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Short Report

Acute hepatitis E infection during the 1988 floods in Khartoum, Sudan

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Following catastrophic floods in August 1988, physicians in Khartoum and Omdurman, Sudan, observed an outbreak of acute hepatitis. During a period of 2 weeks in October 1988 we conducted a seroepidemiological study of icteric patients presenting to an acute-care clinic at the Omdurman Military Hospital. After informed consent was obtained, a standardized questionnaire was administered to each person and a venous blood sample was obtained. Acute and convalescent sera were tested for immunoglobulin (Ig) M and IgG antibody to hepatitis E virus (anti-HEV) using a Western blot assay (He *et al.*, 1993). Specimens were also tested for IgM antibody to the hepatitis B core protein and IgM antibody to hepatitis A virus (anti-HAV) (Abbott).

Fifty-five patients participated in the study, with 16 (29%) returning for follow-up. The average age of patients was 22 years (range 3–48 years), and 47 (85%) were male. The patients had been jaundiced for an average of 9 d (range 1–30 d when the study began). Thirty-two patients (58%) had IgM anti-HEV detected in the acute serum specimens; IgG anti-HEV was also detected in 29 of these 32 specimens. Paired sera were available from 10 of the 32 cases of acute hepatitis E infection. In 3 of the 10 convalescent sera, IgM antibody was no longer detectable. IgG antibody to hepatitis A persisted in all 10, however. Of the remaining 23 patients in whom no IgM anti-HEV was detected, paired sera were available from 6 patients and no IgG anti-HEV seroconversion was observed. There were 3 cases of acute hepatitis B and 3 of acute hepatitis A. Thus, there were 17 study subjects with no serological marker of acute hepatitis A, B or E; however, 10 of these 17 patients had IgG anti-HEV in the acute serum specimen. When the 17 patients with no serological marker of acute hepatitis A, B or E were compared with the 32 cases of acute hepatitis E, there was no significant difference in their age, number of days of jaundice, prior history of jaundice, source of drinking water, symptoms, or physical examination results.

The findings of this study indicate that hepatitis E infection was an important cause of epidemic jaundice following the floods of 1988 in the Khartoum province of Sudan. Other studies have implicated hepatitis E infection as an important cause of epidemic and sporadic jaundice in this region (CENTERS FOR DISEASE CONTROL, 1987; HYAMS *et al.*, 1992a, 1992b). The serological profiles of hepatitis E cases found in this study suggest that the IgG antibody response appears early in this infection and that the IgM antibody response may quickly wane.

In a prior study in Khartoum, acute sporadic hepatitis E infection was found to be frequent among children (average age 6.5 years) (HYAMS *et al.*, 1992b). In this study, hepatitis E infection was found commonly among much older study subjects. Together, these findings suggest that, while this population may be frequently exposed to hepatitis E virus at an early age, symptomatic infection in adults is still common. Longitudinal studies will be needed to elucidate further whether a protective immune response to hepatitis E virus occurs after acute infection in childhood.

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References

- Centers for Disease Control (1987). Enterically transmitted non-A, non-B hepatitis in East Africa. *Mortality and Morbidity Weekly Report*, 36, 241–244.
- He, J., Tam, A. W., Yarbough, P. O., Reyes, G. R. & Carl, M. (1993). Expression and diagnostic utility of hepatitis E virus putative structural proteins expressed in baculovirus. *Journal of Clinical Microbiology*, 31, 2167–2172.
- Hyams, K. C., Purdy, M. A., Manjit, K., McCarthy, M. C., Hussarin, M. A. M., El-Tigani, A., Krawczynski, K., Bradley, D. W. & Carl, M. (1992a). Acute sporadic hepatitis E in Sudanese children: analysis based on a new Western blot assay. *Journal of Infectious Diseases*, 165, 1001–1005.
- Hyams, K. C., McCarthy, M. C., Manjit, K., Purdy, M. A., Bradley, D. W., Mansour, M. M., Gray, S., Watts, D. M. & Carl, M. (1992b). Acute sporadic hepatitis E in children living in Cairo, Egypt. *Journal of Medical Virology*, 37, 274–277.

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