Hepatitis: a new crisis in pedodontics

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Abstract

Pedodontists by and large have not been concerned with viral hepatitis as a health hazard in practice. However, based upon the high incidence of viral hepatitis in children and adolescents from lower socioeconomic areas, of recent immigration from Africa, Asia, or other tropical countries, with certain handicapping conditions, or receiving certain pharmacodynamic therapy, abusing drugs, with male homosexual precocious sexual activity, and from other environments conducive to viral hepatitis many pedodontic practices are at risk for disease transmission. Exposure to viral hepatitis often necessitates direct immune globulin prophylaxis specific to the type of viral hepatitis. All pedodontic practices should review and update their disease control protocol.

Introduction

Pedodontists in the past have demonstrated an alarming lack of concern for the health hazards of viral hepatitis. Two epidemiological factors may have contributed to this paucity of concern. First, pedodontists as a specialty group have had the lowest incidence of viral hepatitis exposure of any specialist or generalist groups in most serological studies of dental professionals. Second, serological evidence indicating exposure to viral hepatitis increases throughout life with children generally exhibiting lower exposure rates than adults. These two factors, although still credible in the 1980’s, must be analyzed more cautiously in view of present disease morbidity data. Children from certain socioeconomic areas, immigrant children from certain African, Asian, and tropical countries, and certain handicapped children have now been shown to have a high incidence of exposure to viral hepatitis. In view of these findings it appears that viral hepatitis may become a new crisis in pedodontics.

Viral Hepatitis

Viral hepatitis may be differentiated into three clinically similar, but immunologically and epidemiologically different types of disease (Table 1). Hepatitis A viral disease (HAV), previously referred to as infectious hepatitis, is sporadic, epidemic within families, has an incubation period of between two and six weeks, and is not dependent upon the route of inoculation. A viremia occurs during the incubational and acute phases of HAV. Rarely does HAV produce a carrier state after the clinical phase. Morbidity is age-related with asymptomatic infection and anicteric illness predominating in children. Mortality is usually less than 1%.

Hepatitis B viral disease (HBV), previously referred to as serum hepatitis, has an incubation period of between six and 26 weeks depending upon the route of inoculation with the oral route usually resulting in the longer incubation times. Again, a viremia occurs during the incubational and acute phases and, occasionally, post-disease patients may be chronic carriers. Morbidity and mortality are variable depending upon severity of viremia and age of patient.

Hepatitis Non-A, Non-B is the third and most recently discovered type. It is thought to be a major

Table 1. Viral hepatitis.

<table>
<thead>
<tr>
<th>Type A</th>
<th>Type B</th>
<th>Type Non-A, Non-B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Agents</td>
<td>Hepatitis A Virus</td>
<td>Hepatitis B Virus</td>
</tr>
<tr>
<td>Incubation</td>
<td>2-6 weeks</td>
<td>6-26 weeks</td>
</tr>
<tr>
<td>Occurrence</td>
<td>Epidemic</td>
<td>Point source</td>
</tr>
<tr>
<td>Carrier State</td>
<td>Rare</td>
<td>Yes</td>
</tr>
</tbody>
</table>

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cause of transfusion related hepatitis and is probably the result of more than one of the causative viruses that have been implicated. The incubation and clinical course are similar to HBV, but serological and immunological studies are exclusive for HBV. Cases of sporadic, clinically obvious hepatitis in urban centers are approximately 25% HAV, 50% HBV, and 25% Non-A, Non-B.\(^1\)

Clinically, all types appear undifferentiated with respect to liver pathology with histological evidence of tissue inflammation and cell necrosis. The major complications and high incidence of mortality can be attributed to massive hepato-cellular necrosis. The clinical course of the disease is characterized by three periods (Table 2).

Table 2. Clinical phases of viral hepatitis.

| I | Anicteric (non-jaundiced) prodromal period with symptoms of loss of appetite, abdominal pain, nausea, general weakness, and malaise (flu-like symptoms). |
| II | Icteric (jaundiced) period with classic signs of jaundice. |
| III | Recovery period with no symptoms but with complete clinical and biochemical recovery. |

The first clinical phase is usually the anicteric (non-jaundiced) prodromal period characterized by loss of appetite, abdominal pain, nausea, general weakness, and malaise. The second clinical phase is the icteric (jaundiced) period characterized by the classic signs and symptoms of jaundice. The final clinical phase is the recovery period which is usually three to six weeks in duration where the patient usually has no symptoms. Modern therapeutic regimens include diet management and reduced physical activity. Complete clinical and biochemical recovery often takes from three to four months with periodic evaluation of liver function and serological tests necessary. Hepatitis has been detected in asymptomatic patients by the use of liver function and serological tests.\(^2\)\(^-\)\(^4\) It is possible for subclinical cases to exist mimicking a flu-like syndrome.

Since 1966, with the discovery of the Blumberg "Australian" antigen, now known as the Hepatitis B surface antigen (HBsAg), it became possible to recognize through serological testing the hepatitis B viral disease (HBV). It further became possible to differentiate B from other Non-B hepatitis diseases and to identify chronic carriers of the B virus.\(^5\)\(^-\)\(^7\) Typically, the HBsAg develops 20 to 100 days after exposure, is detectable in the serum for one to 120 days after development, and then disappears within four months. The serum becomes negative for the HBsAg and, in most instances, antibodies specific to HBsAg (anti-HBs) develop and are detectable in the serum for many years thereafter. Immunity is evidenced in a person by the presence of these anti-HBs which are serum markers for previous HBV infection.\(^8\)\(^,\)\(^9\)

The Dane particle is a 42 nm particle with HBsAg on its surface and hepatitis B core antigen (HBcAg) in its core. It is the intact virus (virion) — a double-shelled particle enclosing a unique circular double-stranded DNA and a specific DNA polymerase. These antigenic determinants are shared by numerous smaller spherical and tubular particles which presumably represent HBV surface antigen produced in gross excess by infected hepatocytes. The Dane particle is the only viral structure known to contain nucleic acid with DNA sequences capable of integrating into chromosomal CNA of HBV-infected liver.

A circulating protein antigen exclusively seen in HBV infection termed the Hepatitis B "e" antigen (NBeAg) is probably a structural antigen of HBV linking surface to core antigen layers, and is viewed as a serum marker of active virus replication and thus infectivity. A carrier of HBV is thought to be a better transmitter, or more infectious, if the carrier also has a positive test for HBeAg.

Only about .01% of HBV particles are intact infectious viruses, but since HBV particles circulate in enormous titers of approximately \(10^{12}\) HBV particles/ml of blood, the observation of approximately \(10^6\) Dane particles/ml of blood is significant. Therefore, the amount of infected blood that can transmit HBG is relatively small (\(10^5\) to \(10^6\) ml).\(^10\)\(^,\)\(^11\)

Sensitive serological tests have now demonstrated that hepatitis can be attributed to hepatitis A virus, hepatitis B virus, and Non-A, Non-B viruses. Non-A, Non-B hepatitis is a viral hepatitis-like syndrome in the absence of serological evidence for hepatitis A virus (HAV), hepatitis B virus (HBV), cytomegalovirus (CMV), or Epstein-Barr virus (EBV). No serological test to date is available on humans for Non-A, Non-B viruses. Evidence, however, does exist indicating that more than one virus is responsible for Non-A, Non-B hepatitis.\(^12\)\(^-\)\(^16\)

Carrier states exist for hepatitis B and Non-A, Non-B hepatitis, but are extremely rare for hepatitis A. Approximately 5-10% of persons infected with hepatitis B will circulate HBsAg for up to two years or for as long as 25 years before seroconversion to antibodies.\(^4\)\(^,\)\(^8\)\(^,\)\(^17\)\(^,\)\(^18\) In general, however, HBsAg rarely persists for more than four months. Studies report the incidence of prolonged or chronic active liver disease to be 20-40% for Non-A, Non-B hepatitis, which appears higher than for hepatitis B and may indicate a greater ability in perpetuating a chronic carrier state.\(^19\) Some carriers have been reported as healthy in the absence of progressive liver disease, but may still be infec-
tious. A defective autoimmune response may prevent carriers from seroconverting to antibodies. It is the propensity to produce chronic hepatitis and chronic carrier states that makes control of hepatitis B and Non-A, Non-B hepatitis extremely frustrating.

The transmission factors are likewise specific according to the type of hepatitis (Table 3). Hepatitis A is essentially transmitted by fecal-oral contact with infectivity decreasing greatly by the time overt clinical hepatitis develops. Therefore, the maximum infectivity time occurs prior to acute illness during incubation. Serological IgM-anti-HAV testing is often performed to detect acute HAV infection. Parenteral transmission of HAV is rare. Hepatitis B and Non-A, Non-B hepatitis are transmitted by both parenteral and non-parenteral contact. Although hepatitis B and Non-A, Non-B can be transmitted within families, true epidemic hepatitis is usually hepatitis A.

Among the body fluids not apparently blood contaminated that can transmit HBV and probably Non-A, Non-B agents are: saliva, nasopharyngeal secretions, tears, breast milk, vaginal secretions, semen, urine and feces. Many of these fluids, however, contain very low levels of the antigen. Saliva was positive for HBsAg in 76% of patients with acute hepatitis B and 80% of the chronic carriers of HBV.20,21 Gingivitis with accompanying micro-leakage of blood into the oral cavity has been cited as a factor also.22-5 It appears that the non-parenteral routes, however, are not as effective in transmission as the parenteral routes. More serological research is necessary before the various non-parenteral routes for Non-A, Non-B agents can be studied and confirmed.

Table 3. Transmission Factors.

<table>
<thead>
<tr>
<th></th>
<th>Parenteral</th>
<th>Non-Parenteral</th>
</tr>
</thead>
<tbody>
<tr>
<td>HAV</td>
<td>Rare</td>
<td>Very common</td>
</tr>
<tr>
<td>HBV</td>
<td>Common</td>
<td>Common, less effective</td>
</tr>
<tr>
<td>HNANB</td>
<td>Common</td>
<td>Common, less effective</td>
</tr>
</tbody>
</table>

Hepatitis B Exposure Studies in Dental Professionals

It is estimated that approximately 3.5% persons in the United States have had hepatitis B with a carrier rate of approximately 0.5-1.0%. Surveys have reported a 6.3% to 20.8% (average 13.6%) range for previous exposure to hepatitis B in general dentists with a carrier rate of approximately 1% to 2%.26-38 It is estimated that in health care workers the incidence of the HBsAg carrier rate is approximately 1% to 2%.17 Surgical dental specialty groups have reported an increase in this exposure rate with oral surgeons having a 27% total exposure rate and a 2% to 3% carrier rate and endodontists having a 9% total exposure rate.8,37,38

Periodontists in unpublished data are reported to have an exposure rate in this same range.39 Orthodontists in unpublished data are reported to have an exposure rate of 8.9%.39 Pedodontists as a group, however, have never been tested but usually have the lowest incidence of dentists tested. Dental auxiliaries appear to have a low exposure incidence.39,40 Physicians and nurses have an average exposure rate of 15% to 13% respectively.41 Physicians specializing in family practice, internal medicine, pediatrics, and obstetrics-gynecology have exposure rates of approximately 18%.42 Other health personnel at high risk, however, are kidney dialysis staff personnel at 28% and clinical laboratory personnel at 19%.41 It now appears that exposure trends develop during professional education as evidenced by the increasing incidence in dental school classes43,44 (Table 4).

Table 4. Hepatitis B Exposure Rate in health professionals.

<table>
<thead>
<tr>
<th>Health Group</th>
<th>Approximate HBV Exposure Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>General Physician</td>
<td>~ 15%</td>
</tr>
<tr>
<td>Surgical Physician</td>
<td>~ 27%</td>
</tr>
<tr>
<td>Pathologist</td>
<td>~ 27%</td>
</tr>
<tr>
<td>Pediatricians</td>
<td>~ 18%</td>
</tr>
<tr>
<td>Administrative Physician</td>
<td>~ 4%</td>
</tr>
<tr>
<td>Kidney Dialysis Staff</td>
<td>~ 28%</td>
</tr>
<tr>
<td>Clinical Lab Staff</td>
<td>~ 19%</td>
</tr>
<tr>
<td>General Dentists</td>
<td>~ 14%</td>
</tr>
<tr>
<td>Oral Surgeons</td>
<td>~ 27%</td>
</tr>
<tr>
<td>Endodontists</td>
<td>~ 9%</td>
</tr>
<tr>
<td>Periodontists</td>
<td>~ 9%</td>
</tr>
<tr>
<td>Orthodontists</td>
<td>~ 9%</td>
</tr>
<tr>
<td>Dental Auxillaries</td>
<td>~ 7%</td>
</tr>
<tr>
<td>Dental Students</td>
<td>~ 5%</td>
</tr>
<tr>
<td>General Population</td>
<td>~ 3.5%</td>
</tr>
</tbody>
</table>

Viral Hepatitis in Children and Adolescents

Hepatitis A accounts for more than 31,000 cases (15 per 100,000) in the United States yearly.45 Sero-epidemiological studies have reported that over half the U.S. population has evidence of past infection by mid-adult life.46 Serological surveys in New York have demonstrated that anti-HA is present in 30% to 75% of adults, depending largely on socioeconomic factors.47 Because of the high proportion of asymptomatic, anicteric, undiagnosed hepatitis in children, age-specific incidence rate for hepatitis A does not accurately reflect this problem in children and adolescents.

Accurate reporting in all age categories is probably no better than one out of every five cases. In children, this is further exaggerated by the low frequency of icteric infection which is 1:3 to 1:10.48 Anti-HA inci-
Hepatitis B may be contracted by young children in several situations. Newborn children may have obtained a hepatitis B infection from a hepatitis B carrier mother by transplacental transmission, direct transmission during birth process, and transmission by mother’s milk. Anicteric cases of hepatitis B are again exaggerated in children with a low frequency of 1:20. It appears that in hepatitis B socioeconomic factors also play a major role in epidemiology.

Currently the U.S. age group most vulnerable to viral hepatitis is young adults (20-24 years) followed by 15-19 year olds and 25-29 year olds. For hepatitis A there is a preceding but smaller peak of incidence in the five to nine year olds. Although there was a lack of hepatitis B cases in persons less than 15 years old prior to 1977, this is no longer true. Substantial geographic differences in incidence have always been reported, with the Pacific areas continuing to demonstrate the highest incidence in the U.S. Seasonal variation for viral hepatitis has, however, diminished remarkably.

Non-A, Non-B hepatitis accounts for approximately 20% of adult sporadic viral hepatitis. However, it accounts for 80% to 90% of all transfusion related hepatitis with the remaining being hepatitis B. The blood products necessary for replacement therapy are clearly recognized as the source of infection with increased risk from commercial donor blood and blood concentrates derived from large donor pools. Until specific serological testing on humans is available, epidemiological interpretations regarding incidence and age specificity are invalid.

Viral Hepatitis in Specific Patient Populations

Based upon the precarious asymptomatic and anicteric nature of viral hepatitis, it is not surprising that many people have no knowledge of their previous infection or present carrier state. Those stating a history of hepatitis usually do not know what type they had. Some people who have previously been refused health care because of admitting to a past episode of hepatitis often withhold this information when seeking health care again.

Studies specific for dental patients have confirmed these facts. There are, however, certain specific
patient populations noted to be at higher risk for hepatitis exposure (Table 5).

In 1972 approximately 70% of hepatitis B infection was precutaneous drug associated with the largest incidence in the 15-29 year age group. Drug abusers may have a carrier rate approaching 50%. Carrier states appear not to be maintained if drug abuse is eliminated. Up to 85% of drug abusers show serological evidence for HBV infection.

Numerous studies have confirmed that patients of institutions for the mentally retarded or severely disabled have a significant increase in the incidence of hepatitis A and hepatitis B. A range of 10% to 30% for prevalence of HBsAg in institutional populations has been reported, with patients having Down’s Syndrome approximating 50%. Patients undergoing hemodialysis (renal dialysis) have greater risk of hepatitis A and hepatitis B exposure. Serological evidence of HBV infection occurred in 50% of the patients and 34% of the staff at 15 national dialysis centers. The carrier rate in the patients, however, was 16%. Kidney and other organ transplant patients and other immunosuppressed patients (leukemia and any immunodeficient disease) have now been shown to be at greater risk for viral hepatitis. Dialysis and transplant carriers now account for approximately 33% of all accidental HBV infection in needlestick episodes.

Patients receiving transfusion, especially those receiving multiple transfusion and/or concentrate transfusion for coagulation disorders, are at great risk for transfusion related hepatitis. Transfusion related hepatitis is approximately 10%-20% hepatitis B and 80%-90% Non-A, Non-B hepatitis. Anti-HBs have been demonstrated in 90% of hemophilia patients treated with these blood products. Post-transfusion hepatitis B has been drastically reduced since initiation of routine blood bank screening of blood donors. Episodes, however, are still reported from blood donor units due to low-level infectious chronic HBsAg carriers in whom the HBsAg titers fluctuate and can escape the radioimmunoassay detection prior to blood donation.

An epidemiological investigation of post-transfusion hepatitis indicates that the frequency of both hepatitis B and Non-A, Non-B hepatitis can be reduced by reducing commercial blood donors in relation to volunteer blood donors (34.6% vs. 6.7%). With the advent of more sophisticated serological tests it might soon be possible to prescreen blood donors for identification and detection of low level titers of HBsAg in blood donors. Male homosexuals who have frequent intimate contact with different partners are at great risk for viral hepatitis. Some studies indicate that over 50% have some serological marker for HBV infection. Male homosexuals currently represent about 16% of the total HBsAg carriers in the U.S.

Other patient categories with higher exposure rates for viral hepatitis include patients with a history of familial association with the disease, chemotherapy or radiation therapy which suppress immunological defenses, tattooing, ear-piercing, acupuncture and other needlesticking procedures from non-professionals, and other liver diseases.

**The Refugee Crisis**

Epidemiological studies in Africa, Asia, and other tropical countries demonstrate that 5%-10% of the general population in these countries have chronic HBV infection with maternal-infant vertical transmission being the dominant mechanism maintaining this high prevalence. Most of these carriers are asymptomatic with no knowledge of previous exposure. There is a preferential persistence in the young males. Since the African or Asian carrier will usually circulate virus titers higher than North American HBsAg carriers, they are likely to be more infectious.

The recent influx of Southeast Asian refugees has dramatized not only the plight of these people but also their special health problems. With approximately 14,000 Indochinese refugees arriving in the United States monthly, an awareness of their specificity for viral hepatitis is mandatory. Over 150,000 Vietnamese refugees have now been resettled in the United States since 1975.

Current U.S. immigration and naturalization laws list the following conditions that do not exclude a refugee from entry if they are adequately treated: tuberculosis, leprosy, chronic alcoholism and drug addiction. By law, health screening of refugees prior to entry for tuberculosis, leprosy, and venereal disease is necessary. By law, viral hepatitis screening is not necessary. The U.S. Department of Health, Education and Welfare, however, is making a concerted effort to have these screenings performed upon entry into the U.S. Concern has been expressed regarding the trans-

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**Table 5. Viral Hepatitis in specific patient population.**

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug abusers (precutaneous)</td>
<td>Infection. Male homosexuals currently represent about 16% of the total HBsAg carriers in the U.S.</td>
</tr>
<tr>
<td>Institutionalized mentally retarded patients, especially those with Down’s Syndrome</td>
<td></td>
</tr>
<tr>
<td>Hemodialysis patients</td>
<td></td>
</tr>
<tr>
<td>Immunosuppressed patients</td>
<td></td>
</tr>
<tr>
<td>Post-transfusion patients, especially hemophilia patients</td>
<td></td>
</tr>
<tr>
<td>Male homosexuals</td>
<td></td>
</tr>
<tr>
<td>Refugees or immigrants from Africa, Asian, and other tropical countries</td>
<td></td>
</tr>
</tbody>
</table>
mission of tuberculosis and hepatitis to the population of the U.S. by these refugees.

Screening data on Indochinese refugees entering North America indicate that 12%-13% of them are positive for HBsAg with most individuals being asymptomatic and unaware of their chronic carrier state. This addition to the U.S. carriers represents only a 4% increase in total carriers for the U.S. and poses only a small risk to the general population. Since the refugees are often screened and identified as chronic carriers, the risk they represent may be less than that from unrecognized carriers in the general population and other high risk patient groups. Testing for HBsAg and entering the results in the refugee's medical record is recommended by the Center for Disease Control of each Indochinese refugee after arrival into the U.S. Since many of these refugees have significant dental disease, the dental professional may be the first health care person requesting this information. To assist with problems in language differentials, the U.S. Department of Health, Education and Welfare has established a toll free number (800-424-0212) for use by health care professionals.

Among adopted Vietnamese children studied, HBsAg and anti-HBs incidence was 27.3% and 30.3% respectively, with other family members having HBsAg and anti-HBs incidence of 0% and 13.5% respectively, with 23.1% of the children being positive for anti-HBs. Transmission of both hepatitis A and hepatitis B has been documented in family units with HBsAg-positive adopted children. Therefore, other family members, especially siblings of adopted refugees, should be considered a high-risk patient group.

Disease Transmission in Dental Practice

In practice, dental personnel are continually exposed to the blood of patients through minor hand abrasions or accidental inoculation by needle or instrument stick. Although not as effective as blood, saliva does appear to be the most efficient transmission vector of the non-parenteral body fluids. Since this fluid contaminates not only dental personnel, but most dental instruments and equipment, there is a need for genuine concern in dental practice regarding hepatitis transmission. Transmission in the dental office has been well documented. Therefore, the dental personnel should be keenly aware of aseptic technique and proper disease transmission safeguards. Numerous articles have been presented in the literature concerning the prevention of transmissible disease. A review of these articles should be paramount for dental personnel. This article is not meant to diminish their importance, but rather to emphasize that the subject of prevention of all transmissible diseases is in itself an entire area for review.

Exposure Protocol for Viral Hepatitis

Exposure to viral hepatitis may involve both parenteral and non-parenteral exposure. The importance of differentiating this exposure for an episode is often critical to exposure protocol. When in doubt regarding the episode of exposure for a particular type of viral hepatitis, assume the exposure regardless of parenteral versus non-parenteral transmission-caused-disease inoculation until proven otherwise.

Immune globulins are sterile, intramuscular solutions containing antibodies derived from human donor blood. One type of immune globulin (ISG) contains varying amounts of antibodies for hepatitis A and hepatitis B depending upon preparation after 1972. Titer levels of antibody for hepatitis B can attain 1:5000 by PHA in some preparations but must be greater than 1:64. The cost is approximately $15-$20 for one dose of this ISG. Neither hepatitis A nor hepatitis B has been transmitted by immune globulin. Immune globulin with high titers of anti-HBs (greater than 1:100,000 by PHA), prepared from donor pools preselected for these antibodies, are now designated as hepatitis B immune globulin (HBIG). The cost is approximately $150 for one dose of this HBIG. Neither hepatitis A nor hepatitis B has been transmitted by HBIG. Prophylactic administration of immune globulins has been both pre-exposure and post-exposure. Immune globulin preparations rarely cause adverse reactions and are not contraindicated during pregnancy.

Although exposure to hepatitis A usually involves non-parenteral, fecal-oral contamination, experimental animal studies have demonstrated parenteral transmission. The importance of this to health care professionals is unknown. Viral excretion of HAV will peak during late incubation and early illness, and drop rapidly with the onset of jaundice. Maximum infectivity will occur usually two weeks, then, before the onset of jaundice. When administered before, or within, one to two weeks after exposure to hepatitis A in appropriate dose, ISG prevents illness in 80-90% of those exposed. The use of ISG more than two weeks after exposure or after onset of clinical illness is not indicated. Pre-exposure prophylaxis may be indicated for exposure to non-human primates and for travel to some foreign countries. Post-exposure prophylaxis is recommended only in cases of close personal contact, institutional contact, and common source contact.

Exposure to hepatitis B is perplexed by the infectivity of its chronic carrier state. Furthermore, the value of HBIG versus the newer preparations of ISG has been debated over the past few years. There is evidence that HBIG, compared with low titer anti-HBs ISG, merely prolongs the incubation period and leads to reduced incidence of passive-active immunity.
Currently the U.S. Public Health Service Advisory Committee on Immunization Practices unequivocally recommends HBIG only for known exposure to blood containing hepatitis B virus by accidental needle-stick or by contact with mucous membranes.46 One dose of HBIG must be administered within a one-week period after this exposure, and a second identical dose administered one month after the first.

A single dose has been further recommended for infants within seven days of birth if born to HBsAg positive mothers with acute hepatitis occurring in the third trimester.46 It has been further recommended that all cases of percutaneous, oral, mucosal, sexual, or neonatal exposure to patients with acute hepatitis B, or in cases of time-limited exposure to chronic carriers having markers denoting infectivity, be administered the HBIG regimen.117

Pre-exposure prophylaxis has been, at times, recommended for patients and staff of hemodialysis units and custodial staffs of institutions for the mentally retarded.46 It should be noted, however, that only 6% of the persons who are accidentally contaminated with needle-stick blood from an HBsAg positive donor develop hepatitis B.116 If HBIG is not available for use in the above-mentioned episodes, ISG can be administered similarly. The theoretical advantage of HBIG over ISG remains controversial.117 The administration of ISG within one week after exposure and again in 1 week after exposure.

Exposure to Non-A, Non-B hepatitis is also perplexed by lack of serological identification and existence of a chronic carrier-state that is infective. Although not scientifically based, any exposure dictates the administration of ISG within one week. It appears from limited studies that ISG has been of benefit in reducing the total incidence of hepatitis, the icteric cases, and the chronic sequelae.116, 118 (Table 6).

**Table 6.** Exposure protocol for viral hepatitis.

<table>
<thead>
<tr>
<th>Type</th>
<th>Immunoglobulin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatitis A</td>
<td>ISG within 1-2 weeks after exposure.</td>
</tr>
<tr>
<td>Hepatitis B</td>
<td>HBIG within 1 week after exposure and again in 1 month. If HBIG is unavailable, ISG may be substituted.</td>
</tr>
<tr>
<td>Hepatitis/Non A, Non B</td>
<td>ISG within 1 week after exposure.</td>
</tr>
</tbody>
</table>

Management of Children in the High Risk Groups

Management of Children and adolescents in the high risk groups may require a specific office/facility protocol dependent upon the level of sophistication of disease transmission prevention. Optimally, all high risk patients should have serological identification and be afforded the highest level of disease transmission prevention. This is often not possible or practical in all circumstances. If serological identification is unknown or uncertain for a given patient in the high risk group, it is advisable to consider this patient seropositive and able to transmit viral disease. The highest level of prevention of disease transmission should therefore be adopted.

Approximately half of seropositive patients may have progressive chronic liver disease with continued hepatocellular necrosis. This is determined by evaluation of liver enzyme function. If oral surgical procedures are to be done on seropositive patients, not only should the highest level of preventive disease transmission procedures be utilized, but liver function should be ascertained to prevent possible problems in bleeding and drug metabolism and/or detoxification. If drug therapy requiring metabolism and/or detoxification in the liver is anticipated on a seropositive pa-
tient, liver function should likewise be ascertained prior to therapy.


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References

HEPATITIS: CRISIS IN PEDIODONTICS

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