Hepatitis B in Families

To the Editor: The paper by Leichtner and co-workers (1) on intensive studies of hepatitis B infection in two families concluded that "horizontal, nonparenteral transmission of hepatitis B, although infrequently recognized, may readily occur in certain American families." Extrapolations from this study may be limited because the index family included four chronic hepatitis B surface antigen (HBsAg) carriers, was composed largely of recent Haitian immigrants, and lived in extremely crowded conditions. Subsequent Annals papers have emphasized homosexual behavior, immunosuppression, parenteral exposure, or contact with clinical specimens in the spread of hepatitis B. Nevertheless, considerable transmission of hepatitis B in the United States occurs in ordinary persons outside the medical setting. In most cases, the major determinant of infection probably is not the presence of a carrier in the family unit.

We have previously reported studies that directly show the importance of the spread of hepatitis B by contact with acutely infected patients. Hepatitis B virus is a common cause of acute icteric hepatitis in Panama City, Panama, although the prevalence of HBsAg carriers is only about 1% (2). When we prospectively studied households contacts of 20 adult patients with hepatitis B, four of 41 children and one of 60 adults developed anti-HBsAg without clinical disease. Two additional adults had mild anicteric hepatitis B (3). Two infected adults were from the 10 conjugal pairs studied. More than 50% of the subjects came from families living at least at the level of working class urban families in this country. Two seroconversions in children and one anicteric case came from a distinctly upper-class family.

Additional evidence for nonparenteral spread of hepatitis B virus in Panama came from studies of isolated rural Indian tribes. Guaymi and Choco Indians living along streams and rivers on the mainland had high prevalence of infection (32% to 80% of adults), but only the Guaymi had a high prevalence of HBsAg carriers (3% to 8%). The evolution of chronic antigenemia was thought to be genetically influenced because of its tribal distribution and clustering in families (4). The role of environmental factors in transmission was clear from studies of island-dwelling Cuna Indians whose anti-HBsAg prevalence was only 10% in adults. Cuna Indians with similar customs living along the same mainland rivers inhabited by the Choco Indians had antibody prevalence rates of 55% in adults. Hepatitis B antibody rates in different Indian groups paralleled the rates for other viruses transmitted by contact or fecal contamination (reovirus and coxsackievirus) (5). There was no evidence of parenteral transmission by tribal practices, nor any indication of vertical infection.

Our studies of the urban family show that persons in contact with patients with acute hepatitis B may become infected during the course of daily activities under conditions that prevail in developed countries. The Indian studies confirm the potential importance of horizontal transmission of hepatitis B by routes of infection that do not entail a defined breach of epithelial barriers and depend on environmental conditions yet to be identified.

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References
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We have previously reported studies that directly show the importance of the spread of hepatitis B by contact with acutely infected patients. Hepatitis B virus is a common cause of acute icteric hepatitis in Panama City, Panama, although the prevalence of HBsAg carriers is only about 1% (2). When we prospectively studied household contacts of 20 adult patients with hepatitis B, four of 41 children and one of 60 adults developed anti-HBsAg without clinical disease. Two additional adults had mild anicteric hepatitis B (3). Two infected adults were from the 10 conjugal pairs studied. More than 50% of the subjects came from families living at least at the level of working class urban families in this country. Two seroconversions in children and one anicteric case came from a distinctly upper-class family.

Additional evidence for nonparenteral spread of hepatitis B virus in Panama came from studies of isolated rural Indian tribes. Guaymi and Choco Indians living along streams and rivers on the mainland had high prevalence of infection (32% to 58% of adults), but only the Guaymi had a high prevalence of HBsAg carriers (3% to 8%). The evolution of chronic antigenemia was thought to be genetically influenced because of its distribution in the country and clustering in families (4). The role of environmental factors in transmission was clear from studies of island-dwelling Cuna Indians whose anti-HBsAg prevalence was only 10% in adults. Cuna Indians with similar customs living along the same mainland rivers inhabited by the Choco Indians had antibody prevalence rates of 55% in adults. Hepatitis B antibody rates in different Indian groups paralleled the rates for other viruses transmitted by contact or fecal contamination (reoviruses and coxsackieviruses) (5). There was no evidence of parenteral transmission by tribal practices, nor any indication of vertical infection.

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Claims for Moxalactam

To the Editor: Advertisements have begun to appear in your journal and in other medical journals for moxalactam (Moxam; Eli Lilly and Company, Indianapolis, Indiana), a new “third-generation cephalosporin.” An unreferenced statement in these advertisements claims that “Moxam alone was more effective than the combination of gentamicin plus clindamycin” in the treatment of surgical infections. However, if one examines the study on which this claim is based (1), somewhat different conclusions are apparent. In a randomized study 156 patients received either moxalactam or gentamicin plus clindamycin, and “elimination of sepsis was achieved in only 57% of those given gentamicin-clindamycin, as compared to 84% of those receiving moxalactam.” Although the authors do not present a statistical analysis of this difference, it is significant (p < 0.005 by chi-square test). However, the combination therapy used in the study was inadequate at best. The authors state that patients received only 5 mg/kg body weight every 8 h or 1050 mg/d of clindamycin for a 70-kg person, instead of 600 mg every 6 h (2400 mg/d) that is commonly used. Perhaps more importantly, patients received gentamicin in a dose of 1 mg/kg body weight every 8 h with no adjustment for age, sex, or renal function. Gentamicin serum levels were apparently not obtained, although dosage adjustments based on such levels are needed to avoid under- or overdosing most patients with gentamicin (2, 3).
A more appropriate conclusion from the study would be