

Funding: This research received no specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Table 1
Characteristics of patients with chronic hepatitis C.

Gender	
Female, No. %	3 (60)
Male, No. %	2 (40)
Age, mean, years	52
Genotype	
1, No. %	3 (60)
3, No. %	1 (20)
Pre-treatment viral load, mean, UI/ml	297,542
Pretreatment	
Sofosbuvir-ledipasvir, No. %	1 (20)
Sofosbuvir-velpatasvir, No. %	3 (60)
Glecaprevir-pibrentasvir, No. %	1 (20)
VIH co-infection, No. %	1 (20)
Percentage of adherence to first treatment	>80%
Liver cirrhosis, No. %	2 (40)

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Risk factors for sarcopenia in cirrhotic patients under evaluation for liver transplantation.

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Introduction and Objectives: Liver cirrhosis is the sixth cause of death in Mexico and is also associated with a significant reduction in quality of life. Malnutrition is common in patients in the final stage of the disease and its presence has been associated with worse clinical outcomes.

Objectives: determine the prognostic factors of sarcopenia in cirrhotic patients in a protocol of liver transplant.

Materials and Patients: Type of study: Retrospective, cross-sectional, analytical, single center. Patients over 18 years of age were included in the evaluation for liver transplantation within the transplant clinic of the Gastroenterology department of the UMAE Centro Médico Nacional Siglo XXI: Study period: January 1, 2022, to June 1, 2023. Statistical analysis It was carried out with dispersion measures for continuous variables and with proportions for categorical variables. Mean and standard deviation or median and interquartile range were used according to the distribution of the variables for normal Student T distribution and for free Mann Whitney U distribution. Dichotomous variables and determining risk were analyzed with the Chi-square test.

Results: 63 patients with liver cirrhosis on a liver transplant protocol were included, with a predominance of female sex (74.6%), whose most frequent etiology was steatotic liver disease associated with metabolic dysfunction (MASLD) (27%), mostly Child- Pugh Functional Class. Pugh B (32%). Median MELD 3.0 was 17(14-22); The most frequent decompensations were ascites in 46 (73%), followed by hepatic encephalopathy in 31 (49%) and variceal hemorrhage in 27 patients (42.9%). Mortality in the evaluated group was 14%, and 3 of them had sarcopenia, which represents 33%. A prevalence of sarcopenia was found in 55.6% of the patients evaluated. In the risk analysis, age > 60 years had an OR of 5.46 95% CI (1.6-17.6)

Conclusions: The risk factor for sarcopenia in patients being evaluated for liver transplantation is age >60 years. Other factors that can influence the development of sarcopenia must be established.

Ethical statement: Risk-free research and approved by the ethics committee.

Declaration of interests: None.

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Table 1
Characteristics of patients undergoing evaluation for liver transplantation. n= 63.

Age, median	52.2 + 11.7
Gender	
Women, No. (%)	47 (74.6)
Men, No. (%)	16 (25.4)
Sarcopenia, No.(%)	35 (55.6)
Etiology	
MASLD, No. (%)	17 (27)
BPC, No. (%)	14 (22)
VHC, No. (%)	7 (11.1)
Overlap Syndrome, No. (%)	7 (11.1)
AIH, No. (%)	6 (9.5)
Other etiology, No. %	12 (20)
Child-Pugh-Turcotte	
A No. (%)	7 (11.1)
B No. (%)	32 (50.8)
C No. (%)	24 (38)
MELD Na, median, percentile	16 (11 - 20)
MELD 3.0 median, percentile	17 (14 - 22)
Diabetes, No. (%)	16 (25.4)
Systemic arterial hypertension, No. (%)	9 (14.3)
Acute decompensation	
Hepatic encephalopathy, No. (%)	31 (49.2)
Spontaneous bacterial peritonitis, No. (%)	5 (7.9)
Ascites, No. (%)	46 (73)
Hemorrhage, No. (%)	27 (42.9)
Thyroid disease, No. (%)	17 (27)
Death, No. (%)	9 (14)
Transplant, No. (%)	4 (6.3)
Body Mass Index, median, percentile	26.17 (23.2 - 29.6)

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Response to L-ornithine L-aspartate in a single intravenous dose in cirrhotic patients with overt hepatic encephalopathy.

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Introduction and Objectives: Hepatic encephalopathy (HE) is a neuropsychiatric syndrome that occurs in patients with acute and chronic liver disease; It is associated with a higher risk of new episodes and higher mortality at one year. L-ornithine L-aspartate (LOLA) is a stable salt that acts on two key ammonia detoxification pathways: urea synthesis and glutamine synthesis. Its intravenous administration has been studied with repeated doses and at high doses.

The objective is to describe the response to the administration of a single intravenous dose of L-ornithine L-aspartate in cirrhotic patients with an acute event of overt hepatic encephalopathy.

Materials and Methods: Type of study: Retrospective, transversal, observational, analytical, single-center.

Were included patients over 18 years of age, treated in the continuous admission service for acute event of manifest hepatic encephalopathy grade II to IV according to the West-Haven criteria, who received treatment based on L-ornithine L aspartate in a dose of 20 g. intravenous infusion for 4 hours, with evaluation of the response at the end of infusion. Study period: January 2022 to December 2023. Statistical analysis was performed with frequencies and percentages;

For the quantitative variables Student's t or Mann-Whitney U according to the distribution of the variables and to show the difference between the degree of hepatic encephalopathy on admission and after the infusion of L-ornithine L-aspartate, Wilcoxon was used.

Results: 72 patients with decompensated liver cirrhosis of any etiology were included, mostly Child-Pugh C functional class (56.9%), with a predominance of female sex (75%), and it was found that the most frequent triggering factor was constipation (22.2. %), followed by urinary tract infection (12.5%). Upon admission, the degree of encephalopathy was classified according to the West-Haven clinical scale of which grade II was the most prevalent, the single intravenous dose of L-ornithine, L-aspartate was effective with a significant response $p < 0.05$

Conclusions: The response to a single intravenous dose of 20 g of L-ornithine L-aspartate is effective for the treatment of hepatic encephalopathy, clinically manifesting in an acute episode.

Ethical statement: Risk-free research and approved by the ethics committee.

Declaration of interests: None.

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Table 1
Characteristics of patients with hepatic encephalopathy.

Age	59 (56.25 – 67.75)
Women	54 (75%)
Men	18 (25%)
Etiology	
BPC	19 (26.4%)
MASLD	19 (26.4%)
Alcohol	11 (15.3%)
Child-Pugh C	41 (56.9%)
MELD	20 points (18.25-29)
WHC severity scale	
Grade II	35 (48.6%)
Grade III	31 (43.1%)
Grade IV	6 (8.3%)
Precipitating factors	
Constipation	16 (22.2%)
UVI	9 (12.5%)
Unidentified	9 (12.5%)

BPC, Primary Biliary Cirrhosis; MASLD, Metabolic dysfunction-associated steatotic liver disease; UVI, Urinary Tract Infection.

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Glycogen storage disease, an uncommon cause of portal hypertension in adulthood.

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Introduction and Objectives: Glycogen storage diseases are inborn errors of metabolism, with an estimated incidence of 1 in 10,000. Type IV represents 3% of this diseases (GBE1 gene 3p14

involvement), presenting with varied clinical features, including a milder form of hepatic involvement, with hepatic integrity described up to 19 years of age.

Materials and Patients: We present a 22-year-old woman with a history of low weight since childhood, she presented episodes of hematemesis and melena, and she underwent panendoscopy, documenting esophageal varices requiring variceal ligation. Extensive studies demonstrated indirect signs of portal hypertension, partial portal vein thrombosis, and multiple liver lesions, located in segments V, VI and VII, with an irregular heterogeneous morphology, partially defined borders, with a peripheral hypodense halo, the hyperdense center even in simple and porta phases, with the largest lesion being $8.2 \times 7.8 \times 8.1$ cm. A defect in the filling of the left branch of the portal vein was identified, as well as compression of the right branch due to mass effect. Differential diagnoses included cholangiocarcinoma, hepatocellular carcinoma, and hepatic tuberculosis.

Results: Infectious-viral or autoimmune etiologies were ruled out through investigation. Percutaneous liver biopsy guided by ultrasound was performed. The histopathological report showed morphological findings suggestive of metabolic deposit disease. Tiny intracytoplasmic granules, PAS positive, F2 fibrosis on the metavir scale (Masson's trichrome staining); all of these findings consistent with glycogen storage disease type IV (branching enzyme deficiency) with non-progressive hepatic subtype was reached. Based on the history and evolution of the patient she was at the advanced stage of the disease with evidence of fibrosis and portal hypertension. She presented a torpid clinical course, with poor oral tolerance, we identified she had cardiomyopathy with left ventricular hypertrophy, manifesting with cardiac arrhythmia, managed with medical treatment.

This was a challenging case, as the diagnosis was made at an advanced stage of the disease, with multiple complications, limiting the prognosis and therapeutic options for the patient. She was referred to the genetics service for further evaluation.

Conclusions: We present a clinical case of a challenging diagnosis, due to the multiple clinical expressions and variants of glycogen storage disease. It can primarily affect the liver, heart, and neuromuscular system, according to enzymatic deficiency, with milder phenotypes having residual enzymatic activity.

Ethical statement: The patient's identity is protected.

Declaration of interests: None.

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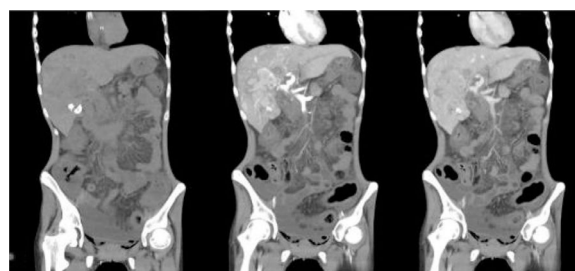


Figure 1. Triphasic abdominal CT scan. Liver, enlarged with multiple lesions located in segments V, VI, and VII, with defined borders, heterogeneous, with peripheral hypodense halo, hyperdense center in all phases, more pronounced in the arterial phase. Left branch of the portal vein, with filling defect attached to the wall, and right branch with decreased caliber.