Preoperative assessment for patients with renal impairment

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Abstract

The kidneys play an essential role in homeostasis. They regulate crucial physiological variables including blood pressure, acid-base, fluid and electrolyte balance. With an ageing and increasingly diabetic population, the prevalence of chronic kidney disease is increasing. Acute kidney injury is also very common in hospitalized patients. It is therefore essential to identify patients with renal impairment and assess its impact on a patient's physiology, with aberrancies such as anuria, metabolic acidosis and hyperkalaemia all being potentially life-threatening if untreated. It is these physiological derangements that lead to the observed association of renal impairment with an increased perioperative morbidity and mortality. Prevention, identification and correction of the physiological consequences of renal impairment are vital in improving postoperative outcomes in these patients. This article aims to provide a context and structure for the preoperative assessment of patients with renal impairment irrespective of its cause.

Keywords Acute kidney injury; chronic kidney disease; preoperative assessment; renal replacement therapy

Royal College of Anaesthetists CPD Matrix: 1A02, 2A03, 2A05, 2A07, 3I00

Learning Objectives

After reading this article, you should know:

- the definitions, causes and staging of both acute kidney injury and chronic kidney disease
- how to perform a structured preoperative assessment of a patient with renal impairment whilst considering the potential complications that may occur
- how to avoid precipitants of acute kidney injury whilst having the abilities to plan a treatment strategy for the consequences of an acute deterioration in renal function

Anatomy and physiology

The kidneys are symmetrically paired organs located in the retroperitoneal space. They receive their blood supply from the renal arteries that branch from the aorta and their venous drainage is via the renal veins that insert into the inferior vena cava. The kidneys receive approximately a quarter of cardiac output and therefore urine output can be a useful indirect marker of end-organ perfusion in a previously healthy individual. Each kidney has approximately one million nephrons, which filter blood leading to the production of urine that leaves the nephron via the collecting ducts. This urine then flows down the ureters into the bladder ready for micturition.

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The kidneys have multiple physiological roles ranging from toxin excretion, homeostasis and hormone synthesis.

Clearance of metabolic waste products

Renal blood flow passes through the glomeruli producing an ultrafiltrate driven by locally mediated arteriolar pressure and concentration gradients. There follows selective reabsorption distally of large amounts of sodium and water and subsequent clearance of toxic metabolites such as urea, creatinine, and multiple drugs breakdown products. The production of this original ultrafiltrate is represented by the estimated glomerular filtration rate (eGFR). The eGFR is calculated by using an individual's serum creatinine and adjusting for age, ethnicity and gender, but not for their size, muscle mass or dietary intake.[1] Creatinine is a product of creatine phosphate metabolism in muscle and therefore patient size, muscle mass and dietary intake can significantly alter its level impacting on the accuracy of the eGFR.

Fluid balance and blood pressure

The renin–angiotensin–aldosterone system (RAAS) is coordinated by the juxtaglomerular cells of the kidney. A reduction in renal blood flow of any cause leads the secretion of renin which in turn converts plasma angiotensinogen into angiotensin I. Angiotensin I is subsequently converted into angiotensin II, which both induces vasoconstriction and stimulates adrenal secretion of aldosterone. Aldosterone then stimulates sodium and water reabsorption, which increases blood volume, blood pressure and ultimately renal perfusion. Various antihypertensive medications block specific steps of this pathway as part of their mechanism of action.

Acid–base balance

The kidneys have a vital role in tandem with the lungs in maintaining an optimal serum pH. The pH is regulated via the variable secretion of protons and fixed acids and/or via bicarbonate excretion. In health the kidneys are key in compensating for acid–base imbalance; however, in renal disease, failure of this homeostatic mechanism itself can result in a metabolic acidosis.

Electrolyte homeostasis

Excretion and reabsorption via tubular symporters, anti-porters and active transporters enables the maintenance of key serum

Please cite this article in press as: Rey de Castro N, Bannard-Smith J, Preoperative assessment for patients with renal impairment, Anaesthesia and intensive care medicine (2018), https://doi.org/10.1016/j.mpaic.2018.02.006

electrolytes. Sodium, potassium, phosphate and calcium are intimately regulated via these mechanisms. Many patients with chronic kidney disease will suffer or need treatment for these electrolyte imbalances. Two such examples are the use of low phosphate/potassium diets or renal replacement therapy to remove excess serum potassium.

Vitamin D

The kidneys have a key role in bone mineralization via their influence on serum calcium reabsorption, phosphate excretion, and vitamin D3 synthesis. The body synthesizes vitamin D3 from 7-dehydrocholesterol. UV radiation converts 7-dehydrochloesterol into cholecalciferol in the skin. The liver then hydroxylates cholecalciferol into 25-hydroxycholecalciferol via the enzyme 25-hydroxylase before a final hydroxylation by 1-alpha-hydroxylase in the kidneys produces 1,25-dihydroxycholecalciferol (vitamin D3). Vitamin D3 is the active form, which increases gastrointestinal calcium absorption, renal calcium re-absorption and osteoclast activation leading to the release of mineralized calcium from bone. Marked derangements of vitamin D synthesis as well as calcium and phosphate homeostasis are seen in chronic renal disease.

Erythropoietin

Interstitial cells in the kidney produce erythropoietin (EPO) at a background rate. This rate increases in response to a persistent impairment in oxygen delivery to the renal interstitium. EPO stimulates erythropoiesis in the bone marrow leading to an increase in serum haemoglobin and therefore oxygen carrying capacity of circulating blood.

Renal impairment

Acute kidney injury (AKI)

Renal impairment can be classified as either acute or chronic. AKI is characterized by a sudden deterioration in renal function irrespective of the cause. The Kidney Disease- Improving Global Outcomes (KDIGO) group have produced an international consensus on the definition and severity classification for AKI[2] (see Table 1). The KDIGO consensus is the most recent of AKI definitions following previous publications by AKIN (Acute Kidney Injury Network, 2004) and the RIFLE (Risk, Injury, Failure, Loss of kidney function, and End-stage kidney disease, 2002) groups.[3,4] KDIGO define AKI by any of the following features:

- an increase in serum creatinine by $\geq$ 26.5 μmol/l with 48 hours
- an increase in serum creatinine by $\geq$ 1.5 times their baseline within the prior 7 days
- urine output <0.5 ml/kg/hr for 6 hours

AKI is extremely common in the inpatient setting. Meta-analysis has shown that approximately one in five adults develop an AKI during an acute admission to hospital and its occurrence is associated with increased mortality.[5] Causes of AKI have classically been divided into those that are pre-renal, renal or post-renal (Table 2). Pre-renal causes are the most common and are related to the impaired perfusion of the kidney. Renal causes are less common and are driven by intrinsic renal pathology, and post-renal causes are associated with the obstruction of urinary outflow from the kidneys leading to a renal insult.

KDIGO 2012[2] severity staging for acute kidney injury

Stage	Serum creatinine	Urine output
1	≥26.5 μmol/l **or** 1.5–1.9 times baseline	<0.5 ml/kg/hr for 6–12 hours
2	2.0–2.9 times baseline	<0.5 ml/kg/hr for ≥12 hours
3	≥353.6 μmol/l **or** 3.0 times baseline **or** Initiation of renal replacement therapy **or** In those <18 years, a decrease in eGFR <35 ml/min per 1.73 m^2	<0.3 ml/kg/hr for ≥24 hours **or** Anuria ≥12 hours

Table 1

Causes of acute kidney injury

	Cause
Pre-renal	**Hypoperfusion** • Hypovolaemic shock • Cardiogenic shock • Distributive shock **Renal artery disruption** • Stenosis • Embolism • Dissection • Surgical clamping
Renal	**Acute Tubular Necrosis** • Ischaemic • Nephrotoxic **Acute Glomerulonephritis** **Acute Interstitial Nephritis** **Vasculitis**
Post-renal	**Intra-luminal** • Renal stones • Blood clot • Tumour **Intra-mural** • Oedema • Stricture **Extra-mural** • Prostatic enlargement • Large pelvic mass

Table 2

Contrast induced AKI (CI-AKI) has previously been cited as a common cause of AKI in hospitalized patients. However, this belief has recently been challenged by evidence that the risk of AKI following CT scan is independent of contrast.[6–8] Many patients labelled as suffering CI-AKI possess other risk factors for AKI and more epidemiological research in this area is needed.[2] It

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