Fluid and electrolyte problems in renal dysfunction

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Learning objectives

After reading this article, you should be able to:

- explain albumin:creatinine ratio
- describe how to treat hyponatraemia of renal disease
- list six treatments of hyperkalaemia

Abstract

The primary function of the kidney is to maintain fluid and electrolyte homeostasis. Each day the kidney must excrete 1500 ml of water and any excess ingested sodium, potassium, magnesium and phosphate. The kidney also plays a key role in calcium homeostasis. Of the total number of patients in intensive care 3–25% will develop acute kidney injury and patients with chronic renal disease will frequently present for surgery. The treatments for renal dysfunction may themselves cause disturbance in fluid and electrolyte homeostasis, particularly the use of diuretics and renal replacement therapy. Loss of normal renal function may lead to major changes in fluid and sodium balance. Volume status assessment will be required in patients with renal dysfunction because they are at risk of both hypovolaemia and hypervolaemia. Loss of renal function will lead to retention of potassium and magnesium with serious systemic and cardiovascular consequences including cardiorespiratory compromise and collapse and cardiac dysrhythmias. Hypocalcaemia will result from reduced renal calcitriol production and phosphate retention. The identification of fluid and electrolyte imbalances and the management of clinically significant changes are required when treating patients with renal dysfunction on an intensive care unit or who present for anaesthesia.

Keywords Calcium; electrolyte; magnesium; potassium; renal; sodium

Royal College of Anaesthetists CPD matrix: 1A02, 1A01, 2A05, 3A03

Renal dysfunction: prevalence and morbidity

Renal dysfunction results in the loss of fluid and electrolyte homeostasis and the accumulation of potentially toxic products of metabolism. Problems may arise as a result of chronic renal dysfunction or as a result of acute renal failure.

Acute kidney injury (AKI) occurs rarely in the community with an incidence of between 140 and 288 per million. It occurs in up to 15% of adults admitted to hospital with the elderly being particularly at risk. The incidence in intensive care patients is much higher and is quoted as 3–25%. Occurrence is quoted at 5–7% of all hospitalized patients to varying degrees. An NHS study has shown severe kidney injury to have a mortality of 30–40%. Many definitions have been applied to AKI but the RIFLE criteria proposed by Bellomo et al.[1,2] have now gained general acceptance.

Chronic renal disease (CRD) has been defined as either a glomerular filtration rate of less than 60 ml/minute/1.73 m^2 for 3 months or more, irrespective of cause, or evidence of kidney damage for more than 3 months based on abnormalities. The incidence of CRD is increasing and more than 550 people/million receive renal replacement therapy (RRT) in the UK which accounts for over 2% of the NHS budget. NICE has suggested that chronic renal disease should be classified to include patients with kidney damage who are at risk of progressing to failure (Table 1). In 2013, the Kidney Disease: Improving Global Outcomes (KDIGO) guidance on the evaluation and management of chronic kidney disease adopted the subdivision of glomerular filtration rate (GFR) categories suggested by the NICE guideline, but also included three albumin:creatinine ratios (ACRs) for each GFR category in an updated classification[3] (Table 2).

Early recognition and treatment of patients at risk of developing AKI on a background of chronic kidney disease (CKD) is extremely important. AKI in CKD patients can lead to a higher mortality than AKI in non-CKD patients and it can also alter the progression of the underlying disease process. Progression to end-stage renal failure has been found to be higher in patients who develop AKI in CKD compared to non-CKD.[4]

Renal dysfunction in the postoperative period

Renal dysfunction and AKI in the postoperative period continues to be a problem. Historically aneurysm repair and cardiac surgery requiring cardiopulmonary bypass have represented a significant risk. Endovascular techniques such as endovascular repair of the aorta and transcutaneous insertion of aortic valves are associated with substantial contrast nephropathy. They are also at risk of micro- and macro-embolization and changes in renal blood flow patterns. The creatinine rise after these insults can be slow and is defined as AKI if there is a 25% relative rise 48 hours after administration of contrast. Prevention measures that have been suggested include hydration, use of statins, antioxidants such as n-acetyl cysteine (orally or as infusions), ascorbic acid and alkalinizing the urine with sodium bicarbonate.

Other high-risk patients are those with existing CKD, patients undergoing emergency surgery who have a degree of AKI at the time of presentation and sepsis.[5]

The most prominent response to anaesthesia and surgery is water and sodium retention which is directly related to the magnitude of surgery. During surgery ADH levels increase 50–100-fold and start decreasing towards the end of surgery or

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Stages of chronic kidney disease (NICE)

Stage	GFR (ml/minute/1.73 m^2)	Description
1	≥90	Normal or increased GFR, with other evidence of kidney damage
2	60–89	Slight decrease in GFR, with other evidence of kidney damage
3A	45–59	Moderate decrease in GFR, with or without other evidence of kidney damage
3B	30–44	Moderate decrease in GFR, with or without other evidence of kidney damage
4	15–29	Severe decrease in GFR, with or without other evidence of kidney damage
5	<15	Established renal failure

GFR, glomerular filtration rate.

Table 1

within 3–5 days. Other reasons for fluid retention may be effects of anaesthesia on renal blood flow, GFR, effects of hypotension and hypovolaemia, increased circulating catecholamines and increased plasma cortisol. To determine whether water imbalances are due to deficiency (diabetes insipidus (DI)) or excess of ADH (syndrome of inappropriate antidiuretic hormone secretion (SIADH)), fluid intake, output, specific gravities and serum electrolytes should be monitored daily. DI occurs more frequently than hyponatraemia, and is characterized by large amounts of dilute urine which can lead to hypernatraemia and may require treatment with desmopressin. SIADH peaks on postoperative day 7 and can present with severe and symptomatic hyponatraemia. Management is mainly by fluid restriction.

Fluid management in CKD

CKD and congestive heart failure (CHF) are closely associated (cardio-renal syndrome) and make fluid management difficult in these patients with conflicting treatment goals.

Preoperative renal dysfunction is a predictor of postoperative renal failure.[6] A preoperative serum creatinine of more than 176 mmol/litre is an independent predictor of systolic and diastolic dysfunction and is associated with major cardiac complications in 9% of cases. CKD is a stronger predictor of mortality in advanced CHF than left ventricular function.

Albumin:creatinine ratio (KDIGO)

ACR category	ACR (mg/mmol)	Terms
A1	<3	Normal to mildly increased[a]
A2	3–30	Moderately increased[b]
A3	>30	Severely increased

ACR, albumin:creatinine ratio; CKD, chronic kidney disease.
[a] Relative to young adult level.
[b] Including nephrotic syndrome (ACR usually >220 mg/mmol).

Table 2

Impacts of treatment of renal dysfunction

Patients with chronic renal disease are frequently on multiple medications[7] to help maintain homeostasis and to treat associated conditions such as hypertension, cardiac disease, and diabetes. While some of these treatments such as bicarbonate and vitamin D supplementation are aimed at restoring normal homeostasis, these and other medications may cause electrolyte and fluid disturbance. Diuretics in particular may result in significant changes in body water, sodium, potassium, and acid/base balance. Care must be taken in particular to medications controlling blood pressure as these can have a profound effect when combined with anaesthesia in the perioperative period.

Patients requiring RRT, particularly haemodialysis, may have profound changes in fluid, electrolyte and acid/base balance at the time of treatment. In such patients total body water status is best estimated by body weight changes. These will be recorded regularly in dialysis patients and many patients will know their own 'dry weight'. It is important for the anaesthetist to know the normal dry weight and the weight just prior to surgery. Failure to achieve dry weight is a common problem especially on short-duration dialysis prescriptions.

Blood electrolyte levels should be measured regularly in patients who are receiving RRT in critical care or those on long-term RRT who require anaesthesia.

Water

Water balance is severely affected by renal dysfunction. Patients with CKD are at risk increased risk of fluid overload and this may be a stimulus for inflammation and rapid progression of renal disease.[8] Patients with oliguria and anuria are at risk of water overload as they will lose only about 1000 ml per day from insensible and other losses. Daily fluid restriction is frequently used to control water levels in such patients. Diuretics are often used to increase fluid excretion in those with some remaining renal function. Conversely, these patients are at risk of dehydration if other water losses increase such as increased insensible loss in sepsis or in patients with diarrhoea. Over-aggressive fluid removal with RRT may also result in dehydration.

Some patients will develop polyuric renal failure (particularly during recovery from AKI). In these patients dehydration may develop if intake is not increased to compensate.

Assessment of hydration status is always required in patients with renal failure. If normal body weight (or 'dry weight' in dialysis patients) is known then weighing the patient will give an idea of hydration status. Each 1000 ml of water weighs 1 kg. Daily weights should be conducted on intensive care patients with renal failure. History of fluid restriction, urine output, RRT, and fluid balance should be taken. Examination may reveal signs of fluid overload or dehydration. Attention should be paid to hydration of mucus membranes, jugular venous pulse, and capillary refill, presence of oedema, blood pressure, heart rate and respiratory symptoms.

Mild dehydration may be treated with oral rehydration. If the enteral route is unavailable or the level of dehydration is more profound intravenous fluid will be required. The nature of fluid