Original Article

Prevalence and Clinical Course of Acute Kidney Injury in Hospitalized Cirrhotic Patients with Spontaneous Bacterial Peritonitis

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Background: Spontaneous bacterial peritonitis (SBP) is a complication of cirrhosis and often followed by liver decompensation. Acute kidney injury (AKI) frequently occurs and is the reason for the increased mortality rate in cirrhotic patients.

Objective: To evaluate the incidence rate of AKI and the prediction of one-month mortality in hospitalized SBP- cirrhotic patients.

Materials and Methods: One hundred and twenty-three cirrhotic patients with SBP were included in a retrospective cohort study. Renal injury was defined by AKIN criteria. The AKI patients were assessed for severity, reversibility, hospital complications, length of hospital stay and mortality. Mean length of time of follow-up for all patients was 30 days. Kaplan-Meier survival for 30 days mortality by Cox regression model was calculated accordingly to the renal injury.

Results: The mean age of patients was 57.8±12.2; 62 (50.4%) were male. Most of them (78.7%) were Child-Pugh C cirrhosis. More than half of the patients (52%) were alcoholic cirrhosis with mean MELD score of 20.6±5.8. Mean length of hospitalization was 15±8.4 days. AKI occurred in 53.7% of the patients (83.3%, 7.6% and 9.1% for AKIN criteria 1, 2 and 3 respectively). The AKI group experienced non-liver related complications, septic shock and death in hospitalization proportionately greater than the other [(7.6% vs. 0%, p = 0.034); (33.3% vs. 14%, p = 0.013); (34.8% vs. 17.5%, p = 0.031), respectively]. Among three AKI sub-groups, there were no significant differences regarding MELD score (p = 0.16), episode of albumin infusion (p = 0.31), reversibility of kidney functions (p = 0.88) or intrahospital 30 days mortality (p = 0.56). The Kaplan-Meier survival showed a significant increase in 30 days mortality of AKI patients as compared to the others. (33.3% vs. 15.7%, p = 0.022). Multivariate cox regression analysis indicated that an AKI episode can predict 30 days mortality to be 2.42 times higher than non-AKI patients after adjusted mean Child-Pugh score. (adjusted HR 2.42, 95% CI 1.11 to 5.25).

Conclusion: AKI is a condition which can predict increased 30 days mortality and is associated with non-liver related complications in decompensated SBP-cirrhotic patients.

Keywords: Spontaneous bacterial peritonitis, Acute kidney injury, Cirrhosis

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Hospitalized cirrhotic patients comprise at least 10% of hospitalized patients and have high mortality due to baseline degree of liver disease or precipitated insults. Spontaneous bacterial peritonitis (SBP) is the most common complication of cirrhotic patients with ascites. After an episode of SBP, worsening of hyperdynamic circulation causes liver decompensation and renal injury, which promotes in-hospital mortality of up to 30% despite resolution of the infection[1,2]. Acute kidney injury (AKI) is common in patients with cirrhosis with ascites due to ineffective circulatory volume. The diagnosis of AKI were made according to the International Club of Ascites (ICA-AKI) new definitions for the diagnosis and management of AKI in patients with cirrhosis which verified renal dysfunction into grades of increasing severity recorded by changes in serum creatinine[3-6]. The aim of our retrospective cohort research was to study the incidence rate of AKI in hospitalized SBP-cirrhotic patients, as well as this group's clinical course of hospitalization, complications, length of hospital stay, AKI reversibility and the prediction of one-month mortality.

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Materials and Methods

Patients

Our retrospective cohort study (COA: 152/2561) enrolled patients aged 18 to 80 years with cirrhosis and SBP who were admitted to Vajira Hospital from January 2012 to December 2018. SBP was diagnosed by ascitic fluid analysis with more than 250 polymorphonuclear cells (PMNs)/mm^3 and the absence of clinical features or laboratory suggestive of secondary bacterial peritonitis. HCC patients

with expected survival of less than 90 days and those HIV positive were excluded. As the urinary output cannot be measured accurately in non-ICU patients, only the definition of change in serum creatinine was considered.

Acute kidney injury in cirrhosis is defined as an acute increase in serum creatinine of ≥0.3 mg/dL within 48 hours or by ≥50% from baseline serum creatinine (SCr). Baseline creatinine was defined as medical records within 3 months prior to admission. If no previous SCr from records existed, admission SCr were used as baseline SCr. Degree of renal injury was evaluated by International Club of Ascites (ICA-AKI) new definitions for the diagnosis and management of AKI in patients with cirrhosis into grades 1, 2 or 3. AKI stage 1 is defined as an acute increase in SCr by ≥0.3 mg/dL within 48 hours or an increase 1.5 to 1.9 times from baseline. AKI stage 2 was defined as increase in SCr 2.0 to 2.9 times from baseline and AKI stage 3 was defined as increase in SCr ≥3 times from baseline or SCr ≥4 mg/dL with an acute increase of at least 0.3 mg/dL or initiation of renal replacement therapy[4]. Patients were classified accordingly to their peak of AKIN stage during hospitalization. AKI reversibility was recorded as three subgroups. The stable AKI was classified if there was an initial impairment in SCr but stabilized during hospitalization, AKI progression if there was an increase in at least 1 AKIN stage or progressive with renal replacement therapy and AKI regression if there was a decrease in at least 1 AKIN stage, decrease in SCr to a value within 0.3 mg/dL above baseline value or returned to baseline values.

Patients were given a physical examination and laboratory work up (complete blood count, liver and renal tests, blood and ascitic fluid cultures, chest x-ray) before starting the treatment. Child Pugh and MELD score were evaluated at baseline. Renal function was monitored daily. When diagnosed with SBP, all patients were treated with intravenous antibiotic for up to 7 days. In cases without a diagnosis of AKI, albumin infusion with antibiotics were used to treat high risk SBP patients on the first and third days to prevent AKI. The antibiotic protocol was deescalated according to the ascitic fluid culture result.

We studied hospital course by monitoring both liver complications such as hepatic encephalopathy and/or variceal bleeding and non-liver complications such as sepsis or pneumonia. Large volume paracentesis was not performed during the treatment to prevent further hemodynamic dysfunction and because it may deteriorate renal function. In cases with a diagnosis of AKI, urine analysis was done to exclude prerenal kidney injury and intravenous albumin infusion was given to patients (1.5 g/kg body weight on day 1 and 1 g/kg on day 3) and serum creatinine was monitored. If diagnosed with type 1 HRS, they were treated with intravenous terlipressin and albumin. The patients were followed-up with hospital courses of 30 days[3-6].

Statistical analysis

Continuous variables data with symmetrical distribution are presented as means and standard deviation (SD). The continuous variables with asymmetric distribution are represented as median and range (interquartile interval, percentile 25 and 75). Percentages were used for the categorical variables. For Group comparisons, categorical variables were analyzed by Chi-squared test and Fisher's exact test and continuous variables were analyzed by Student's t-test and the Wilcoxon-Mann-Whitney test. Survival function was calculated by Kaplan-Meier analysis. Cox regression model, the hazard ratio of mortality was evaluated by adjusted by Child-Pugh scores. A *p*-value of <0.05 was determined to be significant.

Results

A total of one hundred and twenty-three SBP episodes in cirrhotic patients were included in the present study. More than half (59.3%) of patients were nosocomial-SBP. Baseline demographic, clinical and laboratory data are shown in Table 1. The mean age was 57.8 years, male gender (50.4%). Etiology of alcoholic cirrhosis and chronic hepatitis B contributed 52% and 26% respectively. Most of the patients (78.7%) were in status of Child-Pugh C with mean MELD score of 20.6. Mean of baseline GFR, baseline SCr, admission SCr, 48 hours after admission SCr were 55.5 ml/min/1.73 m^2, 1.11, 1.53 and 1.57 mg/dl. One-third (33.3%) of the patients were currently using a beta blocker (Table 1).

Hemoculture and ascites cultures were collected in almost all of the patients. Half of them (50.4%) were blood and/or ascites culture positive. Monomicrobial organisms were found from ascites culture in 37 (30.8%) patients (Table 1). Among blood or ascites culture episodes, nearly half (46.9%) of bacteria isolates were gram negative enterobacteriaceae (*Escherichia coli* and *Klebsiella pneumoniae*).

AKI was diagnosed in more than half of the patients (n = 66; 53.7%), of whom 55 (83.3%), 5 (7.6%) and 6 (9.1%) had AKIN grade 1, 2 and 3, respectively. Baseline BUN, SCr and mean MELD score were significantly higher in the AKI group as compared to the other (33.2 vs. 16.4, $p<0.001$); (1.2 vs. 1.0, $p = 0.017$); (22.2 vs. 18.9, $p = 0.002$) (Table 2). There was no difference in Child Pugh score between groups. The percentage of positive ascites culture AKI patients was no different from the other group (33.3% vs. 28.1% $p = 0.53$) (Table 2).

The mean length of hospitalization of was 15 days. We monitored liver-related complications (hepatic encephalopathy, UGI bleeding) and non-liver related complications (cardiovascular events, mechanical ventilation). Half of the AKI patients (51.5%) developed complications. While there was no difference in liver- related complications, AKI patients had non-liver related complications and septic shock significantly more than non-AKI patients (7.6% vs. 0, $p = 0.03$); (33.3% vs. 14%, $p = 0.013$). During hospitalization, 33 (26.8%) patients did not survive (Table 2). Intrahospital 30 day-mortality rate among the 66 episodes with AKI was 34.8% (23 deaths) as compared with 17.5% (10 deaths) in the 57 episodes without AKI ($p<0.03$).

After diagnosis of AKI, we treated patients by

Table 1. Baseline demographic, laboratory and clinical data of 123 cirrhotic patients with spontaneous bacterial peritonitis

Characteristics	Results
Age (years)	57.8 (12.2)
Male, n (%)	62 (50.4)
Etiology of cirrhosis, n (%)	
Alcoholic	64 (52)
Chronic hepatitis B	32 (26)
Chronic hepatitis C	25 (20.3)
Non-alcoholic fatty liver disease	4 (3.3)
Cryptogenic	10 (8.1)
Child-Pugh C, n (%)	96 (78.7)
Child-Pugh score	10.9 (1.6)
MELD score	20.7 (5.8)
MELD-sodium	23.6 (6.2)
Current use of beta-blocker, n (%)	41 (33.3)
Spontaneous bacterial peritonitis, n (%)	
Community-acquired	50 (40.7)
Hospital-acquired or healthcare-associated	73 (59.3)
Monomicrobial ascitic culture positive	37 (30.8)
Mean arterial pressure (mmHg)	87.7 (16.4)
Serum sodium (mmol/L)	132.2 (4.3)
Serum albumin (g/dL)	2.1 (0.4)
Creatinine at baseline (mg/dL)	1.1 (0.5)
Creatinine at admission (mg/dL)	1.5 (1.1)
Creatinine 48 hours after admission (mg/dL)	1.6 (1.4)
GFR (mL/min/1.73 m^2)	55.5 (30.6)
BUN (mg/dL)	25.4 (20.0)
Acute kidney injury, n (%)	66 (53.7)
Stage 1	55 (83.3)
Stage 2	5 (7.6)
Stage 3	6 (9.1)
Length of hospitalization (days)	15 (8)

Data are expressed as mean (standard deviation) unless specified. AKI = acute kidney injury; BUN = blood urea nitrogen; GFR = glomerular infiltration rate; MELD = model of end stage liver disease

following with diagnosis algorithm of AKI. Albumin infusion was used in 56 (84.8%) AKI episodes. There was no significant difference in mean MELD score of the 66 SBP cirrhotic patients irrespective of AKI stage (p = 0.16). Similarly, there was no significant difference in the frequency of albumin infusion of the 56 patients (p = 0.30). During the follow-up hospital course, AKI episodes were reversible in 48 patients (72.7%), stable in 4 patients (6.1%), and progressive in 14 patients (21.2 %). One of the 14 progressive AKI patients required renal substitution therapy. There was no difference in the opportunity of AKI reversibility or progression in all 3 AKI stage subgroups. Patients with stage 3 AKI had significantly longer hospital stays than those with stage 2 or stage 1 AKI (25, 20, 15 days, p = 0.024) (Table 3). Regardless of AKI reversibility, intrahospital short term mortality was not different among the AKI subgroups (p = 0.56) (Table 3).

Regression analysis factors which impacted 30-day mortality in patients is shown in Table 4. Factors associated with death were determined using Cox proportional hazards models. In the univariate regression analysis, variables that significantly associated with increased death events were AKI (HR 2.4; 95% CI 1.1 to 5.21, p = 0.027). We included only variables which had no multicollinearity problems in multivariate analysis. After multivariate analysis, the hazard ratio of mortality for patients with AKI adjusted by Child-Pugh score was 2.42 (1.1 to 5.25, p = 0.026). During 30 days of follow-up, a statistically significant greater number of non-AKI patients survived as compared to AKI patients (84.2% vs. 66.6%, p = 0.022) (Figure 1).

Discussion

Advanced cirrhosis includes a condition of vasodilatation, a decrease in effective circulatory volume and reduction in renal perfusion. SBP was a common complication and leading cause in hospital admission of cirrhotic ascites. After an episode of SBP, both systemic and splanchnic vasodilatation were more progressive and patients were vulnerable to renal injury. At the time of SBP diagnosis, our patients were in advanced-stage liver disease with a mean MELD score of 20 corresponding with Khan et al, who reported that more than 70% of SBP patients had a baseline MELD score of ≥20[7,8]. The prevalence of AKI as defined by AKIN criteria increases the diagnosis of AKI episodes. Thus, AKI episodes were recorded in over half of our SBP patients (53%) as compared with an estimated prevalence of approximately 20 to 50% among hospitalized cirrhotic patients in previous studies[2-6].

Previously reported non-selective beta-blockade in cirrhotic ascites significantly increased, by three times, the risk of AKI in liver transplant candidates[9]. Due to a narrow therapeutic windows of beta-blocker therapy in liver decompensation, it may precipitate worsening of ascites and increase an occurrence of kidney injury. We followed clinical practice guidelines in selected cases for beta-blocker therapy with an optimal dosage. One-third (1/3) of the patients used beta-blocker with an average dose of 80 mg/day. The frequency and dose of beta-blocker use in our clinical practice was not different regardless of AKI episode. We believed that the use of a proper dosage did not cause a delay in intestinal transit time and did not associate with SBP episode nor AKI occurrence in our study.

As we know, the cause of two-thirds (2/3) of AKI in cirrhosis is prerenal. Most of AKI in our patients had reversibility (72%) because most of them had a good response to plasma volume expansion and suspected volume-

Table 2. Characteristics of 123 cirrhotic patients with spontaneous bacterial peritonitis according to AKI status

Variables	AKI (n = 66)	Non-AKI (n = 57)	*p*-value
Length of hospitalization (days)	16 (9)	14 (7)	0.087
Creatinine at baseline (mg/dL)	1.2 (0.5)	1.0 (0.4)	0.017
GFR (mL/min/1.73 m^2)	42.1 (23.4)	71 (31)	<0.001
BUN (mg/dL)	33.2 (22.6)	16.4 (11)	<0.001
Child Pugh score	10.8 (1.7)	10.9 (1.4)	0.700
MELD score	22.2 (6.3)	18.9 (4.7)	0.002
MELD-sodium	25.4 (5.9)	21.6 (5.8)	0.001
Culture positive monomicrobial organism	21 (33.3)	16 (28.1)	0.533
Current use of beta-blocker	26 (39.4)	15 (26.3)	0.125
Transfer to ICU	8 (12.1)	3 (5.3)	0.184
Septic shock	22 (33.3)	8 (14)	0.013
Organ failure			
0	38 (57.6)	41 (71.9)	0.162
1	15 (22.7)	11 (19.3)	
2 or more	13 (19.7)	5 (8.8)	
Complications			
Liver related	6 (9.1)	6 (10.5)	0.789
Non-liver related	5 (7.6)	0	0.034
Death in hospitalization	23 (34.8)	10 (17.5)	0.031

Data are expressed as mean (standard deviation) and number (%).
AKI = acute kidney injury; BUN = blood urea nitrogen; GFR = glomerular filtration rate; ICU = intensive care unit

Table 3. MELD score, clinical course and complication of SBP-cirrhotic patients by stage of acute kidney injury

	AKI stage 1 (n = 55)	AKI stage 2 (n = 5)	AKI stage 3 (n = 6)	*p*-value
MELD score, mean (SD)	21.5 (5.9)	24 (2.4)	26.3 (10.1)	0.166
Episode of albumin infusion, n (%)	45 (81.8)	5 (100)	6 (100)	0.308
Clinical course of AKI, n (%)				
Reversible (n = 48)	40 (72.7)	4 (80)	4 (66.7)	0.885
Stable (n = 4)	2 (3.6)	1 (20)	1 (16.7)	0.177
Progressive (n = 14)	13 (23.6)	0	1 (16.7)	0.446
No comorbid complications, n (%)	28 (50.9)	3 (60)	1 (16.7)	0.243
Length of hospital stay (days), mean (SD)	15 (8)	20 (10)	25 (15)	0.024
30 days mortality	19 (34.5)	1 (20)	1 (16.7)	0.564

responsive prerenal AKI. Due to a high mean baseline admission creatinine (1.53 mg/dL), 80% of our SBP patients received albumin infusion for two reasons. First, patients received albumin infusion plus intravenous antibiotic preventively of AKI occurrence in high risk SBP (serum bilirubin >4 mg/dL and Scr >1 mg/dl)[10]. Secondly, after withdrawal of nephrotoxic drugs, vasodilators, NSAIDs or diuretics, patients received plasma volume expansion with albumin (1 g/kg, maximum 100 g/day for 2 days) for a step before meeting criteria of hepatorenal syndrome. Together these were reasons for the explanation of AKI reversibility. Because of the high probability of rapid reversibility, episodes of AKI did not have an impact on hospital length-of-stay in the present study. Previous reports indicate that more than half of AKI episodes which were reversible and associated with low mortality, shortened length-of-stay[11]. Tsien et al

Table 4. Regression analysis factors of 30 days mortality

	Univariate analysis			Multivariate analysis		
	HR	**95% CI**	***p*-value**	**Adjusted HR***	**95% CI**	***p*-value**
Age	1	0.97 to 1.03	0.863	-	-	-
MELD at admission	1.05	0.99 to 1.11	0.082	-	-	-
Mean Child Pugh Score	1.22	0.97 to 1.53	0.092	1.22	0.98 to 1.53	0.076
Mean arterial pressure	0.98	0.95 to 1.01	0.152	-	-	-
AKI						
No AKI	1	Reference	1	1	Reference	1
AKI	2.4	1.1 to 5.21	0.027	2.42	1.11 to 5.25	0.026

Adjusted by mean Child-Pugh score.
AKI = acute kidney injury; CI = confidence interval; HR = hazard ratio; MELD = model of end stage liver disease

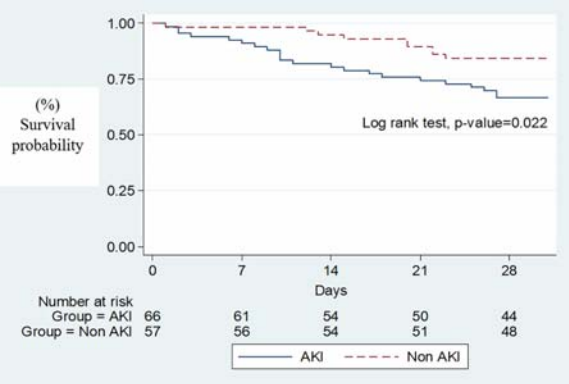

Figure 1. Kaplan-Meier survival curves of spontaneous bacterial peritonitis patients with and without acute kidney injury.

reported that even though most of repeat AKI episodes find reversibility, a gradual increase in serum creatinine were observed during 1 year follow-up and associated with a significant reduction in survival[12].

The authors demonstrated that AKI episodes occurred in all patients who had a baseline mean MELD score greater than 20 and a non-significant higher trend of increased score according to AKI severity. Even though not different in numbers of organ dysfunction, uncontrolled infection, represented as septic shock, was found in 33% of our AKI patients. It has been reported that development of AKI in advanced liver disease is not only related to the MELD score, but the severity of infection and the failure of infection resolution[13]. Regardless of AKI status, we found gram negative isolation which was not multidrug resistance and a good response with broad spectrum antibiotics.

According to severity of kidney injury, each stage can be reversible but the higher stage the greater increase in hospital length-of-stay irrespective of ICU admission. Of 14 progressive AKI patients, only one required renal replacement therapy. However, due to the small number of AKI stage 2 and 3 patients, we cannot draw a conclusion regarding differences in clinical reversibility including complications and mortality according to AKI stage. Apart from the heterogenicity in a proportion of AKI stage in each individual research, previous reported advanced AKIN stages 2 and 3 were distributed to one-third of all AKI stages and independently associated with an increase of hospital acquired complications (hepatic encephalopathy, ICU admission) and mortality in SBP[14].

Other liver-related complications such as hepatic encephalopathy or variceal bleeding occurred in approximately 10% in our patients regardless of AKI event because an SBP episode was a common precipitating insult. We observed only a non-significant increasing trend of AKI patients transferred to intensive care unit and developed multiorgan failure compared to those who did not develop AKI because the number of advanced stages of AKI in our study was too small. Not only episodes of SBP, but most of our patients (75%) were in a stage of liver decompensation and had characteristic of mean BUN >25 mg/dL or mean admission SCr >1.2 mg/dL which is associated with high risk for developing bacterial translocation and other overt infections. An episode of AKI occurrence may imply that these patients develop acute on top chronic liver failure and have potential for occurrence of non-liver related consequences such as sepsis, pneumonia, a cascade of SIR or septic shock which were a reason for higher mortality in the AKI group as supported by the finding that one-third (34.8%) of death events occurred in AKI as compared to only 17.5% from the other. After hospitalization, in-hospital 30 day mortality was one-third (33%) of 66 AKI episodes (22 non-survivors) as compared with 15.7% (9 non-survivors) in the 57 non-AKI, statistically significant according to Kaplan-Meier

survival analysis. Variability in mortality and a consequence of non-liver complications depend on AKI stage and peak SCr[11]. Considering the combines of the AKIN criteria and classical criteria of kidney failure, our AKI stage 1 had a mean SCr >1.68 mg/dl which had a lessor chance of reversibility than AKI stage 1 with a peak SCr ≤1.5 mg/dl. Paino et al mentioned that patients group of SCr >1.5 mg/dl was more at risk for progression and prediction of in-hospital mortality[15]. Correspondingly, Fagundes et al provides a better risk stratification by considering AKI stage with maximum SCr and reported a high 3 months survival (84%) in patients with AKI stage 1 with peak creatinine ≤1.5 mg/dl[16]. Cheyron et al reported in an ICU setting, cirrhotic with AKI had higher baseline MELD scores, more organ failure and longer ICU stay than with those without AKI with a two times increase in mortality rate[17]. Even though we did not see a statistically significant Child-Pugh score in univariate regression analysis of 30 day mortality, this score was accepted as a standard predictor of survival in cirrhotic patients. Our multivariate regression analysis by adjusted Child-Pugh score, showed that an episode of AKI in SBP causes increase short term mortality up to 2.42-fold as compared to non-AKI.

The study was limited firstly by a need for a greater amount of patients in AKI stage 2 and 3 for subgroup comparison in terms of AKI reversibility, hospital length-of-stay and complications including mortality. Secondly, we did not use the application of urinary biomarkers which are helpful for diagnosing causes of AKI more accurately. And finally, we did not study other factors which may have impacted mortality such as failure of SBP resolution, active alcohol dependence or uncontrolled comorbid disease.

Conclusion

Acute kidney injury is a common complication following an episode of spontaneous bacterial peritonitis and is an important predictor for short-term mortality in decompensated cirrhosis.

What is already known on this topic?

Acute kidney injury is a common episode and causes an increase of non-liver related complications in decompensated cirrhosis.

What this study adds?

Even though most AKI in SBP-decompensated cirrhosis have a high probability of recovery, they can cause an increase in short term mortality up to 2.42-fold as compared to non-AKI.

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Conflicts of interest

The authors declare no conflict of interest.

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อุบัติการณ์และการดำเนินโรคทางคลินิกของผู้ป่วยตับแข็งที่เข้ารับการรักษาในโรงพยาบาลและมีภาวะเยื่อบุช่องท้องอักเสบเองจากแบคทีเรีย

สุภัทศรี เศรษฐสินธุ์, อาภา สานติกุล

ภูมิหลัง: เยื่อบุช่องท้องอักเสบเองจากแบคทีเรียเป็นภาวะแทรกซ้อนที่พบในผู้ป่วยตับแข็งและเป็นสาเหตุของสมรรถภาพตับที่แย่ลง ภาวะไตวายเฉียบพลันพบได้บ่อยและเป็นสาเหตุการเพิ่มอัตราการเสียชีวิตในผู้ป่วยตับแข็ง

วัตถุประสงค์: เพื่อศึกษาอุบัติการณ์ภาวะไตวายเฉียบพลันและปัจจัยที่พยากรณ์อัตราการเสียชีวิตที่ 1 เดือน ในผู้ป่วยตับแข็งที่มีภาวะเยื่อบุช่องท้องอักเสบเองจากแบคทีเรียที่เข้ารับการรักษาในโรงพยาบาล

วัสดุและวิธีการ: ศึกษาตามรุ่นย้อนหลังในผู้ป่วย 123 ราย ภาวะไตวายเฉียบพลันใช้คำนิยามตามเกณฑ์ของ AKIN ผู้ป่วยไตวายเฉียบพลันจะได้รับการประเมินความรุนแรง, การดำเนินโรค, ภาวะแทรกซ้อน, ระยะเวลานอนโรงพยาบาล และอัตราเสียชีวิตระหว่างนอนโรงพยาบาลจากการติดตามเฉลี่ย 30 วัน และวิเคราะห์ปัจจัยที่สัมพันธ์กับการเสียชีวิตโดยใช้แบบจำลองความเสี่ยงตามสัดส่วนของ Cox

ผลการศึกษา: อายุเฉลี่ย 57.8±12 ปี, ร้อยละ 50.4 เป็นเพศชายและมีสาเหตุของตับแข็งจากสุรา, ร้อยละ 78.7 มีความรุนแรงของตับแข็งระดับ Child-Pugh C และคะแนน MELD 20.6±5.8 ระยะเวลานอนโรงพยาบาลเฉลี่ย 15±8.4 วัน ร้อยละ 53.7 มีภาวะไตวายเฉียบพลัน (ระดับที่ 1, ระดับที่ 2 และระดับที่ 3 ร้อยละ 83.3, 7.6 และ 9.1 ตามลำดับ) กลุ่มไตวายเฉียบพลันมีภาวะแทรกซ้อนที่ไม่สัมพันธ์กับโรคตับ, ภาวะช็อกเหตุพิษติดเชื้อและเสียชีวิตมากกว่ากลุ่มที่ไม่เกิดไตวายเฉียบพลันอย่างมีนัยสำคัญ [(ร้อยละ 7.6 เทียบกับ 0, ค่าพีเท่ากับ 0.034); (ร้อยละ 33.3 เทียบกับร้อยละ 14, ค่าพีเท่ากับ 0.013); (ร้อยละ 34.8 เทียบกับร้อยละ 17.5, ค่าพีเท่ากับ 0.031)] ศึกษาตามระดับภาวะไตวายเฉียบพลันไม่พบความแตกต่างของคะแนน MELD จำนวนครั้งของการให้แอลบูมิน การดำเนินโรค และอัตราเสียชีวิตในโรงพยาบาลภายใน 30 วัน ในสามกลุ่มย่อย (ค่าพีเท่ากับ 0.16, 0.31, 0.88, 0.56) วิเคราะห์การรอดชีพด้วยวิธี Kapla-Meier พบว่ากลุ่มไตวายเฉียบพลันมีอัตราเสียชีวิตในโรงพยาบาลภายใน 30 วันสูงกว่ากลุ่มที่ไม่เกิดไตวายเฉียบพลันอย่างมีนัยสำคัญ (ร้อยละ 33.3 เทียบกับร้อยละ 15.7, ค่าพีเท่ากับ 0.022) การวิเคราะห์การถดถอยพหุตัวแปรพบว่าการเกิดไตวายเฉียบพลันมีความเสี่ยงต่อการเสียชีวิตระหว่างนอนโรงพยาบาลที่ 30 วัน สูงเป็น 2.42 เท่าเมื่อเทียบกับกลุ่มที่ไม่เกิดไตวายเฉียบพลัน (ช่วงความเชื่อมั่นร้อยละ 95 เท่ากับ 1.11 ถึง 5.25)

สรุป: ไตวายเฉียบพลันในผู้ป่วยตับแข็งระยะท้ายที่มีภาวะเยื่อบุช่องท้องอักเสบเองจากแบคทีเรียจะเพิ่มภาวะแทรกซ้อนที่ไม่สัมพันธ์กับโรคตับ และเพิ่มอัตราการเสียชีวิตที่ 30 วัน