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.

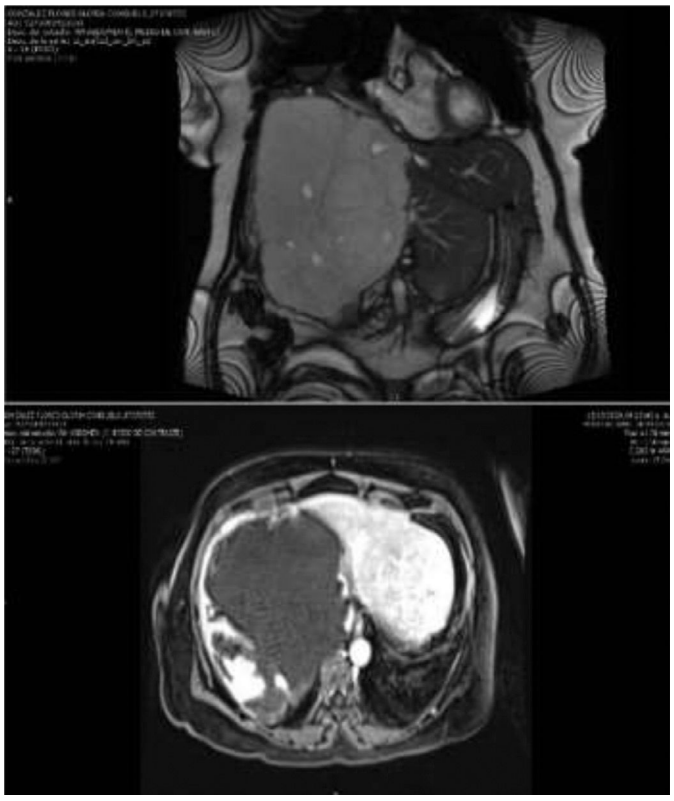

Figure 1.
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Tyrosinemia in a toddler, a case report

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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.

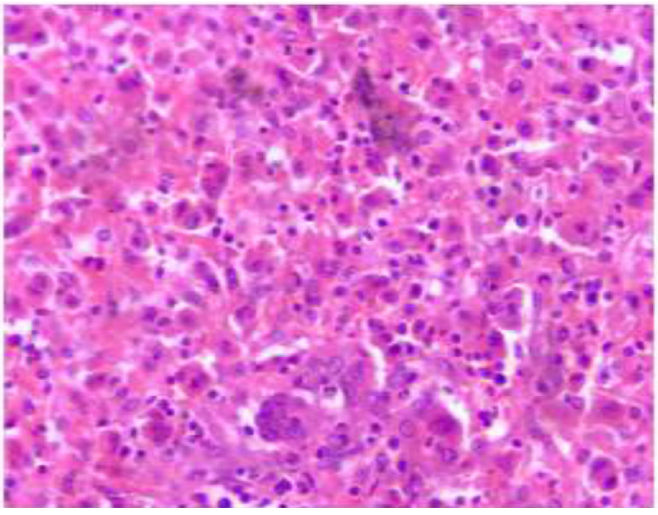
Discussion: Type I tyrosinemia (hepatorenal) is an autosomal recessive aminoacidopathy caused by a deficiency of the enzyme fumarylacetoacetate-hydrolase, generating accumulation of metabolites fumarylacetoacetate and maleylacetoacetate leading to hepatic cell damage. Its prevalence is 1:100,000, debuting 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.

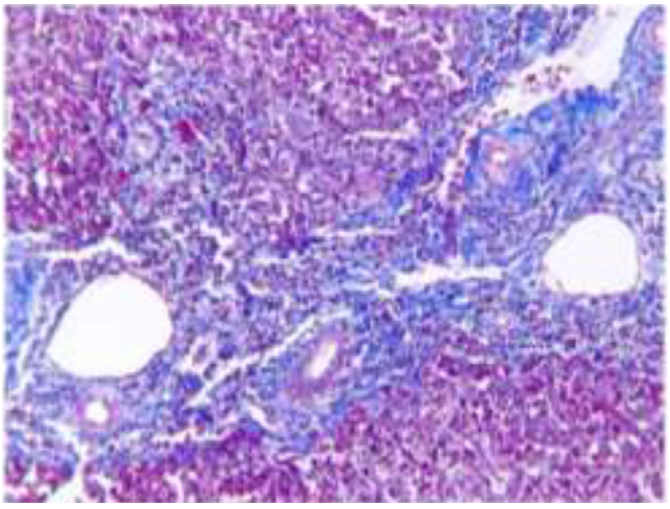
Conclusion: Tyrosinemia belongs to the group of inborn errors of metabolism; although rare, its early diagnosis can be made through newborn metabolic screening, improving its prognosis and survival, as it is unfavorable in advanced stages.

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





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Post-infantile giant cell hepatitis, management, six-year follow-up and re-transplantation, a successful case report during the pandemic

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Introduction and Objectives: HCG is a relatively common histological finding in newborns. In children, it presents with cholestasis, hyperbilirubinemia and inflammation; in the adult population, it remains poorly defined, with only 100 cases published in the literature during the last three decades.

Materials and methods: We present the case of a 20-year-old female patient with a history of herbal medicine and valproate, debuting six years ago with pain in the right hypochondrium, jaundice and fever with progression to liver failure, hepatotropic virus infections and autoimmunity were ruled out. Start liver transplant protocol with incompatible ABO organ, with induction with rituximab, immunoglobulin and basiliximab with post-surgical complications with resolved hemoperitoneum and pulmonary hemorrhage, with subsequent discharge and histopathological report of giant cell hepatitis explant, continuing immunosuppression for six years until readmission due to pruritus with liver biopsy that reported acute cellular rejection and ERCP with choledocho-choledochoanastomosis stenosis with endoscopic rehabilitation, with subsequent biochemical deterioration, starting basiliximab, steroids, plasma exchanges and MARS without improvement, subsequent ABO compatible retransplantation without complications. Currently no rejection data.

Discussion: HCGPI is a progressive, often fatal, disease process with a 50% survival rate without liver transplantation. The high mortality rate is caused by liver failure or sepsis as a result of immunosuppressive therapy.

Conclusion: HCGPI in our patient manifested acutely with rapid evolution toward liver failure. The use of valproate and herbal medicine were factors. Thanks to the possibility of using MARS as a bridge for the transplant, the result was optimal.

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Autoimmune hemolytic anemia as a paraneoplastic syndrome in hepatocarcinoma, case report

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Introduction and Objective: Hemolytic anemia can be associated with various types of solid tumors; however, in hepatocarcinoma, it is extremely rare.

Case Summary: Man 74 years old. Symptoms of two months with dyspnea, asthenia and adynamia. On physical examination, generalized pale skin and sclera, normal heart and lung area, soft, depressible abdomen, with peristalsis present, palpation of the liver edge 5 cm below the costal margin. Laboratories with leukocytes 6.1 103/Al, neutrophils #4.1, lymphocytes #1.3, HB 6.2 g/dL, HTC 16.8%, MCV 103fL, HCM 38pg, platelets 395.00 103/Al; BD 0.5 mg/dL, BI 2.80 mg/dL, BT 3.30 mg/dL, DHL 403 IU/L. Direct Coombs is performed positive dilution 1:128. FSP with anisocytosis, red blood cell agglutination, macrocytosis and macroplatelets, reticulocytes 1.48%, alpha-fetoprotein 12.7 IU/mL. Warm antibodies (IgG) attached to the erythrocyte membrane were documented. Simple and contrast-enhanced abdominopelvic tomography, with images suggestive of multifocal cellular hepatocarcinoma. Liver biopsy, which reports findings of hepatocarcinoma. Management with oral steroid drugs was initiated jointly, reversing the hematological alterations without requiring blood products.

Discussion: There are few cases in the medical literature on hematological alterations associated with solid tumor metastases. In this case, the hematological involvement of the patient was not due to metastasis but to a paraneoplastic syndrome since the first manifestation found was anemia with jaundice secondary to hemolysis.

Conclusion: The diagnosis must be reached by exclusion, ruling out other causes such as primary hematological alterations, metastases, or vascular processes.

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.