Postoperative Care/Critical Care of the Transplant Patient

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KEYWORDS

• Solid-organ abdominal transplantation • Complications of abdominal transplantation
• Posttransplant critical care • Early allograft function

KEY POINTS

- Recipient selection criteria for abdominal solid-organ transplantation are being relaxed to increase patient access.
- Donor selection criteria are being relaxed to expand allograft supply.
- Assessment and optimization of early allograft function are the principal goals of critical care.
- Astute surveillance, early diagnosis, and appropriate treatment of postoperative complications improve outcomes.

INTRODUCTION

The notable achievements in organ transplantation since the introduction of effective immunosuppression have dramatically changed the prognosis for patients with renal, hepatic, pancreatic, and intestinal failure.[1–4] General improvements in outcomes of abdominal solid-organ transplant recipients (ASORs) have resulted in relaxation of candidate eligibility criteria and a large increase in the number of patients awaiting an allograft.[5] Since 2000, the percentage of liver and kidney recipients older than 65 years has increased by 84% and 101%, respectively (**Fig. 1**).[5]

Increasing recipient demand has stimulated relaxation of donor selection criteria, creating a unique allograft qualifier termed expanded or marginal.[6,7] Expanded criteria donor (ECD) allografts imply a greater risk of donor transmitted disease or allograft failure.[8,9] These inferior-quality allografts may be suitable for selected recipients

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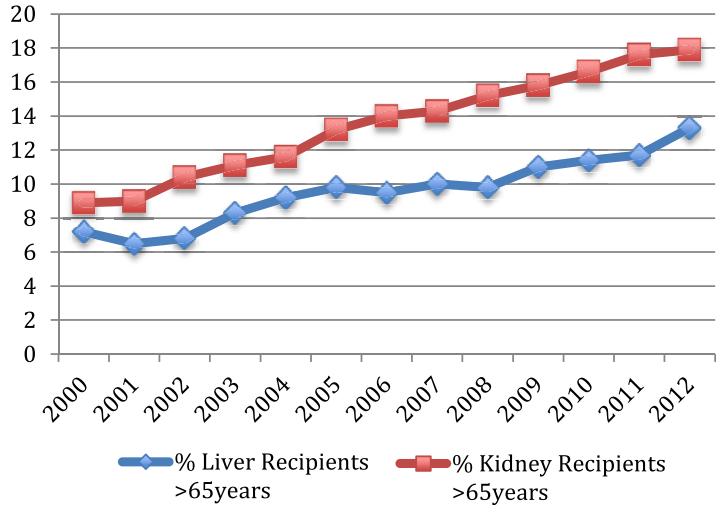

Fig. 1. The increasing percentage of liver and kidney recipients aged more than 65 years. (*Data from* Scientific Registry of Transplant Recipients, United Network for Organ Sharing.)

who would otherwise wait too long for a standard criteria allograft.[10–12] ECD allografts show a higher incidence of delayed graft function and ischemia/reperfusion injury (IRI) in the immediate postoperative period. As the donor and recipient envelopes expand, the risk to ECD recipients has never been higher, whereas potential consequences from complications in this physiologically fragile recipient group have never been greater.

This article describes the 3 principles germane to the management of ASORs: early assessment of allograft function; optimizing support for the transplanted allograft within the context of the recipient's preexisting conditions; and surveillance, recognition, and management of early postoperative complications.

EARLY ASSESSMENT OF ALLOGRAFT FUNCTION

Understanding the donor, recipient, and intraoperative events is paramount to assessment of early allograft function (EAF). All available donor information, including hemodynamic stability, allograft function at recovery, acidosis, vasopressor utilization, sepsis, biopsy data, concomitant organ-system failure, and any history of organ dysfunction, is essential. The mechanism of recovery (donation after brain death [DBD] or donation after cardiac death [DCD]) has the single largest effect on predicting the function of kidney, liver, pancreas, and intestinal allografts. DBD is the most common recovery technique; however, DCD has recently shown significant growth with the potential to expand the donor pool greatly.[13]

The physiologic changes occurring in DBD and DCD are fundamentally different and poorly understood (**Table 1**).[14] Progression to brain death in DBD is secondary to cerebral insult and mass effect. These intracranial events may or may not be associated with systemic ischemia. Recovery occurs in the presence of continuous organ perfusion within the heart-beating brain-dead donor until it is deliberately interrupted by the surgeon, who instantaneously initiates cold allograft perfusion. Because the donor has been legally declared brain dead, the prerecovery period provides an opportunity for donor resuscitation, optimization of organ function, and clinical assessment through additional laboratories or biopsy, before allocation.[15]

Table 1
Significant clinical differences between DBD and DCD

	DBD	DCD
Death criteria	Neurologic	Cardiopulmonary
Donor conditioning	Yes	Minimal
Intraoperative conditioning	Yes	No
Organ assessment	Before perfusion	After perfusion
Preservation	Immediate	Delayed
Injury pattern	Insult-stabilization-conditioning-recovery	Insult-stabilization-withdraw-ischemia-recovery

Abbreviations: DBD, donation after brain death; DCD, donation after cardiac death.

DCD donors do not fulfill brain death criteria so recovery can only begin after declaration of cardiopulmonary death followed by a variable observation period to exclude spontaneous autoresuscitation.[15] The patient's neurologic status limits resuscitation efforts, precludes invasive procedures to assess organ quality, prevents extensive assessment of end-organ function, and impedes allograft optimization before recovery. Additional ischemic injury follows withdrawal of support and cardiopulmonary arrest at recovery. Organ assessment can only occur after procurement has proceeded through cold perfusion with no opportunity for intraoperative organ resuscitation.[14]

The recipient's intraoperative record should be reviewed for events that accentuate IRI, such as hypotension, vasopressor utilization, acidosis, a failed anastomosis that required revision, thrombosis of an anastomosis, or other technical difficulties that may have prolonged warm ischemia. Substantial differences in the hemodynamic environment between donor and recipient can affect outcome in renal transplantation because allografts from hypertensive donors perform poorly in the setting of recipient hypotension.

In renal transplantation, delayed graft function (DGF) is defined as a requirement for dialysis within 1 week after transplantation.[16] Subsequent studies identified DGF as acute renal failure resulting in posttransplant oliguria, enhanced allograft immunogenicity, an increased risk of acute rejection, and decreased long-term survival.[1,17,18] ECD allografts, by definition, show a greater than or equal to 70% risk of failure compared with standard criteria allografts.[6] ECD donors are defined as greater than or equal to 60 years of age or aged 50 to 59 years with 2 of the following 3 characteristics: cerebrovascular accident as the cause of death, a history of hypertension, or a terminal creatinine of greater than 1.5 mg/dL.[6]

The effect of DGF on long-term renal function depends on recovery technique. DGF is observed more frequently among DCD renal allografts[18,19]; however, DGF does not significantly affect long-term renal function, particularly when the DCD donor was less than 50 years of age and cold ischemia time is limited to less than 12 hours.[19] In DBD, DGF is a significant risk factor for acute rejection, primary nonfunction, chronic renal insufficiency, and decreased graft survival.[1,16,20] DGF in the setting of a DBD or living donor renal allograft warrants an immediate investigation for an underlying vascular cause or complication of rejection.

DGF for extrarenal abdominal allografts is less defined. With regard to liver transplantation, estimation of hepatic allograft function is difficult because it depends on the preoperative patient condition as well. Liver allocation is based on the Model for End-Stage Liver Disease (MELD) score,[21] a disease severity score based on total bilirubin, serum creatinine, and International Normalized Ratio; however, up to 25% of patients receive

exceptions whereby their allocation priority MELD is adjusted for specific conditions, such as hepatocellular carcinoma.[22] As a result, liver transplant recipients may range from a noncirrhotic patient diagnosed with alpha-1-antitrypsin deficiency driving in to the hospital from home to a cirrhotic patient intubated in the intensive care unit on vasopressors and hemodialysis. This variability complicates the definition of DGF; however, a recent definition based on bilirubin, transaminases, and International Normalized Ratio has been retrospectively validated, (**Box 1**).[23] Although metabolic demand from the liver cannot be measured directly, metabolic demand can be expected to increase with recipient physiologic MELD, hypothermia, acidosis, large intraoperative blood loss, or past medical history significant for extensive abdominal surgery or retransplantation. If allograft supply cannot satisfy demand or requires extrahepatic support, the allograft is experiencing DGF. If allograft supply plus maximal extrahepatic support cannot satisfy demand, the allograft is showing primary nonfunction. Predictors of DGF include donor age greater than 70 years, cold ischemic time greater than 12 hours, greater than 30% macrovesicular steatosis, DCD, and deteriorating donor physiology at recovery.[7] Donor liver function tests have never been shown to correlate with posttransplant allograft function. MELD score on posttransplant day 5 has recently been shown to be a sensitive predictor of 90-day mortality and allograft failure.[24]

A liver donor risk index (DRI) has been created that incorporates age, cause of death, height, DCD, partial allograft transplantation, and allocation unit as variables in creating a metric to estimate the risk of allograft failure compared with an ideal donor.[25] Although an important first step in creating a metric to quantify allograft risk, the liver DRI is insufficiently powered to be the sole metric in determining allograft suitability.

The surgeon and anesthesiologist should be queried about intraoperative bile production, correction of acidosis, vasopressor requirements, and urine output because these are critical in assessing EAF. Additional early benchmarks include demonstrable mental status, lactic acid clearance, thermoregulation, glucose production, correction of coagulopathy, and preservation of renal function.[26]

EAF in pancreas and small bowel allografts is even less defined. In pancreas and small bowel transplantation, immediate allograft performance is not a physiologic mandate because the principal allograft functions can be temporarily supported.[12,27,28] The risk of these allografts is thrombosis with resulting ischemia and necrosis. The challenge with EAF is to verify function as a marker of allograft viability. Thus, the absence of verified allograft function should prompt an immediate investigation for allograft thrombosis.

Pancreatic and intestinal allografts are low-blood-flow organs with parenchyma that are exquisitely sensitive to ischemia. Pancreatic allografts manifest ischemia as pancreatitis when IRI causes local inflammation that reduces microvascular flow

Box 1
Definition of early allograft dysfunction in liver transplant recipients

INR greater than or equal to 1.6 on postoperative day 7

Bilirubin greater than or equal to 10 mg/dL on postoperative day 7

ALT or AST greater than 2000 IU/mL within the first 7 postoperative days

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; INR, International Normalized Ratio.

Adapted from Olthoff K, Kulik L, Samstein B, et al. Validation of a current definition of early allograft dysfunction in liver transplant recipients and analysis of risk factors. Liver Transpl 2010;16:943–9; with permission.

and promotes thrombosis. Thrombosis accelerates parenchymal ischemia leading to necrosis, superinfection, and additional inflammation.[28,29]

Intestinal allografts show ischemic injury through epithelial sloughing with disarray of Langerhans crypts, which stimulates its own immunologic response in addition to pathogen translocation leading to further inflammation and necrosis. The immunologic destruction of the mucosal lining stimulates fibroblast invasion and fibrosis, which further impede intestinal absorption.[12]

Meticulous surgical technique during recovery and allograft preparation is essential to intestinal and pancreatic allograft outcomes. The extensive microvascular beds within intestines and pancreas are sensitive to increased pressure during recovery or excessive volumes of preservation fluid. Donor risk factors associated with allograft thrombosis include increased donor age, hemodynamic instability, catecholamine requirements, and acidosis. Cold ischemia exceeding 12 hours is an additional independent risk factor for technical failure.[28–30]

The appearance and texture of the pancreas on completion of the procedure is essential because pancreatitis and allograft edema are common at reperfusion. In this setting, osmotic diuretics are frequently administered to decrease edema and improve microvascular perfusion. A similar strategy is used with intestinal allografts to limit edema and bowel distention. The failure of an allograft to respond to intraoperative interventions aimed at reducing edema is a signal to maintain astute surveillance of allograft function perioperatively.

OPTIMAL SUPPORT OF THE TRANSPLANTED ALLOGRAFT

Optimizing the recipient's physiology after transplantation yields the greatest chance of organ recovery. In patients with DGF following renal transplantation, immunosuppressive protocols should be continued with standard target drug trough levels.[31] The traditional practice to lower immunosuppression in the setting of DGF for renal transplant patients is not supported by current literature.

Estimation of hepatic EAF is challenging. An essential strategy is that an ounce of prevention is worth a pound of cure because supplementing early hepatic function and deescalating therapies as the allograft shows sufficient function averts large resuscitations for recipient instability. This strategy includes broad-spectrum antibiotics and antifungals targeted against translocation, intubation until mental status is certain, blood product supplementation to avert coagulopathy, and prevention of hypothermia. Early trophic feeding maintains enterocyte health and decreases bacterial translocation while stimulating enterohepatic circulation to improve biliary physiology.[10]

Optimal support for the pancreatic allograft focuses on detection and prevention of thrombosis. Although never scientifically validated, prophylactic anticoagulation involving low-dose systemic heparin in the operating theater with dose escalation through the first week after transplantation supplemented with aspirin therapy is routine.[28] Volume status, particularly in the setting of combined kidney-pancreas transplantation, is critical to avert venous thrombosis because the newly transplanted kidney produces large amounts of urine, particularly when diuretics have been administered to reduce pancreatic edema.[28,29]

Pancreatitis and pancreatic ischemia typically present as abdominal pain, peritonitis, ileus, and fever.[28,32] Serum amylase and lipase correlate poorly with the severity of allograft pancreatitis, whereas prolonged posttransplant hyperamylasemia is observed in more than 30% of recipients[33]; however, trend analysis is helpful. In particular, a sudden increase in amylase and lipase with a change in exogenous insulin

requirements is a predictor of graft necrosis. The use of Doppler ultrasound is increasing in popularity. This modality is operator and patient habitus dependent; however, when satisfactory imaging is achieved, Doppler ultrasound is sensitive in diagnosing thrombosis.[34] Computed tomography with contrast angiography has been advocated in the diagnosis of vascular complications; however, the authors prefer surgical exploration to computed tomography because most of these patients also have a transplanted renal allograft.

There are even fewer serum markers and radiologic modalities to guide the management of intestinal transplant recipients. In this population, clinicians only have physical examination, ostomy output, and laboratory analysis for hyperkalemia or metabolic acidosis. As in pancreas recipients, prophylactic anticoagulation is routine; however, appropriate volume resuscitation is even more difficult because volume depletion occurs secondary to inflammation and poor fluid absorption from the transplanted intestine in addition to ileostomy losses.[12,35] The differential diagnosis between metabolic acidosis secondary to IRI and luminal losses of bicarbonate is important because luminal bicarbonate losses can be treated with replacement therapy.[35] Intestinal transplant recipients typically show malnutrition and protein deficiency that results in low oncotic pressure and further loss of intravascular fluids.[35] As in liver transplantation, large bolus-type volume resuscitation should be avoided, whereas goal-directed fluid and electrolyte therapy achieves a balanced physiology. Preserving the integrity of the intestinal villi requires early nutritional support through trophic feeding of dilute glucose-containing formulas. As intestinal motility is restored, enteral feedings can be accelerated.

RECOGNITION AND MANAGEMENT OF EARLY COMPLICATIONS

Infectious Complications

Posttransplant infections include bacteremia, fungemia, pneumonia, wound infection, intra-abdominal fluid collections, urinary infections, and *Clostridium difficile* colitis.[36] Prophylactic antimicrobial and antiviral therapies have complemented improved surveillance, detection, and treatment of sepsis, but, despite these efforts, sepsis remains the most common cause of postoperative morbidity and mortality.[37–39] This observation is not a clinical failure but an observation that the microbiology and epidemiology of infectious complications after transplantation continue to change.

Understanding of the changing dynamics of infectious complications in ASORs must begin with the definition of an infectious complication that varies with allograft type. For liver and intestinal allografts, infectious complications after a technically successful surgical procedure represent allograft failure. This concept clarifies the important roles of each of these organs in immune function and pathogen protection. In pancreas and renal transplantation, immediate postoperative infectious complications may represent an iatrogenic event, an existing infection unappreciated at the time of surgery, or surgical complication.[40]

The changing landscape for microbiologic and viral infectious complications has evolved as a result of relaxing ASOR criteria with expansion of the donor pool.[41] The adoption of MELD as the liver allocation scheme fundamentally increased recipient acuity while donor selection criteria have been relaxed to ameliorate organ scarcity.[8] The functional mismatch created by high-acuity recipients, who have an increased requirement for immediate EAF, receiving transplanted allografts with limited immediate hepatic supply creates an opportunity for sepsis and multi–organ-system failure.[42]

In renal and pancreas transplantation, the widespread adoption of induction therapy with antilymphocyte antibody preparations in the immediate postoperative period provides an opportunity for pathogens secondary to abrupt impairment of cellular immunity. Avoiding volume overload, interstitial edema, and undesired areas of hemorrhage limit opportunities for infectious pathogens.

C difficile infection mandates particular attention because of its increasing incidence and morbidity.[43] *C difficile* infection occurs in approximately 19% of liver, 16% of kidney, 9% of intestinal, and 8% of pancreas-kidney recipients compared with less than 1% of patients without transplants.[44,45] *C difficile* may present as classic fulminant colitis with endotoxemic shock or more subtly as a manifestation of nonspecific symptoms including diarrhea.[45] For transplant recipients, the sequelae of diarrhea-induced hypotension, hypovolemia, and electrolyte disorders affect allograft function and predispose to thrombosis.[46] *C difficile* infections peak within 3 months following transplantation and result from antibiotic-induced floral changes, impaired immunity as a consequence of intense immunosuppression, and increased exposure to the health care setting.[47]

Various biomarkers including procalcitonin and C-reactive protein have been widely investigated in the setting of critically ill ASORs. To date, only an increasing C-reactive protein has been found to have clinical value in predicting an infectious event.[48,49]

Pulmonary Complications

Pulmonary complications can be infectious or noninfectious. Pulmonary infectious complications show variability according to allograft type. For renal and pancreas transplant recipients, pulmonary complications typically originate from an operative complication or the high incidence of diabetes, hypertension, coronary artery disease (CAD), and obstructive lung disease within these populations. The risk of aspiration on induction is high, particularly among pancreas recipients, who show diabetic gastroparesis.[28,50] In a multicenter study, the incidence of pneumonia among renal recipients admitted to the intensive care unit with acute respiratory failure was 35% with in-hospital mortality that exceeded 20%.[50] The physiology of cirrhosis is associated with restrictive lung disease secondary to ascites, hepatic hydrothorax, or obesity. Pretransplant hepatopulmonary syndrome persists in the immediate postoperative period and may require prolonged ventilation or oxygen administration. Acute respiratory distress syndrome resulting from bacterial translocation is the principal threat with small bowel transplantation.

Pneumonia is a leading cause of sepsis, prolonged posttransplant hospitalization, and mortality among ASORs. The incidence of hospital-acquired pneumonia within 7 days of liver transplantation approximates 15%.[38] The pathogens were 60% gram-negative and 40% gram-positive with greater than 90% sensitivity to broad-spectrum β-lactams and aminoglycosides. Multivariate analysis identified lactatemia, vasopressor requirements, Simplified Acute Physiology Score II (SAPSII) on admission, and mechanical ventilation exceeding 48 hours as being associated with development of pneumonia.[38] Early diagnostic bronchoscopy with short-term comprehensive empiric antibiotics decreases overall antibiotic exposure, reduces the potential for antibiotic resistance, and minimizes the morbidity associated with diagnostic delay.[51]

Noninfectious pulmonary complications include pleural effusion, atelectasis, diaphragmatic dysfunction, pneumothorax, pulmonary embolism, and pulmonary edema.[52] Pleural effusion, pneumothorax, and atelectasis are easily diagnosed and self-limited if treated early. Acute respiratory failure is common in liver and intestinal transplantation because of upper abdominal surgery, large intravascular volume

shifts, and high transfusion requirements.[52] Acute respiratory failure among kidney and pancreas recipients is typically a complication of antilymphocyte globulin therapy.[53]

When evaluating pulmonary edema, it is essential to distinguish the hydrostatic versus permeability types. Hydrostatic pulmonary edema is common, particularly in liver transplant recipients, in which the incidence has been reported to exceed 50% in some series.[54] This process is self-limited and typically responds to diuretics with little impact on morbidity.

Permeability or noncardiogenic pulmonary edema portends a poor prognosis. Precipitating factors for this type of pulmonary edema include sepsis, gastric aspiration, and multiple transfusions.[55] In an observational analysis the cause of pulmonary edema was evaluated by comparing the protein content of pulmonary fluid with plasma protein content. Most liver recipients had noncardiogenic pulmonary edema, leading the investigators to conclude that the cause is transfusion-related acute lung injury (TRALI).[55]

The correlation of TRALI with posttransplant pulmonary edema supports the 2-event causal model. The initial priming event is a proinflammatory catalyst such as surgical stress or IRI that activates pulmonary endothelial cells and neutrophil sequestration within the pulmonary circulation. Antibodies, lipids, and biologic mediators from transfusion then sequentially activate these primed neutrophils to release a variety of molecules leading to endothelial cell damage, loss of capillary membrane integrity, capillary leak, and interstitial edema.[56]

Treatment of noncardiogenic pulmonary edema should incorporate lung protection strategies using low-tidal-volume ventilation.[57] Positive end-expiratory pressure has been discouraged secondary to theoretic concerns about reduction in venous return and impairment of hepatic venous outflow resulting in allograft congestion; however, emerging data in liver transplantation suggest that positive end-expiratory pressure of up to 15 cm H_2O does not affect Doppler flow velocities within the portal vein, hepatic artery, or hepatic veins.[58]

Cardiac Complications

Cardiovascular (CV) complications are a leading cause of morbidity and mortality, with the highest mortality observed in the immediate posttransplant period.[59–61] Although CV disease is highly prevalent among renal and pancreatic transplant candidates, increasing data refute the conventional concept of a cardioprotective effect of end-stage liver disease. Cirrhotics have a significantly higher prevalence of risk factors for obstructive CAD than the general population.[61,62] A consensus panel of the American Heart Association and American College of Cardiology has released guidelines for the evaluation and management of cardiac disease of kidney and liver transplant candidates.[63] The panel recognized an increased risk among ASORs for cardiac events but acknowledged the need for additional data to facilitate risk stratification.

Current data in liver transplantation suggest that preexisting CAD only contributes a limited component to posttransplant CV morbidity.[64] A notable exception is a subset of candidates with angiographically documented multivessel CAD, even in the absence of severe ($\geq$70%) coronary artery stenosis, which is associated with significantly increased mortality. Additional causes contributing to posttransplant CV morbidity include plaque instability, microvascular thrombosis secondary to transient hypercoagulable states, cirrhotic cardiomyopathy, and uremic cardiomyopathy. The lack of a correlation between obstructive CAD or revascularization before transplantation and CV morbidity has raised speculation that perioperative major adverse cardiac events are not caused by stable plaques propagating inward to create a stenosis, but

by vulnerable plaque rupture or erosion that stimulates thrombus formation and arterial occlusion.

In renal transplant recipients, a single-center observational study determined no survival difference between candidates receiving coronary angiography (CA), coronary intervention, and no invasive cardiac intervention. These results imply that CAD screening may not be necessary and serve only to restrict access to a life-extending renal transplant.[65]

Early data on the sensitivity of cardiac biomarkers in identifying perioperative CV events is promising. In a retrospective study of liver recipients, pretransplant increase of serum troponin I was an independent risk factor for the occurrence of a posttransplant cardiac event.[66] A similar prospective study of kidney and kidney-pancreas recipients determined that pretransplant troponin I was a significant, independent predictor of CV mortality.[67]

Renal Complications

Renal failure following transplantation of a nonrenal organ is a common cause of morbidity and associated with a 4-fold increase in the relative risk of death.[17,68,69] In a landmark study, postoperative acute renal failure, defined as a 50% glomerular filtration rate reduction or requirement for renal replacement therapy (RRT), occurred in 7.6% of ASORs and doubled the risk of chronic renal failure.[17,69] Furthermore, ASORs have an estimated cumulative risk of developing end-stage renal disease that approaches 2% annually.[69] RRT after transplantation increases hospitalization, infectious complications, and mortality.[69] However, recent data suggest that any acute kidney injury (AKI), not just renal injury precipitating RRT, negatively affects long-term morbidity and mortality.[68,70] In a study of liver recipients, posttransplant creatinine changes of as little as 0.5 mg/dL significantly decreased patient and graft survival, with overall mortality corresponding with the severity of renal injury.[68]

Nephrotoxic immunosuppression and infectious prophylaxis compound the risk of AKI. The highest incidence of AKI is observed after intestinal transplantation because of surgical contributions, intense immunosuppression, and underlying allograft injury.[12] As long-term survival following intestinal transplantation increases, the effect of chronic kidney disease on survival has become apparent.[71]

The significance of posttransplant renal function has stimulated identification of biomarkers for earlier detection of perioperative renal injury. The 2 most studied markers are cystatin C and neutrophil gelatinase-associated lipocalin (NGAL). NGAL has shown the most promise through multiple prospective single-center studies of liver transplant recipients.[72–74] A study comparing NGAL with historical renal markers and intensive care unit organ failure scores showed that plasma NGAL was superior to creatinine at predicting AKI, particularly when combined with the APACHE (Acute Physiology and Chronic Health Evaluation) scoring system.[74] Cystatin C is a marker of renal function (not injury) and is independent of muscle mass, age, or sex. Cystatin C may reflect glomerular filtration fraction better than conventional serum markers such as creatinine.

Strategies to optimize posttransplant renal function have been widely disseminated. Modification of immunosuppression to include antibody induction therapy or addition of mycophenolate mofetil with calcineurin inhibitor sparing can be considered in addition to dose adjustment of prophylactic medications in patients with posttransplant AKI. Early detection of chronic calcineurin nephrotoxicity and conversion to renal-sparing immunosuppression is paramount to preserving renal function.

SUMMARY

The maturation of abdominal solid-organ transplantation as a clinical entity has brought new challenges to the intensivists caring for these patients. The relaxation of recipient criteria combined with expansion of the donor pool has opened the field to a wide spectrum of clinical situations. In this environment, donor factors, recipient comorbidities, intraoperative events, and estimation of EAF are essential. Recognition of EAF, optimization of the recipient in support of the allograft, and early treatment of postoperative complications provide a mechanism for serving the largest population of recipients with the widest variety of allografts.

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