EDORIUM Journals

Edorium J Anesth 2018;4:100015A05AM2018.
www.edoriumjournalofanesthesia.com

REVIEW ARTICLE PEER REVIEWED | OPEN ACCESS

Anaesthesia in patients with renal failure

Alice Myers, Zoka Milan

ABSTRACT

Anaesthetists need a working understanding of the implications of renal impairment for the safe conduct of anaesthesia. Patients with renal failure are often encountered when managing elective or emergency surgical cases. In addition, it is fundamental to consider how kidney injury frequently forms part of the clinical picture during resuscitation and optimisation of acutely unwell patients in the emergency department or critical care. The functions of the kidney are multiple, and beyond the scope of this paper. The aim of this review is to summarise current classifications of renal impairment and explore the evidence underpinning relevant practice in anaesthesia. Patient groups include those with chronic renal failure, patients with acute renal failure, and also patients with normal preoperative renal function but who have a high risk of developing peri-operative kidney injury. There is a dearth of strong evidence to guide anaesthetic practice with regards to renal failure. As a result, many of the studies quoted here are small cohorts, case series, or case reports. In general, anaesthetists are still mainly guided by their knowledge of physiology, pharmacology and pathology to safely manage patients with kidney injury.

Keywords: Acute kidney injury, Anaesthesia, Chronic kidney injury

Alice Myers[1], Zoka Milan[2]

Affiliations: [1]Consultant in Anaesthesia and Intensive Care Medicine, Department of Anaesthesia, East Surrey Hospital, UK; [2]Consultant Anaesthetist, Honorary Senior Lecturer, Visiting Professor, Department of Anaesthesia, King's College Hospital, London, UK.

Corresponding Author: Alice Elizabeth Myers, 1 Marlborough Mews, Brixton. London. SW2 5TE; Email: alicemyers@doctors.org.uk

Received: 13 January 2018
Accepted: 10 May 2018
Published: 04 June 2018

How to cite this article

Myers A, Milan Z. Anaesthesia in patients with renal failure. Edorium J Anesth 2018;4:100015A05AM2018.

Article ID: 100015A05AM2018

****

doi: 10.5348/100015A05AM2018RA

INTRODUCTION

Kidney dysfunction is common amongst patients encountered by anaesthetists in various contexts. Acutely, a decrease in blood flow to the kidney can be caused by a multitude of problems: a fall in cardiac output can result from dehydration (particularly in elective patients who have been starved and possibly undergone pre-operative bowel preparation, or those with an acute abdomen leading to vomiting etc), fluid distribution problems (septic states or hypoproteinaemia), or cardiogenic shock. Furthermore, chronic kidney disease may have already been identified by doctors in the community, particularly in elderly and diabetic populations.

Patients with renal failure present multiple challenges for the anaesthetist – for instance, vascular access may be difficult, there may be significant co-morbidities (e.g. cardiovascular disease and diabetes), and drug metabolism can be altered, of fundamental importance is the need to try and preserve remaining renal function and avoid further deterioration. In order to manage these patients optimally, the anaesthetist should have an understanding of renal impairment.

The presence of kidney damage or decreased function for three months or more distinguishes chronic kidney disease (CKD) from acute kidney disease. CKD has been estimated as having a prevalence of between 6.76–8.5% in the adult population in the UK [1]. The estimated prevalence of acute kidney injury (AKI) in the UK varies from 172 to 630 per million population, and 5 to 20% of the critically ill [2].

Understanding the terms

Acute kidney injury (AKI): AKI has been defined by various scoring systems. The RIFLE system was proposed by The Acute Dialysis Quality Initiative (ADQI) group in 2004 and was the predominant classification system used for several years [3]. This stratified AKI as follows:

- Risk: 1.5 x increase in Creatinine or GFR decreased by >25%, urine output <0.5 ml/kg/hr for 6hrs;
- Injury: 2 x increase in creatinine or GFR decreased by >50%, urine output <0.5 ml/kg/hr for 12 hrs;
- Failure: 3 x increase in creatinine or GFR decreased >50%, urine output < 0.3 ml/kg/hr or anuria for 12 hrs;
- Loss: complete loss of kidney function >4 weeks;
- End stage: loss for >3 months.

This was modified by The Acute Kidney Injury Network (AKIN) in 2007 [4].

- Stage 1: creatinine >0.3 mg/dL or increased by factor of 1.5 to 2, urine output <0.5 ml/kg/hr >6 hrs;
- Stage 2: >2 to 3 x increase in creatinine, urine output <0.5 ml/kg/hr >12 hrs;
- Stage 3: 3 x increase in baseline creatinine, urine output <0.3 ml/kg/hr or anuria for 12 hrs.

The most recent and widely used definition comes from the Kidney Disease: Improving Global Outcomes (KDIGO) group [5]. AKI is defined as an increase in serum creatinine by more than 0.3mg/dl within 48 hours; an increase to more than 1.5 times baseline known or presumed to have occurred within the prior seven days; or urine volume less than 0.5 ml/kg/hr for 6 hours.

This is then further staged for severity according to the degree of increase in serum creatinine and the decrease in urine output.

Each of these systems has been subjected to criticism. For instance, the KDIGO system does not take into consideration the multiple aetiologies that can cause AKI, the use of urine output as part of the scoring criteria can be misleading, and baseline creatinine value information is not available in all patients. However, in spite of their flaws, these systems of classification encourage clinicians to be alert to the development of kidney injury and facilitate research and standardisation of clinical care.

Chronic Kidney Disease (CKD): CKD is usually staged according to glomerular filtration rate (GFR) and the KDOQI system [6, 7].

Stage 1: Normal function, GFR (≥90 ml/min/1.73 m^2), with evidence of damage on structural or urinary investigations.

Stage 2: Mild reduction in GFR (60–89 ml/min/1.73 m^2) with kidney damage

Stage 3: Moderate reduction in GFR: stage 3A (GFR 45–59) and stage 3B (GFR 30–44).

Stage 4: Severe reduction in GFR (15–29 ml/min/1.73 m^2) Preparation for renal replacement therapy

Stage 5: End-stage renal disease or established renal failure (GFR <15 ml/min/1.73 m^2) requiring permanent renal replacement therapy

At stages 1 to 3, patients are usually asymptomatic and the problem is often diagnosed by screening in primary care or pre-operative assessment. The most common causes include diabetes, hypertension, vascular disease, primary or secondary glomerular disease etc.

Anaesthetic considerations

Peri-operative renal protection: Thorough pre-operative assessment is of fundamental importance. Specific attention should be paid to the underlying cause of the renal failure (e.g. hypertension, diabetes, etc), its management (drug therapy, dialysis, previous fistulae or transplant procedures etc) and the repercussions of the disease (uraemia, oedema, micro and macrovasacular damage).

Consideration must be given to the surgical technique planned. Procedures that reduce renal perfusion, either through a global reduction in perfusion pressure (as may occur in hypotensive anaesthetic techniques) or regional reduction in blood flow (e.g. during cross-clamping or pneumoperitoneum at laparoscopy), can make the kidneys more vulnerable to damage.

Particular attention should be paid to those in higher risk categories. Work done by Kheterpal et al. looking at general surgical patients suggests risk factors for developing post-operative AKI include being male, increasing age, diabetes, a history of congestive cardiac failure, hypertension, ascites, pre-operative renal insufficiency, emergency surgery and intra-peritoneal surgery. In addition, Kheterpal showed 30 day mortality increased from 8.6–42% for those who developed AKI and composite morbidity increased from 19–66% [8]. Of these risk factors, preoperative renal dysfunction is the only reliable predictor of post operative renal dysfunction [9].

Specific surgical populations such as those undergoing cardiac, vascular or hepatic procedures may be subject to unique sets of risk factors including bypass time, aortic cross clamp time etc. Any surgery directly in the region of the renal tract, or involving the blood supply to the kidney should be assessed on a case by case basis and consideration given to the potential of its impact on renal function.

Several methods are claimed to offer renal protection yet there is limited evidence to support these. The use of 'renal dose dopamine' has fallen from favour since a paper published in the Lancet in 2000 found it conferred no benefit [10]. Intra-venous saline or hydration with bicarbonate have been suggested as ways of preventing contrast-induced nephropathy. Thus far, fluid management strategies remain controversial [11–13]. There is some evidence for using N-acetylcysteine to avoid contrast induced nephropathy but larger studies are needed [14, 15].

In the search for pharmacological agents potentially useful for renal protection, diuretics have been investigated. Mannitol has been shown to increase renal blood flow while preserving glomerular filtration and maintaining the oxygen supply/demand ratio post-operatively in patients with AKI following cardiac surgery [16]. However, mannitol used during minimally invasive partial nephrectomy had no influence on renal recovery [17]. Loop diuretics, in particular furosemide, are frequently employed in the management of patients with acute lung injury. They are generally used to manage fluid balance and reduce pulmonary oedema. It has been postulated that driving urine output can improve renal function. However, available studies do not support the theory that furosemide improves renal function directly [18]. Fenoldapam, a Dopamine D1 selective agonist, has shown some promise in small studies. Meta-analysis suggests that although it does not impact hospital mortality or renal replacement therapy, it may reduce post operative renal failure [19]. Further multi centre clinical trials would be required to definitively establish the benefits.

A Cochrane review in 2013 found no evidence to support any specific protective intra-operative anaesthetic interventions [20]. Judicious and rational fluid administration with appropriate monitoring for fluid responsiveness probably remains the best method of preventing onset of kidney injury peri-operatively.

Pharmacology in renal failure

Patients with renal failure are at increased risk of adverse drug reactions because of alterations in renal handling of substances and their metabolites. Drugs that undergo renal metabolism and excretion can accumulate, and careful dose adjustment should be made – particularly for drugs with a narrow therapeutic index. Firstly, in order to calculate an adjustment in dose, there must be an assessment of the degree of renal dysfunction. Various methods of calculating glomerular filtration exist. Commonly, the Cockroft-Gault formula is used to estimate creatinine clearance. However, newer methods, such as the CKD-EPI (Chronic Kidney Disease Epidemiology Collaborative), may prove to be more accurate [21].

Another complicating issue is the fact that renal dysfunction is associated with systemic changes that impact drug distribution and bioavailability such as retention of acid, salts and water. Protein losing nephropathy or disproportionate increase in body water can effectively reduce drug protein-bound fractions making effects unpredictable. Patients who have received haemodialysis or filtration may have unexpectedly low drug levels due to clearance via the circuit. In summary, CRF can lead to altered drug levels and familiarity with the physiology of renal failure as well as the pharmacology of individual drugs is necessary to understand and predict drug levels [22].

CRF can impact hepatic metabolism via downregulation of the CYP450 system with certain animal studies suggesting a reduction in activity of 40–85% [23]. Phase II reactions may also be reduced [24].

Specific anaesthesia-related pharmacology

Induction agents: Thiopental has an increased volume of distribution and reduced plasma protein binding in CRF leading to an overall higher free drug concentration so should be administered at a reduced rate and with care [25].

Propofol appears to retain a similar pharmacokinetic and dynamic profile in chronic renal failure to that in health. Propofol is not recommended for prolonged infusion in children due the risk of propofol infusion syndrome (metabolic acidosis, hypertriglyceridaemia, cardiac dysfunction, rhabdomyolysis and renal failure). There are case reports of propofol infusion syndrome in adults [26], yet the recommended adult dosage in renal failure and renal replacement therapy remains unchanged.

Ketamine undergoes hepatic metabolism resulting in inactive byproducts. It is apparently safe in renal failure and dose adjustments are not recommended.

Due to its effects on the adrenal cortex, etomidate is rarely used in the UK now. Some practitioners continue to advocate its use due to etomidate's cardiostable profile. Since this drug relies extensively on renal excretion, particular caution should be employed if considering use in a patient with renal failure.

Opiates and opioids: Renal impairment can impact the elimination of parent drugs or their metabolites. Accumulation of opioids can lead to respiratory depression, therefore these patients should be monitored carefully post-operatively and when opiate analgaesia is used. Evidence is weak and inconsistent as to which opiate is safest or most predictable pharmacokinetically / dynamically [27].

Alfentanil, due to its short half-life, has been recommended for severe post-operative pain relief. Regimens may be written with a reduced dose but standard interval [28]. Remifentanil appears to show no significant alteration of effects in CRF and may be a good alternative [29].

Morphine-6-glucuronide is eliminated by the kidney. In chronic renal failure the half-life of this active metabolite of morphine is prolonged from 2 to 27 hours and dose should be reduced accordingly [30].

Fentanyl is mainly metabolised in the liver but renal failure does result in reduced clearance.

Codeine is generally not recommended for use in chronic kidney disease since 90% of clearance is renal dependant. Accumulation of metabolites (codeine-6-glucuronide, norcodeine and morphine metabolites) can lead to respiratory depression and hypotension [31].

There are concerns regarding the use of various opioids in patients with renal impairment. The accumulation of toxic metabolites can lead to unpleasant and occasionally dangerous clinical consequences. Normeperidine can accumulate with the use of meperidine leading to tremors, agitation and seizures [32]. Hydromorphone is metabolized to hydromorphone-3-glucuronide which can lead to myoclonus, delerium and seizures in accumulation. Hydrocodone undergoes approximately 25% renal excretion so dose adjustment is recommended (reduce dose by 50%). Methadone and oxycodone also undergo substantial renal excretion and close monitoring is advised if these agents are used in patients with impaired renal function [31].

Volatile agents: Compound A, a breakdown product of sevofluorane, is known to cause transient renal toxicity in rats though the mechanism is unknown. In humans, the evidence is controversial but studies suggest that it should be safe when used at flow rates of at least 2 l/min if exposure is for more than one hour (and greater than 1 l/min if less than one hour) [33]. There is some debate over whether the depression of cardiac output leads to damage via decreased renal perfusion, or if there is some renal protection achieved through the reduced inflammation that is associated with volatile anaesthesia [34].

Desfluorane and isofluorane have not been shown to cause any deterioration in renal failure [35]. One group has suggested a more favourable post-operative renal profile in hepatectomy patients where desfluorane has been used rather than sevofluorane [36]. However, there is limited evidence to suggest one agent is preferable over any other.

Paralysing agents: Patients with end stage renal failure may experience significant prolongation of the effects of rocuronium, possibly due to decreased renal clearance. It is worth considering avoiding this agent if the patient has severe renal failure. Sugammadex has been used successfully to reverse paralysis with rocuronium but it should be remembered that the resulting complex also undergoes renal elimination [37].

In general, atracurium and cisatracurium can be used at normal dosages though there are concerns that laudanosine may accumulate in renal failure [38]. There is little evidence that this is clinically significant.

Theoretically, suxamethonium can be used at normal dosage – but caution should be used as this drug may cause dangerous exacerbation of hyperkalaemia (particularly if potassium is greater than 5.5mEq/l).

Cardiovascular support: Maintaining cardiac output does not guarantee regional renal perfusion but poor global cardiac output is certainly potentially harmful.

Intra-operatively, since low cardiac output in a fluid optimised patient is often due to vasodilatation caused by anaesthetic agents, vasopressors are commonly used to augment low blood pressure. Although there are theoretical concerns regarding unwanted vasoconstriction in renal vascular beds, it may yet be beneficial vasodilatory states where cardiac output is preserved [39].

Should the anaesthetist wish to use an inotrope, the optimal choice is controversial. As described above, dopamine does not offer renal protection but increasing cardiac output can improve end organ perfusion. Dobutamine is often used in the critical care but since it does cause a significant increase in myocardial oxygen demand it should be used with caution in patients with cardiac disease, which often co-exists with renal disease [40].

Fluid balance and electrolytes: During the pre-operative assessment it should be clearly established if the patient passes urine and, if so, how much. Electrolytes should be measured and if the patient receives haemodialysis, this should be scheduled for 12–24 hours prior to surgery [41]. Liaison with renal physicians is useful at this stage to ensure electrolytes are optimised prior to anaesthesia to minimise risks of arrhythmias etc.

Careful monitoring of electrolyte levels intra-operatively can help address impending problems, especially during long procedures. Insertion of an intra-arterial catheter can facilitate frequent sampling. Hyperkalaemia must be identified and addressed promptly to avoid dysrhythmias. It is useful to know the patient's baseline electrolyte levels as, in established CRF, a higher than normal potassium level may be usual for the patient and well tolerated. If necessary, haemofiltration can be performed intra-operatively.

There is limited evidence to guide the anaesthetist in their choice of intra-venous fluids. There are concerns regarding the use of fluids that contain potassium, yet Ringer's Lactate has been shown to be safe and useful in renal transplant [42]. Of particular benefit, such balanced solutions may avoid the hyperchloraemic metabolic acidosis which is said to develop with the use of 0.9% saline [43].

Regional anaesthesia and Neuraxial blockade: When neuraxial blockade extends up to around the level of T4, there may be reduced sympathetic drive. While care should be taken to maintain cardiac output, there is suggestion that reduced stimulation of the renin-angiotensin axis may be useful in preventing deterioration of existing disease [44]. This does not appear to be beneficial in healthy patients but more work needs to be done to evaluate whether this could reduce post-operative acute kidney injury.

Practicalities

Ventilation: There are few specific concerns with ventilation for patients with renal failure except that positive pressure ventilation and high positive end expiratory pressure can lead to a decrease in cardiac output. This can stimulate release of catecholamines and increased renin-angoitensin activity. Ultimately, this can cause salt and water retention and reduced urine output [45]. This is more likely to be a problem in patients requiring prolonged ventilation post-operatively.

IV access: Patients with established chronic renal failure will have undergone multiple previous episodes of intra-venous access. Vessels are often thrombosed and central access with ultrasound guidance should be considered. Care should be taken when choosing where to site arterial and peripheral venous catheters. It may be useful to discuss this with the surgical team in the pre-operative period in order to establish whether certain vessels need to be preserved for future arterio-venous fistula formation.

Patients may present with access established via a tunneled central venous catheter. These may normally be reserved for haemofiltration/dialysis and, if used in the peri-operative period, the anaesthetist should employ scrupulous asepsis.

At the time of establishing central IV access for anaesthesia, it should be considered whether simultaneous vascath insertion could be beneficial as prompt haemofiltration might be necessary for some.

Monitoring

In addition to standard AAGBI monitoring, invasive arterial catheterisation should be considered allowing careful management of blood pressure and regular electrolyte assessment. The use of central venous pressure monitoring is controversial, but trends can be useful to indicate fluid optimization [46]. Urine output (if the patient produces urine) should be recorded using a urometer.

There is no evidence to show improved survival with the use of cardiac output monitoring, but it should be considered in light of the importance of maintaining renal blood flow. Judicious fluid optimization can be achieved by delivering fluid challenges against objective parameters such as stroke volume variation.

It is worth noting that in renal failure, changes in skin impedance can result in altered train of four pattern with the use of a peripheral nerve stimulator making this a less reliable form of monitoring [47]. The effects of neuromuscular blocking agents should still be assessed with nerve stimulation but the potential for misleading results should be borne in mind. The anaesthetist should always assess the patient clinically for signs of incomplete blockade or reversal.

Miscellaneous

Patients with severe renal failure may be at higher risk of aspiration due to autonomic neuropathy and delayed gastric emptying [41]. Those receiving peritoneal dialysis are at risk of peritonitis and sub-acute bowel obstruction.

Careful attention should be paid to pressure areas as there may be peripheral neuropathy and pre-existing low grade pressure areas.

Relative immunosuppression and delayed wound healing make patients more susceptible to post-operative infection. Prophylactic antibiotics should be considered. Liaison with microbiology is recommended if patients are known to be colonized with opportunistic pathogens, or if they are already on an anti-microbial regime or immunosuppressant drugs.

CONCLUSION

There is still limited strong evidence to guide the anaesthetic management of patients with renal failure. Much of our practice is based on extrapolation from physical and pharmacologic first principles. There are many small studies in the literature but very few large randomized controlled trials. In order to avoid causing or exacerbating renal failure, anaesthetists still rely heavily upon thorough pre-operative assessment, meticulous attention to detail peri-operatively, and careful management post-operatively. While the lack of strong evidence persists it is imperative that anaesthetists have a good understanding of the physiology of renal failure and the challenges it represents.

REFERENCES

1. Harvey G, Oliver K, Humphreys J, Rothwell K, Hegarty J. Improving the identification and management of chronic kidney disease in primary care: Lessons from a staged improvement collaborative. Int J Qual Health Care 2015 Feb;27(1):10–6.
2. Lewington A, Kanagasundaram S. Renal association clinical practice guidelines on acute kidney injury. Nephron Clin Pract 2011;118 Suppl 1:c349–90.
3. Bellomo R, Ronco C, Kellum JA, Mehta RL, Palevsky P; Acute dialysis quality initiative workgroup. Acute renal failure - definition, outcome measures, animal models, fluid therapy and information technology needs: The second international consensus conference of the acute dialysis quality initiative (ADQI) group. Crit Care 2004 Aug;8(4):R204–12.
4. Lopes JA, Jorge S. The RIFLE and AKIN classifications for acute kidney injury: A critical and comprehensive review. Clin Kidney J 2013 Feb;6(1):8–14.
5. KDIGO clinical practice guideline for acute kidney injury. Kidney Int Suppl 2012;2(Suppl 1):8.
6. K/DOQI clinical practice guidelines for chronic kidney disease: Evaluation, classification, and stratification. Am J Kidney Dis 2002 Feb;39(2 Suppl 1):S1–266.

7. Winearls CG, Glassock RJ. Dissecting and refining the staging of chronic kidney disease. Kidney Int 2009 May;75(10):1009–14.
8. Kheterpal S, Tremper KK, Heung M, et al. Development and validation of an acute kidney injury risk index for patients undergoing general surgery: Results from a national data set. Anesthesiology 2009 Mar;110(3):505–15.
9. Novis BK, Roizen MF, Aronson S, Thisted RA. Association of preoperative risk factors with postoperative acute renal failure. Anesth Analg 1994 Jan;78(1):143–9.
10. Bellomo R, Chapman M, Finfer S, Hickling K, Myburgh J. Low-dose dopamine in patients with early renal dysfunction: A placebo-controlled randomised trial. Australian and New Zealand intensive care society (ANZICS) clinical trials group. Lancet 2000 Dec 23–30;356(9248):2139–43.
11. Bellomo R, Ronco C, Kellum JA, et al. Acute renal failure - definition, outcome measures, animal models, fluid therapy and information technology needs: The second international consensus conference of the acute dialysis quality initiative (ADQI) group. Crit Care 2004 Aug;8(4):R204–12.
12. Merten GJ, Burgess WP, Gray LV, et al. Prevention of contrast-induced nephropathy with sodium bicarbonate: A randomized controlled trial. JAMA 2004 May 19;291(19):2328–34.
13. Trivedi HS, Moore H, Nasr S, et al. A randomized prospective trial to assess the role of saline hydration on the development of contrast nephrotoxicity. Nephron Clin Pract 2003 Jan;93(1):C29–34.
14. Pezeshgi A, Parsamanesh N, Farhood G, Mahmoodi K. Evaluation of the protective effect of N-acetylcysteine on contrast media nephropathy. J Renal Inj Prev 2015 Sep 1;4(4):109–12.
15. Jo SH. N-acetylcysteine for prevention of contrast-induced nephropathy: A narrative review. Korean Circ J 2011 Dec;41(12):695–702.
16. Bragadottir G, Redfors B, Ricksten SE. Mannitol increases renal blood flow and maintains filtration fraction and oxygenation in postoperative acute kidney injury: A prospective interventional study. Crit Care 2012 Aug 17;16(4):R159.
17. Power NE, Maschino AC, Savage C, et al. Intraoperative mannitol use does not improve long-term renal function outcomes after minimally invasive partial nephrectomy. Urology 2012 Apr;79(4):821–5.
18. Ho KM, Power BM. Benefits and risks of furosemide in acute kidney injury. Anaesthesia 2010 Mar;65(3):283–93.
19. Gillies MA, Kakar V, Parker RJ, Honoré PM, Ostermann M. Fenoldopam to prevent acute kidney injury after major surgery-a systematic review and meta-analysis. Crit Care 2015 Dec 25;19:449.
20. Zacharias M, Mugawar M, Herbison GP, et al. Interventions for protecting renal function in the perioperative period. Cochrane Database Syst Rev 2013 Sep 11;(9):CD003590.
21. Jones GR. Estimating renal function for drug dosing decisions. Clin Biochem Rev 2011 May;32(2):81–8.
22. Verbeeck RK, Musuamba FT. Pharmacokinetics and dosage adjustment in patients with renal dysfunction. Eur J Clin Pharmacol 2009 Aug;65(8):757–73.
23. Dreisbach AW, Lertora JJ. The effect of chronic renal failure on drug metabolism and transport. Expert Opin Drug Metab Toxicol 2008 Aug;4(8):1065–74.
24. Pichette V, Leblond FA. Drug metabolism in chronic renal failure. Curr Drug Metab 2003 Apr;4(2):91–103.
25. Sear J. Drug handling in renal impairment. Curr Anaesth Crit Care 1992;3(3):133–9.
26. Casserly B, O'Mahony E, Timm EG, Haqqie S, Eisele G, Urizar R. Propofol infusion syndrome: An unusual cause of renal failure. Am J Kidney Dis 2004 Dec;44(6):e98–101.
27. King S, Forbes K, Hanks GW, Ferro CJ, Chambers EJ. A systematic review of the use of opioid medication for those with moderate to severe cancer pain and renal impairment: A European palliative care research collaborative opioid guidelines project. Palliat Med 2011 Jul;25(5):525–52.
28. Tawfic QA, Bellingham G. Postoperative pain management in patients with chronic kidney disease. J Anaesthesiol Clin Pharmacol 2015 Jan–Mar;31(1):6–13.
29. Hoke JF, Shlugman D, Dershwitz M, et al. Pharmacokinetics and pharmacodynamics of remifentanil in persons with renal failure compared with healthy volunteers. Anesthesiology 1997 Sep;87(3):533–41.
30. Craig RG, Hunter JM. Recent developments in the perioperative management of adult patients with chronic kidney disease. Br J Anaesth 2008 Sep;101(3):296–310.
31. Vilay AM, Churchwell MD, Mueller BA. Clinical review: Drug metabolism and nonrenal clearance in acute kidney injury. Crit Care 2008;12(6):235.
32. Pham PC, Khaing K, Sievers TM, et al. 2017 update on pain management in patients with chronic kidney disease. Clin Kidney J 2017 Oct;10(5):688–97.
33. Gentz BA, Malan TP Jr. Renal toxicity with sevoflurane: A storm in a teacup? Drugs 2001;61(15):2155–62.
34. Lee HT, Ota-Setlik A, Fu Y, Nasr SH, Emala CW. Differential protective effects of volatile anesthetics against renal ischemia-reperfusion injury in vivo. Anesthesiology 2004 Dec;101(6):1313–24.
35. Litz RJ, Hübler M, Lorenz W, Meier VK, Albrecht DM. Renal responses to desflurane and isoflurane in patients with renal insufficiency. Anesthesiology 2002 Nov;97(5):1133–6.
36. Ko JS, Gwak MS, Choi SJ, et al. The effects of desflurane and sevoflurane on hepatic and renal functions after right hepatectomy in living donors*. Transpl Int 2010 Jul;23(7):736–44.
37. Staals LM, Snoeck MM, Driessen JJ, et al. Reduced clearance of rocuronium and sugammadex in patients with severe to end-stage renal failure: A pharmacokinetic study. Br J Anaesth 2010 Jan;104(1):31–9.
38. Della Rocca G, Pompei L, Coccia C, et al. Atracurium, cisatracurium, vecuronium and rocuronium in patients with renal failure. [Article in Italian]. Minerva Anestesiol 2003 Jul–Aug;69(7–8):605–11, 612, 5.
39. Bellomo R, Giantomasso DD. Noradrenaline and the kidney: Friends or foes? Crit Care 2001 Dec;5(6):294–8.

40. Overgaard CB, Dzavík V. Inotropes and vasopressors: Review of physiology and clinical use in cardiovascular disease. Circulation 2008 Sep 2;118(10):1047–56.
41. Rang ST, West NL, HoWard J, Cousins J. Anaesthesia for Chronic renal disease and renal transplantation. EAU-EBU Update Series 2006;4(6):246–56.
42. O'Malley CM, Frumento RJ, Hardy MA, et al. A randomized, double-blind comparison of lactated Ringer's solution and 0.9% NaCl during renal transplantation. Anesth Analg 2005 May;100(5):1518–24.
43. O'Malley CMN. Intravenous fluids and renal failure. Transfusion Alternatives in Transfusion Medicine 2003;5(4):416–23.
44. Li Y, Zhu S, Yan M. Combined general/epidural anesthesia (ropivacaine 0.375%) versus general anesthesia for upper abdominal surgery. Anesth Analg 2008 May;106(5):1562–5.
45. Wagener G, Brentjens TE. Anesthetic concerns in patients presenting with renal failure. Anesthesiol Clin 2010 Mar;28(1):39–54.
46. Baxi V, Jain A, Dasgupta D. Anaesthesia for renal transplantation: An update. Indian J Anaesth 2009 Apr;53(2):139–47.
47. McGrath CD, Hunter JM. Monitoring of neuromuscular block. Contin Educ Anaesth Crit Care Pain 2006;6(1):7–12.

****

Author Contributions

Alice Myers – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published
Zoka Milan – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor of Submission

The corresponding author is the guarantor of submission.

Source of Support

None

Conflict of Interest

Authors declare no conflict of interest.