

two populations: with ACLF criteria and without ACLF accord EASL guidelines. Inclusion criteria: Patients with ascites secondary to cirrhosis, without the use of primary SBP prophylaxis, admitted to the hospitalization service without other identified sources of infection. NLR was performed at the time of hospitalization and after antibiotic use. Measures of central tendency for dispersion and Pearson correlation between Child Pugh and Meld Na scores with NLR were applied.

Results: A total of 128 patients were collected, 25 patients fulfilling the inclusion criteria: 15 without ACLF and 10 with ACLF. Mortality in both groups was 40%; in patients without ACLF it corresponded to 20%, whereas in patients with ACLF mortality was 70%, which corresponded to Grade 2 (50%) and Grade 3 (80%). Regarding the NLR index, patients with ACLF did not have higher values compared to those without ACLF. The correlation with the scores was not significant between ACLF - NLR (r=0.35) and Meld Na - NLR (r=0.035). Seventeen patients responded to antibiotic treatment, 13 in the group without ACLF and 4 in the group with ACLF, in all of which there was a decrease in the NLR after ascites fluid control. However, it was found that the index values were correlated before and after the use of antibiotics at 48 hours of treatment (r= 0.88) in comparison with the NLR before and after no response to treatment at 48 hours (r= 0.31).

Conclusions: We found that NLR was not associated with severity, stage and mortality in patients with SBP, however there is a strong relationship between NLR values before and after antibiotic use at 48 hours, which could allow its use as a biomarker for assessment of antibiotic response.

Ethical statement

The protocol was registered and approved by the Ethics Committee. The identity of the patients is protected. Consentment was obtained.

Declaration of interests

None

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Table 1.
Study population characteristics.

	Without ACLF (n=15)	ACLF (n=10)
Gender		
Male	4 (26.6%)	8 (80%)
Female	6 (73.4%)	2 (20%)
Age	49 años (30 -67)	58.2 años (44 -78)
Etiology.		
Alcohol.	6 (73.4%)	6 (60%)
Autoimmune Hepatitis.	0	1 (10%)
Primary biliary cholangitis.	1 (6.6%)	1 (10%)
Overlap Syndrome	2 (13.2%)	0
Hepatitis C virus.	3 (19.8%)	0
Cryptogenic.	3(19.8%)	2 (20%)
CHILD PUGH		
A	1 (6.6%)	0
B	6 (39.6%)	2 (20%)
C	8 (52.8%)	8 (80%)
Meld- Na	19.53 points (8 -28)	27 points (14 - 40)
Mortality.	3 (20%)	7 (70%)
ACLF		
Grade II		4 (40%)
Grade III		6 (60%)
Polymorphonuclear cells at diagnosis	2150 cels/mm ³ (50 -16,200)	1782.6 cels/mm ³ (188 -6180)
Response to treatment.		
Yes.	13 (87%)	4 (40%)
No.	2 (13%)	6 (60%)
Ascites Fluid Proteins.	1.76 g/dl (0.5 - 4.6)	1.84 (0.6 - 4.6)
NLR		
Before treatment.	8.82 (1.86 - 21.16)	23.65 (1.60 - 59.60)
After treatment.	4.87 (1.4 - 14.61)	18.62 (1.2 - 52.8)

Impact of cholemic nephrosis on renal failure in cirrhotic patients

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Introduction and Objectives: : The development of acute kidney injury (AKI) in cirrhotic patients is of multifactorial origin, including urinary tract infection, diuretics, portal hypertension, shock, etc. Another important factor is cholemic nephrosis, it is considered when total bilirubin exceeds 20 mg/dl, this implies that bile pigments damage the distal tubule with deterioration of renal function, increasing morbidity and mortality. This study aimed to evaluate the levels of hyperbilirubinemia in the development of AKI and its association with biomarkers of renal failure.

Materials and Patients: : Retrospective and analytical study of a cohort of cirrhotic patients, to evaluate the development of AKI associated with bilirubin levels. Statistical analysis: A binary logistic regression model was performed considering bilirubin (greater than 20), NGAL (greater than 150) and cystatin (greater than 0.95) as associated factors. The significance of the model was considered with an alpha level less than 0.05.

Results: 109 patients were included, 45 women 64 men, age 54.67 ± 11.6, Child Pugh A: 2, B: 29, C: 78. The binary logistic model was significant W(1)=11.089, p=0.001. The OR for bilirubin was 4.37 (1.168-16.35, 95% CI P=.027), for NGAL OR 2.7 (1.08-6.71, 95% CI; p=.032), cystatin 0.64 (0.35-11.66, CI 95%; p=0.764) not significant.

Conclusions: Hyperbilirubinemia increases the risk of developing AKI by up to 4 times. The useful biomarker for AKI was NGAL.

Ethical statement

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Declaration of interests

None

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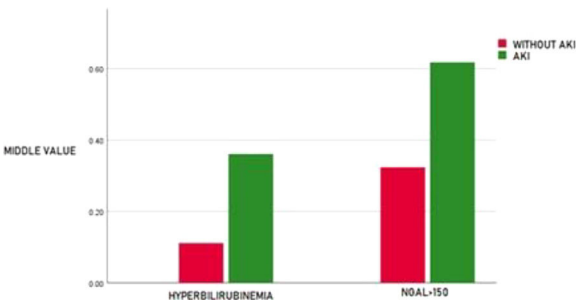


Figure 1. Grouped Bar Graph: mean value of bilirubin and NGAL in patients with AKI

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