

Case report

Hepatocellular Carcinoma in Eleven Year Old Child

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ABSTRACT**Background:**

Hepatocellular carcinoma is a rare tumour in children, mostly associated with hepatitis B virus (HBV) infection, up to the best of our knowledge this is the first documented case in this age group.

Case Report:

We report a case of eleven years old boy who was admitted to Saddam Center for Gastrointestinal and Liver Diseases because of pallor and hepatosplenomegaly. On investigation he was found to have HBV infection chronic liver disease. His liver

biopsy showed established liver cirrhosis with infiltration of hepatocellular carcinoma.

Conclusion:

Chronic liver disease in children can be silent and present in late stage with advanced liver damage and or hepatocellular carcinoma.

Keyword: hepatocellular Carcinoma, children.

Introduction

Hepatocellular carcinoma is the seventh most common cancer in men worldwide⁽¹⁾. Men are generally more susceptible than women to hepatocellular carcinoma (HCC) with a male to female ratio equals to 3:1. The incidence of hepatocellular carcinoma generally rises with age, in some Sub-Saharan countries. However, there is a definite shift of patients toward younger age with a mean age of 33 years, about half of those patients are younger than 30 years. Hepatocellular carcinoma is rare in children⁽²⁾.

In chronic HBV infection acquired during birth or early childhood, which is the type most commonly seen in the Asian population, there is a prolonged phase of immunotolerance. The immune clearance phase is characterized by multiple acute exacerbations preceded by elevations in serum HBV DNA levels, HbeAg concentration and

HbeAg/ anti-Hbe immune complexes. The development of cirrhosis occurs more frequently in patients with episodes of decompensation and with repeated severe acute exacerbations. However, progression to cirrhosis can be relatively silent and can occur in children⁽³⁾.

On gross appearance, there are 3 forms of hepatocellular carcinoma, the nodular, massive or diffuse. The nodular variety account for 75% of hepatocellular carcinoma and usually coexist with cirrhosis, the massive type is most common in non cirrhotic liver in younger patient. The diffusely infiltrating variety is rare. On gross appearance biopsy specimens, well differentiated tumors are light brown, whereas anaplastic tumor are yellow-white or gray. The portal vein and its branches are infiltrated by tumor in as many as 70% of the cases. The hepatic veins and bile ducts are less often

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involved. Microscopically hepatocellular carcinoma can have well differentiated appearance, moderately differentiated or undifferentiated.

The fibrolamellar hepatocellular carcinoma occurs typically in young patients. It does not secrete α - feto protein, has equal sex distribution, it is not caused by hepatitis B or C chronic virus infection and occurs in non cirrhotic liver.

Regarding the etiology of hepatocellular carcinoma, chronic hepatitis B virus is the cause in 80% of cases. Persistent hepatitis B virus infection antedates the development of hepatocellular carcinoma by several many years, early infection carries a considerably greater risk.

Other risk factors for hepatocellular carcinoma are chronic hepatitis C virus infection, Aflatoxin ingestion and liver cirrhosis of other causes.

Hb.	= 10 g/dl.	PT	= 15" (12")
WBC	= 4000 cell/cc.	PTT	= 39" (25-32")
Platelets	= 150000 cell/cc.	SGOT	= 70 IU
ESR	= 15 mm/h	SGPT	= 43 IU
Hypochromic blood film		Alk. Phosphatase	= 20 KAU
		T.S.B.	= 1.2 mg/dl

His viral screen was positive for HbsAg, HBc IgM (twice), negative for HCV and HDV (his family viral screen including mother was negative).

An abdominal ultrasound examination showed hepatomegaly of coarse texture, huge splenomegaly, the portal vein was totally thrombosed while the hepatic veins were patent.

Upper gastrointestinal tract endoscopy showed early grade of esophageal varices. He was put on propranolol and nitrate as prophylactic measure.

A liver biopsy arranged under local anesthesia, macroscopically showed white-yellow fragmented pieces and microscopically showed area of thick fibrous band with regenerative nodules and foci of hepatocellular carcinoma.

One month later, the patient started to develop transudative ascites, progressive elevation of liver enzymes and jaundice and more increase in the liver size with bruit on the liver. He also started to have hematemesis and melena from the grossly enlarged esophageal varices and he was started on sclerotherapy session. The patient died a month later from massive esophageal variceal bleeding.

Discussion

Hepatocellular carcinoma is a highly malignant tumor, seen primarily in older pediatric

aflatoxin ingestion and liver cirrhosis of other causes.

Case Report:

Eleven years old male was admitted to Saddam Center for Gastrointestinal and Liver Diseases in Baghdad in August 2001 with 4 months history of pallor. No previous history of liver disease. Unremarkable past medical history and negative family history of the same condition. On examination he was pale, thin, not jaundiced, his abdominal examination showed hepatomegaly of 16 cm span with nodular surface. Splenomegaly of 8 cm below the costal margin, dilated abdominal wall veins and no leg edema. His investigations on admission were:

Hb. = 10 g/dl. PT = 15" (12") WBC = 4000 cell/cc. PTT = 39" (25-32")
Platelets = 150000 cell/cc. SGOT = 70 IU

Discussion

Hepatocellular carcinoma is a highly malignant tumor, seen primarily in older pediatric patients. It is the third most frequent liver tumor occurs primarily in children older than 10 years of age⁽⁴⁾. Cheah et al. described an exposure time of only 3 years in a 10 years old child who developed hepatocellular carcinoma after blood transfusion⁽⁵⁾, while Chang found that severe liver damage that include cirrhosis and hepatocellular carcinoma may develop insidiously for 2-7 years after infection⁽⁶⁾.

We reported this case because hepatocellular carcinoma rarely diagnosed in Iraqi children. This child had no apparent risk factor for gaining HBV infection (such as blood transfusion or perinatal infection) and probably he got his infection through other less common routes of infection from the community. He developed severe rapid course of disease with rapid deterioration and death. Up to the best of our knowledge. This is the first documented hepatocellular carcinoma following HBV infection in this age group in Iraq.

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