Fluid and electrolyte problems in renal dysfunction

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Learning objectives

After reading this article, you should be able to:

- describe the stages of acute kidney injury
- describe the stages of chronic kidney disease
- list the priorities in treating hyperkalaemia
- describe the management of hyponatremia in renal disease

Abstract

Nephrons are the structural and functional units of the kidneys. The nephrons affect changes to blood plasma via filtration, reabsorption, secretion and excretion. Through these mechanisms the kidneys maintain homeostasis of electrolyte concentrations, fluid volume, osmolality and acid–base balance. In addition to the work of the nephrons, the kidneys have further roles in calcium homeostasis and synthesize the hormones erythropoietin and renin. Acute injury and chronic failure of the kidneys can impact on the kidneys ability to maintain homeostasis and manage fluids and electrolyte balance effectively. This poses challenges to the anaesthetist, perioperative physician and the intensivist in maintaining homeostasis and preventing further injury or damage to the kidneys following surgical or medical stresses. Derangement of electrolytes can be fatal. Recognition and prompt treatment of these abnormalities are essential.

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

Royal College of Anesthetists CPD Matrix: 1AO1, 1AO2, 2AO5, 2A06

Acute kidney injury

Acute kidney injury was previously known as acute renal failure and describes a spectrum of injury to the kidney and not only kidney failure. The definition of acute kidney injury has been subject of debate and change over recent years but is now based mostly on serum creatinine levels with or without the component of measured urine output (Table 1). Acute kidney injury, due to increased vigilance and awareness is now seen in primary care, whereas it was formerly almost exclusively found in secondary care.

The 2009 National Confidential Enquiry into Patient Outcome and Death (NCEPOD)[1] identified potential suboptimal care in patients with acute kidney injury in hospital. It found deficiencies in prevention, recognition, therapy and timely referral to specialist services. Subsequently the UK Department of Health directed the National Institute for Health and Care Excellence (NICE) to develop its first guidelines on acute kidney injury.[2]

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The inpatient mortality associated with acute injury varies significantly based on severity, patient factors and patient admission to an intensive care unit, but is typically 25–30% or more in the United Kingdom.

Chronic kidney disease

Chronic kidney disease (CKD) is an abnormality of the kidney structure or function, present for a period of 3 months or longer. It is often asymptomatic or may present with non-specific symptoms such as fatigue or anorexia. It is often diagnosed in screening patients at risk of CKD, such as those with hypertension or diabetes. CKD is common and affects between 5% and 10% of the world population. Those patients with established CKD are at an increased risk of AKI, particularly in the inpatient setting.

CKD is classified based on cause, glomerular filtration rate (based on serum creatinine) and presence and degree of albuminuria. CKD is classified into 5 GFR categories: G1, G2, G3a, G3b, G4 and G5 (Table 2). There are also three albuminuria categories: A1, A2 and A3. A1 represents a urine albumin to creatinine ration (ACR) of <3 mg/mmol. A2 represents an ACR of 3–30 mg/mmol and A3 is present when the ACR is >30 mg/mmol.

Kidney disease improving global outcomes acute kidney injury staging[6]

Stage	Serum creatinine	Urine output
1	1.5–1.9 times baseline OR ≥0.3 mg/dl (≥26.5 μmol/l) increase	<0.5 ml/kg/h for 6–12 hours
2	2.0–2.9 times baseline	<0.5 ml/kg/h for ≥12 hours
3	3.0 times baseline OR increase in serum creatinine to ≥4.0 mg/dl (≥353.65 μmol/l) OR Initiation of renal replacement therapy OR, In patients <18 years decrease in eGFR to <35 ml/min per 1.73m^2	<0.3 ml/kg/h for ≥24 hours OR Anuria for ≥12 hours

Table 1

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Kidney disease improving global outcomes GFR categories[7]

GFR category	GFR (ml/min/1.73 m^2)	Terms
G1	>90	Normal or high
G2	60–89	Mildly decreased
G3a	45–59	Mildly to moderately decreased
G3b	30–44	Moderately to severely decreased
G4	15–29	Severely decreased
G5	<15	Kidney failure

Table 2

Fluid balance

Fluid balance is significantly affected by renal dysfunction. Fluid overload can become a feature of CKD and can become a stimulus for inflammation and rapid progression of renal disease. Fluid overload is particularly problematic in patients with co-existing cardiac failure as the increased preload can quickly cause pulmonary oedema, and a spiral of worsening heart failure, decreased renal perfusion and increasing oedema can ensue.

Anuric and oliguric patients are particularly at risk of developing fluid overload. Haemodialysis patients are often fluid restricted to 1–1.5 litres a day which accounts for the insensible losses of around 1 litre a day. In patients with some remaining renal function, diuretics are often used to prevent fluid overload. These patients are at risk of dehydration if any intercurrent illness such as diarrhoea or sepsis increases their insensible losses.

Following acute kidney injury, a polyuric phase may be encountered where the water re-absorption of the kidney is impaired along with its electrolyte excretion and re-absorption. These patients are also at risk of dehydration if intake is not increased to match the polyuria.

Hydration status is particularly important in patients with renal impairment. Assessing hydration status can be challenging, and clinical examination should include mucous membranes, skin turgor, jugular venous pulse, chest auscultation, heart rate, blood pressure and oedema status. Confounders to these finding such as hypo-albuminaemia, heart failure and cardiovascular drugs should be considered and reliance on one indicator should be avoided. If the patients normal weight (or 'dry weight' in dialysis patients) is known, then current weight can be a useful guide in determining hydration status. Each litre of water will account for 1 kg above or below 'dry' weight.

Mild dehydration is best corrected using the enteral route. If it is not available or the dehydration is more severe then intravenous fluids should be used. Administration of 0.9% Na^+Cl^- is associated with hyperchloraemic acidosis that may worsen already present metabolic acidosis and can worsen hyperkalaemia. Balanced crystalloid solutions are therefore preferred for rehydration in patients with some remaining renal function. Potassium-containing fluids are usually avoided in anuric patients but can be used cautiously with close monitoring of serum electrolytes.

Diuretics can be helpful in treating fluid overload and often high doses of up to 500 mg of furosemide are required to produce an adequate diuresis. This can be appropriate for fluid overload in chronic kidney disease under specialist guidance, but diuretics should not be used routinely in acute kidney injury and particularly where the cause of renal failure is unknown. Haemodialysis is a rapid and predictable correction of fluid overload. Haemofiltration will remove fluid more slowly but is generally better tolerated from a cardiovascular perspective and easier to deliver to otherwise pre-dialysis patients.

Sodium

Sodium excretion by the kidneys is a function of GFR. Serum sodium levels in renal dysfunction is usually related to fluid shift, and those patients are at risk of both hypo- and hypernatremia. In ascertaining the cause and management of sodium disturbance an assessment of volaemic status is essential.

Hypontraemia (<135 mmol/l) is a common electrolyte abnormality in hospital patients, ranging from 5–30%. Hypernatremia (>145 mmol/l) is much less common, ranging from 1–4%. CKD patients follow a similar pattern of sodium abnormalities. Hyponatremia results from disproportionate sodium loss relative to free water loss or excess free water ingestion. The kidney reabsorbs 99% of filtered sodium, but in patients with renal dysfunction with normal or high urine volumes, a loss of sodium reabsorption capacity may result in hyponatremia with or without hypovolaemia. Polypharmacy and limited nutritional solute intake also contribute towards sodium derangements.

Assessing volaemic status in hyponatremia is essential. In the hypovolaemic patient urinary sodium in helpful in ascertaining a cause. Urinary Na^+ >20 mmol/l is found in the diuretic phase of renal failure, nephrocalcinosis and medullary cystic disease. It is also found with diuretic excess, osmolar diuresis (from uraemia or hyperglycaemia) or with Addison's disease.

A urinary sodium <20 mmol/l is due to excess sodium loss along with water from non-renal sources, such as diarrhoea, vomiting, fistulae, burns and small bowel obstruction.

In the hyponatremic patient with oedema nephrotic syndrome, cardiac failure, liver failure and renal failure should be considered.

In the hyponatremic patient who is clinically euvolaemic then water overload, severe hypothyroidism or glucocorticoid insufficiency is likely unless the urine osmolality is >500 mmol/kg and SIADH is the probable cause.

Hyponatremia is often grouped into mild (serum Na^+ 130–134 mmol/l), moderate (serum Na^+ 120–129 mmol/l) or severe (serum Na^+ <120 mmol/l). Sinister symptoms of hyponatremia such as confusion seizures, coma and respiratory arrest are much more common in severe hyponatremia but should prompt urgent treatment even if serum sodium in >120 mmol/l. These symptoms are uncommon in chronic hyponatremia.

The goals of treatment should be to prevent progressive hyponatremia, decrease intracranial pressure and treat the symptoms of hyponatremia. Furthermore, avoidance of excessively rapid correction of hyponatremia is key to prevent osmotic demyelination syndrome (ODS). ODS is also known as central pontine myelinolysis and is characterised by acute paralysis, dysphagia, dysarthria and other neurological symptoms. The

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