Conclusions: The relevance as Gastroenterologists recognize the disease associated with IgG4 because of the multi-organ involvement as part of the approach to a patient with jaundice syndrome; despite the low prevalence reported in our country, knowing this entity will make its timely treatment and subsequent recognition easier in other 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.

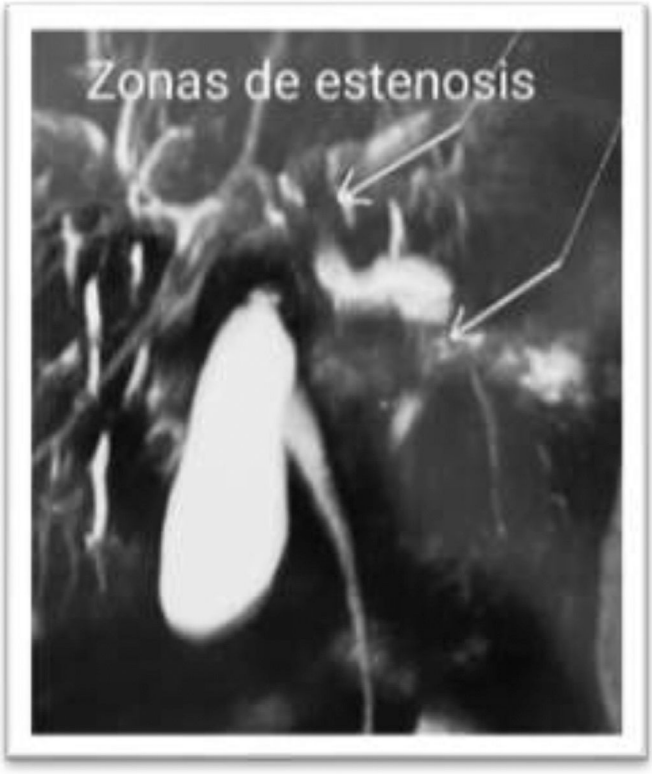

Figure 1.
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Giant simple hepatic cyst, when and how to treat it

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Introduction and Objective: Hepatic cysts are rare, with a prevalence between 0.1 to 0.5%. They are divided into parasitic and non-parasitic, being more common than the last ones. They can be subdivided into simple (<5% of the population) or multiple.

Case Report: 46-year-old woman with no relevant history. She comes due to an increase in abdominal perimeter of 4 months of evolution and weight loss of 9kg in 4 months; asthenia, adynamia, early satiety, postprandial fullness and abdominal pain in the right upper quadrant, oppressive, intensity 9/10, exacerbated by mobilization. CT scan with a giant liver cyst of 219 × 166 × 239mm, a volume of 4544cc. Alkaline phosphatase and GGT >3 times their normal value. She was admitted for percutaneous drainage placement, with a total output of 7480cc and biochemical and clinical improvement, without complications.

Discussion: Simple cysts occur in people over 40 years of age, more frequently in women (4:1 ratio). The differential diagnosis includes liver abscess, tumor, hemangioma, hematoma, parasitic cyst, and polycystosis. They are easy to distinguish by image as they are well-defined; they contain serous fluid and lack septa, papillary projections, and calcifications. They are considered giants when measuring>5 cm and their treatment is only indicated in symptomatic patients, with pain being the usual. Percutaneous drainage has little morbidity and improves compression symptoms. However, recurrence is high (almost 100%), so the administration of a sclerosing agent is recommended.

Conclusions: Conservative procedures have high recurrence rates, so systematized laparoscopic surgery is a good option for definitive treatment.

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.

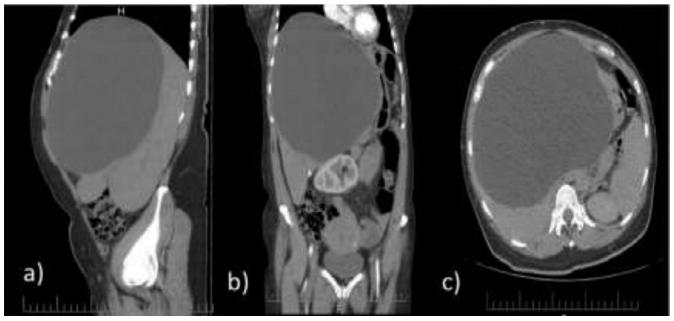

Figure 1.
https://doi.org/10.1016/j.aohep.2022.100843

Symptomatic giant cavernous hemangioma as an indication for liver transplantation

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Introduction and Objective: Hepatic hemangiomas (HH) are the most common primary benign tumors of the liver, more frequent in women, attributed to estrogens. Its size can reach up to 30 cm, being considered giant when it measures >4 cm.

Case Report: A 65-year-old woman with giant cavernous hemangiomas as an incidental finding on liver USG. The abdominal MRI established the diagnosis by spotting two intrahepatic lesions, one of the right lobe occupying all the segments, measuring 18.5 × 16.6 × 15.7 cm, with a volume of 2527.6 cc; another in the left lobe of 7.8 × 7.8 × 6.5 cm, the volume of 206.8 cc; hypointense on T1 sequence, hyperintense on T2, with enhanced contrast medium in the periphery, later it is centripetal, with focal areas without enhancement at 20 minutes.

Physical examination: painful swelling in the left hypochondrium up to the anterior axillary line and epigastrium. Increased alkaline phosphatase and GGT. Surgical management is contraindicated due to the characteristics of the lesion. We decided to send her for a liver transplant.

Discussion: Histologically, they are vascular malformations characterized by caverns covered by a single layer of endothelium. The gold standard is MRI, where we observe peripheral nodular enhancement followed by central enhancement in a well-defined homogeneous mass. Surgical management is indicated in the symptomatic giant HH, with an increase in size or suspicion of malignancy. They

require LT if the interventions such as embolization or resection fail to control the disease.

Conclusions: Giant HHs should be treated if they cause symptoms and may require HT when they are unresectable or have complications such as coagulopathy, risk of rupture, or failure of previous management.

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.

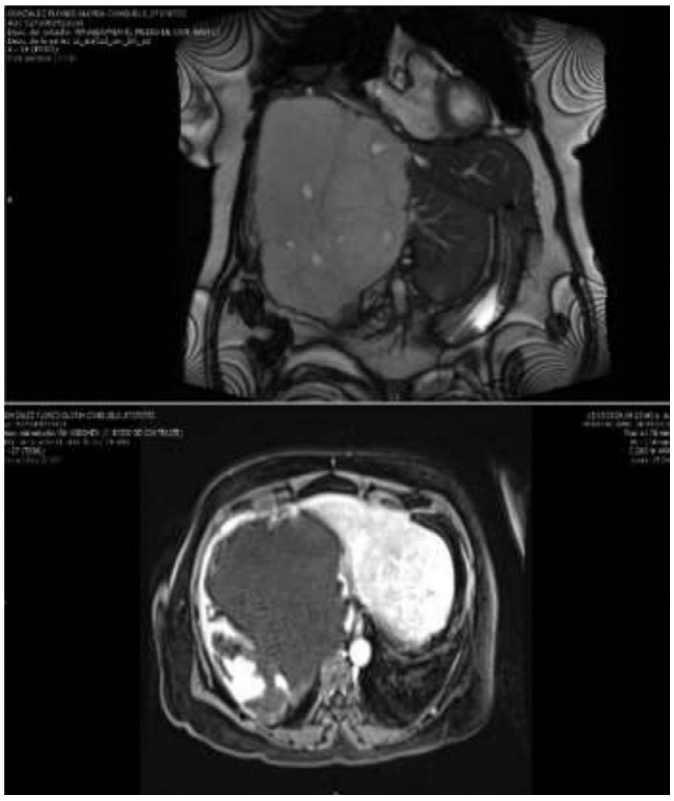

Figure 1.
https://doi.org/10.1016/j.aohep.2022.100844

Tyrosinemia in a toddler, a case report

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Introduction and Objectives: This study aimed to present the case of a toddler with acute-on chronic liver failure probably related to tyrosinemia.

Case Summary: a two-year four-month-old male infant presented with gastroenteritis, which three days later was initiated with jaundice and drowsiness. On physical examination: jaundice, hematemesis, abdominal distention and hepatomegaly ($3 \times 2 \times 2$cm). Laboratory results: pancytopenia, incalculable coagulation test, hydroelectrolytic disorders, hyperbilirubinemia, increased transaminases, hyperammonemia, lactic acidosis, and negative viral hepatitis panel. Abdominal USG: liver with irregular borders, starry sky appearance, increased echogenicity of the right kidney and free fluid compatible with cirrhosis. He died on the second day of hospitalization with a diagnosis of multiple organ failure secondary to fulminant hepatic failure. A liver wedge biopsy reports chronic liver disease, severe acute activity, and fibrosis. Histological image is compatible with tyrosinemia. Newborn metabolic screening, without result.

Discussion: Type I tyrosinemia (hepatorenal) is an autosomal recessive aminoacidopathy caused by a deficiency of the enzyme fumarylacetoacetate-hydrolase, generating accumulation of metabolites fumarylacetoacetate and maleylacetoacetate leading to hepatic cell damage. Its prevalence is 1:100,000, debuting with liver failure, coagulopathy, gastrointestinal bleeding, jaundice, ascites, hepatomegaly, hypoglycemia and peripheral neuropathy. In this case, the patient was admitted with hepatopathy of unknown etiology; most likely, pathologies were ruled out, and finally, with suspicion of a metabolic disorder, he died before confirming the diagnosis with a compatible biopsy and clinical picture.

Conclusion: Tyrosinemia belongs to the group of inborn errors of metabolism; although rare, its early diagnosis can be made through newborn metabolic screening, improving its prognosis and survival, as it is unfavorable in advanced stages.

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

Table 1. Evolution of biochemical data

	02.09.2021	03.09.2021	04.09.2021
Hemoglobin	13.7		10.2
Hematocrit	40.1		32.3
Leukocytes	22.26		3.08
Neutrophils	77%		30%
Band cells	21%		10%
Platelet	211,000		56,000
PT	No coagula		33
PTT	No coagula		47.4
D-dimer	791		
Total bilirubin	15.4	15.4	11.1
Direct bilirubin	8.9	8.4	6.2
Indirect bilirrubin	6.5	7	4.9
AST	2435	1145	753
ALT	2012	1103	680
HDL	655	692	1326
Ammonia	569.6	961	
Sodium	136		152
Potassium	4.2		2.2
Calcium	8.3		11.3
Glucose	9	****	
Urea	19.26	27.82	
Creatinine	0.58	0.80	
Uric acid	8		
Arterial blood gases			Ph:6.76 PCO2: 43.4 HCO3: 6.1 BE: -28.2 Lact: 28.96