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CASE REPORT

Fulminant Hepatitis A with Favourable Outcome

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Abstract

Often benign, hepatitis A can, however, be serious in non-immune people.

However, we report a case of fulminant hepatitis secondary to a hepatitis A virus (HAV) in a 44-year-old male. With no previous history, he had jaundice, asthenia and hepatic encephalopathy. The biological assessment revealed hepatocellular insufficiency, hepatic cytolysis and cholestasis. The outcome was favourable without liver transplantation.

Vaccination against hepatitis A must be widely offered to people at risk and contact of a confirmed case as well as to travelers going to countries with medium to high endemicity of hepatitis A.

Keywords

Hepatitis A, Fulminant hepatitis, Hepatic encephalopathy, Vaccination

Introduction

Hepatitis A is an acute viral infection of the liver caused by the hepatitis A virus (HAV).

Often benign, however, it can be severe in people who have never been vaccinated or previously infected with HAV, or who have chronic liver disease or an immunocompromised ground. It is closely associated with unsafe food and water, unsatisfactory sanitation conditions, poor personal hygiene, oral-anal sex, particularly among men who have sex with men (MSM), recreational drug use and travel to highly endemic areas without vaccination [1].

Our aim is to report a case of fulminant hepatitis secondary to a hepatitis A virus (HAV) with favourable outcome.

Observation

A 44-year-old man was admitted to the Infectious Diseases Department of the Setif University Hospital in April 2022, for management of fulminant hepatitis. Originally from and living in Setif, married and father of 3 children, teacher, with no history of liver disease.

The epidemiological investigation did not find risk factors such as a recent case in the entourage except a trip to Biskra (South of Algeria) one month before the appearance of signs, under precarious hygiene conditions.

No notion of taking drugs or toxic substances. The patient had never been vaccinated against HVA. The onset seems to date back to 5 days before his admission,

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marked by the onset of a flu-like syndrome, asthenia and jaundice, motivated the patient to consult on an outpatient basis where a liver testand prothrombin time (PT) were requested coming back in favor of hepatic cytolysis with a PT of 26%. The abdominal ultrasound showing a biliary sludge without dilation of the bile ducts, hence its orientation and admission.

The Clinical examination on admission found a patient in an altered general condition, eupneic, normotendu, apyretic with grade II hepatic encephalopathy: mental confusion with delirium, amnesia and asterixis. His Glasgow score was 11. Frank mucocutaneous jaundice without pruritus or haemorrhagic syndrome.

His abdomen was tender with hepatomegaly (LA at 14 cm) with discoloured stools and dark urine. Moreover, the rest of the clinical examination was without particularity.

Biological test revealed hepatic cytolysis (AST 445 IU or 11 times the upper limit of normal, ALT 1892 IU or 42 times the upper limit of normal and LDH 1250 IU/l or 5 times the upper limit of normal), hepatocellular insufficiency (Prothrombin time (PT) 26%, International Normalized Ration (INR) 2.85 and Factor V not available) and cholestasis (total bilirubin 186 IU/ml or 16.9 times normal, direct bilirubin 141 IU/ml or 10 times normal, Gamma-GT 180 IU/l or 3 times normal. Urea was 3.32 mmol/l and creatinine was 61.6 umol/l (creatinine clearance estimated at 147 ml/min (normal)). The haemogram showed haemoglobin at 16.4 g/dl, haematocrit at 46.7%, leukocytes at 5290 per microliter including 6320 neutrophils per microliter and platelets at 279,000 per microlitre. Glycemia was at 4.4 mmol/l (lower limit of normal). Disturbed lipid profile: Triglyceride at 3.79 g/l and cholesterol at 1.36 g/l. Lipasemia and amylasemia were correct. The Covid 19 PCR was negative.

A brain computed tomography (CT) scan came back without any particularity, and an abdomino-pelvic ultrasound showing discreet hepatic steatosis with diffuse aerocolia.

The management consisted of symptomatic treatment: Glucose serum, vitamin K and Duphalac.

An etiological assessment was carried out to rule out other causes of fulminant hepatitis, notably infectious (HBV, HCV) or autoimmune. This assessment revealed only positive IgM anti-HAV antibody.

The mandatory declaration form was completed and sent to the DDASS.

The clinical evolution was marked by a gradual return of consciousness to normal, with an improvement in the biological test.

The patient was discharged after a 15-day of hospitalization with regular check-ups. The convalescence was marked by intense asthenia. The assessment returned to normal after 2 months.

Discussion

According to the World Health Organization (WHO), in 2020, HAV infections were responsible for an estimated 3.4 million symptomatic cases and 44,000 deaths [2-4].

The clinical presentation of HAV infection ranges from asymptomatic infection to fulminant, life-threatening hepatitis [5].

Clinical manifestations depend on the age of the host: Less than 30% of infected young children are symptomatic, while around 80% of infected adults present with severe acute hepatitis [5].

Fulminant hepatitis is rare, with a reported incidence of 0.015-0.5% [5].

The risk of fulminant liver failure is higher in elderly patients with chronic liver disease, and liver transplantation may sometimes be necessary [5].

However, fulminant hepatitis due to HAV infection has a better spontaneous recovery rate than fulminant hepatitis of other etiologies [5].

There is no etiological treatment for viral hepatitis A [5]. Prevention is based on universal hygiene rules and vaccination.

Acute liver failure (ALF) is rare in hepatitis A. It corresponds to the loss of liver function occurring in a patient with previously healthy liver parenchyma (our patient), in contrast to acute liver failure occurring in the context of known liver disease (cirrhosis most often), usually referred to as acute-on-chronic hepatic failure.

Liver failure is said to be "severe" if the prothrombin time (PT) is less than 50%. Severe hepatitis is characterized by the coexistence of ALF and encephalopathy [6].

There are several types of classification and denomination. The time between the onset of jaundice and the appearance of encephalopathy is used to characterize fulminant forms, with a delay of less than 15 days, in the case of our patient, and subfilminanates forms, with a delay of between 15 days and 3 months [6,7]. These classifications underline the major prognostic role of the occurrence of neurological symptoms.

The Acute Liver Failure Study Group defined ALF as the combination of an international normalized ratio (INR) greater than 1.5, a prothrombin time (PT) approximately less than 50%, and the existence of encephalopathy within 26 weeks of the onset of symptoms [8], confirming the severity of our patient's clinical presentation. The prognosis is favourable (50% spontaneous survival) in hepatitis A, unlike hepatitis B where the prognosis is often poor.

The cerebral edema, which inconsistently accompanies hepatic encephalopathy, increases with

the severity of the encephalopathy and the rapidity of the onset of acute liver failure [9], is frequently seen in hepatitis B rather than hepatitis A.

The occurrence of fulminant hepatitis during the hepatitis A has already been the subject of numerous publications. In 1989, Sela, et al. published two cases of fulminant liver failure due to hepatitis A in Israel. A 24-year-old woman and a 27-year-old man who developed this complication, one of whom died [10].

A recent outbreak of severe hepatitis A virus infections, was observed at the Vienna General Hospital in Austria, from the first quarter of 2008 to the third quarter of 2018, 578 IgM + HVA were identified of which 38 patients had severe forms requiring hospitalization of 37 of them, six patients required intensive care and one had undergone liver transplantation, most were young men, notably MSM (men who have sex with men) [11].

In 2019, Reno, et al. published a case of a 63-year-old American patient with fatal fulminant hepatitis A with bleeding disorders and cerebral edema following a stay in Lima, Peru [12].

In 2023, Malik, et al. reported the case of a 32-year-old man with grade II hepatic encephalopathy co-infected with hepatitis A and E viruses, whose course was fatal due to lack of liver transplantation [13].

In our case, despite all the criteria of severity: Grade II hepatic encephalopathy, PT < 50% and INR > 1.5. The outcome was favourable, without recourse to liver transplantation, but at the cost of a long convalescence.

Conclusion

The epidemiology of hepatitis "A" epidemic has changed in recent years, this is related to the improvement of socio-economic conditions. It affects adults more and more, and is often accompanied by signs of severity (ALF).

This is why it is so important to emphasize the importance of implementing appropriate prevention and control measures, in particular vaccination, against hepatitis A, which should be widely offered to people at risk: Those in contact with a confirmed case or having oral-anal sex, as well as to travelers to countries with medium to high hepatitis A endemicity. Compliance with hygiene and sanitation rules, while avoiding the consumption of contaminated water and food.

Conflict of Interest

The authors declare no conflicts of interest.

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