Hepatitis B Virus Induced Hepatitis Simultaneously Complicated By Hepatic Encephalopathy And Acute Glomerulonephrirtis In A Nigerian Child

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Citation

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Abstract

We report the case of a 3-year-old male Nigerian child who developed hepatic encephalopathy and acute glomerulonephritis following Hepatitis B virus induced hepatitis. The unusual presentation is discussed.

INTRODUCTION

Infectious hepatitis in children is usually due to Hepatitis A virus and occasionally Hepatitis B virus [HBV]. [1] Infection of the liver with HBV virus can be followed by acute complications such as papular acrodermatitis, acute glomerulonephritis and hepatic encephalopathy. [2] However, these complications are uncommon. Simultaneous complications arising from HBV associated hepatitis are much more rare. We report the case of a 3-year-old boy who developed clinical features consistent with hepatic encephalopathy and acute glomerulonephritis from HBV infection of the liver. To our knowledge this is the first reported case of such a disease. The aim of this article is to draw the attention of practicing physicians to the unusual features of this case with simultaneous acute complications of encephalopathy and glomerulonephritis.

CASE REPORT

OT a 3-year-old boy presented on the 3rd of April 2004 at the children emergency unit of the Ladoke Akintola University of Technology Teaching Hospital, Osogbo, Western Nigeria. The presenting complaints were those of generalized body weakness, jaundice and passage of dark coloured urine, all noticed 2 days before presentation. He had vomited, fed poorly and experienced 2 episodes of generalized tonic clonic convulsions on the day of presentation. He had become unarousable after the convulsions and this had prompted the rushing of the child to Ladoke Akintola University of Technology Teaching Hospital. There was no history of sore throat or skin infections. However, two months prior to the presenting illness the patient had been transfused for severe anaemia due to malaria at the State Specialist Hospital, Osogbo. At that time the patient had responded well to the transfusion and anti-malaria drugs and was discharged home after 3 days of hospitalisation. The blood transfused into him, was donated by his father, screened and cross-matched at the Osun State Government central haematology laboratory.

Immunizations against HBV infections were not received because the vaccines were not available at the times of visit, by the patient to the government owned children's welfare health unit.

At presentation the patient was ill looking, unconscious, moderately jaundiced and pale. He was afebrile with a temperature of 36.5°c. He weighed 15.5kg. There were no significantly enlarged peripheral lymph nodes. The abdomen was full and the liver, was enlarged to 6 cm below the right costal margin. It was tender and soft and the surface smooth. Central nervous system revealed a deeply comatose child. There were no signs of meningeal irritation and both the tone and the deep tendon reflexes were normal. All other systems showed no abnormality on examination.

A diagnosis of hepatic encephalopathy was made and the investigations ordered include full blood count, random blood sugar, liver function tests, blood culture, G-6-PD assay, cerebrospinal fluid for proteins, sugar and microbiology and HbsAg. The father was also tested for HbsAg. The pack cell volume was 34% and the HbsAg was

positive in the patient and father. All other results were within normal range, apart from the total and conjugated bilirubin, which were 111 and 86umol/l respectively. The liver transaminases were not determined because of some technical handicap experienced by the hospital laboratory. Calories were there after supplied by intravenous glucose with a de-emphasis on protein diet. The patient was also put on cimetidine. The recovery was judged to be good after sterilizing the gut with oral neomycin and enema and the patient regained full consciousness 5 days after hospitalisation.

On the 10^{th} day of admission the patient had improved markedly. He was moving about, eating better, his jaundice had virtually cleared and urine colour was less dark; and he was to be discharged the next day. However, on that next day fixed for discharge he was noticed to have developed generalized oedema and some ascites. He had also passed urine, which was darker than before. The blood pressure and the urinary volume were normal. There was no dysuria. A diagnosis of acute glomerulonephritis was therefore made.

Results of the investigation carried out showed that full blood count were normal with a repeat haematocrit still 34%. The blood chemistry report in mmol/l was, urea 1.1, bicarbonate 24, sodium 133, potassium 3.5 and creatinine 44umol/l. Urinalysis report was normal, but urine microscopy revealed 5-10 red blood cells per high power field. Ultrasound studies showed enlarged liver and kidneys as well as moderate ascites and small quantity of pleural effusion.

The patient was thereafter managed with fluid restriction, strict output and input chart, low salt and protein intake, intravenous Frusemide 15 mg every 12 hours and oral Cefuroxime. The oedema gradually resolved and the patient was discharged a week after to follow up in the clinic. At discharge the heamaturia had disappeared but the HBsAg status was still positive.

DISCUSSION

Hepatitis B virus induced hepatitis is dreaded because of the associated chronic sequelae such as chronic hepatitis, cirrhosis and primary liver cell cancer. Acute complications of HBV associated hepatitis are not as common as the chronic complications. Hepatitis B Virus can also rarely cause acute glomerulonephritis. [3] This complication is actually a hypersensitivity reaction to the HBV antigen at the surface of the kidney glomerulus. [4] It is known to present 10 days to 2 weeks after the initial HBV infection. Therefore with the oedema occurring 13 days from the onset of jaundice, the kidney and the liver infections probably took place simultaneously.

Encephalopathy in patients with HBV hepatitis usually arises as a result of hepatic failure and the associated complications of cerebral oedema and sepsis and the outcome may sometimes be fatal. [5,6] Possible differential diagnoses in this patient with hepatic encephalopathy include cerebral malaria, meningitis and septicemia. Differentials of acute glomerulonephritis include hepatorenal syndrome, and heamoglobinuria, from sepsis or malaria. Making a prompt diagnosis can be difficult in resource poor settings where there are many constraints which hamper proper investigations, as happened in this patient. A good clinical acumen with knowledge of the natural history of diseases is indispensable. The good outcome in this case could probably be linked to the prompt diagnosis and good management. All hepatotoxic drugs such as chloramphenicol, which may have been indicated in the management of the differentials, were avoided from the onset.

It is unfortunate however, that a 3-year-old child should experience these complications and also be subjected to the risk of chronic liver and kidney disease. Effective vaccines for HBV have been available since the year 1982. [6] This vaccine can also be obtained in some places in the country, although, they are often unavailable in many hospitals. That our patient was transfused with supposedly HBV negative blood, donated by his father raises serious questions for the blood screening personnel and materials. It also has public health implications, because the father was screened at the State Health central laboratory. Was the father in the window period? Or was the screening improperly conducted? What is the sensitivity of the test kit and method used?

The cost of managing hepatitis B virus hepatitis infection is enormous in terms of the morbidity and mortality. The socio economic implications can obviously not be fully quantified. It is important that hepatitis B infection be prevented everywhere, including and especially in resource poor societies. The government should ensure that potent vaccines are available at the public hospitals. Responsible parenting will ensure that children receive immunization when due. All blood and blood products should be properly screened before administration.

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