

Materials and Methods: Observational, retrospective, comparative, longitudinal study evaluating early mortality (after 15 days) of Mexican patients with decompensating cirrhosis who were given the MELD-LA scale upon admission to assess its predictive capacity. Patients with decompensated cirrhosis of any etiology were included, mortality at 15 days was evaluated, descriptive statistics were performed, and inferential with ROC curves.

Results: Two hundred thirty-eight patients were included, of which 100 were analyzed; 33 men and 66 women, minimum age 27 and maximum 84 years, mean 51 years, SD 11.7. Alcohol was the main cause of cirrhosis, and the most common cause of decompensation was sepsis. The MELD-LA score performed better in predicting mortality at 15 days; sensitivity and specificity were 0.84 and 0.59; providing the highest AUC was 0.72. The results are shown in figure 1.

Conclusions: The MELD-LA score was shown to be an early and objective predictor of hospital mortality in Mexican cirrhotic patients.

Funding: The resources used in this study were from the hospital without any additional financing

Declaration of interest: The authors declare no potential conflicts of interest.

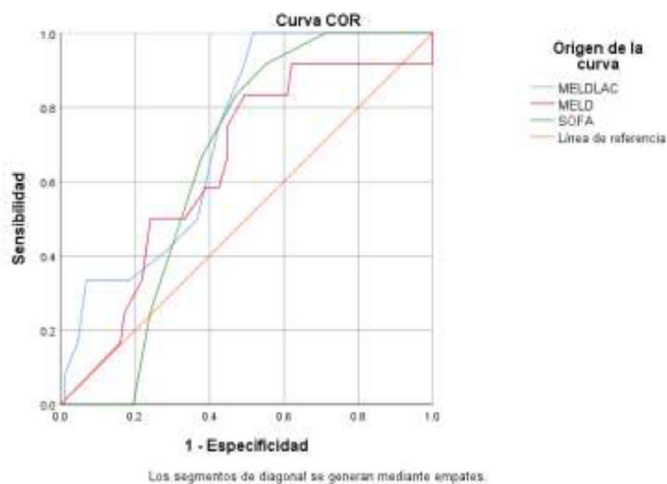


Figure 1.

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Norepinephrine in infusion as an alternative to large volume post-paracentesis albumin in patients with cirrhosis

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Introduction and Objective: Circulatory dysfunction syndrome (DCIP) occurs in patients who undergo large-volume paracentesis. Albumin should usually be administered to prevent it. However, in many cases, this resource is not available. Singh V, Kumar B, *et al.* published a study evaluating norepinephrine in preventing DCIP with promising results. This study aimed to assess whether norepinephrine is a helpful resource in preventing DCIP when albumin is unavailable.

Materials and Methods: A prospective, descriptive, and analytical study includes patients with cirrhosis and grade III ascites. Given norepinephrine as an alternative to albumin to prevent DCPI, patients with kidney injury, shunts, and gastrointestinal bleeding were

excluded. Descriptive statistics were performed, comparing creatine, Ngal, nystatin C, and sodium at days 0, 3, 6, and 28 days, evaluating whether or not DCPI developed. The trial was approved by the research ethics committee, and informed consent was obtained.

Results: Twelve patients were included, one of whom was ruled out due to precordial pain without electrocardiographic changes; 11 patients were analyzed; 9 men (81.8%); age 52.2 ± 4.5 ; and 2 (18.2%) P and 9 (81.8%) Child C; 8 (72.7%) due to alcohol, 2 (18.2%) MAFLD, 1 (9.1%) HCV; the drained liters of ascites were 12.5 L with a range of 9 to 18; Renal function measured by creatinine, Cystatin C and NGAL at 0, 3, 6 and 28 days did not show renal dysfunction. The results are shown in Figure 1.

Conclusions: Norepinephrine promises to be an alternative for the preventive management of DCPI.

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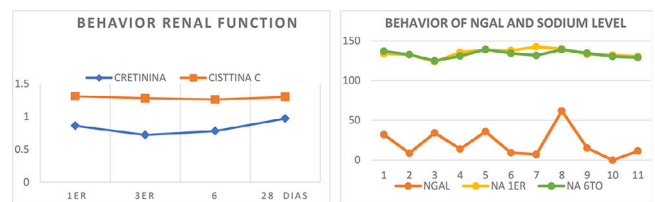


Figure 1

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Management of internal hemorrhoidal bleeding refractory to endoscopic treatment in a patient with liver cirrhosis

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Introduction and Objectives: Hemorrhoidal bleeding associated with portal hypertension is a rare complication. Endoscopic management is the initial treatment recommended. There are no established guidelines in refractory bleeding; we present a patient with hemorrhoidal bleeding refractory to endoscopic treatment.

Clinical case: A 72-year-old woman with decompensated liver cirrhosis and hemorrhoidal disease presented hemorrhoidal bleeding treated with endoscopic band ligation; five days later, rectal bleeding returned. Colonoscopy showed post-ligation ulcers and active bleeding; endoscopic bleeding control with band ligation and sclerotherapy was not achieved (Image 1A-1B). Venography of hemorrhoidal veins and embolization with coils and Histoacryl was performed, achieving bleeding control (Image 1B-C), and hepatic-portal vein gradient was measured (33 mmHg).

Discussion: Symptomatic hemorrhoidal disease in liver cirrhosis has a prevalence of 5%, and it is associated with greater vascular colaterality, coagulopathy and high surgical risk. The treatment options go from endoscopic band ligation, sclerotherapy and arterial or vein embolization, with rebleeding rates between 10-13% in different case

series, being the last option of treatment, the portosystemic derivation, which has shown suboptimal results. There are no established guidelines for its management because the evidence of the different therapeutic options is insufficient.

Conclusion: Embolization of hemorrhoidal vessels with refractory bleeding is a good alternative for bleeding control in patients with liver cirrhosis.

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Figure 1. A Retroflexion colonoscopy shows post-ligation ulcers with active bleeding. 1.B Venography of superior hemorrhoidal veins with dilatation of left system (white arrow) and contrast leakage at distal rectum (black arrows). 1.C-D. Embolization with coils/Histoacryl, showing absence of distal contrast.

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Causes of decompensation of liver cirrhosis and predictors of in-hospital mortality in patients admitted to the gastroenterology service of the Regional Hospital Lic. Adolfo López Mateos in the years 2017 - 2018

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Introduction and Objectives: Identify the main causes of liver cirrhosis decompensation and predictors of in-hospital mortality in patients admitted to the Gastroenterology service of the RH. Lic. Adolfo López Mateos in the years 2017 and 2018.

Material and Methods: Daily digital censuses from 2017 and 2018 were used, and patients diagnosed with decompensated cirrhosis and laboratory studies were included. The statistical software STATA V 16.0 was used, and the incremental model with $p < 0.000$ and the VIF test and goodness of fit with the HOSMER-LEMESHOW test were performed.

Results: Two hundred fifty-five Patients were included, and the most frequent causes of decompensation were; ascites at 35%, encephalopathy at 34% and variceal hemorrhage at 22% (image 1).

The three variables that resulted in a risk for in-hospital mortality were: elevated creatinine levels, encephalopathy, and spontaneous bacterial peritonitis.

Discussion: Ascites as the first cause of liver cirrhosis decompensation is the same reported in international studies; a difference was observed in the second cause, which could be due to the fact that the sample of patients is small. Patients with any degree of encephalopathy, SBP and elevated creatinine levels had higher in-hospital deaths.

Conclusions: Ascites, followed by hepatic encephalopathy and variceal hemorrhage, were the most frequent causes of decompensation in patients with liver cirrhosis admitted to the Gastroenterology service of RH Lic. Adolfo López Mateos in the years 2017 and 2018. The early identification of poor prognosis factors and the initiation of targeted treatment could improve the prognosis of patients.

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Hepatic hydrothorax resistant to diuretics treated with octreotide

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Introduction and Objective: Hepatic hydrothorax is the excessive accumulation of transudate in the pleural cavity, secondary to portal hypertension in cirrhotic patients without existing cardiopulmonary disease. Treatment includes a reduction in ascites production, prevention of ascites transfer to the pleural space, pleural fluid removal, pleural space obliteration, and liver transplantation. This study aimed to report a case of hepatic hydrothorax treated with Octreotide.

Case Summary: A 63-year-old man with liver cirrhosis due to alcohol. It began with edema in the pelvic limbs, increased abdominal perimeter, MRC II dyspnea, orthopnea, and chest pain radiating to the back. EF: scleral jaundice, 60% left pleural effusion. Gii ascites, MPI edema (+). He was classified as Child-Pugh C (12 points), Hb 6.2, Leu 2.2, Plts 37,000, INR 2.1, chest X-ray: 100% left pleural effusion. A 1200 cc cytological/cytochemical thoracentesis was performed: transudate according to Light's criteria, GASA >1.1, BNP 87 mg/dl; spironolactone was started up to 200/80 mg, persists with 100% pleural effusion. Octreotide 100mcg is started for five days. He showed improvement at the end of treatment.

Discussion: In hepatic hydrothorax, sodium restriction as a treatment is insufficient; the use of diuretics is necessary; up to 30-40% do not respond; the placement of chest tubes is associated with high morbidity, clinical deterioration, TIPS, urgent HT or death. Octreotide, a somatostatin analog, directly inhibits RAAS, improving the efficiency of renal plasma flow and natriuresis.

Conclusions: Octreotide can be a useful alternative in hepatic hydrothorax.

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