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Perioperative care of the patient with renal failure

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Chronic kidney disease is increasingly prevalent in the United States [1]. The Third National Health and Nutrition Examination Survey from 1988 to 1994 estimated that 8 million individuals had moderate to severe chronic kidney disease characterized by a glomerular filtration rate (GFR) lower than 60 mL/min/1.73m^2 [1]. According to the 2001 report of the U.S. Renal Data System (USRDS), moreover, the point prevalent count of patients with end-stage renal disease (ESRD) on December 31, 1999, was 328,695 [2]. As the number of Health Maintenance Organizations (HMOs) has escalated, general practitioners and internists have become the central or main physicians for many suffering from chronic kidney disease and ESRD. Additionally, surgeons, intensivists, and hospitalists care for people who undergo surgery—one of the most common therapeutic interventions associated with acute renal failure (ARF). For example, 27% of the 748 cases of ARF reported by Liano and Pascual were thought to originate in the postoperative period [3]. Because of the complexity and pervasiveness of renal failure, it is important that non-nephrologists be acquainted with perioperative care in those afflicted with this disorder. This article addresses the prevention of postoperative ARF as well as the perioperative care of ESRD patients undergoing surgery.

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Postoperative acute renal failure

Epidemiology

ARF is broadly defined as a sudden deterioration of renal function resulting in retention of nitrogenous wastes including urea and creatinine [4]. In many reports, the definition of ARF is based on serum creatinine which is a poor marker of renal function. The degree of creatinine elevation necessary to fulfill the diagnosis of ARF varies among authorities. For some researchers, ARF is present when there is a 25–50% increase in the serum concentration from baseline. Irrespective of a lack of consensus on the definition of ARF, two important facts should be remembered. Firstly, preexisting chronic kidney disease is a strong risk factor for the development of ARF [5–7]. Secondly, significant impairment of renal function, characterized by creatinine levels >1.5–3.0 mg/dL, introduces a serious risk that imposes a major threat to patients who have had surgery [8–10]. Chertow et al reported that acute renal failure requiring renal replacement therapy occurred in 1.1% of 42,773 individuals from 43 Veterans Affairs (VA) medical centers who had cardiac surgery [5] and 0.6% of 87,078 general surgery patients from the National VA Surgical Risk Study [11].

ARF is independently associated with early mortality following cardiac surgery, even after adjustment for comorbidity and postoperative complications. For example, the first aforementioned study also revealed that the 30-day mortality for subjects with acute renal failure was 63.7% compared with 4.4% for those with normal renal function ($P < 0.0001$) [5]. Furthermore, in a recent report by Conlon et al. analyzing data from 2672 patients undergoing coronary artery bypass grafting (CABG), 211 (7.9%) individuals developed surgery-induced ARF. The mortality for patients who contracted ARF was 14% (odds ratio 15, $P = 0.0001$) compared with 1% among those without ARF. In addition, mortality for CABG patients who received some form of dialysis was 28% (odds ratio 20, $P = 0.0001$) as opposed to 1.8% among people who did not require renal replacement therapy [12].

Pathophysiology

Usually, postoperative ARF is categorized as prerenal, intrinsic or renal, and postrenal. This classification may prove useful in determining the physiologic mechanism responsible for the GFR reduction or in establishing a differential diagnosis. Prerenal ARF results from diminished renal perfusion caused by volume depletion and/or hypotension. Intraoperative hormonal changes, secondary to stimulation of the sympathetic nervous system and renin-angiotensin-aldosterone axis, compromise GFR by inducing afferent arteriolar renal vasoconstriction. Simultaneously, angiotensin II modulates its own vasoconstrictive impact by stimulating renal release of prostaglandins. In the postoperative period, decreased glomerular perfusion may be caused by volume depletion, redistribution of extracelluar fluid or cardiac malfunction as in myocardial infarction, congestive heart failure, and tamponade [13,14]. As indicated by several reports, people with the renal or intrinsic type of ARF have diminished baseline GFR because of diabetes, hypertension, or vascular disease. Sustained hypotension and volume depletion, prolonged cardiopulmonary bypass or supra-aortic clamping time, sepsis, and nephrotoxin exposure (aminoglycosides, radiocontrast materials, myoglobin, hemoglobin) may precipitate acute renal failure [5–7,9,15–20]. Postrenal ARF occurs because of tubular obstruction exemplified by sulfonamide and acyclovir crystals or bladder dysfunction [21]. Pelvic or ureteral obstruction caused by blood clots, sloughed papillae, and retroperitoneal hematoma causes ARF only when bilateral or unilateral in a patient with a single functioning kidney.

Clinical and laboratory evaluation

The approach to patients with postoperative ARF necessitates a thorough history and chart review and a comprehensive physical examination in combination with key laboratory measurements such as complete blood count with leukocyte differential, metabolic panel, coagulation profile, microscopic urinalysis, and urine electrolytes. Focused history and record analysis provide important information about volume depletion, hypotension, cardiopulmonary bypass or supraaortic clamping time, arrhythmia, and exposure to endogenous (myoglobin, hemoglobin) and exogenous (drugs) nephrotoxins.

A good physical examination yields valuable clues. Skin inspection for rash, purpura, livedo reticularis, gangrene, and digital cyanosis provides clues to raising the diagnosis of acute interstitial nephritis, renal artery, or atheromatous embolism. A thorough evaluation of the cardiovascular and volume status is the most important facet in the diagnosis and management of ARF, as prerenal azotemia is a correctable condition. Assessing daily fluid intake, output, and body weights is valuable when estimating volume status. Heart rate and blood pressure should be measured in the supine and seated with dangled legs positions whenever possible. Careful evaluation of heart and lungs is paramount. The care of severely ill people with sepsis, peripheral edema, third-spacing losses, or underlying heart disease may require insertion of a Swan-Ganz catheter to measure capillary wedge pressure, cardiac output, and systemic vascular resistance. Abdominal palpation may reveal upper quadrant tenderness secondary to ureteral obstruction or renal infarction, as well as a palpable bladder caused by a blocked bladder catheter or prostatic enlargement. Look for leg edema and muscle tenderness from rhabdomyolysis. Occasionally, patients manifest signs of uremic encephalopathy including confusion, stupor, coma, and seizures. Note that altered mental status is also one of the signs of systemic atheroembolism.

Elevation of BUN and serum creatinine concentration is the hallmark of renal failure. In cases of prerenal azotemia and in some patients with obstructive uropathy, the serum BUN/creatinine ratio is elevated above 20:1 because of enhanced reabsorption of urea. Urinalysis (UA) is the most important test in the diagnostic work up of ARF. A normal UA is compatible with prerenal and postrenal azotemia, whereas the presence of many brown granular casts, renal tubular epithelial cells signals the possibility of ischemic or nephrotoxic ARF. A dipstick reading strongly positive for heme pigments in the absence of a significant number of red blood cells suggests rhabdomyolysis or intravascular hemolysis. Eosinophiluria discerned by Hansel's stain associated with fever, rash, and peripheral eosinophilia are typical manifestations of acute interstitial nephritis [22]. The combination of eosinophiluria and ARF following an arteriographic procedure or in a patient with peripheral vascular disease evokes the diagnosis of atheroembolic renal disease. The fractional excretion of sodium (FE_{Na}), calculated from a random urine specimen, is a useful tool in this setting [23].

FE_{Na} (%) is defined as: {(Urine [Na] /Plasma [Na]) ÷ (Urine [Cr]/Plasma [Cr])} × 100. An $FE_{Na} < 1\%$ favors the diagnosis of prerenal azotemia whereas, in acute tubular necrosis, it is usually $>1\%$. There are exceptions, however. For example, some subjects with ARF secondary to severe burns or underlying liver disease have a $FE_{Na} < 1\%$. Conversely, a patient with prerenal azotemia caused by administration of a loop diuretic may have a $FE_{Na} > 1\%$ [23].

The creatinine clearance can be quickly estimated by using the Cockcroft-Gault equation [24]:

For men: (140-age) × lean body mass (kg)
= creatinine clearance in mL/min
72 × serum creatinine (mg/dL)
For women: 0.85 × value for men

For men, lean body mass (LBM) = 106 lb for the first 60 inches, then 6 lb for each additional inch of height. For women, LBM = 100 lb for the first 60 inches, then 5 lb for each additional inch of height.

Severe renal dysfunction may exist in presence of a normal serum creatinine. Consider the case of an 85-year-old woman who weighs 48 kgs and has a serum creatinine of 1.3 mg/dL. The estimated creatinine clearance by the Cockroft-Gault formula is 24 mL/min, which is compatible with severe renal failure. Renal ultrasonography usually detects dilatation of the collecting system and ureters when obstructive uropathy causes ARF.

Prevention of postoperative acute renal failure

The high mortality rate of postoperative ARF, particularly after cardiac surgery, makes prevention a key objective in the overall management of this renal disease. Before surgical interventions, particularly those capable of inducing renal ischemia, one must identify potential risk factors such as volume depletion, hypotension, sepsis, nephrotoxin exposure, obstructive jaundice, and pre-existing chronic kidney disease. Elective surgery should be delayed until those abnormalities are improved. Correction of certain risk factors reduces the threat and ameliorates the consequences of this devastating complication, meaning postoperative ARF. Chertow et al have designed a preoperative renal risk stratification for subjects scheduled to have coronary artery bypass grafting. The goal is not to withhold or advise against required cardiac surgery, but to target high-risk patients for interventions [5].

Volume depletion and hypotension must be corrected promptly. If persistent, they can induce renal ischemia and tubular cell apoptosis with tubular obstruction of sloughed papillae [25]. Sepsis, by causing hypotension, direct renal vasoconstriction, and release of cytokines can provoke postoperative ARF [26]. Hospital-acquired infections should be prevented and treated whenever possible with non-nephrotoxic drugs [27,28]. Acute renal failure secondary to aminoglycoside nephrotoxicity occurs in 10–20% of patients given these drugs [18]. For most patients, it should not be difficult to avoid aminoglycosides.

Nonsteroidal anti-inflammatory drugs (NSAIDs), including ketorolac, a parenteral compound, can cause hemodynamically-mediated ARF by inhibiting the synthesis of prostaglandins which act to preserve renal blood flow and GFR in subjects with volume depletion, pre-existing renal insufficiency, congestive heart failure, and liver cirrhosis [29,30]. Selective cyclooxygenase (COX)-2 inhibitors, like other NSAIDs, must be used cautiously or not at all in patients with predisposing diseases [31]. Because of their inhibitory effect on the efferent arterioles, angiotensin-converting enzyme inhibitors (ACE-I) and angiotensin receptor blockade (ARB) drugs may worsen ARF and should be withheld.

Radiocontrast nephrotoxicity, perhaps caused by alterations in nitric oxide production and direct toxic effects of contrast agents, is another major cause of ARF [32,33]. Prevention, which is the best treatment for this type of kidney disease, includes avoidance of contrast media in at-risk subjects whenever possible, minimization of contrast load, and hydration before and after radiographic procedure [34,35]. A highly encouraging report indicates that pretreatment with N-acetylcysteine may protect against radiocontrast-induced nephropathy [36].

In the postoperative period, effort should be made to diagnose ARF early, eliminate causative agents, and prevent further insults. For example, we should be alert for tamponade caused by large pericardial effusion after cardiac surgery. Immune complex-mediated glomerulonephritis secondary to visceral abscesses subsides with drainage and appropriate antibiotherapy [27]. Early and aggressive hydration followed by mannitol and sodium bicarbonate infusion may minimize pigment-induced ARF, complicating major vascular surgery [37,38]. Obstructive uropathy can be easily treated by relieving the obstruction.

Throughout the world, physicians including nephrologists have employed several pharmacologic maneuvers with the hope of preventing or improving

ARF, precluding the need for dialysis, and reducing mortality. At this date, no hard data has proven their efficacy in humans, and some may even be harmful. Brown et al, in a controlled and randomized trial, showed that a high dose of furosemide (3 g) given intravenously or orally over 24 hrs prevented or reversed oliguria in 24 of 28 patients of the test group versus 2 of 27 of the control group, but the number of dialyses, duration of renal failure, and mortality were not different in the two groups. Moreover, deafness occurred in two individuals given furosemide and was permanent in one [39].

Many physicians believe that low-dose dopamine affects the outcome of ARF. Recently, Bellomo et al, in a multicenter, randomized, double-blind, placebo-controlled study of 328 patients admitted to 23 intensive care units, found that low-dose dopamine conferred no significant protection from renal dysfunction. There was no difference between the dopamine and placebo groups in peak serum creatinine concentration during treatment (245 versus 249 micromol/L; $P = 0.93$), in the number of patients whose creatinine level exceeded 300 micromol/L (56 versus 56; $P = 0.92$), or who required renal replacement therapy (35 versus 40; $P = 0.55$) [40]. Another group of researchers, in a well-designed trial, concluded that dopamine and furosemide did not have any renoprotective effect during cardiac surgery. Their study suggested, furthermore, that furosemide caused renal dysfunction [41].

Improvement in renal function and histopathology in laboratory animals with renal failure, treated atrial natriuretic peptide (ANP), and prompted the study of anaritide, a synthetic form of ANP, in patients with ARF. The Auriculin Anaritide Acute Renal Failure Study Group conducted a multicenter, randomized, double-blind, placebo-controlled trial of anaritide in 504 critically ill patients with acute tubular necrosis. Study subjects received a 24-hour intravenous infusion of either anaritide or placebo. The primary end point was dialysis-free survival for 21 days after treatment. The rate of dialysis-free survival was not different in the two groups (43% in the anaritide group versus 47% in the placebo group, $P = 0.35$). In a subgroup of 120 subjects with oliguria, dialysis-free survival was 27% in the anaritide group and 8% in the control group ($P = 0.008$). Conversely, nonoliguric patients had inferior survival with anaritide, 48% versus 59% dialysis-free survival with placebo, $P = 0.03$ [42]. A similarly designed trial, enrolling only patients with oliguric acute renal failure, failed to confer any advantage to anaritide [43]. At this time, ANP and its synthetic form, anaritide, are not employed in the management of acute renal failure.

Beside conservative management of ARF complications, renal replacement therapy is widely utilized to manage patients with fluid overload, electrolyte abnormalities, particularly hyperkalemia, or acid-base perturbation. Because the mortality rates of ARF have remained elevated, several clinical trials have examined the role of membrane biocompatibility, timing, type, and adequacy of renal replacement therapy on clinical outcomes. At this writing, none of those dialysis-related variables have been securely linked to improved survival in people affected by ARF [44,45]. Frequently, the decision as to which membrane or technique for dialysis is selected depends on patients' condition, costs, and local circumstances such as the availability of an on-site (in the ICU) skilled nurse for hemodialysis and/or a nephrologist familiar with continuous renal replacement therapy. Whether or not dialysis adequacy has an impact on survival of patients with postoperative ARF, erring in favor of too much dialysis is preferred.

Perioperative care of ESRD patients

According to the 2001 report of the U.S. renal data system, diabetes and hypertension in 1999 were listed as the chief causes of end-stage renal disease in North America, accounting for 68.2% of incident ESRD patients funded by Medicare [2]. Hypertensive and especially diabetic individuals suffering from ESRD have serious comorbid conditions such as myocardial dysfunction, coronary artery, and peripheral vascular diseases. Furthermore, the loss of renal reserve hampers their ability to handle fluids, sodium, and acid loads, eliminate potassium, and excrete and/or metabolize medications including antibiotics, analgesics, and anesthetics. Consequently, they may be unable to compensate for normal stresses of surgery. For instance, hyperkalemia resulting from blood products, muscle trauma, hemolysis, metabolic acidosis, and hematoma resorption occurs frequently after surgery. People with ESRD are immunosuppressed and more susceptible to infections. An imprecise number of uremic patients furthermore have bleeding diathesis secondary to platelet dysfunction. Therefore, it is not surprising that individuals with ESRD have an increased surgical morbidity and mortality whose rates vary according to the burden of associated ailments, and the type and emergent nature of the operation. Kellerman, analyzing data from eight studies, reported that the overall mortality of ESRD patients undergoing general surgery was approximately 4%, ranging from 0–47% in emergency cases [13]. The morbidity rate was 54%, varying from 12–64%. In another review involving 13 studies, the same author indicated mortality and morbidity rates were 10% and 46%, respectively, in patients undergoing cardiac surgery [13].

Clinical and laboratory evaluation

Perioperative care of people with known ESRD begins with an in-depth interview, physical examination, electrocardiogram, and screening laboratory tests including a blood count, metabolic panel, serum magnesium, and phosphorus levels, as well as a coagulation profile. The initial goal is detect comorbid conditions that might adversely impact morbidity and mortality perioperatively. Typically, ESRD patients scheduled to have surgery may become symptomatic with coronary artery disease or myocardial dysfunction, fluid and electrolyte abnormalities, hypotension or uncontrolled hypertension, anemia, and a bleeding diathesis. Therefore, the main objective is to correct or improve those disorders before and/or after surgery. Prevention of infections, adjustment of medications, and glycemic control should also be a concern.

Cardiac evaluation

Cardiac disease is the leading cause of death in both diabetic and nondiabetic patients with ESRD. It accounts for almost 50% of deaths among prevalent ESRD patients whose cardiovascular mortality rates are approximately 10–20 times that of the general population [2,46]. Coronary artery disease (CAD), the key factor in the pathogenesis of cardiac disease, is common among ESRD patients, with a prevalence close to 40% [46]. Congestive heart failure, with a prevalence of 40% among hemodialysis and peritoneal dialysis patients, is an independent predictor of death [46]. Beside coronary artery disease, left ventricular hypertrophy whose prevalence is 75% also constitutes a risk factor for the development of CHF [46]. Clearly, cardiovascular disease can complicate perioperative care in individuals with renal failure.

For the general population, clinicians integrate information from the history, physical examination, and electrocardiogram in order to develop an initial estimate of perioperative risks. In people affected by chronic renal disease, defining a cardiovascular risk profile based on clinical variables is difficult. Although many subjects with both kidney failure and coronary artery disease have a typical history of exertional dyspnea and angina or hypotension-induced chest pain during dialysis, 23–40% have silent ischemia documented by Holter monitoring on and off dialysis [47,48].

Several researchers found, furthermore, that approximately 75% of their diabetic patients with angiographically significant CAD were asymptomatic [49,50]. The inverse situation, namely angina without CAD, is also common. Rostand et al indicated that 47% of their patients complaining of angina had trivial or absent coronary artery occlusion [51]. Assessing functional capacity in ESRD patients is often impossible because of anemia or dialysis-induced weakness, diabetic neuropathy, claudication, or joint and bone pain secondary to renal osteodystrophy and amyloidosis. Also, clinical manifestations of CHF may be different in dialysis patients from those in other patient cohorts. Ultrafiltration, in very compliant subjects, minimizes fluid accumulation and negates the typical symptoms and signs of CHF. Intradialytic hypotension may be the only indication of left ventricular dysfunction [46]. These points should be kept in mind when evaluating ESRD patients.

Because typical manifestations of cardiac disease may be lacking in ESRD patients, we have to turn to noninvasive tests such as echocardiography, thallium stress testing, dipyridamole thallium imaging, combined dipyridamole-exercise thallium scintigraphy, and dobutamine stress echocardiography. Echocardiography in 433 subjects at initiation of renal replacement therapy revealed that 15% had systolic dysfunction, 32% demonstrated left ventricular dilatation with preserved systolic function, and 74% displayed concentric left ventricular hypertrophy [52]. The practicability of exercise testing, even combined with thallium imaging, is limited because patients' physical drawbacks or their inability to reach target heart rate. There are also concerns about difficulties in the interpretation of exercise electrocardiographic tracings in the presence of left ventricular hypertrophy (LVH). Additionally, though thallium stress imaging has a sensitivity of 90% for detection of CAD, its specificity is only 68% [53]. As an alternative to stress testing in ESRD patients, dipyridamole thallium imaging is hampered by widely varying sensitivities and specificities. In nonuremic subjects without diabetes, the sensitivity and specificity of myocardial perfusion imaging have been reported to be 79% and 76% [54]. For ESRD patients, sensitivity and specificity vary respectively from of 37% to 86% and from 73% to 79% [55].

Fortunately, a promising report indicates that the combination of dipyridamole and exercise thallium imaging may be more accurate in dialysis patients [56]. Dipyridamole-exercise thallium imaging and coronary angiography were both performed prospectively in 60 asymptomatic hemodialysis patients. The sensitivity, specificity, positive and negative predictive values, and overall accuracy of thallium imaging were 92, 89, 71, 98, and 90%, respectively. After a median follow-up of 2.8 years, the probability of surviving without a coronary event was significantly higher in patients with normal thallium image than in those with an abnormal test (adjusted risk ratio 9.2; $P < 0.005$) [56]. Dahan et al explain their findings by suggesting that dipyridamole is a less sensitive stimulus for detection of CAD than maximal exercise, but its use with submaximal exercise may be as accurate as maximal exercise alone.

Dobutamine stress echocardiography (DSE) has been very valuable in finding CAD in renal transplant patients when the clinical event rate is used for test validation [57,58]. Conversely, DSE is an imperfect screening test when quantitative coronary angiography (QCA) is used to detect CAD. The sensitivity and specificity of DSE for CAD diagnosis were 52% and 74%, respectively, compared with QCA stenosis of 50% or greater; 75% and 71% compared with QCA stenosis >70%; and 75% and 76% for stenosis >75% by visual estimate [59].

The noninvasive detection of CAD in people affected by ESRD remains problematic. Coronary angiography, the gold standard for the diagnosis of CAD, is invasive and costly and cannot be used as a screening method. It should be reserved for people with a high risk of CAD and for those who would benefit from revascularization.

For the general ESRD population, no published practice guidelines have been devised for perioperative cardiovascular evaluation for noncardiac surgery. De Lemos and Hillis, proposing a diagnostic management strategy for screening for renal transplantation, stratified their patients into groups with low, intermediate, and high risk [60]. Transplant candidates younger than 50 years, without diabetes or symptoms suggestive of CAD or CHF but with a normal EKG, had a low cardiovascular risk and did not require invasive cardiac evaluation. Individuals older than 50 years or diabetic without symptoms of CAD or CHF were at intermediate risk and should have non-invasive testing and subsequently coronary angiography if either dipyridamole thallium imaging or dobutamine echocardiography is positive. All high-risk patients, meaning those with symptoms of CAD, electrocardiographic evidence of previous myocardial infarction, or congestive heart failure should have cardiac catheterization before renal transplantation. This management strategy is derived from studies involving renal transplant candidates who are, in general, healthier than the rest of the ESRD population. Also, renal transplantation is an intermediate cardiac risk procedure. Although the ESRD patient may be able to tolerate the surgical procedure short-term, the long-term prognosis is equally important because of the limited availability of donor kidneys. In emergency cases, one has to weigh the benefits of the surgical procedure against the risk of a fatal or nonfatal cardiac event.

Fluid and electrolyte management

Whether or not dialysis has been initiated, euvolemia should be attained when ESRD patients are being prepared for surgery. For those individuals not on replacement therapy, a euvolemic state can be achieved with diuresis or hydration as appropriate. In other instances, euvolemia is securable with dialysis. Currently, there is no very good measure of adequacy for fluid removal in dialysis patients. In practice, notion of dry weight, the lowest weight tolerated without intradialytic symptoms or hypotension in the absence of overt fluid overload, is employed [61]. Subjects who have a stable dry weight with minimal fluid gain between treatments can undergo emergency surgery without dialytic therapy, provided there is no other indication for dialysis.

Establishing the quantity of fluid to be removed is difficult when fluid overload is accompanied by muscle mass wasting or left ventricular dysfunction exists. Fluid overload is definitely an indication for preoperative dialysis. Cautious volume extraction is preferable to prevent unwanted bouts of intradialytic hypotension. Furthermore, excessive fluid loss may also worsen hypotension secondary to anesthesia-induced vasodilation. At times, ultrafiltration or dialysis must be offered postoperatively in patients receiving a large fluid volume during surgery. It is unusual to find major changes in serum sodium concentration in ESRD. Subjects receiving dialysis have an obvious and ready means for adjusting water surplus or deficit [62].

Hyperkalemia may occur before and after surgery. Dialysis is the treatment of choice in both instances when the serum potassium exceeds

6 mEq/L. Medical treatment of hyperkalemia has to be initiated when surgery is emergent or dialysis is not readily available [62,63]. If the EKG shows signs of a dangerous arrhythmia, cardioprotection or membrane stabilization is accomplished by infusing 10 mL of calcium gluconate with ECG monitoring. Insulin is very effective in driving potassium into the cells of patients with renal failure by stimulating the activity of a Na-K-ATPase pump. Consequently, it increases the net movement of extracellular potassium into the intracellular fluid. Although glucose infusion induces endogenous insulin secretion, it is less effective in the management of hyperkalemia. Patients should be monitored carefully for hypoglycemia. β2-adrenergic agonists also shift potassium into the cells through Na-K-ATPase stimulation. Caution is warranted with β2-agonists because of the risk of tachycardia and arrhythmias which can be dangerous in patients with CAD or with the administration of anesthesia. Serum potassium reduction with sodium bicarbonate is negligible unless there is moderate or severe metabolic acidosis. β2-agonists, sodium bicarbonate, glucose, and insulin drive potassium from one milieu to another and correct hyperkalemia only temporarily. Removing a potassium surfeit is accomplished with sodium polystyrene sulfonate. Forty grams of the resin dissolved in 80 mL of sorbitol is a standard oral dose. Alternatively, 50–100 g in 200 mL of water is given as a retention enema. Oral or rectal dose of resin should be repeated every 2–4 hours. It is important to remember that the resin can cause intestinal necrosis, especially when it is given with sorbitol within the first week after surgery.

Anemia and bleeding diathesis

The ideal hemoglobin level for people with ESRD remains a controversial issue. The Anemia Work Group of the National Kidney Foundation-Kidney Disease Outcome Quality Initiative (NKF-K/DOQI) recommends that the hemoglobin level be maintained between 11 and 12 g/dL [64]. By consensus, transfusion is appropriate for people with hemoglobin levels of 8–10 g/dL when extensive surgery is contemplated or excessive blood loss is a possibility. For elective surgery, the target level of hemoglobin can be reached over weeks by increasing erythropoietin dose, adding intravenous iron if necessary, and by transfusion of packed red blood cells.

Postoperative bleeding occurs for many reasons and a specific cause must be sought. Heparin-induced bleeding is unusual as the drug is withheld during dialysis on the day of surgery. During the postoperative period, dialysis patients undergo heparin-free dialysis for at least 24 hours. Uremic patients may have platelet dysfunction resulting in an increased bleeding tendency manifested by a prolonged bleeding time [65]. Individuals with a prior history of uremic bleeding must be treated before surgery. Dialysis, desmopressin (dDAVP, *l*-desamino-8-*d*-arginine vasopressin) administered intravenously or intranasally at a dose of 0.3 μg/kg, and cryoprecipitate are capable of stopping the bleeding [66]. Raising the hematocrit to 30% improves uremic bleeding [67]. Intravenous conjugated estrogens (0.6 mg/kg) are an adequate alternative when they are given 4 or 5 days before surgery [68].

Hypotension and hypertension

Hypotension or hypertension may afflict dialysis patients. Hypotension can be episodic and intradialytic or persistent [69]. The first type occurs in up to 20% of the dialysis population and is caused by several factors such as rapid or excessive fluid removal, left ventricular and autonomic dysfunction, low sodium concentrate, and intake of antihypertensive drugs before dialysis. Usually, the renal team corrects those abnormalities by adjusting the dry weight, increasing dialysate sodium concentration, and by employing steady ultrafiltration [69]. Treating anemia with erythropoietin, increasing the dialysate calcium concentration, and using cool temperature hemodialysis may improve cardiovascular performance in many dialysis patients [69]. Approximately, 5% of long-term patients suffer from persistent, chronic hypotension which limits fluid removal during hemodialysis. Midodrine, a selective alpha-1 adrenergic agonist, is useful in this condition [70]. During the postoperative period, hypotension may occur because hemorrhage, arrhythmia, pericardial tamponade, or sepsis. Appropriate treatment of the causative factor will improve hypotension.

Fluid retention, augmented sympathoadrenal discharge, endothelin increase, and nitric oxide reduction likely represent the primary factors underlying the hypertension of renal failure [62,71]. Optimization of fluid status with dialysis and ultrafiltration lowers elevated blood pressure. Preoperative anxiety and withholding of antihypertensive drugs worsen hypertension. When fluid removal is not successful or dialysis cannot be performed immediately, labetalol, enalaprilat, or hydralazine can be administered intravenously. In the intensive care units, intravenous nitroprusside can be used for 1 or 2 days. Accumulation of thiocynate, a metabolite of nitroprusside, can occur and cause anorexia, disorientation, and toxic psychosis in ESRD patients.

Drug therapy

Adverse drug response occurs more frequently in uremic patients than in people with normal GFR [72]. Abnormalities of drug metabolism with renal failure consist of prolonged half-life of drugs and active metabolites excreted by the kidneys as well as changes in bioavailability, volume of distribution, and protein binding [73]. Adverse drug reactions can affect all organs including the failing kidneys. It is prudent to abstain from prescribing nephrotoxic drugs for subjects with renal failure. Some reports suggest that the presence of residual renal function is associated with a lower mortality risk in dialysis patients [74,75]. As a rule, before administering any drug to subjects with renal failure, one should determine if a dosage reduction is necessary or if a particular drug should be avoided. The American College of Physicians and the American Society of Internal Medicine published comprehensive guidelines for drug prescribing in renal failure. Medication dosing varies with the degree of renal failure, drug biotransformation, and type of renal replacement therapy [73].

Sedative premedication with benzodiazepines is advised only in reduced doses because chronic renal failure increases the free fraction of those preparations. For example, dialysis patients receiving alprazolam may develop psychomotor and memory abnormalities [76]. Meperidine, whose metabolite is normeperidine, can produce excitatory central nervous system effects including seizures and should be avoided [77]. Caution is warranted with morphine because its conjugation with glucuronic acid results in morphine-6-glucuronide which possesses opioid activity and is excreted by the kidney [78]. Fentanyl is metabolized in the liver, with only 7% excreted unchanged in the urine. It is moderately bound to plasma protein and its volume of distribution is large. Premedication with fentanyl is safe in ESRD [79].

Inhaled anesthetics proffer advantage over intravenous agents because they are eliminated primarily via the lungs and not the kidneys. Halothane, desflurane, and nitrous oxide can be administered to kidney failure patients [79]. Absence of significant change in protein binding and clinical effects of metabolites associated with hepatic metabolism make propofol a suitable agent for the induction of general anesthesia [80,81]. Succinylcholine, used without difficulty in people with renal failure, can cause hyperkalemia—particularly in traumatized, burned, or neurologically injured patients. Succinylcholine, in large doses, should be avoided as its metabolite, succinylmonocholine, is weakly active and excreted by the kidney [79]. Nondepolarizing muscle relaxants like atracurium are the blocking agents of choice for ESRD patients [79].

Antibiotic treatment is common during the perioperative period either for prophylaxis or treatment of infections. Some studies indicate that preoperative antibiotics reduce the risk of infections following vascular access procedures or peritoneal catheter placement [82,83].

The use of low-molecular-weight heparins remains controversial. Data concerning their pharmacokinetic and pharmacodynamic profiles in patients with renal failure are limited. It has been suggested that their doses be decreased by 50% when the GFR is lower than 10 mL/min [73].

Glycemic control

Surgical stress and certain anesthetic agents stimulate the release of counter-regulatory hormones such as glucagon, growth hormone, cortisol, epinephrine, and norepinephrine, whose combined effect worsens insulin deficiency and resistance [84]. Consequently, hyperglycemia and even ketogenesis in type 1 diabetic patients may take place during or after surgery. Additionally, those receiving insulin or oral agents are at risk for hypoglycemia because of necessary preoperative fasting. The goal in managing diabetic uremic patients is to maintain plasma glucose levels between 150 and 200 mg/dL during surgery to protect against hypoglycemia [85]. After surgery, targeting blood glucose levels between 120 and 180 mg/dL reduces morbidity attributed to fluid and electrolyte imbalance, decreases the risk of infection, and perhaps accelerates the wound-healing rate [86]. In planning management, the type of diabetes and surgical procedure, the current therapeutic regimen, and degree of recent glycemic control are considered. Numerous protocols have been suggested for treating diabetic patients undergoing surgery. Generally, no intraoperative treatment is recommended for people treated with diet alone or diet and oral hypoglycemic agents if glycemic control is acceptable (80–200 mg/dL). Subjects receiving insulin or poorly controlled type 2 patients require insulin during the perioperative period. Preoperative insulin recommendations are complex. Key to proper management is reliance on frequent finger stick glucose measurements [85]. For early-morning procedures, insulin can be administered subcutaneously. Continuous insulin infusion is the most rational and physiologic approach for insulin-treated patients undergoing long, complex operative procedures or for people requiring surgery while in ketoacidosis [64,84–87]. After outpatient surgery, a preoperative regimen can be reinstituted when patients resume eating. Diabetic control is difficult in those with gastroparesis or when surgical procedures interdict oral intake.

Summary

Preventing postoperative ARF, especially in subjects with pre-existing chronic kidney disease, and caring for ESRD patients undergoing surgery are challenging and best accomplished by a team comprised of primary care physician, nephrologist, cardiologist, surgeon, anesthesiologist, endocrinologist, and nutritionist. Elimination of risk factors for ARF whenever possible, as well as early diagnosis, may improve the outcome of this devastating illness. Drugs capable of preventing or changing the course of postoperative ARF may be available soon. For uremic patients, a comprehensive approach is necessary to minimize morbidity and mortality imposed by numerous comorbid conditions.

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