

Fulminant hepatitis in a tropical population: clinical course, cause, and early predict

Acharya, KS; Dasarathy, S; Kumer, TL; Sushma, S; Prasanna, KS; Tandon, A; Sreenivas, V; Nijhawan, S; Panda, SK; Nanda, SK; Irshad, M; Joshi, YK; Duttagupta, S; Tandon, RK; Tandon, BN

Date: 1996

Abstract:

The profiles of patients with fulminant hepatic failure (FHF) from developing countries have not been reported earlier. The current study was conducted prospectively, at a single tertiary care center in India, to document the demographic and clinical characteristics, natural course, and causative profile of patients with FHF as well as to define simple prognostic markers in these patients. Four hundred twenty-three consecutive patients with FHF admitted from January 1987 to June 1993 were included in the study. Each patient's serum was tested for various hepatotropic viruses. Univariate Cox's regression for 28 variables, multivariate Cox's proportional hazard regression, stepwise logistic regression, and Kaplan-Meier survival analysis were done to identify independent predictors of outcome at admission. All patients presented with encephalopathy within 4 weeks of onset of symptoms. Hepatotropic viruses were the likely cause in most of these patients. Hepatitis A (HAV), hepatitis B (HBV), hepatitis D (HDV) viruses, and antitubercular drugs could be implicated as the cause of FHF in 1.7% (n= 7), 28% (n= 117), 3.8% (n= 16), and 4.5% (n= 19) patients, respectively. In the remaining 62% (n= 264) of patients the serological evidence of HAV, HBV, or HDV infection was lacking, and none of them had ingested hepatotoxins. FHF was presumed to be caused by non-A, non-B virus(es) infection. Sera of 50 patients from the latter group were tested for hepatitis E virus (HEV) RNA and HCV RNA. In 31 (62%), HEV could be implicated as the causative agent, and isolated HCV RNA could be detected in 7 (19%). Two hundred eighty eight (66%) patients died. Approximately 75% of those who died did so within 72 hours of hospitalisation. One quarter of the female patients with FHF were pregnant. Mortality among pregnant females, nonpregnant females, and male patients with FHF was similar ($P > .1$). Univariate analysis showed that age, size of the liver assessed by percussion, grade of coma, presence of clinical features of cerebral edema, presence of infection, serum bilirubin, and prothrombin time prolongation over controls at admission were related to survival ($P < .01$). The rapidity of onset of encephalopathy and cause of FHF did not influence the outcome. Cox's proportional hazard regression showed age ≥ 40 years, presence of cerebral edema, serum bilirubin ≥ 15 mg/dL, and prothrombin time prolongation of 25 seconds or more over controls were independent predictors of outcome. Ninety-three percent of the patients with three or more of the above prognostic markers died. The sensitivity, specificity, positive predictive value, and the negative predictive value of the presence of three or more of these prognostic factors for mortality was 93%, 80%, 86%, and 89.5%, respectively, with a diagnostic accuracy of 87.3%. We conclude that most of our patients

with FHF might have been caused by hepatotropic viral infection, and non-A, non-B virus(es) seems to be the dominant hepatotropic viral infection among these patients. They presented with encephalopathy within 4 weeks of the onset of symptoms. Pregnancy, cause, and rapidity of onset of encephalopathy did not influence survival. The prognostic model developed in the current study is simple and can be performed at admission.