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Virus B multiplication in fulminant hepatitis

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Hepatitis B virus (HBV) is one of the most important etiological agents of fulminant hepatitis. Pathogenesis of sub-massive hepatic necrosis in fulminenat hepatitis is not known. Bernuau et al,[1] 1986 demonstrated that the continued spread of HBV infection within the liver increases the destroyed hepatic mass and decreases the residual functional hepatocyte mass which leads to acute liver failure and fatal outcome in fulminant hepatitis. The mechanism of the spread of HBV infection in the undestroyed mass is not known. In the present study the presence of HBeAg and HBV specific DNA-polymerase activity - regarded as markers of HBV multiplication were studied in 40 patients of fulminant hepatitis B within one to five days of the onset of hepatic encephalopathy. Etiology of acute HBV infection was established on following criteria: (a) a negative history for intake of hepatotoxic substances (b) positive IgM anti-HBc and (c) negative IgM anti-HAV (both tested by diagnostic EIA-kits from Abbott Labs., U.S.A. Post mortem liver biopsy showed sub-massive and/or massive necrosis. Blood samples were collected from 5th to 15th day of illness. The sera was stored at $-70^{o}C$ and tested for DNA-polymerase by modified technique of Fang et al[2], and HBe markers were done with Abbott's EIA kit.

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HBeAg and DNA -P were detected in 3 (7%) and 21(51%) of the patients respectively. Only 3 (7%) were positive of anti-HBs. 17 patients were negative for all HBV markers i.e, HBeAg, anti-HBs and DNA-P except IgM anti-HBc. Present findings indicate continuation of HBV multiplication in about half of the patients of fulminant B hepatitis and suggest that virus replication inhibiting agents may be beneficial to these patients.

REFERENCES

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