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**Liver transplantation in acute liver failure: A Challenging scenario**

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**Abstract**

Acute liver failure is a critical medical condition defined as rapid development of hepatic dysfunction associated with encephalopathy. The prognosis in these patients is highly variable and depends on the etiology, interval between jaundice and encephalopathy, age and the degree of coagulopathy. Determining prognosis for this population is vital. Unfortunately prognostic models with both high sensitivity and specificity for prediction of death have not been developed. Liver transplantation has dramatically improved survival in patients with acute liver failure. Still, 25% to 45% of patients will survive with medical treatment. The identification of patients who will eventually require liver transplantation should be carefully addressed through the combination of current prognostic models and continuous medical assessment. The concerns of inaccurate selection for transplantation are significant, exposing the recipient to a complex surgery and lifelong immunosuppression. In this challenging scenario, where organ shortage remains one of the main problems, alternatives to conventional orthotopic liver transplantation like living donor liver transplantation, auxiliary liver transplant and ABO-incompatible grafts should be explored. Although overall outcomes after liver transplantation for acute liver failure are improving they are not yet comparable to elective transplantation.

**Key words:** Encephalopathy; Fulminant hepatic failure; Liver transplantation; Outcome and prognostic scores

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**Core tip:** Acute liver failure is the most dramatic clinical situation in which liver transplantation is performed. In this manuscript we describe the timing and benefits of this procedure by analyzing the different prognostic scores and surgical techniques.

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**LIVER TRANSPLANTATION IN ACUTE LIVER FAILURE: A CHALLENGING SCENARIO**

Acute liver failure (ALF) is characterized by a rapid deterioration of the liver function [international normalized ratio (INR) ≥ 1.5] and the development of hepatic encephalopathy in a patient with no previous history of liver disease, with the onset of encephalopathy within 26 wk of jaundice[1]. ALF accounts for 8% of indications for liver transplantation in Europe and 7% in the United States[2,3]. Globally, viral hepatitis are probably responsible for the majority of cases of ALF. Hepatitis A and E are common in developing countries; while hepatitis B is a common cause in some Asian and South American countries[4-6]. In developed countries drug-induced liver injury, especially with paracetamol, accounts for approximately 50% of cases[7]. Before the era of liver transplantation (LT), ALF mortality rates ranged between 80% and 85%[8]. Advances in the field of critical care management and LT however, have dramatically improved survival outcomes for patients with ALF[9,10]. Current LT results are especially good considering the emergency context of the surgical indication, with 1- and 5-year patient survival rates of 80% and 75%, respectively[9,10]. Nevertheless, LT candidate selection in the ALF setting must be carefully addressed. Risks of emergency transplantation in patients with evolving or established multiple organ failure (MOF) must be balanced against survival with continued medical supportive care alone. In this review, we discuss current decision making strategies used to indicate LT in this challenging clinical scenario.

***Timing of liver transplantation***

Several prognostic evaluation systems use different variables correlating with outcome in ALF patients to identify patients with high likelihood of mortality[11-15] (Table 1), When evaluating the accuracy of a prognostic score to identify those patients who will die if a LT is not performed , one should consider the positive and negative predictive values. The positive predictive value is the probability that a positive prognostic test will truly reflect the need of a LT. On the other hand, the negative predictive value will describe which patients will survive without LT. Four variables are considered key determinants to assess prognosis: etiology, interval between jaundice and encephalopathy, age and synthetic markers of disease severity[16,17]. Most importantly, prognostic models need to be able to capture critical data at crucial time points during the course of disease[18]. Many prognostic models are used worldwide, most based on historical cohorts of patients not receiving a transplant. Although details of the systems differ, they share many common variables (Table 2). Unfortunately, prognostic models have limitations, and their predictive accuracy varies[16,19].

 Described in 1989, the King´s College Hospital criteria (KCC) are amongst the most common set of tests applied for patient selection, and were the first to distinguish between paracetamol-induced and other ALF etiologies[11]. The criteria have a clinically acceptable specificity, in that patients fulfilling the criteria are very likely to die if they are not transplanted. Conversely, sensitivity is less, as a certain number of patients not meeting criteria do not survive[20,21]. A recent meta-analysis from 18 studies analyzing KCC performance in patients with non-paracetamol-induced ALF, totaling 1105 patients[22], reported an overall sensitivity of 68% and a specificity of 82%[22]. Interestingly, specificity increased to 88% when criteria were applied dynamically and to 93% in patients with more advanced encephalopathy. Sensitivity fell in studies published after 2005, suggesting modern medical management of ALF may affect KCC performance[22]. Two separate meta-analyses studied paracetamol-induced ALF[20,23] reporting an overall sensitivity of 58%-69% and specificity of 92%-94%. Again, sensitivity was considered to be low as a result of non-dynamic application of the criteria.[23] The model has been refined and both specificity and sensitivity improved, for example, with the inclusion of lactate[24]. In a cohort of patients with paracetamol-induced ALF, the addition of the postresuscitation lactate concentration to the established KCC, increased the ability to identify patients unlikely to survive unless transplanted[24]. The rationale of including lactate as a prognostic marker is based on that hyperlactatemia reflects not only tissue systemic dysfunction but, most importantly, a substantially decreased hepatic clearance of lactate. Two significant points to remember regarding KCC application are: first, its use is clearly most effective in patients with high-grade encephalopathy; and second, KCC were not formulated as part of a static model, but rather as a dynamic evaluation system, therefore their most effective application will arise from continued patient monitoring.

The Clichy criteria were described in 1986, and originated from the study of a cohort of 115 patients with fulminant hepatitis B[12]. The model is based on decreased levels of factor V, age and the presence of grade 3-4 encephalopathy, with a positive predictive value of 82% and a negative predictive value of 98%[12]. Validation studies have shown Clichy criteria to be not only less accurate than originally reported, but also less accurate than KCC for predicting outcome[25,26]. Seeking for a better alternative to the KCC and Clichy criteria, the following scores were also evaluated: the Sequential Organ Failure Assessment (SOFA), and the Acute Physiology and Chronic Health Evaluation II (APACHE II). The rationale of evaluating non-liver-specific scores is based on the uncontrolled inflammatory response observed in patients with ALF. The SOFA and APACHE II scores are strongly associated with MOF and blood lactate levels[27]. In a retrospective study that included 125 patients with paracetamol-induced ALF, assessment of the prognosis was performed with: KCC; Model for End-Stage Liver Disease (MELD); SOFA; and APACHE II. The SOFA score performed better than the other prognostic scores with an area under the curve (AUC) of 0.79, sensitivity of 67% and specificity of 80%[28]. Interestingly, temporal changes in SOFA score were also evaluated. A SOFA score > 6 by 72 h or >7 by 96 h, post-paracetamol overdose, predicted death/transplantation with a negative predictive value of 97% and 99%, respectively[29]. Use of the APACHE II model is of similar sensitivity and specificity as KCC, but cannot be applied as soon after admission as KCC[30].

More recently, interest has been focused on application of the MELD score to predict ALF outcomes in both paracetamol-induced and non-paracetamol-induced groups. However, consistent advantage has failed to be conclusively demonstrated[6,13,19,31,32]. In order to improve the predictive value of MELD scores, a prognostic score substituting M65 (a cell death-associated marker) for bilirubin was described[33]. Fatal outcome predictability with MELD-M65 score was superior to both classic MELD and KCC[33]. Characterization of cell death-related serum factors were further explored. The Acute Liver Failure Study Group proposed an index based on the composite of different clinical markers on admission (INR, coma grade, bilirubin and phosphorus level) and added to an apoptosis marker: M30[34]. This index outperformed MELD and KCC improving sensitivity to 86%, but reducing specificity to only 65%. Markers of cell death or apoptosis are encouraging, although their use is technically more complex and published results still need to be confirmed in further studies. Alternative prognostic variables have also been suggested to improve selection of transplant candidates. A wide variety of blood markers have been proposed including: α-fetoprotein[35,36], serum phosphate[37], apoptosis and necrosis markers[38,39], monocyte HLA-DR expression[40] and Gc-globulin levels.[41] However, widespread clinical application of these blood markers and other prognostic scores are limited due to lack of external validation and of general availability[42-45].

 Ideally, prognostic models should be simple, accurate and rapidly measured with high positive predictive values and negative predictive values which should not lower specificity. In order to improve LT candidate selection, prognostic models should be applied continuously over time, as part of overall clinical monitoring by experienced multidisciplinary transplant teams.

***Liver transplantation***

In the transplant era, overall survival in patients with ALF underwent considerable improvement. Still, nearly 30% of patients with ALF die[6,9,10]. Reasons restricting transplantation of more patients include: organ shortage, clinical complications, substance abuse issues, or involvement of other organ system (*i.e.*, heart failure, malignancy). Outcomes differ between regions mostly due to differences in etiology and to access to a suitable organ. In Argentina, for example, we reported a large ALF series with no cases of paracetamol toxicity, in clear contrast to United States and United Kingdom series[6,7,9]. ALF associated to paracetamol toxicity has not only a better prognosis than most other etiologies, but is often associated with concomitant psychosocial issues, explaining why general transplantation rates for ALF in the US are lower than in Argentina, 24% and 54%, respectively[6,7,46].

 One-year survival following LT in ALF patients ranges between 74% and 84%. These results are worse than those of patients grafted for other indications[7,10,47]. In spite of this fact, outcomes remain better compared to 64% one-year survival described in patients in ICU immediately prior to LT; and to 54% observed in patients on mechanical ventilation at time of organ allocation[7,47]. Most deaths occur in the first 3-mo after surgery, from neurological complications, MOF or sepsis[10,48-50]. Great efforts have been made to find out which factors affect outcome (Table 3). Those better predicting survival without transplant are not the same as those predicting survival after LT. Graft quality and recipient clinical condition were described as the most relevant factors influencing transplant outcome in this setting[10,50,51]. Multivariate analysis of variables in the United Network for Organ Sharing (UNOS) database identified four factors associated with poor outcome: recipient age > 50 years, history of life support, body mass index ≥ 30 kg/m2 and serum creatinine > 2.0 mg/dL[50]. Five-year survival ranged from 47% for those with all four variables, to 83% for those with none. Limitations identified were that the high-risk group accounted for only 2% of the study population; that cumulative effects of adverse graft factors were not considered; and that the analysis included patients who underwent LT, exclusively.

 Data from the European Liver Transplant Registry (ELTR) were analyzed, including 4903 patients with ALF[10]. Despite certain limitations of the multivariate model, authors identified major risk factors with detrimental impact on post-LT mortality as: use of reduced-sized organs, recipient age > 50 years and male gender, donor age over 60 years and incompatible ABO group matching. A prognostic model constructed for patients over 50 years of age foreseeably indicated that presence of multiple risk factors (ABO incompatibility, male recipient, use of partial graft and donor age > 60 years) had negative impact both on patient and graft survival. In this model, a male patient over 50 years of age receiving a graft from a donor older than 60 years for example, would have an estimated 57% risk of death or graft loss at 1-year. Limitations of the ELTR database relate to insufficient information on pre-transplant renal function, encephalopathy grade, brain edema and/or mechanical ventilation, all of which could improve LT prognosis assessment.

 King´s College Hospital presented their experience in 310 patients with ALF over a 10-year period[51]. Four variables associated with 90-day post-LT mortality were observed, namely: recipient age > 45 years, vasopressor requirement, transplantation era and use of high-risk grafts. The latter were defined by the presence of any 2 of the following: donor age > 60 years, liver steatosis, ABO non-identical match and use of non-whole graft. Interestingly, older recipient age presented the strongest link to increased mortality; 90-day survival was only 47%, compared to 80% in the younger cohort. An age-related reduction in physiologic hepatic reserve was proposed to explain the higher mortality rate observed in this group[51].

Based on pre LT evaluation of the factors described, transplant teams should attempt to establish when an outcome might be unacceptable. Recipient and donor factors associated with poor post-LT outcomes are similar in transplants performed electively or secondary to ALF. Particular causes of graft failure or patient death could potentially explain the gap in survival rates in ALF compared to other LT indications, especially during the first 3-mo following surgery. For example, multisystem disorder triggered by ALF, as well as marked activation of systemic inflammatory response can extend into the post-transplant period. In UNOS and ELTR reviews of databases, infection was the most common cause of mortality following LT for ALF (24% and 18%, respectively). Remarkably, in the UNOS data almost 22% of infectious complications were associated to fungal infections. Neurological complications were reported as the second most common cause of death following transplantation (13%). Fortunately, with better intensive care management of these critical patients, intracranial hypertension incidence has fallen dramatically, coinciding with survival improvement observed over time[9,10]. Another important and alarming issue is death or graft failure related to psychosocial problems. According to the ELTR, patients transplanted for paracetamol overdose present ten times higher rates of death or graft failure, resulting from suicide or lack of compliance than patients transplanted for other etiologies[10]. The finding is even more alarming if we consider that in Europe transplantation for paracetamol-induced ALF has increased seven-fold, from 2% (1973-1978) to 14.1% (2004-2008)[10]. Patients with ALF due to paracetamol need very close post transplant monitoring, including improved psychological and social patient care.

***Graft-related issues***

In this challenging scenario, in which patients with ALF quickly deteriorate and organ shortage remains one of the main problems, risk of mortality while on the waiting list should be weighed against risk of complications, or failure resulting from use of an alternative graft. Different LT procedures can be selected depending on donor organ availability including use of: deceased organ donor, living donor liver transplantation (LDLT), auxiliary liver transplant and variable ABO status (Table 4).

**Living donor liver transplantation:** LDLT provides an alternative source of grafts to overcome the problem of organ shortage, accounting for up to 4% of liver transplants in the United States. However, this figure could account for more than 90% of liver transplants in some Asian countries[47,52,53]. The indication in the pediatric ALF population is well established, and in experienced centers patient and graft outcomes are similar to those of conventional cadaver donor transplants[54,55]. ALF is the indication for transplantation in only 1% of patients evaluated for LDLT in the United States[56]. This contrasts sharply with LDLT indication for ALF in Asia, where different groups report rates to be between 6% and 15%[57,58]. Use of living donor grafts in against the clock emergency settings is often complicated by insufficient time for donors to assess their spontaneous willingness to donate, or for transplant teams to evaluate important ethical and medical issues. Pressing donor evaluation raises special concerns regarding possible donor coercion. Potential consequences of expedited donor evaluation could increase donor postoperative complication rates and worsen psychosocial problems. Transplant centers with great expertise in LDLT report living donor complication rates in ALF to be 34%, similar to those of other indications.

 Graft size is a crucial variable in LDLT. Graft to recipient weight ratios (GRWR) < 0.8% are generally associated with poor outcome[59]. The optimal GRWR value would appear to be closer to 1.0%. However, some authors believe smaller grafts can still be used in ALF, given that this is an acute condition and most patients do not have portal hypertension[60]. In countries where wait-list mortality rates are high, or access to deceased donors is limited, LDLT allows better control over surgical procedure timing[61]. Additionally, once donor evaluation is completed, LDLT can be performed at the first sign of patient decompensation, using a good quality graft.

**Auxiliary transplantation:** Auxiliary liver transplantation is an attractive alternative to total transplantation. Partial left or right donor lobe is used, acting as temporary support to replace the damaged recipient liver, while all or part of the native liver remains in situ. The partial graft can be placed below the native liver (heterotopic) or replacing the resected right or left native lobe (auxiliary transplantation). Increased incidence of portal vein thrombosis or primary non-function has been observed with heterotopic transplantation compared to auxiliary partial, or standard LT[62]. Auxiliary transplantation provides temporary support of liver function until spontaneous regeneration and recovery of the native liver occurs, at which time immunosuppressive treatment is withdrawn and the implant atrophies or is removed. The surgical procedure is challenging because it requires partial native liver resection in a critically ill patient and complex vascular reconstruction. Initial reports of auxiliary LT showed relatively high anastomotic complications and retransplantation rates, although recent outcomes have improved substantially[63-65]. In Europe, auxiliary transplantation peaked between 1994-1998 to 4%, but has since fallen to only 1.9% in 2004-2009[10]. Optimal indications for auxiliary transplantation include patient age below 40 years, excellent temporary liver graft and hemodynamic stability. However, it is difficult to predict which patients will present regeneration of the native liver. This appears to be the case in those with hyper-acute presentation and viral or paracetamol etiology, as well as certain histology subtypes (diffuse pattern, map-like necrosis) and timing of hepatectomy[65,66].

**ABO-incompatible graft:** Length of time on the waiting list also influences policies related to use of ABO incompatible grafts. Early results with ABO incompatible LT’s were disappointing because of increased risk of severe cellular and humoral graft rejection, biliary complications and vascular thrombosis. Recent analysis of the ELTR showed the rate of graft loss at 3 mo doubled in ABO mismatched grafts used during emergency transplantation[10]. Different strategies have been implemented to improve results with ABO-incompatible livers and other grafts. Toso *et al*[67] reported acceptable graft and patient survival in 14 patients, using a quadruple immunosuppressive regimen without splenectomy; 64% and 56% of ABO incompatible grafts remained functional after 1- and 5-years, respectively. Recent approaches have been described with promising results. Paul Brousse Hospital reported three patients treated with antigen-specific immunoadsorption and a quadruple immunosuppressive regimen combined or not to anti-CD20 humanized monoclonal antibodies (rituximab)[68]. Keio University proposed a complex but successful protocol for ABO incompatible LDLTs that included: multiple perioperative plasmaphereses together with rituximab, splenectomy and triple systemic immunosuppression. In addition, portal vein infusion therapy was administered after transplant with methylprednisolone, prostaglandin E1 and gabexate mesylate[69]. Thirteen adult patients underwent ABO incompatible LDLTs under this protocol, for which authors reported a 3-year survival of 76%, almost identical to that of ABO compatible cases[69]. Regardless of these promising results, close monitoring of patient’s immune status and adjustment of immunosuppression need to be implemented, as infection remains the major cause of morbidity and mortality. The protocols described should be viewed as important treatment options in selected adult patients with ALF. However, they are very complex and require maximum expertise. Controversy remains as to whether ABO-compatible or incompatible LT can really present similar post-transplant outcomes.

**CONCLUSION**

Despite progressive and constant improvement in ALF survival after LT, high mortality and graft loss rates persist, especially within the first 3-months post-transplant. Current prognostic models are helpful in identifying individuals who will need LT; nonetheless, fine-tuning of these scores are needed to improve identification in patients who would benefit from transplantation. Newer technologies are being developed and enhanced to improve survival. Different extracorporeal support devices have been advocated to supplant liver function in patients with ALF, either to improve native liver regeneration, or to stabilize patients before transplantation. However, conclusive evidence has not been reported[70,71]. Mesenchymal stem cell infusion also appears promising, but several problems remain in relation to use of this therapy, including conflicting data on potential risk of malignant transformation, as well as degree of liver engraftment and their long term efficacy[72].

 With increasing knowledge on encephalopathy pathogenesis, hepatic regeneration and mechanisms of liver cell injury, outcomes should continue to improve. Early referral to a transplant center and prompt treatment of patients with worsening liver failure remain however, the backbone behind outcome improvement.

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|  |
| --- |
| **Table 1 Comparison between different prognostic scoring systems for acute liver failure** |
|  | ***n*** | **Etiologies** | **Parameters** | **Comments** |
| **Laboratory** | **Clinical** |
| Bernuau  *et al*[8] (1986) | 115 | Hepatitis B | Factor V levels | Age | Clichy criteria |
| O´Grady  *et al*[11] (1989) | 332 | Non-paracetamol | Bilirubin, INR  | Age, etiology, jaundice to encephalopathy >7 | First model to differentiate between paracetamol-induced and other etiologies |
| 431 | Paracetamol | Arterial pH, Creatinine, INR, grade 3-4 encephalopathy |  |  |
| Bismuth  *et al*[63] (1996) | 139 | All patients | Factor V levels | Age, grade 3-4 encephalopathy |  |
| Mitchell  *et al*[30] (1998) | 102 | Paracetamol | APACHE II |  | APACHE II score > 15: sensitivity 82%, specificity 98%. Similar to KCC |
| Schmidt and Dalhoff[37] (2002) | 125 | Paracetamol | Serum phosphate > 1.2 mmol/L |  | Applicable from day 2-4 after overdose. Sensitivity 89%, specificity 100%. Superior to KCC |
| Bernal  *et al*[24] (2002) | 210 | Paracetamol | Lactate |  | Addition of post resuscitation lactate to KCC improved sensitivity |
| Larson *et al*[46] (2005) | 275 | Paracetamol | APACHE II |  | APACHE II score > 20: sensitivity 68%, specificity 87%. Superior to KCC |
| Ganzert  *et al*[44] (2005) | 198 | Amanita Phalloides | Prothrombin time < 25%, Creatinine > 1.2 mg/dL  |  | Applicable from day 3 after ingestionSensitivity 100%, specificity 98% |
| Schmidt and Dalhoff[36] (2005) | 239 | Paracetamol | α -fetoprotein |  | Dynamic α-fetoprotein measurement |
| Schiødt  *et al*[41] (2005) | 252 | All patients | Actin-free Gc-globulin |  | Cutoff level 40 mL/L similar prognostic information as KCC in a single measurement admission |
| Taylor  *et al*[45] | 29 | Hepatitis A | ALT ≤ 2600 IU/L, Creatinine ≥ 2.0 mg/dL | Intubation, vasopressors requirement | Superior to MELD score and KCC |
| Schiødt  *et al*[35] | 206 | All patients | α-fetoprotein ratio day 1 and 3 |  | Ratio ≥ 1 indicated better prognosis |
| Antoniades  *et al*[40] | 70 | Paracetamol | Monocyte HLA-DR ≤ 15% |  |  |
| Yantorno  *et al*[13]  | 64 | Non-paracetamol | MELD score |  | MELD superior to KCC and Clichy criteria |
| Dhiman  *et al*[32]  | 144 | Acute viral hepatitis | Creatinine ≥ 1.5 mg/dL, prothrombin time ≥ 35 s | Age ≥ 50, jaundice to encephalopathy >7, cerebral edema, grade 3-4 encephalopathy | Presence of any of 3 variables superior to KCC and MELD score |
| Schimdt and Larsen  *et al* [31] (2007) | 460 | Paracetamol | Serial MELD score |  | MELD score did not provide more information than KCC or INR alone |
| Escudié  *et al*[43] | 27 | Amanita phalloides | INR > 6 at day 4 | Ingestion diarrhea interval < 8 h | Encephalopathy not needed to decide transplantation |
| Volkmann  *et al*[38]   | 70 | All patients | Caspase activation (Measured by Cytokeratin 18 fragments, M30 and M65) |  | Caspase activity might predict spontaneous recovery |
| Mochida  *et al*[15]   | 698 | All patients | Prothrombin time < 10%, Bilirrubin ≥ 18 mg/dL | Age ≥ 45, jaundice to encephalopathy ≥ 11 d | Reevaluates within 5 d if patient remains alive and liver transplantation was not performed. |
| Hadem  *et al*[14] (2008) | 102 | All patients | Bilirubin, lactate | Etiology | BiLE score, better prognostic accuracy than MELD score or KCC. |
| Bechmann  *et al*[33]   | 68 | All patients | Cytokeratin 18 (M65), Creatinine, INR |  | MELD-M65 score |
| Westbrook  *et al* [42]  | 54 | Pregnancy-related | Lactate ≥ 2.8 mg/dL | Encephalopathy | Sensitivity 90%, specificity 86%. Superior to KCC |
| Cholongitas  *et al*[28]  | 125 | Paracetamol | SOFA scoreAPACHE II scoreKK, MELD |  | SOFA score was superior to KCC, MELD and APACHE II |
| Rutherford  *et al*[34]  | 500 | All patients | INR, bilirubin, phosphorus ≥ 3.7 mg/dL, log10 M30 | Encephalopathy grade | ALFSG index sensitivity 86% and specificity 65%; superior to MELD score and KCC. |
| Mendizabal  *et al*[6]  | 154 | Non-paracetamol | MELD score |  | MELD superior to KCC and Clichy criteria |

 ALFSG: Acute liver failure study group, ALT: Alanine aminotransferase; APACHE: Acute physiology and chronic evaluation; INR: International normalized ratio; KCC: King´s College Criteria; MELD: Model for end-stage liver disease.

**Table 2 Variables that correlate with outcome in patients with acute liver failure**

|  |  |
| --- | --- |
| **Clinical / Demographical** | **Serological** |
| AgeEncephalopathyEtiologyCerebral EdemaJaundice to Encephalopathy IntervalMechanical VentilationVasopressors requirement | BilirubinCreatinineINR / Factor VLactatepHGc globulin | PhosphateKetone body ratioα-fetoproteinCell death markers (M30, M65)Monocyte HLA-DR |
| Functional / PhysiologicalAPACHE IIHepatic artery resistance index changes | MorphologicalHepatocyte necrosisLiver volume  |

**Table 3 Determinants associated with poor outcomes following liver transplantation in patients with acute liver failure**

|  |  |  |
| --- | --- | --- |
| **Demographic** | **Graft-related** | **Clinical** |
| Age > 45 yr | Donor > 60 yr | Vasopressors |
| Male Gender | ABO mismatch | Creatinine |
| Non-viral etiology | Steatosis | BMI ≥ 30 |
|  | Reduced liver |  |

BMI: Body mass index.

**Table 4 One-year survival following liver transplantation depending on the type of graft used**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  | **OLT[3]** | **LDLT[35,39]** | **ABO-incompatible[50]** | **Auxiliary LT[45]** |
| Graft | 73% | 70% | 64% | 53% |
| Patient | 79% | 70%-79% | 69% | 62% |

LDLT: Living-donor liver transplantation; LT: Liver transplantation; OLT: Orthotopic liver transplantation.