Factors Related to Survival of Patients With Type 1 Hepatorenal Syndrome Treated With Renal Replacement Therapy

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Abstract. *Background and objectives: The aim of the study was to analyze the etiology, course of dialysis treatment, outcomes and prognostic factors of survival in patients with hepatorenal syndrome (HRS) who received renal replacement therapy (RRT).*

Materials and methods: The retrospective cross-sectional study included patients with HRS who received RRT at the Hospital of Lithuanian University of Health Sciences Kauno klinikos from 2010 to 2015 and met the International Ascites Club criteria for the diagnosis of HRS proposed in 2007. Variables obtained before the initiation of RRT were evaluated by univariate and multivariate analyses to identify prognostic factors of shorter survival.

Results: A total of 61 patients with type 1 HRS and liver cirrhosis (LC) were enrolled into the study. The main etiology of LC was alcoholic liver disease (45.9%). Precipitating factors for HRS were found in 39 cases (63.9%). The most common precipitating factors were a bacterial infection or spontaneous bacterial peritonitis (52.5%) and large volume paracentesis (> 4 L/day) performed without intravenous albumin replacement (18%). None of the patients survived. The mean survival time following the diagnosis of HRS was 18 ± 2.9 days. Independent risk factors associated with decreased survival time were hepatitis C virus (HCV) infection, PaO_2/FiO_2 ratio ⩽ 164, mean arterial pressure (MAP) ⩽ 70 mm Hg, mechanical ventilation at the initiation of RRT, serum urea level > 22 mmol/L and new model for end-stage liver disease (MELD)-Na score > 26 (P < 0.05).

Conclusions: Almost two-thirds of the HRS cases were associated with precipitating factors. Independent risk factors for shorter survival time in patients with type 1 HRS were HCV infection, low PaO_2/FiO_2 ratio, low MAP value, mechanical ventilation at the initiation of RRT, high serum urea level, and high MELD-Na score. The MELD-Na score could provide a better short-term survival prediction than MELD for patients with type 1 HRS.

Introduction

Hepatorenal syndrome (HRS) is a form of acute kidney injury (AKI) resulting from portal hypertension and renal vasoconstriction in the setting of splanchnic arterial vasodilatation in patients with advanced liver cirrhosis (LC) [1]. AKI is one of the most common and severe complications of decompensated LC, which occurs in one-fifth of patients admitted to the hospital [2]. The incidence of HRS in patients with AKI and LC with ascites is 13–45.8% [3–5]. The most effective option to avoid the development of HRS is early elimination of HRS precipitating risk factors. The only option that guarantees complete remission of liver disease is liver transplantation [6]. Other methods of treatment are used as a "bridge to transplantation". These include medications that trigger splanchnic arterial vasoconstriction, implantation of a transjugular intrahepatic portosystemic shunt and/or renal replacement therapy (RRT).

Indications for RRT in patients with HRS are the same as for those with AKI of other origin. However, those with LC are at a higher risk for developing hemodynamic instability due to bleeding, hypotension or arrhythmia; therefore, hemodialysis may become more complicated. The mortality rate in patients with LC who are treated with RRT is by 2–8% higher compared with those with a normal liver function [7]. Based on recommendations, RRT should be applied for HRS only in cases where liver transplantation or the improvement of the kidney function is expected [8]. There is no evidence that would support the idea that RRT prolongs long-term survival of patients without liver transplantation [9]. It is accepted that continuous renal replacement therapy (CRRT) is superior to intermittent hemodialysis (IHD) in the treatment of hemodynamically unstable patients. However, there are no evidence-based recommendations as to which method of RRT

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is the most effective for patients with HRS [10].

The decision on whether to initiate RRT is also complicated by the fact that it is not always possible to accurately determine the etiology of AKI, differentiating between functional and structural renal damage. In cases where AKI develops due to acute tubular necrosis, RRT should always be applied, expecting improvement or recovery of the kidney function. However, it is often unclear whether RRT is effective in patients with HRS that are not suitable for liver transplantation and which method of RRT is the best for them. Therefore, it is essential to analyze various factors that contribute to the development and prognosis of HRS.

The aim of the study was to analyze etiology, course of dialysis treatment, outcomes and prognostic factors of survival in patients with HRS who received RRT.

Material and Methods

This was a retrospective cross-sectional study approved by the Bioethics Centre of the Lithuanian University of Health Sciences (approval No. BEC-MF-24). The study included patients with HRS who received RRT at the Hospital of Lithuanian University of Health Sciences Kauno klinikos from 2010 to 2015. Demographic data, data on liver disease, clinical, laboratory and instrumental test results, methods of applied treatment and outcomes were collected and analyzed.

LC was confirmed histologically or by laboratory and instrumental tests results. The diagnosis of HRS was based on the updated International Ascites Club criteria proposed in 2007 [5]: cirrhosis with ascites; serum creatinine level greater than 133 μmol/L, not decreased for at least 2 days after discontinuation of diuretics and initiation of treatment with albumin (1 g/kg per day, maximum 100 g); exclusion of parenchymal kidney disease (proteinuria greater than 500 mg/d and microhematuria greater than 50 red blood cells per high power field) and/or urinary tract obstruction (abnormal findings in renal ultrasonography); no shock (may be a bacterial infection) or recent treatment with nephrotoxic drugs or vasodilators. Patients who met all HRS diagnostic criteria, except for administration of albumin, were also included into the study (as it is defined in the earlier HRS criteria [11] – volemic expansion with 1.5 L of isotonic saline solution instead of albumin).

All of the patients were categorized as type 1 HRS, as the serum creatinine level doubled from baseline in less than 2 weeks.

For the prognosis of patients with cirrhosis the Child-Pugh, model for end-stage liver disease (MELD) and MELD-Na scores were calculated. Other scores for mortality prediction based on the degree of organ failure were calculated, which included the sequential organ failure assessment score (SOFA) and the chronic liver failure-sequential organ failure assessment (CLIF-SOFA), i.e., the scale that was developed by the European Association for the Study of Liver (EASL) by modifying the SOFA scale [12]. The glomerular filtration rate (GFR) was calculated based on 2009 Chronic Kidney Disease Epidemiology (CKD-Epi) formula [13]. Prognostic scores and GFR were based on the worst values in a 24-hour period before the initiation of RRT.

Statistical analysis

The statistical analysis was performed using the SPSS 22.0 and MedCalc 16.2.1 statistical packages. The normality of data distribution was checked using the Kolmogorov-Smirnov and Shapiro-Wilk tests. Normally distributed data are presented as means together with a standard deviation (SD) and were compared with the Student *t* test. Qualitative values are expressed as absolute values and percentages in brackets. The difference in RRT total duration depending on the RRT method was calculated using a one-way analysis of variance (ANOVA).

The mean survival time was analyzed by the Kaplan-Meier method, and the outcomes are presented as the mean with a standard error (± SE). The mean survival time was calculated from the date of HRS diagnosis until the patient's death. The univariate survival analysis was performed for the analysis of the influence of factors on patients' survival. The log-rank (Mantel-Cox) test was used to determine statistically significant differences between the groups. In addition, multivariate analysis was performed using the Cox proportional hazards model to identify the independent predictors of shorter survival time. Multivariate analysis included factors that were significant in univariate analysis when P was < 0.1. The Youden index was used to find optimal cut-off points for the analyzed factors (a sum of sensitivity and specificity minus 1). The results were considered statistically significant at $P < 0.05$.

Results

Sixty-one patients fulfilled the inclusion criteria. Demographic data, prognostic scores, liver and renal function tests are summarized in Table 1. The mean duration from the diagnosis of LC to the development of HRS was 1.9 (3.1) years. Almost two-thirds of HRS cases were associated with precipitating factors, which included an inpatient bacterial infection or spontaneous bacterial peritonitis in 32 cases (52.5%), higher than 4 liters paracenteses without the administration of albumin in 11 cases (18%), excessive alcohol intake 1 month before hospitalization in 9 cases (14.18%), gastrointestinal bleeding in 5 cases (8.2%), and any surgical intervention less than 1 month before the diagnosis of HRS in one case.

RRT was initiated 7.7 days after the diagnosis of HRS. Regarding the modality of RRT, 45 patients

Table 1. Demographic data, prognostic scores, liver and renal function tests of 61 patients with hepatorenal syndrome

Variable	n (%) or mean ± SD
Age (year)	53.1 (12.7)
Sex	
Male	30 (49.2%)
Female	31 (50.8%)
Etiology of liver cirrhosis	
Alcoholic hepatitis	28 (45.9%)
VHC	16 (26.2%)
VHB	2 (0.03%)
Primary biliary cholangitis	1 (0.02%)
Esophageal varices	
Absent	51 (47.9%)
F1	20 (32.1%)
F2	9 (14.18 %)
F3	11 (18%)
Hepatocellular carcinoma	6 (9.8%)
Portal vein thrombosis	6 (9.8%)
Spontaneous bacterial peritonitis	23 (37.7%)
MELD score	26.8 (5.9)
Child-Pugh score	12.4 (5.3)
MELD-Na score	29.4 (5.3)
SOFA score	12.7 (3.1)
CLIF-SOFA score	17.1 (3.9)
GFR (mL/min/1.73 m^2)	13.1 (9.9)
Hemoglobin (g/L)	95.7 (21)
White blood cells ($\times 10^9$/L)	15.9 (8.4)
Platelets ($\times 10^9$/L)	141.8 (97.3)
Serum sodium (mmol/L)	129.5 (5.9)
Potassium (mmol/L)	4.9 (1.1)
Blood urea nitrogen (mmol/L)	27.8 (12.7)
Serum creatinine (μmol/L)	455.2 (205.5)
Total serum bilirubin (μmol/L)	231.2 (201.5)
Serum albumin (g/L)	24.6 (5.5)
INR	2.02 (0.7)

VHC, viral hepatitis C; VHB, viral hepatitis B; MELD, model for end-stage liver disease; SOFA, sequential organ failure assessment score; CLIF-SOFA, chronic liver failure-sequential organ failure assessment; GFR, glomerular filtration rate; INR, international normalized ratio.

(73.8%) received IHD, 9 patients (14.8%) received CRRT and 7 patients (11.5%) received both IHD and CRRT. The average number of RRT procedures performed for each patient was 5.4 (7), while the total mean duration of RRT for one patient was 27.4 (52.5) hours.

Twenty-seven patients (44.3%) required vasopressor support, and 27 patients were mechanically ventilated at the initiation of RRT. Fifty-three patients (86.9%) were oligoanuric and 28 (45.9%) had radiologically confirmed pulmonary edema. Overall, forty-one patients (67.2%) developed shock before or after the initiation of RRT.

None of the patients survived. The mean survival time following the diagnosis of HRS was 18 days. The main causes of death included septic shock in 21 cases (34.4%) and progressive liver failure in 17 cases (27.8%). Less common causes were progressive respiratory failure, uncontrolled gastrointestinal bleeding, and intracerebral hemorrhage.

In the univariate analysis, it was established that the mean survival time was statistically significantly shorter in cases when albumin was not administered, when CRRT was applied, and when on the first day of RRT the patient was mechanically ventilated or on vasopressors (Table 2).

The multivariate analysis was performed with all factors that were used in the univariate analysis (Table 3). Independent risk factors for shorter survival time in patients with type 1 HRS included viral hepatitis C (VHC) infection, $PaO_2/FiO_2 \leqslant 164$, mean arterial pressure (MAP) $\leqslant$ 70 mm Hg, mechanical ventilation at the initiation of RRT, serum urea level > 22 mmol/L, and MELD-Na score > 26.

Discussion

In critically ill patients, RRT can be applied as long as the function of kidneys recovers or a patient dies due to underlying disease or its complications. In this case, J. M. Belcher suggests that hemodialysis can be considered as one of the life support methods, and the decision to initiate RRT should be based not only on laboratory values but also on the individual clinical case [14]. RRT is highly recommended for patients with type 1 HRS who are likely to undergo liver transplantation; however, the benefit to other patients remains unknown [15, 16]. In our study, none of the patients underwent liver transplantation, and all of them were diagnosed with type 1 HRS (which has a significantly worse prognosis). HRS develops in patients with advanced liver disease, yet it is not always possible to distinguish the exact cause of AKI in patients with LC. Therefore, there are more studies focusing on AKI in patients with LC than on HRS itself [4, 9, 17, 18]. However, the prognosis of AKI in patients with LC is poor, irrespective of the cause of AKI. For example, in a prospective study, no statistically significant difference in survival of the patients who had AKI caused by HRS or acute tubular necrosis was observed [18].

There is a lack of studies regarding RRT in HRS patients who are not candidates for liver transplantation [16, 19, 20]. There is one similar study that was conducted with 30 patients with Child-Pugh C LC that developed HRS. All the patients were treated with RRT. Only 26.7% of them survived 30 days from the diagnosis of HRS with a median survival time of 21 days [16]. In our study, almost all (98.4%) patients were Child-Pugh C. In our cohort, the 30-day survival was also low (16.4%) and the mean survival time from the diagnosis of HRS was 18 days.

More than 70% of cases of type 1 HRS are associated with precipitating factors [21]. The results in our cohort were similar to those observed in the

Table 2. Univariate analysis of predictive parameters for shorter survival in patients with cirrhosis and type 1 HRS

Variable		Mean survival time	
		Days ± SE	*P* value
1	2	3	4
Factors related to the patient and his/her condition			
Age (year)	⩽ 42 > 42	27 ± 7.9 16.1 ± 2.9	0.139
Sex	Male Female	21.2 ± 5.2 15.2 ± 3	0.303
Shock	None Septic Hypovolemic	25.8 ± 7.6 13.4 ± 2.7 17.2 ± 4.8	0.195
Vasopressor at the initiation of RRT	Yes No	11.7 ± 2.5 22.8 ± 4.6	**0.030**
Mechanical ventilation at the initiation of RRT	Yes No	11.6 ± 2.8 23.4 ± 4.7	0.017
Diuresis status	Anuria (< 100 mL) Oligoanuria (100–500 mL) Neoliguria (> 500 mL)	15.4 ± 3.3 17.3 ± 5.2 27 ± 10.5	0.426
Factors related to liver disease			
Etiology of ESLD – VHC	Yes No	7.3 ± 1 21.5 ± 3.7	**0.006**
Etiology of ESLD – VHB	Yes No	17 ± 6.1 18 ± 3	0.751
Etiology of ESLD – alcoholic	Yes No	18.8 ± 5.4 17.7 ± 3.1	0.838
Etiology of ESLD – cryptogenic	Yes No	23.1 ± 4.7 16 ± 3.8	0.170
SBP	Yes No	18.8 ± 4.2 16.8 ± 3.6	0.502
Portal vein thrombosis	Yes No	14.3 ± 6.8 18.4 ± 3.2	0.526
TIPS	Yes No	23 ± 4.6 17.6 ± 3.1	0.282
Factors related to treatment of hepatorenal syndrome			
Modality of RRT	IHD CCRT Both IHD and CCRT	20 ± 3.7 4.4 ± 1.1 23 ± 7.4	< **0.001**
Albumin admission	Yes No	21.6 ± 3.9 12.6 ± 3.4	**0.047**
Laboratory and hemodynamic variable values at the initiation of RRT			
Hemoglobin (g/L)	⩽ 110 > 110	8.3 ± 2.6 20.5 ± 3.5	0.019
White blood cells ($\times 10^9$/L)	⩽ 9 > 9	20 ± 6.5 16.3 ± 2.8	0.710
Thrombocytes ($\times 10^9$/L)	⩽ 100 > 100	15 ± 2.8 22.2 ± 5.8	0.298
Serum sodium (mmol/L)	⩽ 126 > 126	12.4 ± 3.4 20.7 ± 4	0.173
Serum potassium (mmol/L)	⩽ 4.8 > 4.8	13.2 ± 2.7 23.2 ± 5.1	0.107
Serum creatinine (μmol/L)	⩽ 400 > 400	21.6 ± 4.7 13.5 ± 3.1	0.106
Glomerular filtration rate (mL/min/1.73 m^2)	⩽ 15 > 15	11.5 ± 3.1 20.7 ± 3.9	0.120
Blood urea nitrogen (mmol/L)	⩽ 22 > 22	22 ± 4.2 11.1 ± 3.1	**0.015**
Total serum bilirubin (μmol/L)	⩽ 100 > 100	17.2 ± 5.1 17.6 ± 3.1	0.652
Serum albumin (g/L)	⩽ 30 > 30	17.9 ± 3.2 21.3 ± 7.4	0.560
International normalized ratio	⩽ 1.8 > 1.8	24.1 ± 5.1 12.4 ± 2.6	**0.042**

Table 2 continued

1	2	3	4
Aspartate transaminase	Normal at least 3 times above the normal range	22.2 ± 5 13.3 ± 2.5	0.197
Alanine transaminase	Normal above the normal range	25.7 ± 5.5 11 ± 1.8	**0.012**
Gamma-glutamyltransferase	Normal above the normal range	25.1 ± 8.2 15.2 ± 2.5	0.202
Alkaline phosphatase	Normal above the normal range	16.1 ± 6 17.4 ± 3.1	0.691
Arterial blood pH	⩽ 7.3 > 7.3	11.4 ± 2.9 17.2 ± 4	0.153
Standard base excess (mmol/L)	⩽ –21 > –21	4.6 ± 1.2 15.8 ± 2.8	**0.002**
PaO_2 (mm Hg)	⩽ 99 > 99	10.9 ± 2.5 20.2 ± 5.5	**0.048**
$PaCO_2$ (mm Hg)	⩽ 26 > 26	17.7 ± 3.7 10.2 ± 3	0.051
PaO_2/FiO_2 (mm Hg)	⩽ 164 > 164	9.6 ± 2.7 18.4 ± 4.2	**0.019**
Mean arterial pressure (mm Hg)	⩽ 70 > 70	7.4 ± 1.7 23.8 ± 4.2	**< 0.001**
Central venous pressure (cm H_2O)	⩽ 15 > 15	22.7 ± 5.4 11.8 ± 1.7	0.387
Prognostic scale values at the initiation of RRT			
MELD score	⩽ 23 > 23	23.1 ± 8.2 16.1 ± 2.6	0.595
MELD-Na score	⩽ 26 > 26	20.4 ± 3.6 10.3 ± 4	0.057
Child-Pugh score	⩽ 12 > 12	24.3 ± 5.5 13.4 ± 2.9	0.066
SOFA score	⩽ 12 > 12	21.5 ± 4.7 14.4 ± 3.3	0.193
CLIF-SOFA score	⩽ 16 > 16	21.8 ± 4.7 14 ± 3.3	0.120

ESLD, end-stage liver disease; VHC, viral hepatitis C; VHB, viral hepatitis B; SBP, spontaneous bacterial peritonitis; TIPS, transjugular intrahepatic portosystemic shunt; RRT, renal replacement therapy; CRRT, continuous renal replacement therapies; IHD, intermittent haemodialysis; PaO_2, partial pressure of arterial oxygen; $PaCO_2$, partial pressure of carbon dioxide; PaO_2/FiO_2, ratio of partial pressure arterial oxygen and fraction of inspired oxygen; MELD, model for end-stage liver disease; SOFA, sequential organ failure assessment score; CLIF-SOFA, chronic liver failure-sequential organ failure assessment.

Table 3. Independent risk factors for shorter survival time in patients with type 1 HRS

Variable parameter	Hazard ratio	95% confidence interval	*P* value
VHC infection	7.9	2.9–21.1	< 0.001
PaO_2/FiO_2 ⩽ 164	7.3	2.3–23.3	0.001
MAP ⩽ 70 mm Hg	6.1	2.4–15.7	< 0.001
Mechanical ventilation at the initiation of RRT	4.1	1.4–12.3	0.012
Serum urea level > 22 mmol/L	3.1	1.4–6.6	0.005
MELD-Na score > 26	2.4	1–5.6	0.041

VHC, viral hepatitis C; RRT, renal replacement therapy; PaO_2/FiO_2, ratio of partial pressure arterial oxygen and fraction of inspired oxygen; MAP, mean arterial pressure; MELD-Na, model for end-stage liver disease-Na.

literature. Factors that may have precipitated the development of HRS were confirmed in almost 64% of cases. In more than half of the patients, it was a bacterial infection. According to the literature, one-third of patients with LC and ascites that are diagnosed with a bacterial infection during hospitalization develop AKI [22]. In almost 20% of our patients, HRS was associated with paracentesis larger than 4 L without the administration of albumin. It is commonly known that HRS develops in 21% of patients undergoing large volume paracentesis (4–6 L per day) without the administration of albumin [23].

Half of our patients with HRS were treated without the administration of albumin. In the univariate analysis, the survival of these patients was relatively shorter compared with those who received intravenous albumin (12.6 ± 3.4 days vs. 21.6 ± 3.9 days, $P = 0.047$). However, the multivariate analysis did not reveal plasma volume expansion with albumin as an independent prognostic survival factor. Prevention of HRS plays an important role in management of patients with LC, since type 1 HRS is often associated with a precipitating factor and the prognosis is poor.

There were 2 studies with no statistically significant differences in the outcomes of type 1 HRS, irrespective of whether RRT was performed [19, 20]. A study by Zhang et al. has indicated that the only independent risk factor that influences 180-day survival was the etiology of LC: 71.4% of those who survived 180 days had viral hepatitis, 23.8% had alcoholic LC, and the rest (4.8%) had LC of other etiology [20]. Another study also suggested significant differences based on the etiology of LC: the lowest survival rate was found in patients with alcoholic liver disease (8 days) [15]. The results of our study also confirmed the presumption that the etiology of LC had an impact on the survival of HRS patients. The multivariate analysis revealed that VHC LC was an independent risk factor of shorter survival. The mean survival of patients who were diagnosed with VHC was 7.3 ± 1 days, while those who had LC of other etiologies survived 21.5 ± 3.7 days ($P = 0.006$). Etiologies such as viral hepatitis B (VHB), alcoholic and cryptogenic LC did not influence the patients' survival. In the above-mentioned studies, viral LC was considered as one category, while in our study VHB and VHC were distinguished into 2 groups (therefore, the results cannot be compared). Although a variety of studies confirm the relation between the etiology of LC and survival of patients with HRS, the exact cause that contributes to the worst prognosis still remains controversial.

In our cohort, independent factors related with shorter survival of patients were MAP $\leqslant$ 70 mm Hg ($P < 0.001$), PaO_2 / FiO_2 $\leqslant$ 164 ($P = 0.001$) and mechanical ventilation on the first day of RRT ($P = 0.012$). Other studies that investigated the treatment of HRS with RRT show similar results [16, 19]. For example, Witzke et al. found that none of the patients who were treated with CRRT and mechanical ventilation survived 30 days [16]. Consequently, this leads to the conclusion that the shorter survival of HRS patients treated with RRT is related to their critical condition. The results of our study also support this assumption: the patients who were treated with IHD survived longer than those who underwent CRRT (20 ± 3.7 vs. 4.4 ± 1.1 days, $P < 0.001$). These differences were observed because hemodynamically unstable patients were treated with CRRT. Similar results were found in the above-mentioned study where on the first day of RRT the condition of patients treated with CRRT was worse (i.e., the Acute Physiology and Chronic Health Evaluation (APACHE II) and MELD scores were higher) [16]. For this reason, the influence of RRT modality on the survival of patients cannot be estimated. However, the findings of our study contribute to the above-mentioned presupposition that RRT does not prolong the survival of hemodynamically unstable critically ill patients [14, 16, 19].

The survival rate of the patients who had higher prognostic scores (MELD, MELD-Na, Child-Pugh, SOFA, and CLIF-SOFA) was lower, but there was no statistically significant difference based on an established cut-off value ($P > 0.05$). There are several studies that confirm the prognostic importance of the MELD score for life expectancy in patients with HRS [4, 18, 22, 24–26]. However, it remains unknown whether these results can be applied to the population of patients with type 1 HRS treated by RRT, as type 1 HRS is a specific form of AKI with a very poor prognosis. The reason for this is that in the former studies AKI was not always caused by HRS. Alessandria et al. determined that the MELD score was an independent factor that contributed to the shorter survival of patients [24]. The authors indicated that the prognosis of patients with type 1 HRS was poor and was not associated with the MELD score; while on the contrary, the survival of patients with type 2 HRS was associated with the MELD score.

The novelty of our study is the analysis of the prognostic value of the MELD-Na score for patients diagnosed with type 1 HRS. The MELD-Na score is calculated based on serum sodium concentration, and since 2016 it has been recommended by the American Organ Procurement and Transplantation Network. According to the univariate analysis, of all prognostic scales the MELD-Na score was closest to the selected reliability level (patients with MELD-Na $\leqslant$ 26 survived 20.4 ± 3.6 days, while those with $>$ 26 scores survived 10.3 ± 4 days, $P = 0.057$). The multivariate analysis also revealed that, of all the prognostic scales, the MELD-Na score $>$ 26 was an independent risk factor that contributed to shorter life expectancy of patients.

Conclusions

Almost two-thirds of HRS cases were associated with precipitating factors such as a bacterial infection or large volume paracenteses without the administration of albumin. Independent risk factors for shorter survival time in patients with type 1 HRS were HCV infection, low PaO_2/FiO_2 ratio, low MAP value, mechanical ventilation at the initiation of RRT, high serum urea level, and high MELD-Na

score. Therefore, in critically ill patients with type 1 HRS, RRT does not provide an improved long-term survival benefit. Moreover, the etiology of LC might influence the survival of type 1 HRS patients with the worst survival encountered in VHC patients. The MELD-Na score could provide a better short-term survival prediction than the MELD for patients with type 1 HRS.

Conflict of Interests

None of the authors has any conflicts of interest to declare.

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