# Letters to the Editor

## Hepatitis B Infection in a Vaccinated Renal Dialysis Staff Worker

**To the Editor:**

Published studies of double-blind trials of different hepatitis B vaccines have not reported any instances of subsequent hepatitis B infection with hepatitis B surface antigenemia ($HB_sAg$) and elevated levels of transaminases among healthy, presumed immunocompetent vaccine recipients who had developed an optimal antibody response (ie, >10 S/N units by radioimmunoassay) to the surface antigen (anti-$HB_s$).[1-7] However, data from follow-up of vaccinated homosexual males have revealed that $HB_sAg$ developed and transaminases became elevated in some whose maximum postvaccination anti-$HB_s$ response was suboptimal (ie, 2.1 to <10 S/N units).[7,8] We report here a case of subclinical hepatitis B with $HB_sAg$ and elevated transaminase levels in a vaccinated dialysis staff technician.

The technician was a previously healthy, non-obese 31-year-old woman who began work at a regional dialysis unit in 1980. She was negative for $HB_sAg$ and anti-$HB_s$ on a serial testing for the 15 months before she received hepatitis B vaccine (HEPTAVAX B,* Merck Sharp and Dohme, West Point, PA) in November 1982. The vaccine was given in the buttock as 20 μg injections at 0, 1, and 7 months. Anti-$HB_s$ was detected 6 months after the first dose and was still evident at 9 and 12 months. The actual unit values were unavailable from the reference laboratory, and therefore it could not be determined if she was an optimal responder. In routine testing done 21 months after the first dose, anti-$HB_s$ was no longer detectable (ie, <2.1 S/N units) and $HB_sAg$ had developed. The technician had no fatigue, malaise, arthralgia, jaundice, or abdominal pain. At work she had rotated through the separate unit for dialysis patients who are known chronic carriers of hepatitis B virus (HBV), but she was unaware of percutaneous or mucous membrane exposure to blood or secretions. Moreover, she denied non-work-related exposure to any of the known risk factors for HBV infection. Her physical examination was and continues to be normal. Serum transaminase levels were elevated for 3 months (peak SGPT level, 688 mIU/ml) before returning to normal. $HB_sAg$ persisted for 5 months before disappearing coincidentally with the return of anti-$HB_s$ (4 S/N units).

Although vaccine-induced anti-HBs is protective against HBV infection, it is unclear what minimum level is necessary for protection and what the duration of protection might be. Recent studies suggest that peak postvaccination anti-$HB_s$ levels are lower in individuals injected in the buttock compared to individuals injected in the arm.[9] It is known that the peak level of antibody following vaccination correlates with the duration of detectable antibody.[7] Vaccinated individuals might be protected even after their anti-HBs has dropped to undetectable levels, since on exposure to HBV an anamnestic response may prevent infection or limit it to seroconversion to hepatitis B core antigen only. However, such an anamnestic response may be lacking or insufficient to prevent HBV infection in those with a suboptimal anti-$HB_s$ vaccine response.[8]

The duration of protection can be determined only by observing when breakthrough infections such as this begin to occur. The paucity of known similar cases suggests that breakthrough infections with $HB_sAg$ and elevated transaminase levels are uncommon or unrecognized. Follow-up of several vaccine trial cohorts is underway to examine the relation between degree of response and duration of protection.[7,8] Guidelines for the use of booster injections should await the results of these studies.

*Use of trade name is for identification only and does not indicate endorsement by the Public Health Service or the U.S. Department of Health and Human Services.*

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