dysfunction ultimately resulting in ESKD. Critically, CKD is clinically silent in up to 90% of patients until it has reached an advanced stage, and patients who reach ESKD without prior contact with nephrology services experience greater co-morbidity and poorer survival following initiation of renal replacement therapy. There is therefore an opportunity to detect patients with asymptomatic CKD by screening, with the aim of applying therapies to ameliorate disease progression.

Chronic Kidney Disease (CKD) draws heavily on patients’ daily functioning [5]. The disease, treatment and associated demands have a great impact on physical and emotional wellbeing and interfere with patients’ social roles. Patients with CKD who are being prepared for, or receive renal replacement therapy often experience difficulties in participating in various domains of life, such as paid work, sports and other social and leisure activities. For CKD patients on dialysis it seems in particular difficult to perform paid work, and it is notable that people who are being prepared for renal replacement therapy (pre-dialysis patients) already experience work related problems. Restrictions with respect to performing daily activities, including work, might impede people’s feelings of autonomy and self-esteem.

Essential trace elements play a vital role in cellular metabolism and the maintenance of homeostasis, by acting as key cofactors for enzymes [6]. Their intracellular and plasma concentrations are regulated by gastrointestinal absorption and renal and gastrointestinal excretion to prevent deficiency and toxicity. Patients with chronic kidney disease are potentially at risk of both essential trace element deficiencies and toxicity due to the failure to excrete other nonessential elements, leading to accumulation within the kidney which may cause chronic kidney damage, resulting in hypertension, proteinuria, and progression of kidney disease. Environmental exposure to chemical elements varies throughout the world due to differences in the composition of topsoil and surface water. Epidemiologic studies suggest possible linkages between environmental contaminations and increased local populations.

The level of GFR is accepted as the most useful index of kidney function in health and disease [7]. A decrease in GFR precedes the onset of kidney failure; therefore a persistently reduced GFR is a specific diagnostic criterion for chronic kidney disease (CKD). CKD is defined as GFR <60 mL/min per 1.73 m<sup>2</sup> in addition to markers

of kidney damage. The severity of CKD is also determined by the level of GFR. Kidney failure is defined as GFR  $<15$  mL/min per  $1.73$  m<sup>2</sup>. Kidney failure is associated with uremic symptoms and laboratory findings, such as anemia, malnutrition, bone and mineral disorders, neuropathy, and decreased quality of life. There is a graded relationship between the severity of these signs and symptoms at intermediate reductions in GFR in patients with kidney disease. The level of GFR is also associated with progression to kidney failure and cardiovascular disease. In addition, drug dosages will need to be adjusted for the level of GFR.

Decreased kidney function is associated with complications of all organ systems [8]. The major outcomes of CKD, regardless of the specific diagnosis (i.e. type of kidney disease), include progression to kidney failure, complications from decreased kidney function, and development of cardiovascular disease. Increasing evidence shows that early detection and treatment often can prevent or delay some of these adverse outcomes. Referral to a nephrologist depends on practice patterns, which are not uniform across health care system or geographic regions, even within countries. Most cases of nonprogressive CKD can be managed without referral to the nephrologist. One indication that is common to most guidelines is patients with severely decreased GFR (estimated GFR, eGFR,  $<30$  ml/min/ $1.73$  m<sup>2</sup>). There are fewer consensus about referral for patients with higher eGFR. Nephrologists can assist primary care physicians and other specialists in the diagnosis and care of patients at all stages of CKD. This includes determination of the cause of CKD, recommendations for specific therapy, suggestions for treatments to slow progression in patients who have not responded to conventional therapies, identification and treatment for kidney disease-related complications and preparation for renal replacement therapy.

### Markers

Markers of kidney damage include proteinuria, hematuria, and other abnormalities of the urinary sediment, and radiologic evidence of damage [9]. The most common cause of CKD in adults are diabetes and hypertension, and, therefore, the most common marker for kidney damage is increased excretion of protein, and specifically of albumin. Measurement of protein excretion is useful in a variety of clinical settings, particularly to establish the diagnosis and to follow the course of glomerular disease.

In normal subjects, low molecular weight proteins and small amounts of albumin are filtered. The actual amount of albumin filtered each day in humans is controversial. The majority view is that no more than about 2 to 4 g of albumin per day are filtered normally, but some investigators claim that as much as 200 g of albumin are filtered each day (with the bulk of this filtered albumin “reclaimed” in the early proximal tubule). The filtered proteins enter the proximal tubule where they are almost completely reabsorbed and then catabolized by the proximal tubular cells. Some of the catabolized proteins (including albumin) are excreted as peptides in the urine. These are not detected by dipstick or the immuno-nephelometric albumin-specific assays, but are detected by chromatographic assays. The net result is the normal daily protein excretion of less than 150 mg (usually 40 to 80 mg), of which approximately about 4 to 7 mg is intact, immuno-reactive albumin.

### Proteinuria

Proteinuria is an early marker of kidney damage in many forms of renal disease, such as diabetic nephropathy and glomerulonephritis

[4]. Persistent proteinuria has a strong positive correlation with the subsequent development of ESKD. Proteinuria was the strongest predictor of subsequent need for dialysis, with an adjusted odds ratio (OR) of 14.9 (95% confidence interval (CI) 10.920.2). The presence of proteinuria at the time diabetes was identified was the strongest risk factor for reaching ESKD (relative risk (RR) 12.1, CI 4.3-34). There is also evidence from controlled trials that proteinuria is a risk factor for CKD progression. In the modification of diet in renal disease (MDRD) trial, there was a positive correlation between baseline proteinuria and the rate of decline in GFR. This association was independent of other risk factors for decline in GFR such as blood pressure.

### Ultrasound

Ultrasound examinations are used extensively now in the investigation of renal, ureteric, bladder, prostatic, and scrotal pathology [10]. They may be regarded as an extension of examination. Whether an ultrasound examination is undertaken by an ultrasonographer, radiologist, or urologist, the person who undertakes the examination has the advantage of seeing the images in real time, while the doctor has only a few still images. The report thus is of prime importance, and the skill of the person who undertakes the examination is paramount. Limitations of ultrasound vary in different situations.

In the kidney, ultrasound is better than computed tomography at identifying renal cysts, but it may fail to distinguish between parapelvic cysts and hydronephrosis. Although renal stones may give the classic appearance of a bright echo with a black shadow behind, this is not always the case. Ultrasound is a poor way of screening for renal stones. Assessment of the size of a stone using ultrasound is not very accurate. On occasions, if a stone fills the renal pelvis or the entire collecting system, it is possible to miss it on ultrasound. If the patient is obese, ultrasound becomes more difficult.

### Conclusion

Kidney disease affects overall health and kidney failure is life-threatening. One of the healthiest ways to maintain good kidney health is with foods rich in antioxidants and nutrients. Symptoms of kidney damage often go unnoticed because they usually do not produce pain. If kidney damage is detected early, treatment will be applied that can slow or stop further kidney damage and even repair the damage. Chronic kidney disease and the risk of loss of renal function can be detected at an early stage by examination of urine and blood. Protein in the urine indicates the risk of deterioration of the kidney, and from creatinine in the blood can be calculated the degree of existing kidney damage. The primary goal of treatment is to reduce protein leakage in the urine by choosing blood pressure medications. All other kidney-preserving measures will be applied. People with kidney disease must regularly check their health status.

### References

1. Baker L (1998) Nephrology, Cavendish Publishing Limited 1-5.
2. Arroyo JP, Schweickert AJ (2013) Back to Basics in Physiology-Fluids in the Renal and Cardiovascular Systems, Academic Press, Elsevier, Oxford, 77.
3. Ponikowski P, Ronco C, Anker SD (2010) Cardiorenal syndromes--recommendations from clinical practice guidelines: the cardiologist's view Contrib Nephrol 165: 145-152,
4. Francis R, Johnson D (2012) Screening for Chronic Kidney Disease. Chronic Kidney Disease and Renal Transplantation, InTech pp 1-16.

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5. Jansen DL, Rijken M, Heijmans JWM, Kaptein AA, Groenewegen PP, et al., (2012) Psychological and Social Aspects of Living with Chronic Kidney Disease. Chronic Kidney Disease and Renal Transplantation, Prof. Manisha Sahay (Ed), InTech pp 1-30.
  6. Davenport A (2020) Trace elements in chronic kidney disease. In Chronic renal disease, Elsevier pp 703-717.
  7. Stevens LA (2012) Measurement of Glomerular Filtration Rate. Nephrology Secrets, Third Edition, Lerma EV, Nissenson A (Ed) Elsevier, pp 26.
  8. Malovrh M (2015) Patients with chronic kidney disease: safety aspects in the preoperative management. Contrib Nephrol 184: 13-23.
  9. Kazmi W H, Danial K (2012) Chronic Kidney Disease Update. Topics in Renal Biopsy and Pathology, Prof Muhammed Mubarak (Ed), InTech, pp 200-201.
  10. Whitfield HN (2006) ABC of urology: Urological evaluation. BMJ 333: 432-5.

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