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ORIGINAL ARTICLE

# Assignment of steatotic livers by the Mayo model for end-stage liver disease

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## Summary

Prognosis after liver transplantation depends on a combination of recipient and donor variables. The purpose of this study is to define an allocation system of steatotic donor livers relative to recipient model for end-stage liver disease (MELD) score. We reviewed 500 consecutive OLT, computing the MELD score for each recipient. Fatty infiltration in grafts was categorized in no steatosis, 10–30%, 30–60% and ≥60% steatosis. MELD score did not affect preservation injury and graft dysfunction, which were increased with fat content. Recipient and graft survivals lowered when increasing MELD score. Outcome in low-risk recipients (MELD ≤9) was not altered with steatosis, except those with ≥60%. Survival functions in moderate-risk recipients (MELD 10–19) were moderately affected with 10–30% steatosis and severely with those with >30. Exactly 30–60% steatotic grafts work poorly in high-risk recipients (MELD ≥20), and very poorly with ≥60% steatosis. Prognosis of candidates is optimally influenced when divergence of recipient–donor risks is presented.

## Introduction

The growing discrepancy between the available donor organs and the need for liver transplantation has led to continued reevaluation of selection and listing criteria as well as allocation and distribution policies for donor organs. The most appropriate use of scarce livers continues to evolve and be debated. A main problem in liver transplantation is to assign a single liver donor for one of the recipients listed, combining the principles of efficacy and justice. However, there is an absence of planned strategies to correlate recipient and donor factors and, subsequently, the assignment of a marginal donor for the best recipient is not resolved.

The model for end-stage liver disease (MELD) has been proposed as a replacement for the Child-Turcotte-Pugh (CTP) classification to stratify patients for priorization for orthotopic liver transplantation (OLT) [1]. This disease severity index relies on three objective parameters [total serum bilirubin, serum creatinine and International Normalized Ratio (INR) for prothombin time] and has been validated in patients with diverse etiology and severity of liver disease [2]. The MELD is a good guideline for organ allocation because this scale can score transplant candidates in the order of medical urgency, promoting efficient use of scarce donor organs and avoiding futile transplants. Unfortunately, MELD can only evaluate the recipient prognosis, and allocation algorithms may include also marginal donor variables such as liver steatosis. There is a lack of evidence about how this model can work with higher- and lower-risk donors.

Liver steatosis is the most important variable in multivariate analysis of factors determining graft function after transplantation [3], with substantial risk for primary nonfunction (PNF) or very poor function after reperfusion [4]. The decision to use steatotic livers in a specific patient should be considered in the context of additional risk factors. Although moderate steatosis in otherwise low-risk donors and recipients results in favorable outcome, steatosis combined with additional risk factors should be considered with caution [5,6]. The aim of the present study was to correlate the MELD score of liver transplantation recipients with steatosis in liver grafts to obtain a more real prediction of patient prognosis and post-transplantation liver function.

## Patients and methods

The last 500 consecutive nonfulminant OLT performed were reviewed. Pediatric candidates and those with hepatocellular carcinoma were excluded because original MELD is not appropriate in these groups and specific scales of risk were not still available at the moment of the analysis [7]. The MELD score applied is a slight modification of the risk score used in the original TIPS model obtained by Malinchoc *et al.* [8]. In this analysis, we have applied a recent MELD modification excluding the causes of end-stage liver disease [2]. MELD score was computed for each candidate at two moments, when listed and when transplanted, introducing the values of creatinine, bilirubin and INR in an on-line worksheet available over the Internet at http://www.mayoclinic.org/gi-rst/mayomodel5.html. MELD values were categorized in five groups according to different survival risks in the model: group I: ≤9; group II: 10–19; group III: 20–29; group IV: 30–39; and group V: ≥40 [1].

Procurement of liver grafts was performed following the technique described by Starzl *et al.* [9]: the University of Wisconsin (UW) chilled solution was infused for cooling and preserving through an aortic cannula, with simultaneous crossclamp of the supraceliac aorta. In addition, a portal venous infusion was carried out through a separately cannulated inferior or superior mesenteric vein. The venous bed of the liver was decompressed by a venotomy of the suprahepatic vena cava. The total amount of UW-infusate was guided by blanching of the graft and estimation by palpation of the degree of cooling.

Fatty infiltration in liver grafts was differentiated in macrovesicular and microvesicular steatosis. Two biopsies were obtained from each graft: before hepatectomy of heart-beating donors and after reperfusion of the organ in the recipient. These specimens were fixed conventionally in formaldehyde and then embedded in paraffin; 3–4-μm deparaffinated sections were hematoxylin–eosin (H&E)-stained. Fat droplets displacing the hepatocyte nucleus and occupying the majority of the cytosol were considered macrovesicular steatosis [10] and categorized semiquantitatively in four groups: no steatosis, mild steatosis (<30%), moderate steatosis (30–60%) and severe steatosis (>60%) [11]. Biopsy specimens were reviewed independently by two pathologists.

The histological features of liver preservation injury (LPI) were the severity, type and location of necrosis, inflammation and the location and severity of hepatocelular swelling and cytoaggregation. Cytoaggregation refers to a rounding-up of the hepatocyte, so that the cell assumes a rounded appearance. Preservation injury was categorized according to the severity of inflammation and the severity of necrosis, cytoaggregation and hepatocyte swelling and divided into four grades [5,12].

Primary nonfunction was defined as a nonrecoverable hepatocellular function necessitating emergency retransplantation within 72 h. Delayed nonfunction (DNF) was defined as a graft function necessitating emergency retransplantation within 72 h and the first postoperative month [13]. Initial poor function (IPF) was defined by criteria similar to those of Makowka *et al.* [14] and characterized by peak serum values of AST > 1500 U/l or ALT > 1000 U/l on any day during the first postoperative week.

Survival curves were analyzed by the Kaplan–Meier method and compared with the log-rank test. Recipient survival at 1, 2 and 3 months were compared longitudinally in a crosstab designed with columns of MELD categories and rows of macrosteatosis groups. Comparison of percentages was calculated with the paired-data *t*-test.

## Results

### Demographic and clinical characteristics

Demographic and clinical characteristics of the study population are listed in Table 1. Median age of recipients was 52 years (range, 18–72 years). Thirty-four patients underwent retransplantation for PNF and DNF. Mean

**Table 1.** Demographic and clinical profile of study patients.

| Characteristic/variable | |
|---|---|
| Men | 368 (79) |
| Age (year) | 52 (18–72) |
| Cause of liver disease | |
| Alcoholic liver disease | 125 (25) |
| Chronic hepatitis C | 111 (22.3) |
| Cholestatic liver disease | 43 (8.6) |
| Chronic hepatitis B | 34 (6.9) |
| Cryptogenic cirrhosis | 29 (5.8) |
| Other | 158 (31.6) |
| Laboratory parameters | |
| Serum bilirubin (mg/dl) | 7.5 (0.3–52) |
| Serum creatinine (mg/dl) | 1.38 (0.1–11.0) |
| INR for prothrombin time | 1.6 (1.0–4.5) |
| Child's score | |
| 5–6 (A) | 0 (0) |
| 7–9 (B) | 19 (4) |
| 10–15 (C) | 447 (96) |
| MELD score | |
| ≤9 | 108 (20.6) |
| 10–19 | 242 (48.4) |
| 20–29 | 138 (27.6) |
| 30–39 | 8 (1.6) |
| ≥40 | 4 (0.8) |
| Retransplants | 34 |

Values expressed as number (%) or median (range).

**Table 2.** Distribution of steatotic grafts by MELD categories and distribution of primary (PNF) and delayed nonfunction (DNF).

| MELD category/ Grade of liver steatosis | MELD ≤9 ($n = 108$) | MELD 10–19 ($n = 242$) | MELD ≥20 ($n = 150$) |
|---|---|---|---|
| No steatosis ($n = 255$) | 25 (10) | 130 (51) | 100 (39) |
| PNF | 0 (0) | 0 (0) | 0 (0) |
| DNF | 0 (0) | 0 (0) | 1 (0.4) |
| Steatosis 10–30% ($n = 160$) | 22 (14) | 66 (41) | 72 (45) |
| PNF | 1 (0.6) | 0 (0) | 0 (0) |
| DNF | 1 (0.6) | 1 (0.6) | 2 (1.2) |
| Steatosis 30–60% ($n = 67$) | 19 (13.7) | 28 (42) | 20 (45.3) |
| PNF | 0 (0) | 1 (1.5) | 0 (0) |
| DNF | 5 (7.5)* | 5 (7.5)* | 6 (8.9)* |
| Steatosis >60 ($n = 18$) | 4 (22.2) | 8 (45.6) | 4 (22.2) |
| PNF | 1 (5.5) | 1 (5.5) | 1 (5.5) |
| DNF | 2 (11)* | 3 (16.6)** | 3 (16.6)** |

Values expressed as number (%).
*$P < 0.05$, **$P < 0.01$.

**Table 3.** Distribution of cold and warm ischemia times for the MELD and steatosis subgroups.

| MELD category/ Grade of liver steatosis | MELD ≤9 ($n = 108$) | MELD 10–19 ($n = 242$) | MELD ≥20 ($n = 150$) |
|---|---|---|---|
| No steatosis ($n = 255$) | | | |
| Cold ischemia times | 202 ± 53 | 199 ± 52 | 168 ± 35 |
| Warm ischemia times | 42 ± 17 | 52 ± 17 | 45 ± 13 |
| Steatosis 10–30% ($n = 160$) | | | |
| Cold ischemia times | 183 ± 82 | 191 ± 38 | 175 ± 22 |
| Warm ischemia times | 53 ± 22 | 45 ± 13 | 39 ± 10 |
| Steatosis 30–60% ($n = 67$) | | | |
| Cold ischemia times | 166 ± 59 | 178 ± 62 | 125 ± 54* |
| Warm ischemia times | 48 ± 20 | 41 ± 20 | 38 ± 16 |
| Steatosis >60% ($n = 18$) | | | |
| Cold ischemia times | 162 ± 70 | 132 ± 42* | 98 ± 23** |
| Warm ischemia times | 49 ± 17 | 42 ± 15 | 35 ± 12 |

Values expressed as mean ± SD (min).
*$P < 0.05$, **$P < 0.01$.

serum bilirubin was 7.5 mg/dl, mean serum creatinine 1.38 mg/dl and INR for prothrombin time 1.61. Most patients were Child's class C ($n = 447$, 96%) and 4% were Child's class B.

The mean MELD score at the moment of inclusion in the waiting list was 15.2 (range, −3 to 54) and at the moment of OLT was 16.4 (range, −3 to 58). MELD score increased 1.2 as candidates listing until OLT (mean time 5.6 months, $P = 0.3$). MELD scores ≤9 at the inclusion in waiting list experienced a mean increase of 0.8 until OLT; MELD scores 10–19 increased 2.3; and MELD ≥20 was increased in 4.1 ($P = 0.6$). Of the patients 20.6% had MELD scores ≤9; 48.4% with scores 10–19 and 27.6% with scores 20–29. Because MELD categories IV and V (30–39 and ≥40, respectively) were infrequent, these patients were grouped together with MELD category 20–29 for the proposal of statistical analysis (MELD category ≥20).

Fatty infiltration in biopsy specimens revealed no macrovesicular steatosis in 255 grafts (51%), mild macrosteatosis in 160 (32%), 67 with moderate (13.5%) and 18 with severe macrovesicular steatosis (3.5%). Distribution of steatotic livers by MELD categories was not essentially different ($\Xi^2 = 15.99$; $P = 0.067$) (Table 2).

Cold and warm ischemia times for the whole cohort were 185 ± 76 and 40 ± 13 min, respectively. Table 3 summarizes cold and warm ischemia times for the MELD and steatosis subgroups. These two times were similar for each category, except a decreased cold ischemia times for those transplants with combination of extreme risk factors (MELD ≥20 and fat infiltration ≥30%). It suggests an effort for brief harvesting times in the donor combined with rapid explantation in the recipient with the objective of diminishing the deletereous effect of prolonged cold ischemia times on moderate to severe steatotic livers.

### Liver preservation injury and graft function

Moderate to severe LPI was a common event when increasing the grade of macrovesicular steatosis. Fifty-six (22%) of grafts without macrosteatosis presented moderate to severe LPI; 58 grafts (36%) with macrosteatosis <30% ($P = 0.07$); 44 grafts (65%) with 30–60% macrosteatosis ($P = 0.03$); and 14 (78%) with macrosteatosis >60% ($P = 0.002$).

Primary nonfunction was an uncommon event in our series ($n = 5$; 1%). However, severe graft dysfunction necessitating retransplantation within the first month post-transplantation (DNF) was relatively frequent ($n = 29$; 5.8%). Table 2 depicts DNF with steatotic grafts by MELD categories. DNF was not affected by MELD categories, but an increase in the grade of graft steatosis was significantly associated with more DNF episodes, especially for grafts with macrosteatosis >30% (1% with no steatosis; 4.5% with steatosis <30%; 23% with steatosis 30–60%; and 41% with steatosis >60%; $\Xi^2 = 10.46$; $P = 0.015$).

### Recipient and graft survivals

The MELD scale performed well in predicting death within 3 months in the original report of Kamath *et al.* [1]. This is the reason of computing survival curves for 1, 2 and 3 months in this study. Recipient survival and graft survival by MELD categories are presented in Fig. 1. Recipient survival for MELD ≤9 was 98%, 94% and 89%, respectively; 96%, 90% and 85% for MELD 10–19; and

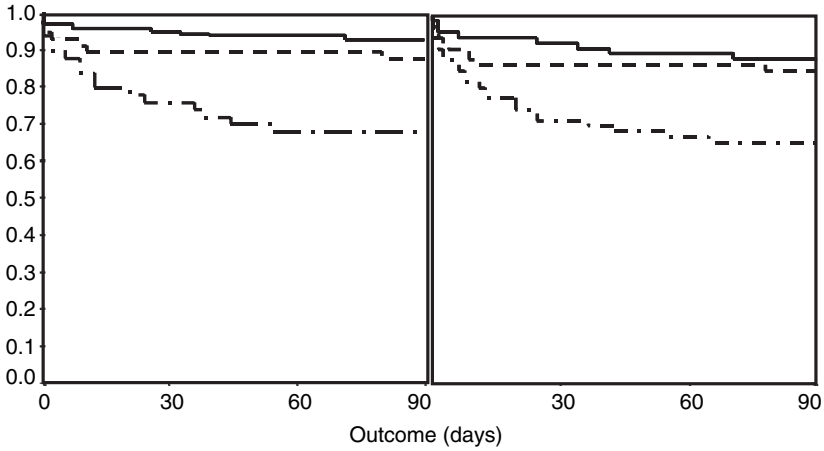


**Figure 1** Overall recipient (left; $P = 0.0031$) and graft (right; $P = 0.002$) cumulative survivals by MELD categories. — MELD <9; - - - MELD 10–19; — · — MELD >20.

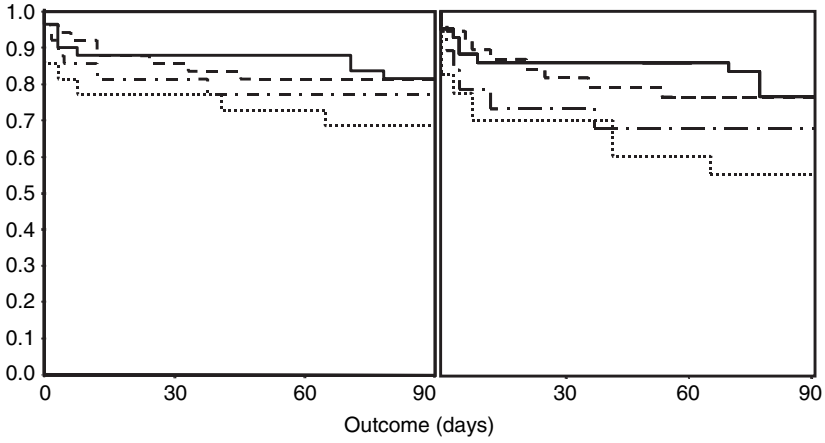


**Figure 2** Overall recipient (left; $P = 0.17$) and graft (right; $P = 0.001$) cumulative survivals by grade of liver steatosis. — No steatosis; - - - 10–30% steatosis; — · — 30–60% steatosis; ····· >60% steatosis.

80%, 73% and 69% for MELD ≥20 (log-rank = 13.88; $P = 0.0031$). Graft survival for MELD ≤9 was 98%, 92% and 88%, respectively; 92%, 88% and 84% for MELD 10–19; and 77%, 67% and 65% for MELD ≥20 (log-rank = 34.07; $P = 0.002$).

Recipient and graft survival with different grades of steatotic livers are shown in Fig. 2. Recipient survival was not statistically different (log-rank = 4.92; $P = 0.17$). However, graft survival was severely affected with livers with high grade of fat content. In comparison with OLT with livers without steatosis (graft survival at 1, 2 and 3 months was 90%, 90% and 84%, respectively), graft survival decreased with livers with 30–60% of macrosteatosis (78%, 72% and 72%, respectively) and with livers with >60% of fat content (69%, 64% and 58%, respectively) (log-rank = 56.20; $P = 0.001$).

### Recipient survival crosstab of OLT with steatotic livers by MELD categories

The effect of increasing grades of liver steatosis and MELD score on recipient survival is depicted in Fig. 3. Recipients with MELD score <9 were not affected with livers with increasing fat content, except those with >60% steatosis (log-rank = 0.11; $P = 0.74$). Recipients with MELD score 10–19 were subjected to low risk with livers with no steatosis, moderate risk with livers with 10–30% steatosis, and high risk with livers >30% of fat content (log-rank = 35.03; $P = 0.003$). Recipient survival with MELD score >20 was not affected with livers with steatosis <30%; however, there was a moderate increase of recipient losses in the group with 30–60% steatosis, and a substantial increase in the group with >60% steatosis (log-rank = 56.20; $P = 0.0001$). Table 4 summarizes recipient survival at 1, 2 and 3 months, combining the potential effect of liver steatosis on MELD categories and a classification of cells in low, moderate and high risk OLT.

## Discussion

The main obstacle in OLT is the scarcity of liver donors, with a growing acceptance of candidates in waiting lists. The primary principle underlying organ allocation in OLT is to offer livers to recipients in greatest need who

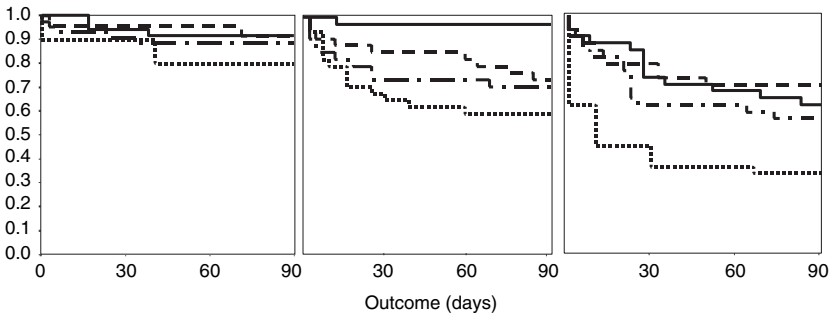


**Figure 3** Potential effect on recipient cumulative survival of steatotic livers by MELD categories. MELD <9 (left; $P = 0.74$), MELD 10–19 (middle; $P = 0.003$) and MELD >20 (right; $P = 0.0001$). — No steatosis; - - - 10–30% steatosis; — · — 30–60% steatosis; ····· >60% steatosis.

**Table 4.** Recipient survival crosstab at 1, 2 and 3 months with steatotic livers by MELD categories.

| | MELD category | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | ≤9 | | | 10–19 | | | ≥20 | | |
| Recipient survival (months) | 1 | 2 | 3 | 1 | 2 | 3 | 1 | 2 | 3 |
| No steatosis | 96* | 94* | 94* | 95* | 95* | 95* | 76* | 70* | 62* |
| Steatosis <30% | 98* | 98* | 92* | 88** | 78** | 78** | 80* | 72* | 60* |
| Steatosis 30–60% | 90* | 88* | 88* | 70*** | 70*** | 68*** | 58** | 58** | 52** |
| Steatosis >60% | 86*** | 72*** | 72*** | 68*** | 60*** | 60*** | 46*** | 38*** | 36*** |

All values without indicators represent low risk (not statistically significant).
*Low risk (no statistically significant); **Moderate risk ($P < 0.05$); ***High risk ($P < 0.01$).

have a substantial risk of dying. However, organs have not been allocated according to uniform medical criteria and the adoption of the CTP classification cannot differentiate the sickest patients with needs of priorization. Another concern is that time on the waiting list has served as the tiebreaker for allocation of the organ and organs have been offered based on accumulated waiting time. Freeman and Edwards [15] concluded that waiting time was not an appropriate measure for the fairness of the organ allocation system and recommended the use of a more objective system to ensure equitable allocation of livers. In February 2002, UNOS instituted MELD to stratify potential recipients of donor livers.

The model for end-stage liver disease is a disease severity index for patients with end-stage liver disease awaiting liver transplantation. This model can predict mortality in patients undergoing TIPS and in several groups of patients with liver diseases including hospitalized and ambulatory patients with cirrhosis [1]. MELD is also superior to CTP score in estimating pre-OLT disease severity in UNOS status 2A patients and thus may help risk stratify status 2A or decompensated status 2B OLT candidates and optimize the timing of OLT [2]. Recently, Wiesner *et al.* [16] showed that MELD accurately predicted 3-month mortality in adult patients who were listed, and is a valid measure of disease severity for patients with chronic liver disease on the waiting list. Despite the current enthusiasm for MELD and the suggestion that use of MELD in organ allocation may lower waiting list mortality, several concerns have been raised. One of them is the need for analyses of impact of MELD on overall patient survival. The impact of MELD on post-transplant outcomes has not been analyzed. This analysis must include: (1) an evaluation of the capability of MELD for predicting early post-transplant mortality; and (2) the influence of donor factors on MELD capability for prognosis.

To answer the first of these questions, a major finding of the present study is that MELD score was able to accurately estimate 3-month mortality in transplanted patients. Our results demonstrate that an increase in MELD score is accompanied with higher rates of recipient and graft losses, irrespective of donor variables. MELD score has a real ability to predict outcome after OLT. This result contrasts with that reported by Brown *et al.* [2], in which neither scoring system, MELD and CTP, could not be predictive of short-term survival post-OLT, suggesting that perhaps outcome after OLT is most directly related to severity of illness at OLT. Allocation of donor livers to the sickest patients may yield significant increases in post-transplant mortality and, subsequently, this policy of liver allocation can reduce waiting list mortality, but may suppose a waste of grafts. In our study, the effect of MELD scale on post-transplant mortality was independent of the moment of computing MELD values. There was not a significant evolution of MELD score in waiting list until OLT procedure.

The second concern is relative to the influence of donor variables on the validation of MELD score for post-transplant prognosis. In the present study, we have tested the role of increasing grades of macrovesicular steatosis in each MELD category. The significance of steatosis on graft function is underscored by its emergence as the most important variable in multivariate analysis of factors determining graft function after OLT [3]. Moderate to severe steatosis is also the most important factor for the appearance of severe LPI [17]. In the present study, recipient survival is not statistically different with livers with different fat content. Graft survival has a dismal prognosis with increasing grades of macrosteatosis. This effect is revealed by higher rates of DNF, ranging from 1% with nonsteatotic grafts to 41% with >60% steatosis. However, distribution of PNF and DNF was not different between groups of MELD scores, suggesting that fat content is a primary factor for graft dysfunction but not related to recipient clinical status.

A major finding of the present study reflects the disparity of recipient survival in each MELD category with different fat content in the grafts. MELD allocation system is intended to identify the patients at higher risk for death within 3–6 months and allocate livers accordingly. MELD allocation schema will result in the allocation of livers that produces outcomes that can satisfy the recipients, transplant community and National Health Systems or insurers, leading to a less capricious and more efficient use of livers [18]. This policy follows the principle of the 'sickest-first' (higher MELD scores): as the patients on the list deteriorate, this may not result in optimal survival of patients at risk with end-stage liver disease. The sickest-first policy is hope-preserving, but may be inefficient, especially under conditions of severe shortage, resulting in organ wastage and excessive morbidity [19]. According to both factors, those from recipients and donors, the final decision should be carried out by doctors, who care for their patients and not by computers (MELD score). Indeed, MELD allocation schema is adequate under conditions of 'optimal shortage'. In those communities with severe shortage (and predictable over-use of extreme marginal donors), MELD score only works partially. A good example may be the use of steatotic grafts: the adverse effects of steatosis on graft and patient survival are more evident in sicker liver recipients. A clinical consensus exists that grafts with severe steatosis should be discarded and grafts with mild steatosis should be used. The question if grafts with moderate steatosis can be used is unresolved. Should such grafts be discarded? Should such livers be transplanted into patients in no urgent need or into patients in urgent need of a transplant but in whom survival of such grafts is poor? [20]. In daily practice, the distribution of steatotic grafts allocated to different clinical status of recipients must follow the principle of divergence of risk factors: grafts with moderate to severe steatosis must not be transplanted in otherwise high-risk patients. This principle can be adopted also with grafts with moderate steatosis and patients with moderate risk. Rules for allocation of steatotic livers in listed candidates must be as follows: (1) Low-risk candidates (MELD ≤9): in this group livers with steatosis >60% must be discarded. (2) Moderate-risk candidates (MELD 10–19): grafts with steatosis >30% must be discarded. Graft with steatosis 10–30% can be used, but this decision depends on the pressure of waiting list. (3) High-risk candidates (MELD ≥20): grafts with <30% of fat content can be safely transplanted; those with >60% of fat content must be discarded; and livers with steatosis 30–60% can be used considering local waiting list mortality. As donor livers with moderate steatosis is the most stressing group to allocate, networks of organ sharing must prioritize these grafts to appropriate low- to moderate-risk recipients (MELD <20), considering additional factors as cold ischemia time, longer waiting lists and time of listing. Three basic situations can be faced:

(**a**) Lists with severe shortage: there is a growing number of sicker recipients and longer waiting times. In this case, the use of livers with moderate steatosis may result in organ wastage and recipient poor prognosis. The policy of organ sharing network must give priority to the assignment of grafts without or with low fat content.

(**b**) Lists with low shortage: there is a little proportion of higher versus lower MELD scores. The number of livers is also reasonable and the turn-over of the waiting list is relatively high. The policy of allocation can assign a greater proportion of fatty livers, giving priority of ''good'' livers to the candidates of the previous group.

(**c**) Lists with moderate shortage: in this group, there is a combination of higher and lower MELD scores. Waiting list can be expanded in addition to the number of available donors. In this case, the assignment of both low and moderate fatty livers may be safe, giving priority to candidates in group (a).

Steatosis is but one of many factors that contributes to patient mortality. The conclusions would be strengthened by multivariate analysis of all those factors identified by univariate analysis as predictive of mortality. This would include, amongst others, cold ischemia times, ventilatory status, doses of inotropic drugs, components of the MELD as well as steatosis grade. It would help to delineate the impact of steatosis and MELD score. Obviously, a larger cohort may be necessary to define this impact.

In conclusion, MELD is a good index for predicting post-transplant outcome. However, its capability of prediction is modulated by donor factors as macrovesicular steatosis. The policy of allocation of steatotic livers must

be a conjunction of recipient MELD score and the grade of fat content in the liver.

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