Section: General Medicine

Original Research Article

A STUDY COMPARING THE LYMPHOCYTE/MONOCYTE RATIO WITH AARC AND CLIF C – ACLF SCORES IN ASSESSING THE CLINICAL OUTCOMES OF PATIENTS WITH ACUTE OR CHRONIC LIVER FAILURE.

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Abstract

Background: AARC and CLIF C-ACF scores were used to determine the survival of patients with ACLF and CLIF C-ACLF. The present study was carried out to compare the lymphocyte/monocyte ratio with the AARC and CLIF C – ACLF scores in assessing the clinical outcomes of patients with ACLF. **Material & Methods:** All enrolled patients were classified into Group 1 (n=40) if ACLF was defined by APASL or Group 2 (n=55) if the EASL-AASLD definition defined ACLF. The CTP score/MELD Na/AARC / LMR ratio was calculated in group 1, and the CTP score/MELD Na/CLIF ACLF score/LMR ratio was calculated in group 2. Both groups were followed up for mortality, the need for transplantation, readmission, and decompensation. **Results:** The APASL cohort's overall mortality rate was 37.5%. Of the patients who died, 40% had ACLF of grade 2. LMR was equivalent to a good predictor of mortality and showed higher statistical significance. The LMR ratio negatively correlated with MELD Na, with an r-value of -0.301. In the EASL-AASLD cohort, the overall mortality rate was 30.9%. 47.1% of the expired patients belong to ACLF grade 3. LMR was equivalently a good predictor of mortality and showed a higher statistical significance, with a p-value of 0.0005. The LMR ratio negatively correlated with the MELD Na and CLIF ACLF scores, with r values of -0.314 and -0.464, respectively. **Conclusion:** The present study showed that the LMR predicts the outcome in patients with ACLF and is comparable to the traditional ACLF scores given by the APASL and EASL-AASLD.

INTRODUCTION

Acute chronic liver failure (ACLF) is a distinct entity in the spectrum of chronic liver disease, with a rapid downhill course and poor outcome in response to acute insult. Patients with ACLF have underlying chronic liver disease, which becomes aggravated due to an acute precipitant.[1] Mitigating the acute insult may lead to spontaneous recovery in many cases. The potential of reversibility without liver transplantation or early recognition of the need for transplantation is the main reason for classifying these patients into distinct groups.[2]

The two most widely used definitions of ACLF are the European Association for the Study of Liver (EASL) Chronic Liver Failure/American Association (AASLD) consortium and the Asia Pacific for Study of Liver (APASL) ACLF research consortium. According to the former, in the EASL AASLD definition, ACLF is defined as an acute deterioration of preexisting chronic liver disease, usually related to a precipitating event and associated with increased mortality at 12 weeks due to multisystem organ failure. As per the latter, ACLF is an acute hepatic insult manifesting as jaundice (serum bilirubin ≥5 mg/dl) and coagulopathy (INR ≥1.5 or prothrombin activity ≤40%) complicated within four weeks by clinical ascites and encephalopathy in a patient with previously diagnosed or undiagnosed chronic liver disease/cirrhosis, and is associated with high 28-day mortality.[3-4]

Numerous studies have been conducted to elucidate the role of various inflammatory markers in

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determining patient survival. Among these markers, the ratio of neutrophils to monocytes, ratio of neutrophils to platelets, distribution of red cell width (RDW), and its ratio with other blood cells have been well studied. However, the most studied inflammatory marker in the last few years has been the lymphocyte–to–monocyte ratio (LMR).[5] This inflammatory marker has been shown to have an absolute role in determining the survival of patients with various diseases such as cancer, cardiovascular disease, gastrointestinal diseases (Crohn's disease), and colorectal carcinoma. Many recent studies have shown that the LMR is a good prognostic marker for patients with hepatocellular carcinoma. This marker is extensively studied because it is cost-effective and easy to calculate and interpret.[5-6]

Although AARC and CLIF C-ACF scores are used to determine the survival of patients with ACLF, CLIF C-ACLF scores are difficult to calculate without a personal digital assistant. In contrast, five parameters are required to interpret and calculate the AARC score.[7] This has led to the need to identify markers that are easy to obtain, calculate, and interpret. The LMR has been widely used to predict patient outcomes in chronic diseases. However, its role in patients with ACLF has not been well studied, and to date, only two studies have assessed its role in determining the outcomes in patients with liver cirrhosis and ACLF.[8-9] Hence, the present study was carried out to compare the lymphocyte/monocyte ratio with the AARC and CLIF C – ACLF scores in assessing the clinical outcome of patients with acute or chronic liver failure.

MATERIALS AND METHODS

This prospective observational study included 150 patients with acute decompensation underlying chronic liver disease admitted to the Institute of Internal Medicine/Institute of Hepatology, Madras Medical College, Rajiv Gandhi Government General Hospital, and Chennai for six months. The patients were randomly divided into two groups: Group I if APASL defined ACLF in 40 patients, and Group II, if ACLF was defined by the EASL-AASLD definition in 55 patients. Institutional ethics committee permission and informed consent were obtained from all participants before the start of the study.

Inclusion Criteria

Patients fulfilling the definition of ACLF as defined by either of the two definitions given by the APASL or EASL-AASLD and aged between 18 and 80 years were included in the present study.

Exclusion Criteria

Patients with acute decompensation do not amount to ACLF, and Patients diagnosed with liver cirrhosis are admitted because of other medical illnesses, such as diabetes mellitus, ischaemic heart disease, or cerebrovascular accident. Patients with hepatocellular carcinoma and any other concurrent ailments that could alter LMR, such as haematological malignancies, autoimmune diseases, or chronic infections (tuberculosis). Patients were administered antibiotics in the last 14 days (because antibiotics can alter the blood counts in the complete picture of blood). Pregnancy, post-liver transplant, and death within 24 hours of admission were excluded from the study.

Group 1: ACLF according to the APASL definition of the Asian Pacific Association of Study of Liver Diseases (APASL) group, defines ACLF as Acute hepatic insult manifesting as jaundice (serum bilirubin ≥5 mg/dl), coagulopathy (INR ≥1.5, prothrombin activity ≤30%), complicated within four weeks by development of ascites and encephalopathy with diagnosed or undiagnosed underlying chronic liver disease which is associated with high 28-day mortality. AARC score and Grading of ACLF severity were graded based on the APASL ACLF Research Consortium (AARC) score. AARC score was validated, and the dynamic score incorporated five variables: S. Bilirubin, S. Creaatinine, INR, S. Lactate, and Grade of HE as per West Haven grading.[3]

Group 2: ACLF by EASL-AASLD Definition: The American Association for the Study of Liver Disease (AASLD) and European Association for the Study of the Liver (EASL) working definition of ACLF as follows the acute deterioration of preexisting chronic liver disease, usually a precipitating event, and is associated with increased mortality at 28 days due to multisystem organ failure. The CLIF C ACLF score, which ranged from 0 to 100, was calculated using an online application on the CLIF Consortium website. It was calculated by dividing the total white cell count by the total number of white cells and compared to the lymphocyte monocyte ratio (LMR) using absolute lymphocyte count / absolute monocyte count.9,10

All patients underwent detailed clinical evaluation, including history, physical examination, and routine biochemical and imaging evaluations. Investigations of the aetiology of cirrhosis and the cause of acute deterioration were performed on a case-by-case basis. Patients were diagnosed with chronic liver disease with a proper history, clinical examination, and investigations, which included viral markers (HBsAg, Anti HCV, total anti-Hbc, AIH markers (ANA/SMA/LKM), serum ceruloplasmin, iron profile, celiac work-up, NAFLD work-up, and radiological investigations for cirrhosis, as otherwise indicated.

For acute insults, patients work up for hepatic insults and infections, leading to acute decompensation. The combination of an aspartate aminotransferase level that was elevated (but < 300U/ml) and a ratio of the aspartate aminotransferase level to the alanine aminotransferase level that was > 2, a total serum bilirubin level of > 5 mg/dl, an elevated INR, and neutrophilia in a patient with ascites and a history of

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heavy alcohol use was considered as a case of alcoholic hepatitis until proven otherwise. Clinical examination included a thorough general physical examination, vital signs, and systemic examination. Laboratory investigations included a complete haemogram, serum electrolytes, renal and liver function tests, and a coagulogram. The CTP and MELD/MELD Na scores determined the severity of cirrhosis.

Statistical Analysis

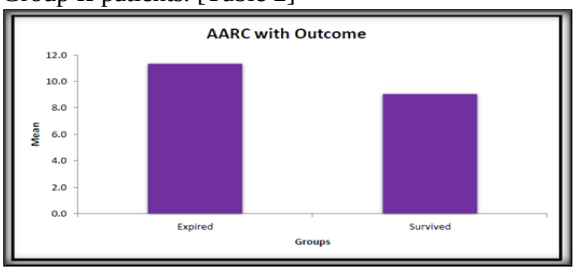
The collected data were analysed with IBM.SPSS statistics software 23.0 Version. Descriptive statistics and frequency analysis percentage analysis were used for categorical variables, and mean & SD were used for continuous variables. The unpaired sample t-test was used to find significant differences between bivariate samples in independent groups. Pearson's correlation coefficient was used to assess the relationships between variables. The Chi-Square test was used to determine the significance of categorical data. The probability value ≤0.05 is considered significant in all statistical tools.

RESULTS

In the present study, 95 patients participated, of whom 40 were randomly selected in Group I, and 55 were included in Group II. Male predominance was reported in both groups (77.5% in Group I and 70.9% in Group II) (Table 1). Alcohol and primary hepatotropic infections were the most common acute insults (57.5 %) in Group I, whereas alcohol and UGI bleeding were the major contributing insults (56.4 %) in Group II. Almost all patients in both groups were CTP class C (Group I: 97.5%; Group II: 98.2%). Mortality was observed in 37.5% of Group I and 30.9% of Group II patients, mainly from Grade 3 ACLF. Although alcohol was the most common acute insult infection, either primarily caused hepatic failure or patients who developed sepsis in the hospital had a higher mortality rate (46.7%) in Group I and drugs in Group II (35.2%). [Table 1]

The total count had a higher statistical significance, with the mean count being higher in the expired patients than in those who survived (Group I: mean 15960, p=0.0003; Group II: mean 16.2*109, p=0.0005). The absolute lymphocyte count and monocyte count also had a higher statistical significance, with both higher counts in the expired group (Group I: p = 0.0002 and p = 0.01; Group II: p = 0.0005 and p = 0.0005). The total bilirubin and creatinine levels were significantly higher in the expired group than in the surviving group (Group I: p= 0.009 and 0.06, respectively; Group II: p= 0.004 and 0.037, respectively). No statistical significance was observed with serum albumin, INR, serum lactate, and sodium levels in Group I patients and total bilirubin, creatinine, and sodium levels in Group II patients. [Tables 1 and 2]

Traditional CTP, MELD, MELD Na, and AARC scores were good predictors of mortality. Statistical significance was observed with p-values of 0.005, 0.018, 0.023, and 0.015 in Group I patients (Figure 1). Traditional CTP, MELD, MELD Na, and CLIF C ACLF scores were good predictors of mortality. It showed statistical significance with p-values of 0.0005, 0.018, 0.008, and 0.0005 in Group II patients (Figure 1, Tables 1 and 2). LMR was equivalently a good predictor of mortality and showed a higher statistical significance in both patient groups (Group I: p = 0.0005; Group II: p = 0.0005) (Figure 2). In addition, the LMR ratio showed a negative correlation with MELD Na in both groups, with an r-value of -0.301 for Group I patients and -0.314 and -0.464, respectively, for Group II patients. [Table 2]



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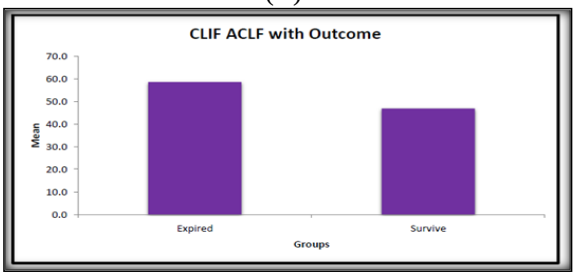
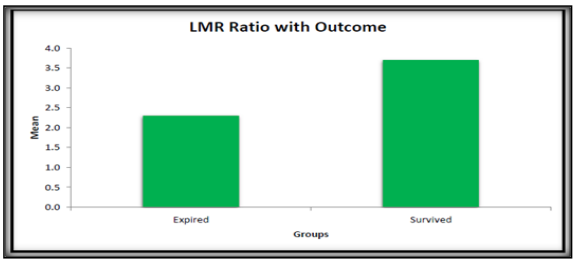


(B)

Figure 1: Observation of outcome for (A) AARC of Group I patients and (B) CLIF-ACLF of Group II patients



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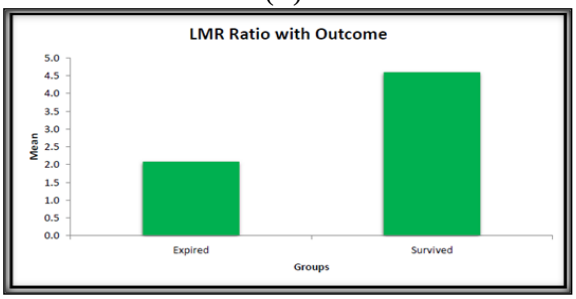

(B)

Figure 2: Observation of LMR ratio of patients (A) Group I (B) Group II

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Table 1: Observation of demographic and other evaluation parameters of both group patients

Parameters		Frequency N (%)	
		Group I (N=40)	Group II (N=55)
Gender	Male	31 (77.5%)	16 (29.1%)
	Female	9 (22.5%)	39 (70.9%)
Acute precipitating events	Alcohol	13 (32.5%)	17 (30.9%)
	Autoimmune	1 (2.5%)	12 (21.8%)
	Drugs	6 (15%)	1 (1.8%)
	Infection	10 (25%)	11 (20%)
	UGI bleed	10 (25%)	14 (25.5%)
Incidence of hepatic encephalopathy	Grade 0	4 (10%)	-
	Grade 1	5 (12%)	26 (47.5%)
	Grade 2	14 (35%)	14 (25.5%)
	Grade 3	11 (27.5%)	15 (27.3%)
	Grade 4	6 (15%)	-
CTP classification in ACLF	Grade B	1 (2.5%)	1 (1.8%)
	Grade C	39 (97.5%)	54 (98.2%)
Incidence of various grades of ACLF	Grade 1	9 (22.5%)	-
	Grade 2	19 (47.5%)	-
	Grade 3	12 (30%)	-
Outcome of patients with ACLF	Survived	25 (62.5%)	38 (69.1%)
	Expired	15 (37.5%)	17 (30.9%)

Table 2: Observation of different evaluation variables of patients in both groups

Group I	**ACLF Grade**			**P-value**
Acute insult	**I**	**II**	**III**	
Alcohol	9(34.6%)	4(18.6%)	4 (26.7%)	0.686
Drugs	0 (0%)	0 (0%)	1 (6.7%)	
Infection	5 (19.2%)	3 (21.4%)	3 (20%)	
UGI Bleed	8 (30.8%)	2 (14.3%)	4 (26.7%)	
Unknown	4 (15.4%)	5 (35.7%)	3 (20%)	
Group II				
Acute insult	**I**	**II**	**III**	
Alcohol	9 (34.6%)	4 (28.6%)	4 (26.7%)	0.686
Drugs	0 (0%)	0(0%)	1 (6.7%)	
Infection	5 (19.2%)	3 (21.4%)	3 (20%)	
UGI Bleed	8 (30.8%)	2 (14.3%)	4 (26.7%)	
Unknown	4 (15.4%)	5 (35.7%)	3 (10%)	
Group I	**Outcome**			-
Acute insult	**Survived**	**Expired**		-
Alcohol	12 (48%)	1 (6.7%)	-	0.030
Autoimmune	1 (4%)	0(0%)	-	
Drugs	4 (16%)	2 (13.3%)	-	
Infection	3 (12%)	7 (46.7%)	-	
UGI Bleed	5 (20%)	5 (33.3%)	-	
Group II				
Acute insult	**Survived**	**Expired**		-
Alcohol	12(31.6%)	5 (29.4%)	-	0.340
Autoimmune	1 (2.6%)	0(0%)	-	
Drugs	5 (13.2%)	6 (35.3%)		
Infection	10 (26.3%)	4 (23.5%)	-	
UGI Bleed	10 (26.3%)	2 (11.8%)	-	
Group I				p-value
Total count	25	15	-	0.0003
Mean ±SD	10768.0 ± 3610.7	15960.0 ±4635.9	-	
Hb	25	15	-	0.037
Mean ±SD	11.1±2.0	9.6±2.7	-	
Abs Lympho count	25	15	-	0.0002
Mean ±SD	1924.4± 439.2	1465.4±262.7	-	
Absolute lymphocyte count	25	15	-	0.010
Mean ±SD	551.6 ±117.2	644.0± 78.4	-	
Total bilirubin	25	15	-	0.009
Mean ±SD	15.1 ±7.3	21.6 ±7.1	-	
Serum albumin	25	15	-	0.279
Mean ±SD	2.3± 0.3	2.4± 0.3	-	
MELD SCORE	25	15	-	0.018
Mean ±SD	21.4± 11.1	30.9± 12.7	-	
MELD NA SCORE	25	15	-	0.23
Mean ±SD	27.2 ±8.0	33.6 ±8.9	-	
AARC	25	15	-	0.015
Mean ±SD	9±2.7	11.3±2.8	-	
LMR Ratio	25	15	-	0.0005

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Mean ±SD	3.7 ±1.3	2.3 ±0.5	-	
Group II			-	
Total count	38	17	-	0.0005
Mean ±SD	9.3± 3.6	16.2 ±5.6		
Absolute lymphocyte count	38	17	-	0.0005
Mean ±SD	2082.1 ±369.1	1423.5± 407.6	-	
Abs Mono count	38	17	-	0.0005
Mean ±SD	474.7 ±108.9	692.4 ±74.6	-	
Albumin	38	17	-	0.004
Mean ±SD	2.4 ±0.3	2.1 ±0.3	-	
INR	38	17	-	0.037
Mean ±SD	2.1 ±0.6	2.5± 0.6	-	
CTP Score	38	17	-	0.0005
Mean ±SD	12.3 ±1.8	14.2± 1.2	-	
MELD	38	17	-	0.018
Mean ±SD	28.9 ±5.2	34.0 ±7.5	-	
MELD Na	38	17	-	0.008
Mean ±SD	32.3 ±4.6	36.1± 5.1	-	
CLIF ACLF	38	17	-	0.0005
Mean ±SD	46.8 ±7.6	58.5 ±9.3	-	
LMR Ratio	38	17	-	0.0005
Mean ±SD	4.6 ±1.3	2.1 ±0.6	-	

DISCUSSION

The present study included 40 patients in Group I (if APASL defined ACLF) and 55 in Group II (if ACLF was defined by EASL –AASLD). The diseased populations in the present study were homogenous, with chronic liver disease/cirrhosis presenting for the first time as acute hepatic decompensation in response to an acute hepatic insult, suggesting a wider applicability. Interestingly, severe alcoholic hepatitis was the most common acute insult in Asia, unlike the earlier belief that HBV was the predominant aetiology in Asia.

Male predominance was reported in both groups (77.5% in Group I and 70.9% in Group II). Alcohol and Primary hepatotrophic infections were the most common acute insults (57.5 %) in Group I, whereas alcohol and UGI bleeding were the major contributing insults (56.4 %) in Group II. These findings in the present study are following earlier reported studies.[11]

Almost all patients in both groups were CTP class C (Group I: 97.5%; Group II: 98.2%). Mortality was observed in 37.5% of Group I and 30.9% of Group II patients, mainly from Grade 3 ACLF. Garg et al., in their investigation, also reported similar findings where more than 90% of patients belong to CTP class 3.[12] The total count had a higher statistical significance, with the mean count being higher in the expired patients than in those who survived (Group I: mean 15960, p=0.0003; Group II: mean 16.2*109, p=0.0005). Traditional CTP, MELD, MELD Na, and AARC scores were good predictors of mortality. It showed statistical significance with p-values of 0.005, 0.018, 0.023, and 0.015 in Group I patients. Choudhury et al., in their study, also showed good prediction of mortality of ACLF by AARC score.[13] The traditional CTP, MELD, MELD Na and CLIF C ACLF scores were good mortality predictors and showed statistical significance with a p-value of 0.0005, 0.018, 0.008 and 0.0005, respectively, in Group II patients. Jalan et al. also reported similar findings in their study.[14]

Our study found that the LMR was significantly lower in the expired group than in those who survived in Groups 1 and 2. LMR, CTP, MELD, and MELD Na predicted both groups' outcomes very well, with a statistical significance of 101. The AARC and CLIF ACLF scores were also good predictors of mortality in Groups 1 and 2, respectively. Jamil et al. have studied the prognostic value of LMR in patients with liver cirrhosis, and a comparison of LMR with CTP and meld score has been made.[15] The study found that LMR, MELD and CTP were all good scores in predicting mortality. According to the study, MELD had more predictive power than the LMR and CTP scores. LMR was significantly lower in the study group than in the control group. The LMR was inversely correlated with both the MELD and CTP scores. Zhu et al. studied the LMR ratio in acute or chronic liver failure patients. They studied LMR in ACLF and compared it with that in patients with CHB and healthy controls. They found that the LMR was significantly reduced in the study group compared to that in the control group (CHB and HC). They also found an inverse correlation between the MELD and LMR. The combined use of LMR and MELD augmented the predictive value.[9]

Limitations of the study

Our study was a single-centre study with a relatively small sample size. The findings of this study need to be confirmed by large multicentre studies. The prognostic value of the LMR was not dynamically assessed during the patients' course, which requires further study.

CONCLUSION

We conclude that LMR can be easily calculated from the differential white cell counts upon admission, predicts the outcome in patients with ACLF, and is comparable to the traditional ACLF

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scores given by APASL and EASL-AASLD. Further prospective studies involving more patients are required to define better the relationship between LMR and the outcomes of patients with ACLF.

REFERENCES

1. Moreau R, Jalan R, Gines P, Pavesi M, Angeli P, Cordoba J, et al. Acute-on-chronic liver failure is a distinct syndrome that develops in patients with acute decompensation of cirrhosis. Gastroenterology 2013; 144:1426-1437.e9. https://doi.org/10.1053/j.gastro.2013.02.042.
2. Jalan R, Yurdaydin C, Bajaj JS, Acharya SK, Arroyo V, Lin H-C, et al. Toward an improved definition of acute-on-chronic liver failure. Gastroenterology 2014; 147:4–10. https://doi.org/10.1053/j.gastro.2014.05.005.
3. Zheng M-H, Wu S-J, Shi K-Q, Yan H-D, Li H, Zhu G-Q, et al. Conditional survival estimate of acute-on-chronic hepatitis B liver failure: A dynamic prediction based on a multicenter cohort. Oncotarget 2015; 6:23261–71. https://doi.org/10.18632/oncotarget.4666.
4. Sarin SK, Kedarisetty CK, Abbas Z, Amarapurkar D, Bihari C, Chan AC, et al. Acute-on-chronic liver failure: consensus recommendations of the Asian Pacific Association for the Study of the Liver (APASL) 2014. Hepatol Int 2014; 8:453–71. https://doi.org/10.1007/s12072-014-9580-2.
5. Berry PA, Antoniades CG, Carey I, McPhail MJW, Hussain MJ, Davies ET, et al. Severity of the compensatory anti-inflammatory response determined by monocyte HLA-DR expression may assist outcome prediction in cirrhosis. Intensive Care Med 2011; 37:453–60. https://doi.org/10.1007/s00134-010-2099-7.
6. Agrawal S, Duseja A, Gupta T, Dhiman RK, Chawla Y. Simple organ failure count versus CANONIC grading system for predicting mortality in acute- on- chronic liver failure. J Gastroenterol Hepatol 2015; 30:575–81. https://doi.org/10.1111/jgh.12778.
7. Sarin SK, Choudhury A. Acute-on-chronic liver failure. Curr Gastroenterol Rep 2016;18. https://doi.org/10.1007/s11894-016-0535-8.
8. Triantafyllou E, Woollard KJ, McPhail MJW, Antoniades CG, Possamai LA. The role of monocytes and macrophages in acute and acute-on-chronic liver failure. Front Immunol 2018;9. https://doi.org/10.3389/fimmu.2018.02948.
9. Zhu S, Waili Y, Qi X, Chen Y, Lou Y. Lymphocyte–monocyte ratio at admission predicts possible outcomes in patients with acute-on-chronic liver failure. Eur J Gastroenterol Hepatol 2017; 29:31–5. https://doi.org/10.1097/meg.0000000000000767.
10. Jalan R, Pavesi M, Saliba F, Amorós A, Fernandez J, Holland-Fischer P, et al. The CLIF Consortium Acute Decompensation score (CLIF-C ADs) for prognosis of hospitalised cirrhotic patients without acute-on-chronic liver failure. J Hepatol 2015; 62:831–40. https://doi.org/10.1016/j.jhep.2014.11.012.
11. Yang WB, Chen EQ, Bi HX, Bai L, Chen XB, Feng P, et al. Different models in predicting the short-term prognosis of patients with hepatitis B virus-related acute-on-chronic liver failure. Ann Hepatol 2012; 11:311–9. https://pubmed.ncbi.nlm.nih.gov/22481448/.
12. Garg H, Kumar A, Garg V, Sharma P, Sharma BC, Sarin SK. Clinical profile and predictors of mortality in patients of acute-on-chronic liver failure. Dig Liver Dis 2012; 44:166–71. https://doi.org/10.1016/j.dld.2011.08.029.
13. Choudhury A, Jindal A, Maiwall R, Sharma MK, Sharma BC, Pamecha V, et al. Liver failure determines the outcome in patients of acute-on-chronic liver failure (ACLF): comparison of APASL ACLF research consortium (AARC) and CLIF-SOFA models. Hepatol Int 2017; 11:461–71. https://doi.org/10.1007/s12072-017-9816-z.
14. Jalan R, Saliba F, Pavesi M, Amoros A, Moreau R, Ginès P, et al. Development and validation of a prognostic score to predict mortality in patients with acute-on-chronic liver failure. J Hepatol 2014; 61:1038–47. https://doi.org/10.1016/j.jhep.2014.06.012.
15. Jamil J, Durrani AA. Assessing the outcome of patients with liver cirrhosis during hospital stay: A comparison of lymphocyte/monocyte ratio with MELD and Child-Pugh scores. Turk J Gastroenterol 2018; 29:308-15. https://doi.org/10.5152/tjg.2018.17631.

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