The Impact of MELD Allocation on Simultaneous Liver–Kidney Transplantation

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Model for End-Stage Liver Disease (MELD) allocation has improved the process for ranking patients on the liver transplant list. One unintended consequence has been an increase in the number of simultaneous liver-kidney (SLK) transplants. Some have argued that the system unfairly advantages patients with kidney disease and that some kidneys are being prematurely placed in SLK transplantation. This review summarizes the MELD score, assessment of kidney function in cirrhosis, the impact of kidney function in liver disease, and changes in kidney function status in liver transplant recipients in the MELD era. Finally, recommendations regarding who should receive SLK transplants are reviewed.

Introduction

Serum creatinine is heavily weighted in the equation used to calculate Model for End-Stage Liver Disease (MELD) scores, so impaired kidney function has become increasingly present in liver transplant recipients. Moreover, hepatorenal syndrome has become a more important condition in liver transplantation, and more liver transplant candidates with intrinsic chronic kidney disease (CKD) are undergoing transplantation. In fact, a liver transplant candidate with end-stage renal disease (ESRD) on dialysis starts with a MELD score of 20 even before the International Normalized Ratio (INR) and serum bilirubin levels are added to the MELD score calculation. Currently, about 10% of liver recipients are on dialysis at the time of transplant, receive simultaneous liver-kidney (SLK) transplants, or both (Table 1) [1].

According to the guidelines of the United Network for Organ Sharing (UNOS), listing for kidney transplantation alone (KTA) requires a calculated glomerular filtration rate (GFR) of less than 20 mL/min. There is no GFR threshold for SLK listing, however, so SLK and KTA recipients differ in their degree of renal function impairment at transplantation. In fact, only about 60% of those receiving SLK transplants are on dialysis at transplantation. These facts are disturbing to those involved primarily with kidney transplantation. All organs are precious and need to be placed wisely. With the MELD score, the subsequent increased priority of liver transplant candidates with significantly impaired renal function, and the increase in the number of SLK transplants, a call has been made to develop a consistent evaluation process and selection criteria for SLK transplants in liver transplant candidates with impaired kidney function.

The Development of the MELD Score

The system for allocation of donor livers for transplantation has evolved over the past 25 years. In 1984, the US government passed the National Organ Transplantation Act, which established the Organ Procurement and Transplantation Network (OPTN) and responded to the need to allocate organs fairly for transplantation. The OPTN has operated under contract with UNOS since 1986. Originally, patients were prioritized based on time on the wait list, reflecting the principles of kidney allocation. Subsequently, it was recognized that a priority system for distributing organs to the sickest patient was needed. Levels of disease severity were created, which aimed to provide higher priority to the most emergent cases. Status 1 patients were emergency cases. The remaining status designations focused on the patient's physical location as a surrogate for disease severity: in the intensive care unit (status 2), in the hospital (status 3), or at home (status 4). The biases and inadequacies of this system were soon recognized. A system that included the Child-Turcotte-Pugh (CTP) score along with waiting time and physical location followed, but this was similarly identified as too

Table 1. Liver/kidney transplantation in the United States, February 2002–June 2005

Transplant group	Patients, *n* (%)
LTA, no hemodialysis	11,055 (89.9)
LTA, hemodialysis	556 (4.5)
SLK, no hemodialysis	277 (2.3)
SLK, hemodialysis	406 (3.3)
Total	12,294 (100.0)

LTA—liver transplantation alone; SLK—simultaneous liver–kidney transplantation.
(*Data from* Scientific Registry of Transplant Recipients [SRTR] 2005 report [1].)

subjective and not discriminating enough [2]. In addition, Freeman and Edwards [3] reported that waiting time did not correlate with mortality. Deaths on the wait list continued to increase. An objective, fair, and accurate system to allocate organs was still needed.

In 1998, the Department of Health and Human Services issued the Final Rule, stating that standard criteria should be developed for placing patients on the wait list and for assessing listed patients' medical status, and that waiting time should be discounted. Livers were to be allocated using a more continuous system and in order of medical urgency, while still avoiding futile transplantations [4].

As part of the Final Rule mandate, UNOS formed an ad hoc Liver Allocation Committee. MELD immediately caught the committee's attention. MELD was first developed in 2000 as a means to predict survival after transjugular intrahepatic portosystemic shunt (TIPS) placement [5,6]. The original model used INR, serum creatinine, serum bilirubin, and a correction for disease etiology (alcoholic or cholestatic liver disease received fewer points than other causes) to estimate 3-month survival. The committee put MELD through extensive testing as a predictor of short-term pretransplant survival in multiple, large, diverse cohorts of patients with a wide variety of liver diseases. These studies found that etiology of liver disease was not a significant variable in predicting survival in the model, but the remaining objective laboratory values provided a high degree of concordance with 3-month and 1-year mortality. These findings led to acceptance of the model as a predictor of survival in patients with liver disease, regardless of etiology and without considering waiting time. In 2002, the MELD score was adopted as the method for prioritizing patients on the liver transplant wait list. The MELD score equation is $0.957 \times \text{Log}_e(\text{creatinine mg/dL}) + 0.378 \times \text{Log}_e(\text{bilirubin mg/dL}) + 1.120 \times \text{Log}_e(\text{INR}) + 0.643$. The score is multiplied by 10 and rounded to the nearest whole number. Laboratory values less than 1.0 are set to 1.0 for the purposes of MELD score calculation. In the model, serum creatinine is capped at 4 mg/dL, and those who have been dialyzed twice within a week are assigned a creatinine of 4 mg/dL.

The MELD score provided a standardized, objective, continuous model and nearly eliminated waiting time as a factor in ranking. After MELD was implemented, the national liver transplant wait list decreased by 12% in the first year [7], mortality on the wait list declined by 15% [8], median waiting time decreased by more than 200 days (from 656 to 416 days) [9], and, importantly, there have been no decreases in 1-year patient and graft survival rates or in the number of transplants performed [10••]. MELD allocation also has not increased the risk of renal failure after liver transplantation [11].

Assessment of Kidney Function in Liver Disease

Although allocation by MELD represents a significant improvement in prioritization of liver transplant candidates, it is not without criticisms. In examining the MELD equation, one can easily see that creatinine has the greatest impact on the overall score, reflecting the influence of kidney dysfunction on survival in liver failure patients.

In assessing kidney function, the main aim is to determine the GFR, the sum of the filtration rates in all of the functioning nephrons. GFR can be directly measured using clearance of exogenous markers (eg, inulin, I^{125} iothalamate, iohexol) or may be estimated using serum creatinine or equations based on serum creatinine (eg, the Cockroft-Gault, Modification of Diet in Renal Disease [MDRD], or Nankivell equations). Of these estimates, the MDRD equation has been found to be the most accurate but still shows significant differences when compared with measured GFR. The six-variable MDRD equation includes urea, albumin, ethnicity, gender, age, and body surface area. Gonwa et al. [12] evaluated measured GFR (by I^{125} iothalamate) and estimated GFR (using MDRD, Cockroft-Gault and Nankivell) before and after liver transplantation in over 1400 patients. Although MDRD had the highest correlation with measured GFR, only two thirds of the estimates were within 30% of true GFR, highlighting the fact that our assessment of kidney function through standard estimation techniques is lacking. These inadequacies in measurement of kidney function are combined with great variability in definitions of renal failure and lack of standardization of assays used to measure serum creatinine.

Creatinine is an end product of metabolism. Creatine is released from the liver, taken up by muscle, and dehydrated to creatinine. Serum creatinine is affected by many factors not related to the kidneys (total muscle mass, hydration, dietary intake of creatinine). Some have argued that it is inadequate and too imprecise for measuring renal function in patients with cirrhosis. Creatinine may be an inaccurate indirect measurement in patients with cirrhosis for several reasons: 1) in cirrhosis, production of hepatic creatine is decreased; 2) edema results in wider distribution of creati-

nine and thus lower serum creatinine; and 3) the use of certain medications (eg, cephalosporins) alters tubular creatinine secretion [13]. In addition, serum creatinine does not always correlate with measured GFR, and it has been shown to have low sensitivity for detection of CKD.

Fluctuations in serum creatinine can significantly alter the MELD score. These variations (due, for example, to dehydration or overdiuresis, blood loss, or nephrotoxic agents) may be spurious and reversible and thus not meaningful. To avoid giving unfair advantage to those with transient elevations in creatinine values, OPTN rules require serial testing of laboratory values, every 7 days for those with a MELD score higher than 25 and every 30 days for a MELD score 19 to 24.

Despite these criticisms, serum creatinine is unlikely to be replaced by other measures because its measurement is inexpensive and widely available. The MDRD equations may provide for better estimates of GFR in cirrhosis, but these estimates still use creatinine in the calculation. Direct measurements of GFR are less widely available and much more expensive.

Impact of Pretransplant Kidney Function on Liver Transplantation Outcomes

It is widely known that CKD is a risk factor for morbidity and mortality. Renal insufficiency is common in patients with liver disease both before and after transplantation. About one third of patients undergoing liver transplantation have impaired kidney function as defined by GFR less than 56 mL/min [14]. Hemodynamic effects in cirrhosis and primary disease account for the association between liver disease and renal dysfunction. Hepatorenal syndrome accounts for some of this association, but the diagnosis of this potentially reversible condition is sometimes difficult.

Using the Scientific Registry of Transplant Recipients (SRTR) database, Nair et al. [14] examined the impact of renal function on survival in more than 20,000 patients undergoing liver transplantation. They defined three categories of renal dysfunction based on creatinine clearance (CrCl): mild (40–69.9 mL/min), moderate (20–39.9 mL/min), or severe (< 20 mL/min); CrCl of 70 mL/min or higher was considered normal. Normal renal function or mild dysfunction was found in 89% of patients. Moderate or severe renal dysfunction was associated with primary graft nonfunction, higher 30-day mortality, and worse 1-year, 2-year, and 5-year graft and patient survival. Patients needing renal replacement therapy also have worse outcomes [15,16]. Analyses of large numbers of patients from the SRTR database previously showed that SLK transplantation results in better outcomes than liver transplantation alone (LTA) in patients whose serum creatinine is less than 2 mg/dL [17].

In fact, in almost every published survival model, pretransplant renal function is the most robust predictor of posttransplant survival. Patients with pretransplant renal dysfunction are also more likely to experience more significant posttransplant renal impairment [18], which significantly affects outcomes.

Kidney Function after Liver Transplantation

Recipients of any solid organ transplant are at risk for posttransplant CKD. About one quarter of liver transplant patients experience acute kidney injury (AKI) immediately after surgery, but estimates vary widely, owing in part to variability in definition of AKI. Those with AKI who require renal replacement therapy have worse outcomes [18]. Mortality is high in patients with AKI after liver transplantation; one report cites mortality rates up to 90% [19]. On average, recipients lose about 40% to 50% of their kidney function after liver transplantation because of a combination of the effects of calcineurin inhibitors, hemodynamic factors, progression of preexisting kidney disease, and the development of hypertension and diabetes mellitus. During the first 10 years after transplantation, up to one quarter will develop severe CKD and about 10% will develop ESRD. Risk factors for development of CKD include older age, use of calcineurin inhibitors, pretransplant hepatorenal syndrome, preexisting kidney disease, hypertension, hepatitis C, and diabetes mellitus [20,21].

In their landmark paper, Ojo and colleagues [20] showed that liver transplant patients fare worse in terms of kidney function than recipients of other nonkidney solid organ transplants (heart, lung, and heart-lung) (Fig. 1). At 5 years, 18.1% of liver transplant patients are classified as having chronic renal failure, versus 15.8% for lung transplants, 10.9% of heart recipients, and 6.9% for heart-lung transplants. Creatinine clearance of 30 to 59 mL/min was associated with a 2.5-fold risk of developing chronic renal failure, and those with CrCl of 29 mL/min or less had a relative risk of 3.8 compared with those with CrCl greater than 90 mL/min. The mortality rate for those developing CKD was 4.5-fold higher than for those who did not.

MELD Allocation and Kidney Function Before and After Liver Transplantation

With the institution of MELD allocation, the degree of renal impairment in recipients at the time of liver transplantation is much greater than it was in the pre-MELD era. An evaluation of the SRTR database in the post-MELD era revealed a 41% increase in patients on dialysis at the time of liver transplantation and a 177% increase in combined liver and kidney transplantation [22]. In 2005, 7.8% of all patients receiving LTA were on dialysis. Among those who underwent SLK transplantation, only 59% were on dialysis, compared with about 80%

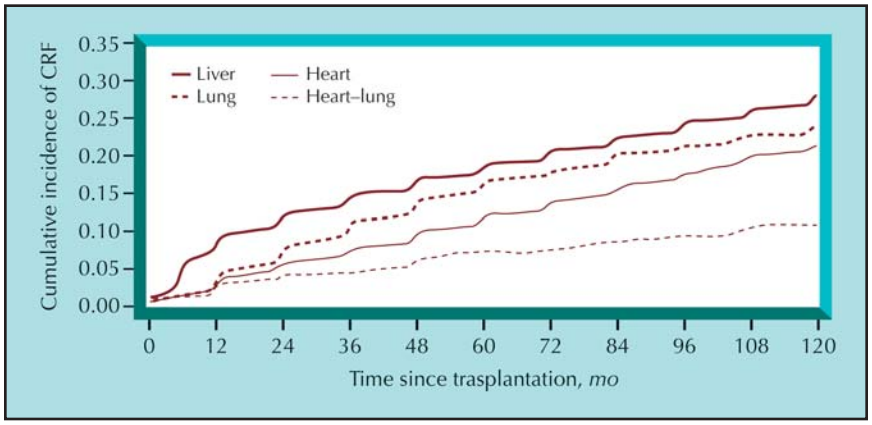

Figure 1. Cumulative incidence of chronic renal failure (CRF) in recipients of nonrenal transplants, 1990 to 2000. (*Adapted from* Ojo et al. [20].)

to 85% of those who received KTA [23,24]. Preoperative need for dialysis has previously been shown to result in decreased posttransplant survival [25,26]. A review of the UNOS database supports the role of kidney function as an important predictor of survival in the post-MELD era for patients who received a deceased-donor liver only, but it is not true for SLK transplant recipients [10••]. Patients who received LTA and were not on dialysis had the highest 3-year survival from 2002 to 2005, more than 80%. Those who underwent LTA and were on dialysis fared the worst, with 3-year survival just over 65%. Survival for SLK transplant recipients was 70%.

MELD Allocation and SLK Transplantation

Combined liver–kidney transplantation has been performed for more than two decades [27]. One of the unexpected consequences of MELD allocation for liver transplantation has been an increase in SLK transplants. In 2001, the year before MELD allocation was adopted, there were 226 candidates for SLK transplantation, and 134 combined transplants were performed. The number of SLK transplants (about 85 to 135 per year) had not varied much since 1994. In 2002, the year MELD was instituted, there were 364 candidates and 210 combined transplant procedures. By 2006, these numbers had grown to 585 candidates and 400 procedures, a 300% increase in SLK transplants within 5 years. Currently about 6% of all deceased-donor liver transplants are SLK transplants [28].

The weight placed on serum creatinine has resulted in a higher rate of SLK transplants but no change in survival. Although some analyses have not shown an adverse effect on outcomes of SLK transplants after MELD was instituted [10••], Locke and colleagues [29••] recently reported that SLK transplantation may be overused and that prioritizing kidneys to liver transplant patients is wasting resources. They suggest that patients who are too old and too ill are receiving kidneys via SLK transplantation to the detriment of other patients awaiting KTA. Their group evaluated data from the UNOS database on more than 19,000 liver transplants, nearly 34,000 kidney transplants, and 1000 SLK transplants between 1987 and 2006. They found that in the MELD era, there was no benefit in the SLK group compared with LTA; in fact, 1-year survival after SLK transplantation declined in the post-MELD years, from a high of 87% in 2002 to 76% in 2005. During the same period, survival with LTA was stable. However, a subgroup of SLK patients who had been on dialysis for at least 3 months before transplantation did have better 1-year patient and liver-allograft survival compared with LTA patients (84.5% vs 70.8%, $P = 0.008$). These authors also reported that SLK transplant recipients in the post-MELD era were older, more ill, and more frequently suffering from hepatorenal syndrome. Interestingly, SLK transplant recipients are likely to receive higher quality liver allografts than LTA recipients, with the donor risk index being 1.55 for SLK and 1.66 for LTA. They found that kidney graft survival was worse in SLK transplant recipients (77.2%) than with KTA (89.3%), even in patients on long-term dialysis. Finally, a MELD score higher than 23 was associated with increased kidney graft loss in SLK transplant recipients.

Who Should Receive SLK Transplants?

The MELD score and the drive to perform SLK transplantation reflect the significant impact of kidney disease on liver disease mortality and transplant outcomes. Though patients with CKD may be easier to sort out, determining which patients with AKI should receive SLK transplants can be challenging. When considering SLK transplantation, several questions must be considered: Which scenario is worse—no functioning kidneys or three functioning kidneys? What is the optimal timing for kidney transplantation? Is it better to perform SLK transplantation or to wait for evidence of nonrecovery of kidney function, then transplant a kidney after the liver? Can we predict beforehand which patients not on dialysis will be more likely to develop CKD and thus be more appropriate candidates for transplantation? Can kidney biopsy data help to guide the decision? Is biopsy safe?

Are we providing patients who undergo SLK transplantation a net benefit similar to that of KTA? In some studies, renal allograft survival for SLK transplants has been shown to be substantially lower than in KTA recipients [30,31••,32]. In a report from the University of California, Los Angeles, SLK transplant recipients experienced more early graft loss than KTA recipients [32]. Fong et al. [30] reported on 800 SLK transplants and 800 paired kidneys given to KTA and kidney–pancreas recipients between 1987 and 2001. Increased mortality due to infection resulted in lower graft and patient survival with SLK transplantation.

Perhaps one should wait to perform kidney transplantation until significant kidney disease develops in patients who have less-clear indications for SLK transplantation. Is there an advantage to transplanting the kidney simultaneously with the liver, or are outcomes just as good with kidney after liver transplantation (KALT)? The immunoprotective effect of the liver allograft on the kidney may result in better kidney outcomes. This hypothesis was evaluated in 1136 SLK transplant recipients who were compared with 352 patients who received KALT [31••]. They found that the renal half-life of the kidney allografts placed after liver transplantation was 5 years shorter than for those in the SLK group, and there was a 10% reduction in 1-year and 3-year rejection-free renal graft survival in the KALT group. This same group has also shown less renal allograft loss from chronic rejection in SLK patients than in those receiving a kidney alone [30]. However, one other report found a possible benefit of delaying kidney transplantation until after renal recovery following LTA [33]. The conflicting nature of these reports calls for more detailed study and discussion.

Will the kidneys recover? It would be ideal to perform SLK transplants only in patients who have CKD or are very likely to develop CKD after liver transplantation. Studies evaluating predictive risk factors for nonrecovery of renal function have not clearly identified which patients should be considered for SLK transplantation. Many patients with cirrhosis who develop acute elevations in creatinine have functional renal insufficiency (ie, hepatorenal syndrome). Several issues with hepatorenal syndrome need to be considered when evaluating patients as candidates for SLK transplantation or LTA. The first is estimating the chance of complete recovery of kidney function after liver transplantation. Part of this question relates to whether the kidneys are "normal" in the first place. It has been known for years that patients with a clinical diagnosis of hepatorenal syndrome who receive LTA often do not recover normal renal function [26]. Data from a recent study evaluating the effects of terlipressin on hepatorenal syndrome showed that 13% of the placebo group had reversal of the syndrome, compared with 34% of the treatment group ($P = 0.008$). Duration of serum creatinine elevation has been shown to predict serum creatinine 6 to 12 months after LTA [34]. Most centers consider length of time on dialysis in selecting candidates for SLK transplantation. Ruiz et al. [32] showed that of 80 patients who were on dialysis for less than 4 weeks before liver transplantation, only 3 required long-term dialysis after LTA.

Renal biopsies are not routinely obtained in these patients, who have significant coagulopathy, but complication rates of the procedure are not well defined. Therapy and consideration as a candidate for SLK transplantation may be altered based on renal pathology. One report by Pichler et al. [35] of 26 liver transplant candidates who had renal insufficiency of unknown etiology for at least 4 weeks showed that kidney biopsy was safe. This report and others found variable histology that did not correlate with urinary or serum findings. The most important contribution of a renal biopsy in these patients is prediction of progression to CKD by identifying fixed renal scarring (interstitial fibrosis, arterial hyalinosis, and glomerulosclerosis) [36]. More than 30% to 50% glomerulosclerosis, arterial hyalinosis, tubular atrophy, or interstitial fibrosis is commonly used as a criterion for SLK transplantation [37,38]. In their series of 26 patients, Pichler et al. [35] recommended SLK transplantation in 10 patients who had more than 40% global glomerulosclerosis, more than 30% of the interstitium composed of interstitial fibrosis, or severe glomerular injury. A consensus panel recommended that kidney biopsies should be performed more commonly and that more data should be collected on complications and on the impact of biopsies on prioritization for SLK transplantation versus LTA [39••].

In 2006, the transplant community met to review post-MELD data on the impact of renal function on the wait list and outcomes for liver transplantation and results of SLK transplantation. The meeting report was published in 2007 [39••]. It recommended a thorough evaluation of patients if creatinine is near or above the upper limit of normal (Table 2). Selection for SLK transplantation was deemed appropriate if measured CrCl is less than 30 mL/min. In patients with acute kidney dysfunction due to hepatorenal syndrome or AKI, renal dysfunction requiring dialysis was considered evidence that renal recovery is less likely. The report recommended 6 weeks of dialysis as a valid selection criterion for SLK transplantation. In patients with AKI not requiring dialysis, SLK transplantation is not justified; LTA recipients with an estimated GFR higher than 30 mL/min have a 1-year posttransplant survival of about 82%, and only 1.5% of these 1648 patients were listed for a kidney transplant within a year after liver transplantation. In candidates with kidney failure of unknown cause, a kidney biopsy showing fixed damage warrants listing for SLK transplantation.

Conclusions

Liver allocation based on MELD scores has succeeded in directing more livers to the recipients most in need, lowering wait-list mortality rates without affecting graft

Table 2. Evaluation for simultaneous liver–kidney transplantation

I. All liver transplant candidates
Serum creatinine
Urinalysis
Dipstick for chemistry and protein
Protein:creatinine ratio
Albumin:creatinine ratio
24-hour urine for volume and protein excretion
II. Further evaluation if
Serum creatinine > 0.8 mg/dL and/or
Urinalysis shows abnormal number of WBCs, RBCs, or casts, and/or
Urinalysis shows abnormal amount of albuminuria or proteinuria
III. Further evaluation includes
Urine electrolytes and creatinine, to calculate fractional excretion of sodium
Anatomic evaluation: ultrasound, CT scan, or MRI to rule out obstruction, stone, or cystic disease and determine differential renal size and cortical thickness
GFR measurement such as iothalamate clearance; if not available, measure 24-hr creatinine clearance
Serology as indicated by history, examination, cause of liver disease, and prior laboratory testing
Measurement of renal blood flow (ultrasound duplex Doppler, PAH clearance, blood oxygen level–dependent MRI)
IV. Kidney biopsy if urinalysis suggests primary renal disease or if the cause of kidney dysfunction is unclear or reversibility is uncertain.

GFR—glomerular filtration rate; PAH—para-aminohippurate; RBCs—red blood cells; WBCs—white blood cells.
(*Adapted from* Davis et al. [39••].)

survival rates. However, MELD allocation has increased the importance of impaired peri-transplant renal function. Going forward, we need to better manage impairment of renal function before and after liver transplantation if we are going to improve outcomes. We also need to better define which recipients need and will benefit from SLK transplantation.

Disclosures

No potential conflicts of interest relevant to this article were reported.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

- • Of importance
- •• Of major importance

1. **Scientific Registry of Transplant Recipients (SRTR) 2005 report: transplants from 2/27/02–6/30/05.** Available at www.unos.org.
2. United Network for Organ Sharing: **Policy 3.6. Allocation of livers.** Available at http://www.unos.org/PoliciesandBylaws2/policies/pdfs/policy_8.pdf. Accessed November 19, 2008.
3. Freeman RB Jr, Edwards EB: **Liver transplant waiting time does not correlate with waiting list mortality: implications for liver allocation policy.** *Liver Transpl* 2000, **6:**543–552.
4. Institute of Medicine: **Analysis of waiting times.** In *Committee on Organ Transplantation. Assessing current policies and the potential impact of the DHHS final rule.* Washington, DC: National Academy Press; 1999:57–78.
5. Malinchoc M, Kamath PS, Gordon FD, et al.: **A model to predict poor survival in patients undergoing transjugular intrahepatic portosystemic shunts.** *Hepatology* 2000, **31:**864–871.
6. Kamath PS, Wiesner RH, Malinchoc M, et al.: **A model to predict survival in patients with end-stage liver disease.** *Hepatology* 2001, **33:**464–470.
7. Wiesner RH, Edwards E, Freeman R, et al.: **Model for end-stage liver disease (MELD) and allocation of donor livers.** *Gastroenterology* 2003, **124:**91–96.
8. Fink MA, Angus PW, Gow PJ, et al.: **Liver transplant recipient selection: MELD versus clinical judgement.** *Liver Transpl* 2005, **11:**621–626.
9. Wiesner R, Lake JR, Freeman RB, Gish RG: **Model for end-stage liver disease (MELD) exception guidelines.** *Liver Transpl* 2006, **12(Suppl):**S85–S87.
10. •• Gonwa TA, McBride MA, Anderson K, et al.: **Continued influence of preoperative renal function on outcome of orthotopic liver transplant (OLTX) in the US: Where will MELD lead us?** *Am J Transplant* 2006, **6:**2651–2659.
A large data set was used to evaluate the effect of renal function on outcomes in patients receiving either deceased-donor LTA (DDLTA) or SLK before and after the implementation of MELD allocation. Preoperative kidney function was a predictor of survival in DDLTA but not in SLK. Survival in SLK patients was superior to that of LTA patients receiving renal replacement therapy (RRT) at transplant. These data suggest that only patients requiring RRT should receive a kidney and that patients with decreased GFR not on RRT should undergo a kidney biopsy.
11. Machicao VI, Srinivas TR, Hemming AW, et al.: **Impact of implementation of the MELD scoring system on the prevalence and incidence of chronic renal disease following liver transplantation.** *Liver Transpl* 2006, **12:**754–761.

12. Gonwa TA, Jennings L, Mai ML, et al.: **Estimation of glomerular filtration rates before and after orthotopic liver transplantation: evaluation of current equations.** *Liver Transpl* 2004, **10:**301–309.
13. Cholongitas E, Shusang V, Marelli L, et al.: **Review article: renal function assessment in cirrhosis—difficulties and alternative measurements.** *Aliment Pharmacol Ther* 2007, **26:**969–978.
14. Nair S, Verma S, Thuluvath PJ: **Pretransplant renal function predicts survival in patients undergoing orthotopic liver transplantation.** *Hepatology* 2002, **35:**1179–1185.
15. Menon KVN, Nyberg SL. Harmsen WS, et al.: **MELD and other factors associated with survival after liver transplantation.** *Am J Transplant* 2004, **4:**819–825.
16. Gonwa TA, Mai ML, Klintmalm GB: **Chronic renal failure after transplantation of a nonrenal organ [letter].** *N Engl J Med* 2003, **349:**2563–2565.
17. Nair SP, Krishnan M, Scheel P, Thuluvath PJ: **Renal allograft survival in patients who had simultaneous liver and kidney transplantation compared with those who had kidney transplantation alone.** *Transplant Proc* 2001, **33:**1139–1140.
18. Afonso RC, Hidalgo R, Zurstrassen MP, et al.: **Impact of renal failure on liver transplantation survival.** *Transplant Proc* 2008, **40:**808–810.
19. Richardson D: **Dialysis in non-renal organ (liver) transplantation.** *Nephron* 2001, **88:**296–306.
20. Ojo AO, Held PJ, Port FK, et al.: **Chronic renal failure after transplantation of a nonrenal organ.** *N Engl J Med* 2003, **349:**931–940.
21. Velidedeoglu E, Bloom RD, Crawford MD, et al.: **Early kidney dysfunction post liver transplantation predicts late chronic kidney disease.** *Transplantation* 2004, **77:**553–556.
22. Dellon ES, Galanko JA, Medapalli RK, Russo MW: **Impact of dialysis and older age on survival after liver transplantation.** *Am J Transplant* 2006, **6:**2183–2190.
23. Kasiske BL, Snyder JJ, Matas AJ, et al.: **Preemptive kidney transplantation: the advantage and the advantaged.** *J Am Soc Nephrol* 2002, **13:**1358–1364.
24. Danovitch GM, Cohen DJ, Weir MR, et al.: **Current status of kidney and pancreas transplantation in the United States, 1994–2003.** *Am J Transplant* 2005, **5:**904–915.
25. Lafayette RA, Pare G, Schmidt CH, et al.: **Pretransplant renal dysfunction predicts poorer outcome in liver transplantation.** *Clin Nephrol* 1997, **48:**159–164.
26. Gonwa TA, Klintmalm GB, Levy M, et al.: **Impact of pretransplant renal function on survival after liver transplantation.** *Transplantation* 1995, **59:**361–365.
27. Margreiter R, Kramar R, Huber C, et al.: **Combined liver and kidney transplantation [case report].** *Lancet* 1984, **1(8385):**1077–1078.
28. Pomfret EA, Fryer JP, Sima CS, et al.: **Liver and intestine transplantation in the United States, 1996-2005.** *Am J Transplant* 2007, **7:**1376–1389.
29.•• Locke JE, Warren DS, Singer AL, et al.: **Declining outcomes in simultaneous liver-kidney transplantation in the MELD era: ineffective usage of renal allografts.** *Transplantation* 2008, **85:**935–942.
This group evaluated over 40,000 transplants (LTA, KTA, SLK) between 1987 and 2006. They found a decline in patient survival after SLK and no benefit in the SLK cohort compared with the LTA cohort. The authors suggest that SLK may waste limited resources.
30. Fong TL, Bunnapradist S, Jordan SC, et al.: **Analysis of the United Network for Organ Sharing database comparing renal allografts and patient survival in combined liver-kidney transplantation with the collateral allografts in kidney alone of kidney-pancreas transplantation.** *Transplantation* 2003, **76:**348–353.
31.•• Simpson N, Cho YW, Cicciarelli JC, et al.: **Comparison of renal allograft outcomes in combined liver-kidney transplantation versus subsequent kidney transplantation in liver transplant recipients: analysis of UNOS database.** *Transplantation* 2006, **82:**1298–1303.
About 1500 SLK and KALT procedures were evaluated. The liver allograft provided protection to the kidney allograft in SLK but not in KALT.
32. Ruiz R, Kunitake H, Wilkinson AH, et al.: **Long-term analysis of combined liver-kidney transplantation at a single center.** *Arch Surg* 2006, **141:**735–741.
33. Ruiz R, Barri YM, Jennings LW, et al.: **Hepatorenal syndrome: a proposal for kidney after liver transplantation (KALT).** *Liver Transpl* 2007, **13:**838–847.
34. Campbell MS, Kotlyar DS, Brensinger CM, et al.: **Renal function after orthotopic liver transplantation is predicted by duration of pretransplantation creatinine elevation.** *Liver Transpl* 2005, **11:**1048–1055.
35. Pichler R, Dittrich MO, Anderson AE, et al.: **Prediction of benefit from simultaneous liver-kidney transplantation versus liver-alone transplantation: potential role for native kidney biopsy.** *J Am Soc Nephrol* 2006, **17:**2919–2927.
36. Coppo R, D'Amico G: **Factors predicting progression of IgA nephropathies.** *J Nephrol* 2005, **18:**503–512.
37. Davis CL, Gonwa TA, Wilkinson AH: **Pathophysiology of renal disease associated with liver disorders: implications for liver transplantation. Part I.** *Liver Transpl* 2002, **8:**91–109.
38. Davis CL, Gonwa TA, Wilkinson AH: **Identification of patients best suited for combined liver-kidney transplantation: part II.** *Liver Transpl* 2002, **8:**193–211.
39.•• Davis CL, Feng S, Sung R, et al.: **Simultaneous liver-kidney transplantation: evaluation to decision making.** *Am J Transplant* 2007, **7:**1702–1709.
This meeting report from a consensus conference of hepatologists, transplant surgeons, nephrologists, and coordinators reviews post-MELD outcomes in liver wait list and SLK transplantation and makes recommendations for evaluation and selection for SLK transplantation in liver transplant candidates with renal dysfunction.