Hepatitis B and Delta Viruses in Fulminant Hepatitis

Priti Desai, D D Banker
Sir Harkishandas Narottandas Medical Research Society, Raja Ramnath Ray Road, Bombay 400 004

Abstract
From June 1985 to 1989, we studied 39 cases with fulminant viral hepatitis. These included 32 cases due to hepatitis B, of whom 27 died. Twenty of the 32 cases were positive for delta antibody, and all of them died. Four cases who died were negative for IgM anti-HAV, HBSAg and IgM anti-HBe and were classified as NANNB. Thus, a total of 31 patients died. Hepatitis B and delta virus infection were the major risk factors for fulminant hepatitis and eventual death.

Key words: Viral hepatitis.

Introduction
Fulminant hepatitis is the most severe form of acute viral hepatitis and may rapidly result in death. The progress may be so rapid that jaundice may be slight. Prognosis is related to the depth of coma; recovery is rare.

Fulminant viral hepatitis is usually due to hepatitis B (HBV) or non A non B (HANAB) viruses. It is much less common with hepatitis A virus (HAV). Investigators from different parts of the world have reported varied incidence and etiology of fulminant viral hepatitis. Thus, HBV has been found responsible in 40% to 60% of cases or in as low as 12.5% while HANAB incidence has been variously reported as less than 1%, 12.5, 22.5% or as high as 80.9%. When associated with hepatitis delta virus (HDV), HBV is more likely to result in fulminant hepatitis. HAV incidence in fulminant viral hepatitis has varied from 1% to 6.9%. We therefore looked at our patients with fulminant hepatitis to find the frequency of different viral markers in these cases.

Material and Methods
From June 1985-89, 39 patients (32 males, 7 females; aged 18 to 62 years, mean 30) with fulminant hepatitis were studied from Sir HN and Kasturba Hospitals from central Bombay. Detailed clinical notes were recorded and serological tests performed; serum bilirubin, SGOT, SGPT and prothrombin time were estimated. The sera were tested for IgM anti-HAV, HBSAg and IgM anti-HBe (ELISA) with Abbott kits. The 32 HBSAg positive sera were tested for the presence of delta antibody, 20 by RIA and 12 by ELISA (Abbott).

Results
Serologically, all 39 patients showed bilirubin levels of 5 mg/dl (86 µmol/L) or more, with an increase in aminotransferases more than twice the upper limit of normal. In two cases bilirubin was as high as 45 mg/dl (770 µmol/L) and SGPT was 1500 U/L (25 µkat/L). Thirty seven patients had prolonged prothrombin time. All the 39 sera were IgM anti-HAV negative. Thirty two of the 39 sera were HBSAg and IgM anti-HBe positive, indicating that these were acute HBV cases. Twenty and nine of these 32 patients showed the presence of delta antibody and antigen respectively. The remaining seven cases showing absence of all parameters were classified as HANAB.

Thirty one of the 39 cases proved fatal within 10 days; of these, 27 were due to HBV and 4 due to HANAB. All 20 delta virus positive patients died; 7 of 12 delta negative HBV patients died (chi square with Yates’s correction: 6.96, P<0.01). Two pregnant females with fulminant hepatitis died. Five renal failure patients requiring repeated hemodialysis who developed acute HBV hepatitis had a fulminant course and died.

Discussion
Fulminant hepatitis associated with delta infection was described in 1962 in Italy and the rest of Europe, where 59% of patients with fulminant HBV hepatitis had concomitant delta infection; approximately half of these patients had delta-virus-hepatitis B coinfection and half were HBV carriers with delta virus superinfection. Craig et al12 found that the prevalence of delta markers in fulminant viral hepatitis was higher (33-8%) than in the non-fulminant group (4-2%). Saracco et al16 in their recent worldwide study of 377 cases with fulminant hepatitis found that 35% were caused by coinfection or superinfection of HBV with hepatitis delta virus.

It is significant that all 20 HBV patients with concomitant HDV infection died while 5 of the 12 delta negative HBV patients survived. These findings highlight the importance of combined HBV and HDV infections in fulminant hepatitis. Delta virus has earlier been reported as endemic in Western17 and Northern18,19 India. Bal et al16 however did not find delta anti-
bodies in their series of fulminant hepatitis cases reported from Bombay.

This study demonstrates the role of delta infection in fulminant HBV infection. Hepatitis B and delta virus infection were the major risk factors for fulminant hepatitis and eventual death.

References
18 Pal SK. The endemicity of delta agent infection in and around Chandigarh, Northern India. *Virus Information Exchange Newsletter for South East Asia and the Western Pacific* 1985; 2: 56.

210 INDIAN J GASTROENTEROL Vol 9 No 3 JULY 1990 FULMINANT HEPATITIS RISK FACTORS—DESAI AND BANKER