Turk J Gastroenterol *2016; 27: 349-53*

Cirrhosis with ascites: Is the presence of hemorrhagic ascites an indicator of poor prognosis?

LIVER

Hakan Yıldız, Meral Akdoğan, Nuretdin Suna, Erkin Öztaş, Ufuk B. Kuzu, Zülfükar Bilge, Onur Aydınlı, İsmail Taşkıran

Department of Gastroenterology, Türkiye Yüksek İhtisas Hospital, Ankara, Turkey

Original Article

ABSTRACT

Background/Aims: Hemorrhagic ascites in patients with cirrhosis is described as a RBC (Red Blood cell) >50,000/mm^3 and leads to increased morbidity and mortality. Positive red blood cells at a level of less than 50,000/mm^3 (10,000-50,000) may be encountered in the ascites but it is not known whether this is clinically significant or not. This study aimed to examine the outcome of hemorrhagic ascites in patients with advanced cirrhosis.

Materials and Methods: Data from 329 cirrhotic patients with ascites who received paracentesis at least once due to ascites was retrospectively analyzed from the period of 2007-2013 from the Türkiye Yüksek İhtisas Hospital, Department of Gastroenterology. Patients were divided according to the number of RBC, with greater than 10,000/mm^3 being described as hemorrhagic ascites, and less than 10,000/mm^3 described as the normal or control group. Patient data included: number of accepted intensive unit service stays, acute kidney injury (AKI), hepatic encephalopathy (HES), model for end-liver disease (MELD) score, Child Pugh score (CPS), degree of esophageal varices, spleen size and mortality rates.

Results: Patients were defined as having hemorrhagic ascites with a RBC count greater than 10,000/mm^3 in 118 (35.9%) patients and as a non-hemorrhagic ascites group with less than 10,000/mm^3 in 211 (64.1%) patients. The hemorrhagic ascites group had advanced liver disease symptoms compared to the control group. Meld score in the hemorrhagic group was statistically higher than in the control group (21.5±8.3 vs. 17.3±6.6; p value: 0.001). The median value of bilirubin was 5.9 (0.45-33) in the hemorrhagic ascites group and 4.01 (0.39-33) in the non-hemorrhagic group (p value: 0.001). Using multivariate logistic regression analysis, hemorrhagic ascites was also an independent predictor of mortality (HR 2.7 1.4-6.3), with other mortality indicators being HCC (HR 3.1 1.5-6.4) and HRS (HR 2.6 1.2-5.5).

Conclusion: Patients with hemorrhagic ascites had higher HRS, SBP and admissions to the intensive care unit. We believe that the presence of hemorrhagic ascites can be used as a marker for advanced liver disease and for predicting mortality.

Keywords: Ascites, cirrhosis, hemorrhage, spontaneous

INTRODUCTION

Ascites is the most common complication of cirrhosis and is frequently a cause of admission to the hospital (1). Development of ascites is an indicator of poor prognosis in patients with cirrhosis. The mortality rate of these cases is approximately 40% and 50% at 1 and 2 years, respectively (2). As is known, hemorrhagic ascites is described as a red blood cell (RBC) count greater than 50,000/mm^3 (McGibbon, Chen, Peltekian, & Zanten, 2007). RBC count in normal ascitic fluid is less than 1000/mm^3. Ascitic fluid will be pink colored at a level of approximately 10,000 RBCs/mm^3. Hemorrhagic ascites in cirrhosis is described as having increased morbidity and mortality, but these definitions are commonly related to hepatocellular cancer (HCC) and ruptured varices (4,5).

When patients with chronic liver disease (CLD) with ascites are admitted to the hospital, an ascites fluid sample obtained by paracentesis is sent to the laboratory to measure white blood cell count. Meanwhile, a positive red blood cell count of less than 50,000/mm^3 (between 10,000-50,000/mm^3) in the ascites may be encountered. It is not known whether this is clinically significant or

Address for Correspondence: Hakan Yıldız, E-mail: 12yhakan@gmail.com

Received: January 19, 2016 **Accepted:** April 13, 2016 **Available Online Date:** April 28, 2016

© Copyright 2016 by The Turkish Society of Gastroenterology • Available online at www.turkjgastroenterol.org • DOI: 10.5152/tjg.2016.160042

not. However, we have observed frequently that patients with hemorrhagic ascites have advanced liver disease, poor prognosis and an increased rate of mortality. There are limited studies on cirrhotic patients with hemorrhagic ascites. Moreover, most of these studies were conducted in small case series and only patients with a RBC count ≥50,000/mm^3 were included (4,6-8). We postulated that patients with hemorrhagic ascites and cirrhosis would have a poor prognosis and increased mortality rate. Thus we thought that hemorrhagic ascites may be an important indicator for defining patient prognosis. In this study we aimed to evaluate the effect on prognosis and the relationship of hemorrhagic ascites with advanced cirrhosis.

MATERIALS AND METHODS

Data from 329 cirrhotic patients with ascites who had paracentesis at least once due to ascites was retrospectively analyzed for the period of 2007-2013 from the Türkiye Yüksek İhtisas Hospital, Department of Gastroenterology. Patient data included the number of accepted intensive unit service stays, acute kidney injury (AKI), hepatic encephalopathy (HES), model for end-liver disease (MELD) score, Child Pugh score (CPS), degree of esophageal varices, spleen size, thrombosis of portal veins and prevalence of hepatocellular cancer. Death records of patients were obtained from hospital records and the national death record service. Patient laboratory parameters from venous blood samples and ascites fluid were also obtained from the medical data records, such as creatinine, international normalized ratio (INR), albumin (Alb), alpha fetoprotein (AFP), total bilirubin, alanine aminotransferase (ALT), thrombosis count (PLT), lactate dehydrogenases (LDH), white blood cells (WBCs), and RBC. The study was approved by the ethics committee of Türkiye Yüksek İhtisas Hospital.

Cirrhosis was defined according to clinical features and history consistent with chronic liver disease, as well as a record of complications of CLD (esophageal varices bleeding, hepatic encephalopathy, and ascites) and/or imaging consistent with cirrhosis and/or liver biopsy compatible with cirrhosis. The etiology of cirrhosis was determined according to the following criteria: Hepatitis C, patient with /dL positive HBs-Ag or HBV-RNA, and alcoholic cirrhosis was determined by the intake of alcohol and cirrhosis. Other causes were defined as autoimmune hepatitis, primary biliary cirrhosis, and cryptogenic.

In patients with cirrhosis and ascites, acute kidney injury was determined when creatinine was greater than 1.5 mg/dL after all diuretics were stopped and sufficient fluid support was given (Mehta RL, 2007). Hepatic encephalopathy was determined by West Haven Criteria (Ferenci P, 2002). Grade I was defined as trivial lack of awareness, euphoria or anxiety, and shortened attention span; Grade II was defined as lethargy or apathy; Grade III was defined as somnolence to semi-stupor, but responsive to verbal stimuli, confusion, gross disorientation; Grade IV was defined as coma. Spontaneous bacterial peritonitis was determined by a positive ascetic fluid bacterial culture or neutrophil count greater than 250/mm^3. A corrected PMN was counted if there is bloody fluid: one PMN is subtracted from the absolute PMN count for every 250 red cells/mm^3. Ascites fluid was classified as: USG grade 1 (mild); disorder of shape, level 2 (middle); causing breathing problems, level 3 (tense ascites) (Moore KP, 2003).

We chose 10,000/mm^3 of RBC as a threshold value for hemorrhagic ascites because of our clinical observations and published data of Urrunage et al. (Urrunaga, Nathalie H; Singal, Amit G; Cuthbert, Jennifer A.; Rockey, Don C;, 2013). Patients were classified by the number of RBC with greater than 10,000/mm^3 described as hemorrhagic ascites, and less than 10,000/mm^3 described as normal or control group.

The etiology of hemorrhagic ascites was classified according to the following reasons. Hemorrhagic ascites due to HCC: imaging suggests that the hemoperitoneum was likely secondary to HCC, including bleeding from a liver mass, localized hematoma or "sentinel clot" close to the tumor, a liver mass ≥5 cm or a mass of any size close to the surface (1 cm) (5,13,14).

Iatrogenic hemorrhagic ascites: after medical procedures were performed (paracentesis, diagnostic or therapeutic, transjugular intrahepatic Porto systemic shunt, liver biopsy), iatrogenic hemorrhagic ascites was defined as the detection of hemoperitoneum in the patient after the medical procedure (diagnostic or therapeutic paracentesis, transjugular intrahepatic portosystemic shunt, or liver biopsy). When no cause was found, it was accepted as spontaneous hemorrhagic ascites (15,16).

Statistical analysis

Statistical analyses were performed using the SPSS software version 21 (SPSS Inc.; Chicago, Illinois, USA). Variables were investigated using visual (histograms, probability plots) and analytical methods (Kolmogorov-smirnov/Shapiro-Wilk's test) to determine whether or not they were normally distributed. Descriptive analyses are presented as mean and standard deviation for normally distributed and median and interquartile range (IQR) for non-normally distributed and ordinal variables. Univariate analyses to identify variables associated with patient outcome between hemorrhagic and non-hemorrhagic groups was investigated using Chi square, Fisher exact, Student t and Mann-Whitney U tests, where appropriate. For multivariate analyses, the possible factors were identified. Kaplan-Meier survival estimates were used for patients for survival rate comparison between hemorrhagic and non-hemorrhagic groups. A 5% type-I error level was used to infer statistical significance.

RESULTS

In total, 329 patients (F/M: 129/210) were included in this study. Mean age of the patients was 58.7±13.7 years. The etiology of cirrhosis in patients was 35% Hepatitis B, 22% Hepatitis C, 10% alcohol and 10% others. Hemorrhagic ascites with a RBC count ≥10.000/ mm^3 was detected in 118 (35.9%) cases and non-

hemorrhagic ascites was detected in 211 (64.1%) cases. In the hemorrhagic ascites group, the symptoms were statistically different from the control group, such as abdominal swelling 65 (55%) vs. 42 (19.9%), unconsciousness 56 (47.4%) vs. 50 (23.6%), and pain 38 (32.2%) vs. 39 (18.4%). P values were, respectively, 0.001, 0001 and 0.005 (Table 1).

The study group had more symptoms of advanced liver disease than the control group. Meld score in the hemorrhagic group was statistically higher than controls (21.5±8.3 vs. 17.3±6.6, p=0.001). The mean value of bilirubin was 5.9 (0.45-33) in the hemorrhagic ascites group and was 4.01 (0.39-33) in controls (p=0.001). Additionally, patients with hemorrhagic ascites had higher levels of creatinine (1.5±1.1 vs 1.1±0.6) upon presentation than the control group. Alb, ALT, LDH, AFP, INR and PLT showed no statistical difference between the two groups (Table 1).

Table 1. Demographic and clinical characteristics

Variables	Hemorrhagic ascites (n: 118) (+), mean±standard deviation	Non-Hemorrhagic ascites (n: 211) (-), mean±standard deviation	p
Gender female (%)	44 (37.3%)	75 (35.5%)	0.752
Age (years)	58.3±14.4	59.1±12.8	0.005
Etiology of cirrhosis			
Hepatitis B	44 (37.9%)	79 (37.6%)	0.937
Hepatitis C	13 (11.2%)	39 (13.8%)	
Alcohol	9 (7.8%)	13 (6.2%)	
Autoimmune	17 (14.7%)	33 (15.7%)	
Others*	33 (28.4%)	56 (26.7%)	
Symptoms and signs			
Abdominal distension (%)	65 (55%)	42 (19.9%)	0.001
Abdominal pain (%)	38 (32.2%)	39 (18.4%)	0.005
Unconsciousness (%)	56 (47.4%)	50 (23.6%)	0.001
Laboratory values			
Creatinine (mg/dL)	1.5±1	1.1±0.6	0.001
Biliburin (mg/dL),range	5.9 (0.45-33)	4.01 (0.39-33)	0.001
ALT (U/L)	57.1±78.6	47.1±62.8	0.153
Albumin (g/dL)	2.6±0.4	2.7±0.5	0.969
INR	1.7±0.6	1.6±0.4	0.11
MELD score°	21.5±8.3	17.3±6.6	0.001
Child-Pugh score°	10.4±2.1	9.8±2.2	0.038
WBCx10^3	8.7±6.3	6.7±4.4	0.03
Pltx10^9	108±63.7	110±91	0.3

ALT: alanine aminotransferase; INR: international normalized ratio; MELD: model for end-liver disease; PLT: thrombosis count; WBC: White blood cells

Size of spleen, the degree of esophageal varices and ascites between the groups were statistically different. Mean size of the spleen was 151±34.5 mm in the hemorrhagic ascites group and 140±21.8 mm in controls (p=0.002). When the study patients were compared with controls, while first degree of esophageal varices was less frequent than the control group (28.9% vs. 50.6%), 2nd degree (56.6% vs 39.7%) and 3rd degree (14.5% vs. 4.8%) esophageal varices were higher in study subjects than the controls. On admission to the hospital, the comparison of the degree of ascites between the study group and controls showed that first, second and third degree were 5.1%, 41% and 53.8% in study subjects, and 14.8%, 44.5% and 40.7% in controls, respectively. Although patients with hemorrhagic ascites were less common with first-degree ascites, second and third degree ascites were more common in the hemorrhagic ascites group (Table 2).

In the hemorrhagic ascites group, co-existence of AKI and HES were more common, as well as admission to the ICU and SBP compared to the control group (p=0.001, 0.001, 0.001, 0002). Portal vein thrombosis was found to be 19.5% and 16.2% in patients with or without hemorrhagic ascites (p=0.449) (Table 3).

Table 2. Indirect indicators of portal pressure

Variables	Hemorrhagic ascites (n: 118) (+), mean±standard deviation	Non-Hemorrhagic ascites (n: 211) (-), mean±standard deviation	p
Size of spleen, mean±SD	151±34.5	140±21.8	0.002
Degree of ascites (%)			0.01
1st degree	6 (5.1)	31 (14.8)	
2nd degree	48 (41)	93 (44.5)	
3rd degree	63 (53.8)	85 (40.7)	
Degree of varices (%)			0.005
1st degree	24 (28.9)	88 (50.6)	
2nd degree	47 (56.6)	69 (39.7)	
3rd degree	12 (14.5)	17 (9.89	

SD: standard deviation

Table 3. Complications and outcomes

Complications, (%)	Hemorrhagic ascites (+)	Non-Hemorrhagic ascites (-)	p
AKI	61 (52.1)	49 (23.3)	0.001
SBP	49 (41.5)	53 (25.1)	0.002
Admission to the ICU	77 (65.8)	93 (44.5)	0.001
HES	60 (50.8)	53 (24)	0.004
Varices bleeding	16 (13.6)	24 (11.4)	0.571
Portal vein thrombosis	23 (19.5)	34 (16.2)	0.449

AKI: acute kidney injury; ICU: intensive care unit; HES: hepatic encephalopathy

The causes of hemorrhagic ascites in the study group were relevant to spontaneous hemorrhagic ascites in 87 (82.3%) patients, HCC in 18 (15.1%) patients and iatrogenic in 3 (2.5%) patients.

The duration of median survival in patients with hemorrhagic ascites was 8.1 months and it was 19.3 months in the non-hemorrhagic group. Mortality rate in the hemorrhagic ascites group was 34.1% at the first month, 85.7% at the first year, and 93.4% at the third year. These mortality rates were less than the non-hemorrhagic ascites group, which had a mortality rate at one month of 6.8%, at one year of 54.7%, and at three years of 79.8%. Between groups, a significant difference was found statistically as shown in Figure 1 (p=0.001).

Using multivariate logistic regression analysis, hemorrhagic ascites with RBC≥10,000/mm^3 was also an independent predictor of mortality (HR 2.7 1.4-6.3) and other mortality indicators were found to be HCC (HR 3.1 1.5-6.4) and HRS (HR 2.6 1.2-5.5) (Table 4).

Only 20 (6.1%) patients had greater than 50,000/mm^3 RBC in the ascites fluid. Patients with hemorrhagic ascites were divided two groups, which were 10,000-50,000/mm^{3}and more than 50,000/mm^3. There were twenty patients (16.9%) who had RBC ≥50,000/mm^3. These two groups were compared for AKI, HES, SBP and admission to ICU and showed no statistical difference from each other in p values, with values of 0.544, 0.516, 0.115, and 0.996 (Table 5).

DISCUSSION

Poor prognostic indicators of liver cirrhosis are hyponatremia, increased serum creatinine, low arterial pressure, low urine sodium, High MELD and CPS score (2,17,18). These indicators are widely used for patients with cirrhosis for describing patient prognosis. In most cirrhotic patients, spontaneous hemorrhagic ascites are incidental and do not present any bleeding signs such as tachycardia, hypotension, or syncope. But some researchers suggested that hemorrhagic ascites may represent a poor prognostic sign because it is associated with an increased risk of encephalopathy, acute renal injury and a high mortality rate (4,5).

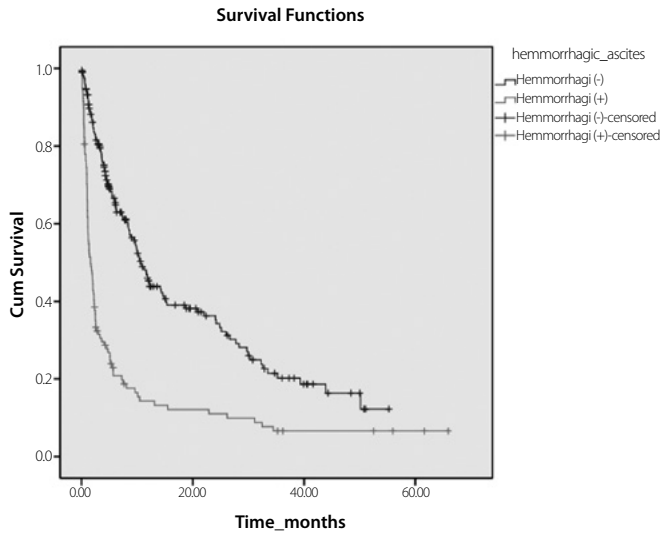


Figure 1. Survival of patients with hemorrhagic ascites. The blue line is defined as hemorrhagic ascites negative and the green line is hemorrhagic ascites positive. In the hemorrhagic ascites group, the median survival was 8.2 months and in the control group the median survival was 19.3 months.

The etiology of spontaneous hemorrhagic ascites is not clearly understood, but two mechanisms have been postulated for cirrhotic patients with ascites (4). The first theory is that intra-abdominal bleeding is due to an organ, a small peritoneal vessel, or an abdominal cavity varix. The second theory is that it is connected with increased portal or splenic pressure. Increased pressure can lead to erythrocyte leakage from the vessels into the peritoneal cavity (10). In our study, increased spleen size and esophageal varices in patients with hemorrhagic ascites compared to the control group may support this second theory.

This study showed that high mortality rates are remarkable in cirrhotic patients with hemorrhagic ascites. Urrunaga et al. (Urrunaga, Nathalie H; Singal, Amit G; Cuthbert, Jennifer A.; Rock-

Table 4. Complications and outcomes

Variable	Multivariate analysis (HR) (CI)	p
Hemorrhagic ascites	2.7 (1.4-6.3)	0.002
History of HCC	3.1 (1.5-6.4)	0.004
AKI	2.6 (1.2-5.5)	0.026
HES	2.3 (1.1-4.7)	0.014
Gender	0.8 (0.4-1.6)	0.697
SBP	1.1 (0.6-2.2)	0.604
Age	1.03 (0.99-1.034)	0.221
MELD score	1.054 (0.99-1.23)	0.101

AKI: acute kidney injury; HES: hepatic encephalopathy; HCC: hepatocellular carcinoma; SBP: spontaneous bacterial peritonitis; MELD: model for end-liver disease

Table 5. Complications

Complications, (%)	Ascites RBC 10,000-50,000 n: 98	Ascites RBC >50,000 n: 20	p
AKI	51 (52.6)	12 (60)	0.544
SBP	40 (40.8))	12 (60)	0.115
Admission to the ICU	63 (64.9)	13 (65)	0.996
HES	51 (52)	12 (60)	0.516

AKI: acute kidney injury; HES: hepatic encephalopathy; SBP: spontaneous bacterial peritonitis; RBC red blood cell; ICU: intensive care unit

ey, Don C;, 2013) reported that mortality rates in cirrhotic patients with hemorrhagic ascites are a median of 1 year and for our patients it was 8.1 months. These rates can be relevant to advanced liver disease and MELD score (20 (6-46) vs 18 (6-46)).

When we compared the patients with hemorrhagic ascites according to RBC count (10,000-50000/mm^3 and >50,000/mm^3), the rate of AKI, HES, SBP and admission to ICU were similar between the two groups. Thus we thought that we could use RBC≥10,000/mm^3 for hemorrhagic ascites evaluation in cirrhotic patients according to the results of our study.

Spontaneous hemorrhagic ascites often have a clinically less severe course. Most patients complain of abdominal swelling and weakness, but cirrhotic patients with ruptured varices or HCC rupture can present with hypotension, decreased level of hemoglobin, and rapid swelling in the abdomen (4,5,19). This life-threatening situation is encountered in less than 0.5% of cirrhotic patient with ascites (20). In our study, we encountered two (0.6%) patients and they died 3 and 7 days after diagnosis.

The drawbacks of this study were that it was retrospective, and patients who underwent first time paracentesis could not be excluded for iatrogenic hemorrhagic ascites. But paracentesis was performed by three experienced gastroenterologist fellows and ascites samples for the 1st degree ascites were taken under ultrasound guidance and first paracentesis value included in the study, others paracentesis(second or third) were excluded from the study. Also, paracentesis is very rarely associated with bleeding complications (0.01%) (21,22).

In conclusion, according to our results, patients with hemorrhagic ascites had higher rates of HRS, SBP and admission to the intensive care unit. We believe that the presence of hemorrhagic ascites can be used as a marker of advanced liver disease and help predict mortality.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Türkiye Yüksek İhtisas Hospital Hospital.

Informed Consent: N/A.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - H.Y.; Design - H.Y., E.Ö.; Supervision - M.A.; Materials - H.Y.; Data Collection and/or Processing - H.Y., N.S., U.K., Z.B., O.A.; Analysis and/or Interpretation - M.A., E.Ö.; Literature Review - I.T.; Writer - H.Y. U.K.; Critical Review - M.A., E.Ö.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

REFERENCES

1. Runyon BA; AASLD Practice Guidelines Committee. Management of adult patients with ascites due update. Hepatology 2009; 49: 2087-107. [CrossRef]
2. Guevara M, Gines P. Prognosis in patients with cirrhosis In: Ginès P, Arroyo V, Rodés J, Schrier RW, editors. Ascites and renal dysfunction in liver disease: pathogenesis, diagnosis and treatment. Malden: Blackwell 2005; 23: 260-70.
3. McGibbon A, Chen GI, Peltekian KM, van Zanten SV. An evidence-based manual for abdominal paracentesis. Dig Dis Sci 2007; 52: 3307-15. [CrossRef]
4. DeSitter L, Rector WG. The significance of bloody ascites in patients with cirrhosis. Am J Gastroenterol 1984; 79: 136-8.
5. Akriviadis EA. Hemoperitoneum in patients with ascites. Am J Gastroenterol 1997; 92: 567-75.
6. Mallory A, Schaefer J. Complications of diagnostic paracentesis. JAMA 1978; 239: 628-30. [CrossRef]
7. McVay PA, Toy PT. Lack of increased bleeding after paracentesis and thoracentesis in patients with mild coagulation abnormalities. Transfusion 1991; 31: 164-71. [CrossRef]
8. Liebowitz HR. Hazards of abdominal paracentesis in the cirrhotic patient. NY State J Med 1962; 62: 2223-9.
9. Mehta RL, Kellum JA, Shah SV, et al. Acute kidney injury network: report of an initiative to improve outcomes in acute kidney injury. Cri Care 2007; 11: R31. [CrossRef]
10. Ferenci P, Lockwood A, Mullen K, et al. Hepatic encephalopathy–definition, nomenclature, diagnosis, and quantification: final report of the working party at the 11th world congresses of gastroenterology, Vienna, 1998. Hepatology 2002; 35: 716-21. [CrossRef]
11. Moore KP, Wong F, Gines P, et al. The management of ascites in cirrhosis: report on the consensus conference of the International Ascites Club. Hepatology 2003; 38: 258-66. [CrossRef]
12. Urrunaga NH, Singal AG, Cuthbert JA, et al. Hemorrhagic ascites. Clinical presentation and outcomes in patients with cirrhosis. Journal of Hepatology 2013; 58: 1113-8. [CrossRef]
13. Clarkston W, Icciardi M, Kirkpatrick S. Acute hemoperitoneum from rupture of a hepatocellular carcinoma. J Clin Gastroenterol 1988; 10: 221-5. [CrossRef]
14. Tarantino L, Sordelli I, Calise F, Ripa C, Perrotta M, Sperlongano P. Prognosis of patients with spontaneous rupture of hepatocellular carcinoma in cirrhosis. Uptades Surg 2011; 63: 25-30. [CrossRef]
15. Pache I, B.ilodeau M. Severe haemorrhage following abdominal paracentesis for ascites in patients with liver disease. Aliment Pharmacol Ther 2005; 21: 525-9. [CrossRef]
16. Martinet O, Reis ED, Mosimann F. Delayed hemoperitoneum following largevolume paracentesis in a patient with cirrhosis and ascites. Dig Dis Sci 2000; 45: 357-8. [CrossRef]
17. Llach J, Ginès P, Arroyo V, et al. Prognostic value of arterial pressure, endogenous vasoactive systems and renal function in cirrhotic patients admitted to the hospital for the treatment of ascites. Gastroenterology 1988; 94: 482-7. [CrossRef]
18. Salpeter SR, Luo EJ, Malter DS, Stuart B. Systematic review of noncancer presentations with a median survival of 6 months or less. Am J Med 2012; 125: 512.e1. [CrossRef]
19. Norton ID, Andrews JC, Kamath PS. Management of ectopic varices. Hepatology 1998; 28: 1154-8. [CrossRef]
20. Ma YJ, Chen EQ, Lu JJ, Tan MZ, Tang H. Hemoperitoneum in cirrhotic patients without abdominal trauma or tumor. Hepatobiliary Pancreat Dis Int 2011; 10: 644-8. [CrossRef]
21. Runyon BA. Paracentesis of ascitic fluid: a safe procedure. Arch Intern Med 1986; 146: 2259-61. [CrossRef]
22. McVay PA, Toy PT. Lack of increased bleeding after paracentesis and thoracocentesis in patients with mild coagulation abnormalities. Transfusion 1991; 13: 164-71. [CrossRef]