Presentation of a case of fulminant hepatitis due to Hepatitis A, in a 3 – year - old female preschool

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Abstract

Introduction.

Hepatitis A Virus is one of the most widespread infections in the world, associated with poor hygienic - sanitary conditions. It generates around 10 million cases annually, being a third of the pediatric population. Transmission is via fecal oral, enteric from person to person. It is more frequent in childhood, almost always benign and self-limited.

We find normally that in young children hepatitis A virus (HVA) infections are usually subclinical events. However, is also associated with progressive hepatic failure and even death in some patients. Fulminant hepatic failure (FHF) also called acute liver failure, is defined as a sever liver failure (Including encephalopathy or decreed drain function) that occurs with six weeks of onset of jaundice. The liver becomes damaged as a result. Other children may experience acute liver failure because of an overactive immune system (an autoimmune responded where the body attacks the liver for any reason). The early symptoms of the PFHF include: Ascites, abdominal pain, dark urine, fatigue, fever, itching, jaundice, loss of appetite, nausea, pale stool, tendency to bruise or bleed easily. The etiology of fulminant hepatic failure in children differs around the world, In the developing world and I certain communities in developed countries, hepatitis A is the most important etiological agent causing Fulminant hepatic failure in children. With the advent of liver transplantation as an effective therapy for FHF, the importance of early realia be prognostic evaluation of patients has become critical for many diseases like this who start with something apparently without control.

Etiology of Fulminant hepatic failure and patient age may be important prognostic factors. In metabolic diseases in which the liver is persistently exposed to a toxic insult liver injury is considered irreversible and transplantation is often necessary. In a acute self-limited insulted, such as hepatitis A, or pracet Bibliography amol overdose, exposure to liver injury may be limited and liver failure is potentially reversible. Fulminant hepatic failure was defined as the presence of acute liver failure in the cases in which the symptoms and biochemical and histological features were similar to those of viral hepatitis but in which no viral markers were detected and no history of toxin or drug exposure was found , the etiology of FHF was classified as non A - non B - non C hepatitis. Inborn metabolic defect was confirmed my measuring the relevant diagnostic metabolites in the urine or plasma or by tissue measurements of enzyme activity or mutation detection as appropriate. In some cases, no specific diagnosis was reached and a metabolic disease was considered likely if there was a history of parental consanguinity or symptoms - free interval, followed by clinical signs of intoxication including vomiting, lethargy, coma, lactic acidosis, hypoglycemia or hyperammonemia.

The followings definitions were used for staging of Encephalopathy hepatic. Grade 1 - Alert, mood changes, slow mentation, Grade II – Lethargic and even very often they are confused. Grade III - sporous, obeys simple commands, Grade IV - Unarousable, increased or flaccid extensive plantar response or absent reflexes. Time to the Encephalopathy was defined as time from first symptom to onset of encephalopathy. In conclusion, FHF is a rare but fatal condition. Children with severe coagulopathy, lower transaminases on admission and a prolonged duration of illness before the onset to require Liver transplantation (LT). LT is lifesaving in those with progressive disease and early recognition and referral to a specialist center for consideration for LT is essential.

Objective. Describe the evolution of fulminant Hepatitis A, treated in my environment. Private Hospital Los Cabo's Mexico.

Summary. 3-year-old female, G1, term. She was not immunized against Hepatitis A. She attended daycare.

He came to the office for headache, abdominal pain, conjunctival jaundice and coluria.

Physical examination. Weight 15 kg Height 112 cm TC 36.4 C, Sat O2 98 % Glasgow of 15 points, no hepatomegaly. With no data of bleeding on physical examination hemodialy stable. To the 5th. The day of evolution begins with Grade II encephalopathy data, so he goes to the emergency room. She is lethargic, changes in the rhythm of sleep, hallucinations, she is transferred to the Intensive Care Unit, she is managed with general measures. She evolves torpidly to Grade IV encephalopathy with data of cerebral edema, it is decided to perform a liver transplant, on the 9 th day of evolution, 24 hours after the transplant surgery, he died of kidney failure and upper gastrointestinal and pulmonary bleeding.

Usg. abdominal, shows sonographic changes related to acute liver disease

Laboratories

bh 11.2 hto 11.2 leucos 11,800 neutros 43 %, linfos 47 %, monos 10 % plaq 116,000 bt 12.3 bd 8.3 bi 4.5 ast 2015 alt 3082
IgM Hep. A positivo IgG Hep. A negativo.
bh hb 10, hto 31, leucos 15,700, neutros 49, linfos 44, plaq 139,000 AST 4263, ALT 2849 ALP612, GGT 170, BT 16, BD 11.6 BI 7.1 Prot. Tot. 8:9 Alb 3.2
na. 140, k2.7, cL102, p. 3.3
Cr.0.2 Au > 5.2 bUN 8.5 TP 31 TTP 25 INR 3.0

Discussion.

Hepatic failure is a multisystemic disease with severe impairment of liver function, INR > 1.5 or prothrombin activity less than 50 % of acute onset with or without encephalopathy. Encephalopathy, hypoglycemia and hypoalbuminemia and very high bilirubin levels suggest poor outcome.

Conclusion. Fulminant hepatitis is the most feared complication in children, from 0.1 to 5 % of hepatitis A, with mortality higher than 80 % even in patients with transplant of this organ.

Bibliography

Alonso EM. Superina RA, Whitington PF. Fulminant hepatitis and acute liver failure. In. Kelly DA, editor. Diseases of the liver and biliary system in children, first edition. Oxford: Backwell Science, 1999. p. 77 - 94
Jimenez-Gomez J, Ibarra I. Acute liver failure, En. Lopez Herce J et al. Pediatric Intensive Care Manual Third Edition. Madrid: pUBLIMED, 2009. p. 487 - 495
Rivera - Penera T, Moreno J, Skaff C, Mc Diarmid S. Vargas J, Ament ME, Delayed encephalopathy in fulminant hepatic failure in the pediatric population and the role of liver trnasplantation J Pediatr Gastroenterol Nutr 1997, 24 : 128 - 34
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Biography

Dra. Laura Elena Castañeda Ramirez is a pediatrician with over 20 years of experience. She started her career as a general physician and then she starts the specialization in pediatrician. With many years working for the public sector in Mexico, Dra. Laura Castañeda is currently working as the boss of pediatrician in Hospiten Cabos San Lucas, México.

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Notes/Comments: