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SUPPLEMENT

Perspectives on the Control of Viral Hepatitis, Type B

A joint statement by the Committee on Viral Hepatitis, Division of Medical Sciences, National Academy of Sciences-National Research Council, and the Public Health Service Advisory Committee on Immunization Practices

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE / PUBLIC HEALTH SERVICE

Perspectives on the Control of Viral Hepatitis, Type B

PREAMBLE

In 1972 the Committee on Viral Hepatitis of the Division of Medical Sciences, National Academy of Sciences-National Research Council, published a statement on the public health implications of hepatitis B surface antigen in human serum.[1] The statement was revised and updated in 1974.[2] These commentaries dealt with the significance of hepatitis B surface antigen and suggested ways to manage hepatitis B patients and other persons found to have this antigen in their blood.

Recent data on the diagnosis, epidemiology, and management of hepatitis B have helped clarify its complex natural history. Important questions remain to be answered, but there is sufficient new information to warrant thorough review of the subject and interpretation of its health significance.

Traditional prevention and control of hepatitis A involve good hygienic practices and passive prophylaxis (immune serum globulin, human—ISG) for close personal contacts of hepatitis patients. For control of hepatitis B, in view of its only partially understood natural history and epidemiology, we rely on establishing personal and environmental barriers to transmission. At present, ISG appears to be of limited value as a preventive of hepatitis B; therefore, considerable effort is being directed toward the development of biologics for both active and passive immunization.

In order fully to consider the current implications of hepatitis B, both in clinical and public health practices, the Committee on Viral Hepatitis is joined in the following statement by the Public Health Service Advisory Committee on Immunization Practices. Since 1968, this latter group has considered the control of viral hepatitis in its recommendations on use of ISG and has become increasingly cognizant of the need for hepatitis B control in addressing the community management of viral hepatitis.

The following perspectives and recommendations are based on facts known about hepatitis B, sound general principles of containment of infectious diseases, and judgments on the interpretation and significance of antigenemia in hepatitis patients and other persons.

EXTENT OF THE PROBLEM

Viral hepatitis, type B, is of major public health importance in the United States. The number of cases reported have been increasing steadily since 1966, the first year hepatitis B was differentiated from hepatitis A in reports to the Center for Disease Control (CDC). In 1975 approximately 55,000 cases of viral hepatitis were reported, more than 20% of them diagnosed by reporting physicians as type B. This proportion is clearly an underestimate since data from national surveillance and studies of hospitalized adult patients indicate that up to 50% of acute icteric viral hepatitis in adults may be caused by hepatitis B virus (HBV).

CLINICAL AND LABORATORY ASPECTS

Hepatitis B cannot readily be distinguished from other types of viral or toxic hepatitis, and tests of liver function do not differentiate them.

Typical acute icteric hepatitis B has an incubation period of approximately 2-3 months (range, 6 weeks-6 months). The prodromal phase begins with malaise, anorexia, nausea, vomiting, fatigue, and mental depression. Elevated serum transaminases, glutamic oxalacetic (SGOT) and glutamic pyruvic (SGPT), develop early in the prodrome. Symptoms often begin insidiously rather than abruptly; urticaria and/or arthralgia may occur. Icterus appears in 1-2 weeks or somewhat later and is usually accompanied by anorexia; icterus generally persists for 1-2 weeks. Full recovery may take several months; the patient is often fatigued but otherwise has a sense of well being.

Icteric hepatitis B may be associated with considerable debility; on rare occasions, it progresses to fulminant hepatic necrosis. The death-to-case ratio of icteric hepatitis B reported to CDC is 1%; depending on age and underlying health, it may occasionally be as high as 10%.

Anicteric hepatitis B is at least 2-3 times more common than the icteric form. It is often diagnosed as gastroenteritis or some other viral syndrome.

Hepatitis B Surface Antigen (HB_sAg)

Patients with acute hepatitis B have hepatitis B surface antigen (HB_sAg) detectable in blood, usually for 1-8 weeks. This antigen is protein from the outer coat or surface of HBV. Its presence indicates a potential for infectivity.

HB_sAg was first identified by immunodiffusion. This is a simple, inexpensive laboratory technique with great specificity, but one that is time-consuming (1-3 days) and relatively insensitive. So-called second generation procedures including counterimmuno-electrophoresis, complement fixation, and passive hemagglutination are 2 to 10 times more sensitive than immunodiffusion and much faster (30 minutes to several hours). Third generation tests such as radioimmunoassay and reversed passive hemagglutination are at least 100 times more sensitive than immunodiffusion and are recommended for HB_sAg screening. Sensitive techniques are currently

used in most blood banks and many health department and hospital laboratories.

HB_sAg appears during the incubation period, preceding clinical or biochemical evidence of infection by several weeks. It persists during early symptomatic illness and usually disappears before liver function returns to normal. Antibody to HB_sAg (anti-HB_s) is first detectable late in convalescence and indicates complete or partial immunity to subsequent hepatitis B infection.

Five to ten percent of hepatitis B patients have HB_sAg in their blood for many months, even years. some of them progress to chronic active hepatitis and cirrhosis. Patients who are asymptomatic or have mild disease are more likely to have persisting antigenemia than those with icterus. Persistent antigenemia is also seen in persons with impaired or immature cellular or humoral immune systems. These include hemodialysis patients, children with Down's syndrome in institutions for the mentally retarded, patients on immunosuppression, and infants whose mothers had hepatitis B during pregnancy.

EPIDEMIOLOGY

Hepatitis B used to be called "serum hepatitis" and was thought to be transmitted only by parenteral routes such as inoculation or transfusion of blood or blood products. Recent evidence indicates that HBV is spread in other ways as well: Experimental studies have established that oral administration of serum containing HBV can cause hepatitis; observations in non-experimental settings have shown that this can also occur naturally. Although HB_sAg has been detected in many human biologic fluids during acute infection, transmission of disease by saliva or other body fluids containing antigen has not yet been convincingly demonstrated. Evidence for person-to-person spread of hepatitis B includes the spread of infection among mentally retarded persons in institutions and transmission of disease to household or other intimate contacts of patients with hepatitis B or persons with persistent antigenemia.

Although adult volunteer blood donors are not representative of the United States population, 1-5 per 1,000 of them have HB_sAg and thereby are considered potentially infectious. This and other observations on the prevalence of antigenemia in selected groups indicates that there is only a very small risk of hepatitis B for most people. However, HBV infection is more likely for those whose occupation (e.g., hospital, laboratory, and other health care personnel) or household exposure results in close and continued contact with HB_sAg-positive persons. For example, a high risk of acquiring hepatitis B has been reported for patients and staff in hemodialysis units, hematology-oncology units, surgery units, and for workers in clinical pathology laboratories and plasma fractionation facilities.

Transmission of HBV is either direct—when an infectious individual is in physical contact with a susceptible person—or indirect—when the infectious and susceptible persons are separated and HBV is transferred by contaminated substance or object such as blood or needle.

Direct transmission is the principal mechanism of infection for household contacts of patients with acute hepatitis B (primarily spouses) and for workers in certain occupations. HBV can enter the body orally, through mucous membranes, or percutaneously. Blood or serum is most often responsible for infection. (Since HB_sAg has also been demonstrated in saliva and other body substances, these might also transmit the agent.)

Indirect transmission is the principal mechanism of infection for persons exposed to HBV by blood transfusion, sharing contaminated needles in self-injection of drugs, and other such parenteral means. Tattooing, ear piercing, using multiple dose syringes, and handling blood-stained towels have been implicated. Oral exposures such as mouth pipetting infective substances have also caused hepatitis B. Airborne and vector-borne mechanisms of spread of HBV have been postulated but never proven.

CONTROL AND PREVENTION

Widespread application of serologic tests for HB_sAg have underscored the significance of hepatitis B as a clinical and public health problem. HB_sAg in the blood of a patient or of an apparently healthy person raises questions not only of the presence of liver disease but also of the risk of transmitting infection to others. Only limited data are presently available to assess the extent of this risk, but it is clear that not all HB_sAg-positive persons spread infection. The likelihood of transmitting HBV to susceptible contacts is related to many factors including personal hygiene, aseptic technique, protective clothing, state of mind, etc.

Recommendations for limiting the spread of hepatitis B have emphasized minimizing exposure to sources of infection. They have focused on improving personal and environmental hygiene, disease surveillance, public education, and the safety of blood transfusion. These have been partially successful.

Several prospective studies of transfused patients show that the incidence of transfusion-associated hepatitis B has been significantly reduced by the required testing of every unit of blood for HB_sAg by a method of third generation sensitivity (either RIA or RPHA). Nevertheless, there remain a small number of cases presumably due to transfusion of blood contaminated with HBV but with insufficient HB_sAg for detection with the most sensitive tests currently available.

An additional benefit of the advent of HB_sAg testing of blood donors has been definitive identification of a 3-10 times greater prevalence of HB_sAg contamination of blood collected from paid donors than in blood from volunteer donors. The term "paid donor" is used here to describe a donor whose economic status or life-style greatly increases the likelihood that his blood is infective. It does not include donors who are paid for regularly contributing low-risk blood for special pur-

poses. Most cases of transfusion-associated hepatitis appear to be caused by an agent, or agents, other than HAV or HBV. They are reasonably called non-A, non-B hepatitis cases. The risk of non-A, non-B hepatitis is also much greater from paid donor blood than from volunteer donor blood. Therefore, currently the most effective means of further reducing the risk of transfusion-associated hepatitis is elimination, to the extent feasible, of blood from paid donors.

Eliminating commercialism in acquiring blood for transfusion is one of the key objectives of the National Blood Policy. (This policy was announced by HEW in 1973 and is supported by the American Blood Commission, formed in 1975.) In order to provide information regarding relative safety to physicians prescribing and patients receiving blood transfusions, the Food and Drug Administration published a proposal in the Federal Register in November 1975.[3] This proposal would require that all units of blood be labeled to identify the donors as paid or volunteer. Labels would also state that blood from paid donors is associated with a higher risk of hepatitis than blood from volunteer donors. Such labeling requirements are already in effect in California and Illinois. In Illinois there has been a dramatic reduction in the use of blood from paid donors and, based on one study, an equally dramatic reduction in the incidence of transfusion-associated hepatitis.

Definitive control or prevention of hepatitis B awaits development of biologics for active and passive immunization or other specific measures. In the meantime, we must continue to rely on general measures to reduce the chances of exposure and on sound practices for containing infection when the risk of hepatitis B is great.

General Recommendations

The following recommendations offer guidance to those living or working where there is a risk of hepatitis B infection; they can be applied to all settings or environments. (Recommendations tailored to specific settings will be presented later.)

Personal Hygiene: Good personal hygiene is the keystone of protection against hepatitis B infection. The single most important practice is careful handwashing. This combined with the common-sense avoidance of likely sources of infection are fundamental in hepatitis B control.

Disinfection and Sterilization: Most data on inactivation of HBV are from studies conducted prior to the discovery of the HB_sAg in 1965. And many of these studies are deficient in that they involved too few volunteers. In recent years there have been limited opportunities to study the effects of physical and chemical treatments on HBV infectivity. This is because animals that can serve as models for HBV studies are in short supply and HBV has never been adapted to a tissue culture system.

HBV in blood plasma or serum appears to be quite stable and is capable of withstanding wide ranges of temperature and humidity and a variety of chemical agents. Its infectivity persisted for 15 years at -20°C, 6 months at room temperature, and 4 hours at 60°C. In a 1:10 dilution of serum, infectivity was destroyed by boiling for one minute although antigenicity was not altered.

The following methods for disinfection or sterilization are recommended either because of proven effectiveness in volunteer studies, demonstrated destruction of immunological reactivity of HB_sAg, or, on an empirical basis, because of known biocidal activity of the particular treatment.

Heat sterilization is the treatment of choice for instruments and other objects that can conveniently be handled this way. The importance of thoroughly cleansing instruments, containers, or surfaces, etc. to remove adherent material before treatment cannot be overemphasized. HBV has been shown or is expected to be inactivated by heat under each of the following conditions: 1) boiling in water (100°C) for 10 minutes, 2) steam under pressure (autoclaving) at 121°C and 15 pounds per square inch pressure for 15 minutes, or 3) dry heat (160°C) for 2 hours.

Alternatives to heat, presumed to be effective, include some chemical disinfectants: 1) solutions of sodium hypochlorite, 0.5% to 1.0% (5,000-10,000 ppm available chlorine) for 30 minutes, 2) 40% aqueous formalin (16% aqueous formaldehyde) for 12 hours; formalin, 20% in 70% alcohol, 18 hours, 3) 2% aqueous alkalinized glutaraldehyde for 10 hours, and 4) gas sterilization with ethylene oxide (check manufacturer's recommendations).

Immune Serum Globulin: There have been conflicting results from the many well-designed studies evaluating the efficacy of ISG in hepatitis B prophylaxis. Most commercial lots available before 1972 appeared to be ineffective in preventing or modifying parenterally acquired hepatitis B. The lack of effect of these ISG preparations was presumed to be due to the absence or low levels of anti-HB_s. By contrast, more than 90% of samples of ISG manufactured after 1972 were found to have some anti-HB_s. Although there was considerable variation in the titers of anti-HB_s among lots tested, titers were generally greater than in pre-1972 lots.

ISG with detectable but low levels of anti-HB_s has been shown to reduce the clinical severity and complications of HBV infection when the virus inoculum was small and the exposure either percutaneous or oral. Therefore, ISG with anti-HB_s could be of value in hepatitis B prophylaxis under some conditions of exposure.

ISG with high anti-HB_s titers (1:200,000-1:500,000 passive hemagglutination–PHA), hepatitis B immune globulin (HBIG), has been evaluated in a few investigational trials in settings with various kinds of exposure and found to provide significantly greater protection against hepatitis B than ISG with intermediate or low anti-HB_s titers ($\leq$ 1:5,000 PHA). It is expected that

HBIG will be licensed for specified uses; guidelines for use are under development.

As has been stated, standard ISG appears to provide only limited protection against hepatitis B under some conditions of exposure. It is therefore recommended that ISG manufactured after 1972 be offered to individuals who clearly have had an oral or percutaneous exposure to known HB_sAg-positive blood or fluids (e.g. accidental ingestion or accidental needle punctures). The best available data on dosage suggest that, for an adult, a single 5 ml intramuscular injection may be of benefit.

Reporting and Education: Every case of hepatitis should be tested for HB_sAg and reported promptly to the local or state health department. Reporting of confirmed cases permits more accurate hepatitis surveillance, identification of changes in epidemiologic trends, and community-wide prevention and control measures.

HB_sAg-positive persons and their close contacts should be fully informed about HBV and how to limit its spread. This education is especially important for health care professionals since they often have continual exposure to sources of possible infection (patients, blood, body fluids, etc.).

Recommendations for Minimizing Transmission in Specific Settings

The risk of acquiring hepatitis B is greatest for persons who frequently encounter hepatitis patients or specimens containing HB_sAg. Generally speaking, the highest risk involves a few specific household and hospital settings. Before considering them in detail, and to avoid redundancy, some general comments can be made.

Regardless of the setting, patients and human biological specimens should be managed carefully because some will present an unrecognized hepatitis risk. To protect susceptible patients and staff, blood and other specimens from hepatitis patients and HB_sAg-positive persons should be labeled as such and optimally be enclosed in impermeable bags. Charts of these patients should be flagged (e.g. "Hepatitis B," "blood and instrument precautions," etc.). All persons involved, patients and staff, should practice careful handwashing and personal hygiene.

Specific recommendations should be tailored to the particular setting and reflect the unique aspects of each environment:

Household: Within the household, spouses and other intimate contacts of patients with acute hepatitis B or of asymptomatic HB_sAg-positive persons appear to have the greatest risk of infection. It is important that all household contacts know how hepatitis B is transmitted and that blood and possibly other body fluids, if HB_sAg-positive, might spread HBV infection. In addition to practicing good personal hygiene (especially handwashing), they also need to handle and dispose of blood-contaminated articles carefully and avoid practices which might increase the opportunity for infection such as sharing razors, toothbrushes, towels, washcloths, or other personal items.

Hospitals: *General Patient Care Area:* Hepatitis B has spread from patients to staff in intensive care units, transplant units, hematology-oncology wards, and general medical-pediatric and surgical wards. Nevertheless, patients with acute hepatitis or HB_sAg-positive persons in these environments generally need *not* be placed in isolation; they can be cared for in semi-private or ward accommodations providing blood and instruments are handled with the precautions discussed earlier. When handling blood or blood-contaminated objects from HB_sAg-positive patients, staff should wear gloves and possibly other protective clothing as well. During procedures which could result in splattering or splashing infective material, a surgical-type mask or facial covering to protect eyes, nose, and mouth has value. Disposable needles and syringes, proper sterile technique, and adequate sterilization and chemical disinfection procedures are important to control percutaneous spread.

Laboratories: Clinical biochemistry and hematology-serology laboratories, hepatitis research laboratories, and autopsy laboratories are recognized to be settings where hepatitis B transmission occurs. Common exposures are accidentally pricking the skin with instruments contaminated with HBV, pipetting infective fluids, contaminating cuts or scratches with infective blood or splashing it in eyes or mouth. Contamination may result from shaking specimens, homogenizing, opening screw-cap bottles, blowing the last drop of fluid from a pipette, pouring fluids, and centrifuging. Mouth pipetting, smoking, and eating in the laboratory are dangerous practices and should be forbidden. Protective clothing, including gloves and facial coverings (if there is danger of splashing), may be appropriate. This is especially important when performing autopsies on HB_sAg-positive persons. Gloves and other protective clothing must be properly used (changed frequently, especially when contaminated or torn) and should not be considered a substitute for careful technique and good personal practices. Work areas should be thoroughly cleaned and disinfected daily. All laboratory accidents resulting in HB_sAg exposure must be promptly reviewed with regard to need for environmental decontamination and personal prophylaxis.

Laboratories should specify that potentially infective specimens be properly packaged and labeled. It may be useful for them to have a designated safety officer who keeps records of laboratory accidents, educates personnel in methods of control and prevention, and periodically evaluates and updates safety procedures.

Hemodialysis Units: Hemodialysis units present a great risk of hepatitis B for patients and staff. Most infections in patients are subclinical, and a large percentage of them result in persistent HB_sAg-positivity. Staff commonly have overt hepatitis, often hampering operations.

HBV can be introduced into the units by patients, staff, or infective blood, plasma, or blood products. Once seeded, HBV can be spread among patients and staff by personal contact or parenteral exposure.

Surveillance: Continuous surveillance of patients and staff for HBV infection is essential. Those who are seronegative should be tested periodically for HB_sAg, anti-HB_s, and, possibly, SGOT and/or SGPT. The frequency of testing depends on whether hepatitis B is occurring. If no hepatitis B infections among patients or staff develop in 6-12 months of testing, routine sampling intervals of 2-4 months would be reasonable. If antigen or antibody does appear, HBV spread is probably occurring, and more frequent (e.g., monthly) sampling should be undertaken.

It is useful to screen new hemodialysis patients and staff for HB_sAg and anti-HB_s before admission to the unit. This provides baseline information on their susceptibility or potential infectiousness and the need to institute precautions.

Records: Detailed records of patient management (e.g., machine and other equipment assignments and dates of use) will be essential to determine whether any infections are related to specific procedures. Records should also include complete descriptions of mishaps (needle punctures, membrane leaks-ruptures, etc.).

Staff: Highest quality aseptic technique is fundamental to prevent HBV spread. Protective clothing should be used but changed on a regular basis and whenever obviously contaminated. It should not be worn outside the unit. Using gloves during procedures where there is contact with blood such as handling shunts, drawing blood, or cleaning or dismantling dialysis machines, has been shown to decrease the risk of HBV infection. A fresh pair of gloves should be used with each patient. However, gloves are not a substitute for good technique and proper personal hygiene. Surgical-type masks or other facial coverings may decrease the risk of infection when splattering of blood occurs. Abrasions, lacerations, and other breaks in the skin should be bandaged to protect them from contact with infectious material.

Operating Procedures: Operating procedures should minimize the amount of close personal contact among patients and staff. Overcrowding is to be avoided, and individual equipment and supplies used whenever possible. Patients and staff should not eat, drink, or smoke in the immediate hemodialysis area.

Susceptible patients should be separated from known HB_sAg-positive patients or those whose antigen status is unknown. If practical, staff should be assigned to attend HB_sAg-positive or HB_sAg-negative patients, but not both, during the same shift. Staff with anti-HB_s might preferentially be assigned to HB_sAg-positive patients. Until their antigen/antibody status is known, new patients should be managed in areas separate from these being used for chronic hemodialysis.

After each use, equipment which cannot be heat or gas sterilized should be washed to remove adherent material and cleaned with a disinfectant solution. Nonpermeable disposable diaphragms can prevent contamination of equipment such as venous pressure monitors.

Institutions for the Mentally Retarded: Viral hepatitis, both sporadic and epidemic, has long been known to occur in custodial institutions for the mentally retarded and poses a risk for both patients and staff. The risk of infection appears to increase with the duration of instutionalization. Practical precautions are needed to reduce transmission of HBV from HB_sAg-positive mentally retarded residents to persons such as teachers, classmates, and parents who come in close personal contact. In general and until more definitive information on the infectiousness of persons with HB_sAg becomes available, it is important to avoid placing unwarranted limitations or restrictions on HB_sAg-positive retarded persons.

Surveillance: Serologic surveys of institutionalized persons are not considered necessary as a routine procedure. However, in institutions where hepatitis B infection has been shown to be endemic, periodic screening of residents will be helpful in identifying high-risk areas, in investigating hepatitis outbreaks, and in evaluating the effectiveness of control measures.

Since persons who are antigen-positive may become negative even after 6 or more months of positivity, each HB_sAg-positive person should be retested periodically to determine whether the antigen persists. Once seroconversion to anti-HB_s occurs, any specific hepatitis precautions can be removed.

Control Measures: Parents and personnel responsible for the care of institutionalized mentally retarded persons must be aware of the need for good hygienic practices. This is especially important after caring for open wounds or having contact with blood or blood-contaminated fluids and before eating or handling food. Personal toiletry articles should not be shared. No special procedures need be observed for laundering clothing or linens, although all blood-contaminated items should be handled with appropriate precautions (gloves, etc.). No other restrictions need be imposed on antigen-positive residents when they participate in routine activities such as special education programs and in nursery, day care, or foster care facilities.

Physicians, dentists, laboratory workers, and others providing care for known antigen-positive persons should be so advised so that adequate precautions can be exercised during patient contact or specimen handling. Because of the likelihood of contact with unrecognized HB_sAg-positive persons or specimens, health workers in institutions should always be alert to the risk of hepatitis B and use appropriate protective measures.

Community Placement Programs, Nursing Homes: Because residents of institutions for the retarded more frequently are HB_sAg-positive than non-institutionalized populations, they should be tested for HB_sAg before

transfer to other institutions or discharge to community placement programs or nursing homes. Information on their antigen status will alert health personnel and others who have close contact with them in community programs to use precautions when there is a risk of hepatitis B. HB_sAg-positive persons should be given the same consideration for placement programs or nursing homes as those who are negative. Parents and personnel in contact with antigen-positive retarded persons should be informed of the risk of hepatitis B and given instruction in control and prevention. (HB_sAg-positive residents in community programs should be managed as are those in institutions; see above.)

Management of High-Risk Persons and Populations

Certain persons and groups, particularly those involved in health care, are at greater risk than the general population of acquiring hepatitis B; this is because of occupational and environmental exposures. Since most hepatitis B infections are subclinical and 5%-10% of those infected may develop persistent HB_sAg, there may also be an increased risk of hepatitis B for the general population brought into contact with HB_sAg-positive individuals in health care settings. Precautions against spreading HBV infection currently depend on awareness of personal risks and use of good hygienic practices.

Management of persons with persistent HB_sAg who work in environments and under circumstances where there are many chances for transmitting HBV infection (e.g., regular contact with blood, involvement with parenteral and surgical procedures, etc.) require special consideration. As has been emphasized, the presence of HB_sAg is sufficient reason for personal precautions, but not necessarily evidence of a substantial hepatitis risk for contacts or associates. It is extremely important to prevent misunderstanding and unreasonable management of persons or population groups with HB_sAg. Since it now appears that individuals who have persisting HB_sAg may become negative even after 6 or more months of positivity, each antigen-positive person should be retested periodically (e.g., every 6 months) to determine whether antigen persists.

General Recommendations: Persons working where there is a high risk of hepatitis B infection (especially hemodialysis or hematology-oncology units and clinical laboratories) need to be fully aware of the risks and to use good hygienic practices to minimize any chance for infection. It is reasonable to keep such "high risk" population groups under serologic surveillance for hepatitis B infection in order to be able to detect and investigate problems as quickly as possible. On the other hand, there is no need to routinely test health professionals and hospital employees not working in high-risk areas.

Health personnel observed to be HB_sAg-positive should not be restricted from patient contact solely on the basis of this serologic finding. Rather, their personal procedures and practices should always reflect an awareness of the potential for transmitting HBV and include rigorous efforts to reduce any chance that transmission might occur. Knowing that contact with blood or serum containing HBV is the likely cause of hepatitis B infections, scrupulous aseptic technique, avoidance of personal hand injuries, and use of gloves in office-based minor surgery, dental procedures, wound dressing, etc. have obvious value.

Health personnel clearly associated epidemiologically with HBV transmission obviously pose a greater risk for patients and associates and must be evaluated carefully with respect to continuing risks. In these instances, more restrictive measures (e.g., limiting or eliminating some types of procedures or contact with patients) may be needed. Obviously, each such episode will have to be dealt with separately and recommendations and control measures tailored to the specific conditions.

Health Care Personnel (Dentists, Nurses, Physicians, Technicians, Etc.): Recent investigations of hepatitis B among contacts of antigen-positive health personnel demonstrate that HBV transmission does occur but seems to be very rare. There appears to be considerable variation in the likelihood that persons with persistent HB_sAg will spread infection. The risk depends in part on the kind and extent of contact with susceptibles. In the few instances where epidemiologic evidence linked health workers in specific hospital or dental environments to hepatitis B cases, infection seemed to have been caused by a presumably minute amount of blood or serum which was transferred during routine procedures. Minor hand injuries have generally been thought to be the source of infective blood which then is introduced by oral or percutaneous routes.

Food Handlers: There is no evidence that HB_sAg-positive food handlers pose a health risk to the general public, and transmission of hepatitis B by food has not been documented. Nonetheless, it is prudent to restrict food handlers with acute hepatitis B from working while ill. Food handlers with persistent HB_sAg, like all antigen-positive persons, should be educated about HBV transmission, the need for attention to good personal hygiene, avoidance of hand injuries, etc.

Pregnant Women: Women with hepatitis B infection during pregnancy sometimes transmit the infection to their infants. The risk is highest when an acute illness occurs during the third trimester. Infected newborns who may be HB_sAg-negative at birth generally become HB_sAg-positive 1 to 2 months later in the absence of clinical hepatitis. Most infants who become HB_s-Ag-positive develop persistent HB_sAg, and many eventually have histopathologic evidence of inflammatory liver disease. For these reasons, seronegative pregnant women working in high-risk environments should be transferred to work areas where the risk is lower for the duration of pregnancy.

References

1. MMWR 21(16):133-134, 1972
2. MMWR 23(14):125-126, 1974
3. Federal Register, Vol 40(221):53040, November 14, 1975

SELECTED BIBLIOGRAPHY

General

Chalmers, TC, Alter HJ: Management of the asymptomatic carrier of the hepatitis-associated (Australia) antigen. N Engl J Med 285:613-167, 1971

Cossart YE: Epidemiology of serum hepatitis. Br Med Bull 28:156-162, 1972

Heathcote J, Cameron CH, Dane DS: Hepatitis B antigen in saliva and semen. Lancet 1:71-73, 1974

Krugman S, Giles JP: Viral hepatitis: New light on an old disease. JAMA 212:1019-1029, 1970

Mosley, JW, Galambos JT: Viral Hepatitis. In Diseases of the Liver, 3rd ed, edited by Schiff L. Philadelphia, JB Lippincott Co, 1969

Ogra P: Immunologic aspects of hepatitis-associated antigen and antibody in human body fluids. J Immunol 110:1197-1205, 1973

Proceedings of a symposium on viral hepatitis. Am J Med Sci 270:1-412, 1975

Villarejos VM, Visona KA, Guttierrez A, et al: Role of saliva, urine, and feces in the transmission of type B hepatitis. N Engl J Med 291:1375-1378, 1974

Viral hepatitis: Report of a WHO Meeting. WHO Technical Report Series No. 570, Geneva, 1975

Ward R, Wright A, Borchert P, Kline E: Hepatitis B antigen in saliva and mouth washing. Lancet 2:726-727, 1972

Disinfection, Sterilization, and Handwashing

Krugman S, Giles JP, Hammond J: Hepatitus Virus: Effect of heat on the infectivity and antigenicity of the MS-1 and MS-2 strains. J Infect Dis 122:432-436, 1970

Mallison GF: Disinfection in hospitals. APIC Newsletter 2:1-3, 1974*

Perkins JJ: Principles and Methods of Sterilization in Health Sciences. Springfield, ILL, CC Thomas, 1970, pp 89-90

Spaulding EH and Groschel DHM: C. Hospital Disinfectants and Antiseptics. In Chapter 91, Control of Hospital-Associated Infections. Manual of Clinical Microbiology, Washington DC 1974

Steere AC and Mallison GF: Handwashing practices for the prevention of nosocomial infections. Ann Intern Med 83:683-690, 1975

Passive Immunization

Grady GF, Lee VA: Hepatitis B immune globulin: Prevention of hepatitis from accidental exposures among medical personnel: A preliminary report of a cooperative multicenter trial. N Engl J Med 293:1067-1070, 1975

Kohler PF, Dubois RS, Merrill DA, et al: Prevention of chronic neonatal hepatitis B virus infection with antibody to the hepatitis B surface antigen. N Engl J Med 291:1378-1380, 1974

Krugman S, Giles JP, Hammond J: Viral hepatitis, type B (MS-2 strain). Prevention with specific hepatitis B immune globulin. JAMA 218:1665-1670, 1971

Prince AM, Szmuness W, Mann MK, et al: Hepatitis B "Immune" globulin: Effectiveness in prevention of dialysis-associated hepatitis. N Engl J Med 293:1063-1067, 1975

Prophylactic gamma globulin for prevention of endemic hepatitis. Effects of gamma globulin upon incidence of viral hepatitis and other infectious diseases in US soldiers abroad. Arch Intern Med 128:723-738, 1971

Public Health Service Advisory Committee on Immunization Practices: Immune serum globulin for protection against viral hepatitis. MMWR 21(25): Supplement 1972

Redeker AG, Mosley JW, Gocke DJ, et al: Hepatitis B immune globulin as a prophylactic measure for spouses exposed to acute type B hepatitis. N Engl J Med 293:1055-1059, 1975

Seeff LB, Zimmerman HJ, Wright EC, et al: Efficacy of hepatitis B immune globulin after accidental exposure. Preliminary report of the Veterans Administration Cooperative Study. Lancet 2:939-941, 1975

Surgenor D MacN, Chalmers TC, Conrad ME, et al: Clinical trials of hepatitis B immune globulin. Development of policies and materials for the 1972-1975 studies sponsored by the National Heart and Lung Institute, N Engl J Med 293:1060-1062, 1975

Szmuness W, Prince AM, Goodman M, et al: Hepatitis B immune globulin in prevention of nonparenterally transmitted hepatitis B. N Engl J Med 290:701-706, 1974

Household and Community

Heathcote J, Sherlock S: Spread of acute type B hepatitis in London. Lancet 1:1468-1470, 1973

Szmuness W, Much MI, Prince AM, et al: On the role of sexual behavior in the spread of hepatitis B infection. Ann Intern Med 83:489-495, 1975

Szmuness W, Prince AM, Hirsch RL, Brotman B: Familial clustering of hepatitis B infection. N Engl J Med 209:1162-1166, 1973

Wright RA: Hepatitis B and the HB_sAg carrier. An outbreak related to sexual contact. JAMA 232:717-721, 1975

Health Personnel (Hospitals)

Alter HJ, Chalmers TC, Freeman BM, et al: Health care workers positive for hepatitis B surface antigen. Are their contacts at risk? N Engl J Med 292:454-457, 1975

Bryan JA, Carr HE, Gregg MB: An outbreak of nonparenterally transmitted hepatitis B. JAMA 223:279-283, 1973

Lewis TL, Alter HJ, Chalmers TC, et al: A comparison of the frequency of hepatitis B antigen and antibody in hospital and non-hospital personnel. N Engl J Med 289:647-651, 1973

Pattison CP, Maynard JE, Berquist KR, et al: Epidemiology of hepatitis in hospital personnel, Am J Epidemiol 101:59-64, 1974

Rosenberg JL, Jones DP, Lipitz LR, et al: Viral hepatitis—an occupational hazard to surgeons. JAMA 223:395-400, 1973

Snydman DR, Bryan JA, Dixon RE: Prevention of nosocomial viral hepatitis, type B (hepatitis B). Ann Intern Med 83:838-845, 1975

Trumbull ML and Greiner DJ: Homologous serum jaundice, an occupational hazard to medical personnel. JAMA 145:965-967, 1951

Wands JR, Walker JA, Davis TT, et al: Hepatitis B in an oncology unit. N Engl J Med 291:1371-1375, 1974

Williams SV, Huff JC, Feinglass EJ, et al: Epidemic viral hepatitis, type B, in hospital personnel. Am J Med 57:904-911, 1974

*Available on request to Bureau of Epidemiology, Center for Disease Control, Atlanta, Georgia 30333

Health Personnel (Laboratories)

Grist NR: Survey of hepatitis in laboratories. J Clin Pathol 27:84, 1974

Pattison CP, Boyer K, Maynard JE, et al: Epidemic hepatitis in a clinical laboratory. JAMA 230:854-857, 1974

Percy-Robb IW, Proffitt J, Whitby LG: Precautions adopted in a clinical chemistry laboratory as a result of an outbreak of serum hepatitis affecting laboratory personnel. Am J Clin Pathol 23:751-756, 1970

Safety in pathology laboratories, 1972. A handbook prepared by the Working Party of the Central Pathology Committee. Dept of Health and Social Security. Alexander Fleming House. Elephant and Castle, London SE1 6 BY

Schmidt NJ, Lennette EH: Safety precautions for performing tests for hepatitis-associated "Australia" antigen and antibodies. Am J Clin Pathol 57:526-530, 1972

Sutnick AI, London WT, Millman I, et al: Ergasteric hepatitis: Endemic hepatitis associated with Australia antigen in the research laboratory. Ann Intern Med 75:35-40, 1971

Watson D, Langley DJ, Chrystie IL, et al: Hepatitis B antigen and safety in pathology laboratories. Lancet 1:985-988, 1973

Health Personnel (Dentists)

Feldman RE and Schiff ER: Hepatitis in dental professionals. JAMA 232:1228-1230, 1975

Levin ML, Maddrey WK, Wand JR, and Mendleloff AL: Hepatitis B transmission by dentists. JAMA 228:1139-1140, 1974

Mosley JW, Edwards VM, Casey G, Redeker AG, and White E: Hepatitis B virus infection in dentists. N Engl J Med 293:729-734, 1975

Williams SV, Pattison CP, Berquist KR: Dental infection with hepatitis B. JAMA 232:1231-1233, 1975

Workshop on Viral Hepatitis in Dental Practice. J Am Dent Assoc 92:153-159, 1976

Health Personnel (Renal Hemodialysis)

Garibaldi RA, Bryan JA, Forrest JN, et al: Hemodialysis associated hepatitis. JAMA 225:384-389, 1973

Garibaldi RA, Hatch FE, Bisno AL, et al: Nonparenteral serum hepatitis. JAMA 220:963-966, 1972

Hawe BJ, Goldsmith HJ, and Jones PO: Dialysis-associated hepatitis: Prevention and control. Br Med J 1:540-543, 1971

Hepatitis and the treatment of chronic renal failure. Report of an Advisory Group, 1970-1972. Dept of Health and Social Security. Welsh office.

Marmion BP and Tonkin RW: Control of hepatitis in dialysis units. Br Med Bull 28:169-179, 1972

Pattison CP, Maynard JE, Berquist KR, et al: Serological and epidemiological studies of hepatitis B in hemodialysis units. Lancet 2:172-174, 1973

Szmuness W, Prince AM, Grady GF, et al: Hepatitis B infection: A point prevalence study in 15 US hemodialysis centers. JAMA 227:901-906, 1974

Institutions

Chance ER: Prevalence of hepatitis associated antigen (HAA) in an institution for the mentally retarded. Am J Ment Defic 77:1-5, 1972

MacQuarrie MB, Forghani B, Wolochow DA: Hepatitis B transmitted by a human bite. JAMA 230:723-724, 1974

Szmuness W, Pick R, Prince AM: The serum hepatitis virus specific antigen (SH): A preliminary report on epidemiologic studies in an institution for the mentally retarded. Am J Epidemiol 92:51-61, 1970

Szmuness W, Prince AM, Ellwig GF, Pich R: Development and distribution of hemagglutinating antibody against the hepatitis-associated antigen carrier state. N Engl J Med 287:1280-1282, 1972

Pregnancy and Infancy

Aziz MA, Khan G, Khanum T, et al: Transplacental and postnatal transmission of the hepatitis-associated antigen. J Infect Dis 127:110-112, 1973

Merrill DA, Dubois RS, Kohler PF: Neonatal onset of the hepatitis-associated antigen carrier state. N Engl J Med 287: 1280-1282, 1972

Schweitzer IL, Dunn AE, Peters RL, and Spears RL: Viral hepatitis B in neonates and infants. Am J Med 55:762-771, 1973

Schweitzer IL, Wing A, McPeak C, et al: Hepatitis and hepatitis-associated antigen in 56 mother-infant pairs. JAMA 220:1092-1095, 1972

Stevens CE, Beasley RP, Tsui J, et al: Vertical transmission of hepatitis B antigen in Taiwan. N Engl J Med 292:771-774, 1975

Transfusion-Associated Hepatitis

Alter HJ, Holland PV, Purcell RH, et al: Post-transfusion hepatitis after exclusion of commercial and hepatitis-B-antigen-positive donors. Ann Intern Med 77:691-699, 1972

Goldfield M, Black HC, Bill J, et al: The consequences of administering blood pretested for HB_sAg by third generation techniques: A progress report. Am J Med Sci 270:335-342, 1975

Seeff LB, Zimmerman HJ, Wright EC, McCollum RW: VA cooperative study of post-transfusion hepatitis, 1969-1974: Incidence and characteristics of hepatitis and responsible risk factors. Am J Med Sci 270:355-362, 1975

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