Renal dysfunction and fluid and electrolyte disturbances

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Purpose of review

To examine recent developments in preventing and treating postoperative acute renal dysfunction. To review contemporary issues concerning perioperative fluid and electrolyte management.

Recent findings

Renal dysfunction remains a major postoperative morbidity despite the advent of intermittent and continuous renal replacement therapies. It is also associated with increased mortality. Newer techniques, such as off-pump coronary artery bypass surgery, which promised to reduce the incidence of postoperative renal injury, have failed to do so. In addition, newer techniques such as endovascular repair of aortic disorder and the transcutaneous insertion of aortic valves are associated with substantial degrees of kidney injury partially due to the use of much intravenous contrast material. Therefore, the present-day approach to preventing contrast-induced nephropathy is reviewed. Electrolyte disturbances are especially problematic after certain types of surgery, such as trans-sphenoidal adenomectomy and surgeries requiring the use of large volumes of irrigating solutions.

Summary

Renal dysfunction, along with fluid and electrolyte disturbances, is a major issue that needs to be considered as part of perioperative management. Moreover, it is the prevention of clinically significant renal injury and electrolyte disturbances that is the cornerstone of contemporary anesthetic and surgical care.

Keywords

acute kidney injury, electrolytes, magnesium, postoperative care, risk factors, sodium

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Introduction

The perioperative period is characterized by changes in cardiovascular and fluid homeostasis that can affect renal function and electrolyte balance. The consequences of renal, fluid balance and electrolyte problems during and after surgery have short-term and long-term consequences, including increased mortality. It is, thus, important to identify before surgery the patients at risk for postoperative renal and electrolyte problems. Moreover, it is necessary to continually take steps during and immediately after surgery to prevent perioperative renal failure [1]. This review will examine contemporary issues surrounding postoperative renal failure and electrolyte imbalance.

Incidence and risks

Renal dysfunction is among the most problematic complications that occur during the intraoperative and postoperative periods. In a prospective study of 4158 noncardiac surgical patients older than 70 years in 23 Australian and New Zealand hospitals, acute postoperative renal dysfunction occurred in 244 and was significantly associated with 30-day postoperative mortality [2]. However, preoperative renal impairment was not associated with 30-day mortality. In another study of 11 166 noncardiac surgery PACU/ICU patients with no preoperative history of renal insufficiency, 7.5% developed acute kidney dysfunction. Independent preoperative predictive factors were American Society of Anesthesiology Physical Status Classification, Revised Cardiac Risk Index, high-risk surgery and congestive heart failure. Patients who developed renal dysfunction had longer PACU/ICU stays as well as higher PACU, hospital and 6-month mortality rates [3]. Of 1158 patients undergoing vascular surgery, 558 had preoperative left ventricular dysfunction, 71 of whom developed postoperative acute kidney injury (AKI). These patients had a higher cardiovascular mortality (2.2 years follow-up) and higher rates of AKI than patients without left ventricular dysfunction [4]. A more focused study examined 35 302 diabetic veterans undergoing their first noncardiac surgery. 17.8% of them experienced at least some degree of renal postoperative insufficiency emphasizing the vulnerability of diabetic patients to renal failure. Long-term (median 3.7 years) follow-up

of these diabetic patients revealed that the duration of the AKI was independently associated with long-term mortality. Interestingly, the patients with severe renal injury of short duration had much lower mortality than those with milder injury of medium or long duration [5••]. Because of the enhanced morbidity and mortality associated with AKI, there is major interest in its prevention [6••].

AKI occurs either as an isolated complication or as part of multiple organ system dysfunction. Isolated postoperative renal dysfunction is often secondary to drug toxicity, rhabdomyolysis and cardiopulmonary bypass [7]. Such patients have a lower mortality rate than those with sepsis or systemic inflammatory response- associated acute kidney failure. The latter cause of AKI is often part of multiple organ system dysfunction, that is secondary to an uncontrolled inflammatory response such as occurs with shock, sepsis and trauma. In such patients, mortality is increased [8]. Despite recent advances in dialectic and other therapies, there has been little reduction in mortality and such patients often have prolonged hospital stays [9,10]. Sepsis often causes AKI, whereas, alternately, the immune changes secondary to AKI itself increases vulnerability to developing sepsis [11]. In a study of 611 ICU patients, 28% had sepsis before AKI and 40% became septic a median of 5 days after developing AKI [11]. Therefore, there are many associations between sepsis and acute renal injury.

Cardiac surgery

AKI is an extremely problematic complication of cardiac surgery because of its association with increased morbidity and mortality. Of 12 096 patients undergoing cardiac surgery, 2.1% received renal replacement therapy and 3.9% had severe kidney injury [12]. This increased mortality and morbidity is reflected in the observations that postoperative elevated creatinine concentrations and continuous or intermittent renal replacement therapy (Continuous veno-veno hemofiltration or hemodialysis) are associated with greater postoperative mortality [13]. AKI, defined as a 25% reduction in glomerular filtration rate (GFR), occurs in more than 30% of cardiac surgery patients. It is associated with a greater than four-fold increase in the odds of death [14••]. When the renal dysfunction leads to renal replacement therapy, mortality rates exceed 60% [14••]. The renal dysfunction is often caused by the effects of the surgery and cardiopulmonary bypass, but may also be caused by administration of intravenous contrast material for preoperative coronary angiography. Cardiopulmonary bypass results in loss of pulsatile flow, which enhances both renal vasoconstriction and inflammatory responses with the attendant release of cytokines. In addition, microemboli smaller than 40 υm have been implicated as causing renal dysfunction by blocking renal arterioles [15]. The subject of cardiopulmonary bypass-associated AKI was recently reviewed comprehensively by Kumar and Suneja [15]. A recent systematic review concluded that no pharmacologic agent examined for its renoprotective properties in adults undergoing cardiac surgery with bypass was able to reduce mortality [14••]. Fenoldopam, atrial natriuretic peptide and brain natriuretic peptide showed some evidence of renal protection, whereas dopamine, diuretics and calcium channel antagonists did not [14••].

Key points

- Renal dysfunction remains a major postoperative morbidity despite the advent of intermittent and continuous renal replacement therapies.
- Newer percutaneous techniques are associated with the administration of contrast material increasing the risk for contrast nephropathy.
- Electrolyte disturbances are especially problematic after certain types of surgeries, such as transsphenoidal adenectomies and procedures requiring the use of much irrigation fluid.

The interest in off-bypass coronary artery bypass surgery affords the ability to examine the effects of cardiopulmonary bypass on renal function. Among the touted advantages of off-pump surgery were its association with less renal dysfunction. However, this has not been shown by all studies. In a retrospective study of patients with pre-existing renal dysfunction, in 2711 who underwent on-bypass and 158 who underwent off-bypass coronary revascularization, off-bypass coronary grafting was not associated with a decreased risk of postoperative renal dysfunction [16]. Diabetes and peripheral vascular disease, along with decreased preoperative creatinine clearance, were independent risk factors for developing postoperative renal dysfunction [16]. Another risk factor for post-off-pump renal failure was an elevated preoperative C-reactive protein concentration (CRP), which reflects a pre-existing proinflammatory state, such as occurs in the metabolic syndrome [17]. In a retrospective study of 740 patients, 84 of 320 (26%) with and 44 of 420 (57%) without the metabolic syndrome developed postoperative kidney injury after off-pump surgery, as defined by a 50% increase in serum creatinine or an increase of 0.3 mg/dl or greater [18]. When the components of the metabolic syndrome were examined, diabetes and chronic renal disease were independent risk factors for postoperative acute kidney dysfunction [18]. In a prospective study of 71 diabetic patients randomized to on-bypass and off-bypass coronary artery bypass grafting (CABG), there were no differences between the two groups in either the postoperative serum or urinary creatinine or creatinine clearance, but there was a smaller increase in proteinuria and microalbuminuria in

the off-pump group indicating less kidney injury [19]. Although renal failure does occur after off-pump coronary revascularization, a meta-analysis that included randomized trials and observational studies concluded that it is still unclear whether off-pump surgery reduces the incidence of postoperative renal injury compared with on-pump surgery [20].

Noncardiac surgery

The use of endovascular, rather than open, techniques to repair abdominal aortic aneurysms has aroused much interest in reducing postprocedure renal dysfunction through the use of preprocedure renal protection strategies. These patients have the potential for a quadruple insult to the kidneys: intravenous contrast material, microembolization and macroembolization to the kidneys, changes in renal blood flow patterns due to graft placement in the area of the renal arteries and the systemic inflammatory response syndrome. Usually, the insult is mild and clinically insignificant (small mean increase in serum creatinine of 0.03–0.10 mg/dl) and is seen more often with suprarenal than infrarenal graft fixation likely due to more microembolization and renal artery trauma [21]. Preoperative strategies to reduce the incidence and extent of renal dysfunction include those designed to prevent intravenous contrast material nephropathy. Furthermore, statins may reduce the development of renal failure after suprarenal grafting possibly by attenuating the inflammatory response [21].

Repair of thoracic and thoracoabdominal aneurysms can be complicated by renal dysfunction. Among the touted advantages of endovascular repair are its avoidance of open thoracotomy and cardiopulmonary bypass with the attendant aortic cross-clamping and circulatory arrest. Therefore, it was hoped that the endovascular approach would result in less renal failure. Following thoracic endovascular repair, there is a reported incidence of acute renal failure of 1.5–34%. In a recently reported retrospective study of 175 consecutive patients who underwent 210 procedures at two centers, 13.3% had significant worsening of their renal failure at 1-month follow-up [22]. Risk factors included increased age, male sex, African–American race, diabetes mellitus, hypertension, preoperative stroke and a proximal aneurysm. Interestingly, obesity was nephroprotective. In a series of 509 patients who underwent open thoracoabdominal aneurysm repair, acute renal failure occurred in 9.8% and 5.9% were discharged home on dialysis [23]. The contemporary literature does not demonstrate less renal dysfunction following endovascular stent grafting. Following thoracic endovascular repair, short-term results are encouraging, as it was associated with significantly less mortality and morbidity, but long-term results need to be further investigated [24]. Routine use of intravascular ultrasound and reduced contrast might result in lower rates of renal insufficiency [25].

Contrast nephropathy

Preoperative diagnostic procedures, such as computed tomography scans and cardiac catheterizations, and intraoperative procedures, such as endovascular repair of aortic abdominal aneurysms, require the administration of contrast material. In general, contrast nephropathy is characterized by an increase in serum creatinine within 24 h of contrast administration and usually peaks within 3–5 days. Most often, this creatinine increase does not progress to fulminate renal failure, but when it does, many studies indicate it can increase morbidity and mortality [26]. As the creatinine increase can be slow, contrast-induced AKI is defined as a rise in creatinine of 0.5 mg/dl or a 25% relative rise at 48 h after contrast administration [27•]. Therefore, patients who received contrast material within a few days of surgery may be in the throughs of developing renal dysfunction. This issue was examined in a retrospective study of the incidence of renal dysfunction after off-pump coronary revascularization. Interestingly, there was no difference in the incidence between patients with at least two risk factors for developing contrast nephropathy who had coronary arteriography within 2 days of surgery, compared with those who had it for more than 2 days prior to surgery [28•]. However, other studies have indicated an increased incidence of postoperative renal injury among patients who undergo coronary arteriography immediately prior to coronary revascularization, especially when subjected to cardiopulmonary bypass [29].

Patients receiving contrast material during surgery could develop nephropathy during the early postoperative period. Therefore, it is especially important to prevent its occurrence in high-risk patients such as those with diabetes, poor hydration status and chronic renal failure. The incidence of postoperative kidney injury after open abdominal aneurysm repair is 2–6%, whereas that after endovascular repair is 6–39% [30]. This requires steps to reduce the risk of postoperative renal dysfunction such as using carbon dioxide angiography to replace or reduce the dose of iodinated contrast materials [31]. Furthermore, there has been emphasis on the pretreatment of patients with intravenous fluids to reduce the concentration and contact time of contrast material within the tubule lumen, administration of antioxidants such as N-acetylcysteine or ascorbic acid and alkalinizing the urine with sodium bicarbonate. Recent studies have examined the efficacy of these techniques. The efficacy of N-acetylcysteine in preventing contrast-induced nephropathy has not been consistently demonstrated likely because of differences among studies in the route of administration, doses, populations and different changes in serum creatinine

as the main endpoint [6••]. When administered orally (1200 mg orally twice daily on the day before and the day after the procedure), it was reported to be more effective than ascorbic acid (3 + 2 g before and 2 g twice after the procedure [32]). A multicenter prospective trial [33] showed that intravenous N-acetylcysteine (600 mg twice daily) along with high-dose hydration (normal saline 1 ml/kg per h) on the days before, on and after the procedure was better at reducing contrast nephropathy than high-dose hydration alone and better than the control group (hydration with 1 ml/kg per h 12 h prior and 12 h following the procedure). Despite the results of these and other studies, an expert panel of the European Society of Intensive Care Medicine did not recommend the routine use of N-acetylcysteine, because most studies used an elevation in creatinine as their endpoint and not mortality or the need for renal replacement therapy [6••]. Recent studies of the use of sodium bicarbonate to alkalinize the urine and thus protect against oxygen-free radial injury and saline hydration (75 ml of 8.4% sodium bicarbonate added to 1 litre of isotonic saline administered for 1 h before and 6 h after the procedure) did not show reduced appearance of nephropathy any more than when saline was used alone [34]. This along with other studies has not definitively demonstrated the efficacy of adding sodium bicarbonate to a hydration regimen [26]. Other studies have failed to show that low or isoosmolar, as opposed to high-osmolarity contrast media, reduces contrast nephropathy. However, among patients with pre-existing renal dysfunction undergoing intra-arterial contrast injection, low or iso-osmotic contrast material might be indicated [26]. Furthermore, a meta-analysis of the chronic administration of statins, with their anti-oxidative and anti-inflammatory properties, does not conclusively find them to be protective [35]. The most recommended modality to prevent contrast-induced nephropathy is hydration, although some centers add bicarbonate and/or N-acetylcysteine. Therefore, during the intraoperative and immediate postoperative periods, patients receiving contrast material should be well hydrated with lost fluid volumes promptly restored.

Fluids and electrolyte dysfunction

Up to 30% of surgical inpatients develop complications related to fluid and electrolyte therapy [36]. Fluid shifts during the perioperative period and the physiological responses to surgical stress have significant implications for perioperative fluid management. Many patients are dehydrated before surgery owing to prolonged fasting, the use of purgatives, enemas and diuretics. Intraoperative fluid losses are frequently underestimated and excess losses, both surgical and third-space, persist into the early postoperative period. Therefore, hypovolemia is often present leading to thirst and vasopressin secretion.

The most prominent response to anesthesia and surgery during the perioperative period is sodium and water retention. In general, the tendency to retain water is directly related to the magnitude of surgery. The factors that may contribute to this fluid retention include the effects of anesthetic agents on renal blood flow and GFR, the effects of intraoperative hypotension or hypovolemia on renal function, an increased sympathetic tone and circulating catecholamines causing renal vasoconstriction, the salt-retaining and water-retaining effects of increased plasma cortisol and aldosterone concentrations and an increased ADH activity. During surgery, ADH concentrations can increase as much as 50–100-fold. They often begin to fall toward the end of the surgery or within 3–5 days. This response is partly related to drugs, pain and other factors attributable to the stress of surgery. In neurosurgical patients, inappropriate ADH and cerebral salt-wasting syndrome also occur [37]. However, it is mostly a physiological response to the loss of intravascular fluid into cells or by its sequestration and immobilization in the tissues (i.e. 'third-space'). The difference is important because it determines the choice between fluid loading and restriction as the most physiological approach to fluid therapy in the perioperative period [38]. In children, isotonic saline solution administration, as opposed to fluid restriction, decreased the magnitude and incidence of postoperative hyponatremia [38].

Intravenous fluids are commonly administered during the perioperative period. Concerns about renal dysfunction should not prevent more restrictive approaches to fluid administration [39]. Unless clearly indicated, a more restrictive approach should be taken to maintain fluid administration than the 'mandatory 3 litres per day' often administered. When assessing volume status and the relative risks of insufficient or excessive volume replacement, pre-existing cardiorespiratory and renal disorders and the severity of pathophysiological changes associated with the acute illness itself must be taken into account. When fluid overload is suspected, fluid restriction and/or diuretic therapy may be necessary. In more complex clinical situations, attempts at achieving negative fluid balance are not necessarily sufficient, effective or appropriate, as much fluid may be located extravascularly, whereas the intravascular space is depleted. In such a situation, although there may be total body fluid overload, there is a deficit of intravascular fluid [40]. Some have proposed using hypertonic saline to reduce the total volume of fluid required without affecting kidney function. However, the serum sodium will rise and must be monitored closely [41].

In a prospective observational study performed upon 57 patients undergoing trans-sphenoidal adenomectomy, water and electrolyte disturbances occurred in the

majority of patients and were usually transient. Monitoring for water imbalances, due to deficiency or excess of ADH [diabetes insipidus or syndrome of inappropriate antidiuretic hormone hypersecretion (SIADH), respectively], is accomplished by continuously monitoring fluid intake, urine output and specific gravities, coupled with daily serum electrolyte measurements. Diabetes insipidus occurs more frequently than hyponatremia [42] and is characterized by excess volumes of inappropriately dilute urine, which can lead to hypernatremia. Most patients maintain adequate fluid intake and euvolemia, but desmopressin therapy is required for some. SIADH, which peaks in incidence on postoperative day 7, presents with hyponatremia that can be severe and symptomatic. Management consists of fluid restriction [43].

Changes in the serum sodium during the perioperative period can increase the risk of hospital mortality. A retrospective study was performed using data from 151 486 adults admitted consecutively over a period of 10 years (1998–2007) to 77 Austrian medical, surgical and mixed ICUs [44]. The results suggest that both hyponatremia and hypernatremia present on admission to the ICU are independent risk factors for poor prognosis. Another study performed from 2000 to 2006 in three medical–surgical ICUs in Canada included more than 8000 adult patients with normal sodium concentrations on ICU admission. It showed that ICU-acquired hyponatremia and hypernatremia are common in critically ill patients and are associated with elevated risk of hospital mortality [45].

Many endoscopic surgical procedures involve using irrigating fluid to dilate the operating field and wash away debris and blood. A potential complication of such irrigation is systemic absorption of the fluid. Excessive absorption of fluid can cause intravascular overload and/or electrolyte disturbances to the point that symptoms are produced [46]. The ideal irrigant for endoscopic resection would be a nonconductive solution that does not interfere with electrosurgical resection, has a high degree of translucency, has an osmolarity similar to the serum and causes only minimal side-effects when absorbed [47]. A prospective controlled trial was done on 360 patients undergoing transurethral resection of the prostate using three types of irrigating fluids (1.5%, glycine, 5% glucose and normal saline). Each group included 120 patients [48]. Normal saline and 5% glucose were associated with lower perioperative morbidity including a lower incidence of transurethral resection syndrome, lower urinary catheterization duration and shorter hospital stay as compared with 1.5% glycine. There was transient postoperative hyperglycemia in the 5% glucose group. A study included 96 patients undergoing percutaneous nephrolithotomy (PCNL) using half-strength saline irrigation fluids and intravenous infusion solutions with hypotonic sodium. Half-strength saline irrigation fluid did not result in considerable reductions in postoperative serum sodium concentrations in most simple PCNL procedures. Using intravenous infusion solutions with hypotonic sodium is discouraged in PCNL patients with a high probability of absorbing large volumes of hypotonic irrigation solution [49].

Conclusion

Despite the advent and extensive use of intermittent and continuous dialectic therapies, acute renal dysfunction remains a major cause of postoperative mortality and morbidity. This persistence of high morbidity and mortality occurs in spite of the many routine interventions designed to prevent renal dysfunction, including the preoperative identification of patients at risk of developing acute renal dysfunction (e.g. diabetes, chronic renal failure) and using intraoperative modalities such as aggressive hydration, avoidance of nephrotoxic drugs (e.g. aminoglycosides) and additional physiologic monitoring, for example measurement of central pressures. The inability to reduce the incidence of these poor outcomes can be attributed to three factors: the first is our still rudimentary understanding of the pathophysiology, cause and triggers of kidney injury. As a result, it is still unknown why some patients develop perioperative renal dysfunction and others do not. The second factor is the introduction of new diagnostic and therapeutic modalities. For example, gadolinium-based MRI contrast media that was originally thought to be well tolerated and lack nephrotoxic effects can induce a usually reversible decrease in renal function in patients with altered baseline renal function [50]. A prime example of new therapeutic modalities is the increasing use of endovascular grafts with the concomitant use of radiocontrast material. The final factor is the aging population with its higher incidence of diabetes mellitus, hypertension and obesity. Therefore, more investigation is needed to develop methods to prevent and reduce the poor outcomes of perioperative renal dysfunction.

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Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 406).

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