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Viral hepatitis and the surgeon

G. Y. MINUK, A. J. COHEN, N. ASSY & M. MOSER

Liver Diseases Unit, Departments of Medicine, Surgery and Pharmacology, University of Manitoba, Winnipeg, Manitoba, Canada

Abstract

Background. Viral hepatitis is an infection of the liver caused by one or more of six known (HAV-HGV) hepatotropic viruses. It is a common problem among health care workers and their patients. Surgeons are at particular risk of both acquiring and transmitting some of these viruses from and to their patients. Unfortunately, specific immunoprophylaxis for viral hepatitis is presently limited to protecting against the spread of hepatitis A and B viral infections, leaving a high degree of vigilance and careful surgical technique as the only means available to prevent the transmission of other viruses relative to the surgeon. The purpose of this paper is to review the various forms of viral hepatitis including the nature of the virus, serologic testing, clinical features, epidemiology (with specific reference to those issues that arise in surgical practice), treatment and prevention.

Key Words: *Viral hepatitis, hepatitis, liver disease, hepatitis A, hepatitis B, hepatitis C, epidemiology*

Hepatitis A

The hepatitis A virus is a 27 nanometre single-stranded RNA virus that belongs to the Picorna family of viruses [1]. The serologic diagnosis of an acute hepatitis A infection is relatively straightforward. A positive IgM antibody to hepatitis A virus (IgM anti-HAV) indicates that infection with this virus has taken place within the past 3–6 months [2]. Shortly after the appearance of the IgM anti-HAV, IgG anti-HAV (often referred to as "total" anti-HAV) appears in the circulation. Unlike IgM anti-HAV, the IgG antibody persists for decades and indicates long-standing immunity against future HAV infections. Thus, individuals with acute HAV infections are IgM anti-HAV positive and those who are susceptible to HAV and therefore candidates for immunoprophylaxis are IgG anti-HAV (or total anti-HAV) negative.

The majority of acute HAV infections are subclinical [3]. When symptoms do appear, they tend to be mild and nonspecific in nature. Most commonly they include fever, general malaise, fatigue, abdominal discomfort and change in bowel habits. When severe, dark urine, pale stool and jaundice may appear. The severity of acute HAV infections is proportional to the age of the patient, with younger patients tending to have milder disease than the elderly [4]. Indeed, overall mortality rates are only 0.1% in the general population as opposed to 1–2% in the elderly [5]. Hepatitis A infections do not progress to chronic liver disease (defined as hepatitis persisting beyond 6 months). Rarely, when the acute hepatitis is severe, patients may develop cholestasis (manifest by pruritus) which can persist for as long as 1 year following the initial injury [6].

For all intents and purposes, HAV is spread through fecal contamination of food or drinking water [7]. Although the virus is present in blood, the limited amount of circulating virus and short duration of viremia render parenteral transmission of this virus extremely uncommon. Feces of infected individuals tend to contain the virus for a 2-week period prior to the onset of illness and for at least 2 weeks and perhaps as long as 3 months thereafter [8]. There are several reports of patients in pediatric hospitals and neonatal nurseries in particular who have transmitted HAV to health care workers [9–12]. Nurses appear to be at greatest risk due to their responsibility for changing diapers [13–17]. To date, there have not been reports of surgeons (colorectal or other) acquiring HAV from infected patients. Similarly, there have not been reports of surgeons transmitting HAV infections to their patients.

Treatment of acute HAV infections is supportive, as the majority of cases resolve spontaneously without residual damage or sequelae. In those rare cases that progress to fulminant hepatic failure, liver transplantation should be considered. Both passive and active immunoprophylaxis for HAV is available [18]. Passive immunoprophylaxis consists of immune serum globulin (ISG) which provides prompt protection against HAV and should therefore be administered as soon as possible to susceptible individuals identified in hospital

Correspondence: G. Y. Minuk, John Buhler Research Centre, University of Manitoba, Room 803F, 715 McDermot Avenue, Winnipeg, Manitoba R3E 3P4, Canada. E-mail: gminuk@cc.umanitoba.ca

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Table I. Markers of hepatitis B infection and immunity

Marker	Indicates
HBsAg (surface antigen)	Hepatitis B infection
Anti-HBs	Exposure to HBsAg in the past, immunity to HBV infection
HBcAg (core antigen)*	Presence of replicating viral particles in hepatocytes
IgM anti-HBc	Exposure to the intact virus within 6 months
Anti-HBc	Exposure to the intact virus at some time in life
HBeAg (early antigen)	Active viral replication
Anti-HBe	Nonreplicating virus
HBV-DNA $> 10^5$ copies/ml	Active viral replication

* Limited to testing in the liver, not serum.

outbreaks. The half-life of ISG is relatively short and therefore protection is limited to 3–4 months duration. Commercially available HAV vaccines have been developed which result in more long-term protective antibodies (at least 5 years). Most vaccinated individuals develop protective antibody levels after a single administration of a potent formulation [19]. Although the response to HAV vaccines is prompt, ISG is still recommended for post-exposure prophylaxis. The response rate to the HAV vaccine is excellent, often in the $>95\%$ range [20].

Hepatitis B

The hepatitis B virus is a 42 nanometre, double-stranded DNA virus that belongs to the Hepadna (Hepatitis DNA) family of viruses [21]. Although the serology associated with this virus is often considered complex, on most occasions, the following interpretations are valid (Table I) [22]. The presence of hepatitis B surface antigen (HBsAg) in the blood indicates the presence of an HBV infection. On the other hand, antibody to HBsAg (anti-HBs) indicates immunity to HBV infection. The antibody to the hepatitis B core antigen (anti-HBc) indicates that the individual has been exposed to the intact hepatitis B virus at some time in their life. IgM anti-HBc indicates that the exposure took place within the past 3–6 months. The presence of early antigen (HBeAg) or antibody to HBeAg (anti-HBe) reflects viral replicative activity such that HBeAg positive patients have active viral replication (all body fluids should be considered potentially infectious), whereas HBeAg negative but anti-HBe positive patients usually have inactive viral replication (generally, only the patient's blood is considered infectious to others). Unfortunately, there are sufficient exceptions to the latter interpretation (in particular a mutation to the pre-core gene that results in the absence of serum HBeAg despite active viral replication and viremia) that all body fluids from an HBsAg positive individual regardless of their HBeAg/anti-HBe status, should be considered infectious [23]. Quantitative HBV-DNA testing (which tends to parallel HBeAg results) is a more accurate marker of viral load and infectivity.

The natural history of HBV infections in adults can be described in terms of three phases—acute hepatitis, chronic hepatitis and an "inactive disease" state—with the majority of adult patients only experiencing the first phase. By definition, the acute hepatitis phase represents the first 6 months of the infection. During this phase, patients are often asymptomatic or have nonspecific complaints similar to those described with mild HAV infections [24,25]. HBsAg, IgM anti-HBc and HBeAg testing are frequently positive during this phase of the illness. In 90–95% of adults the acute hepatitis resolves spontaneously and patients develop natural immunity (seroconvert from HBsAg to anti-HBs positive). The more severe the acute hepatitis, the more likely this is to occur [24]. Unfortunately, only 5–10% of infants follow that course. Children and adolescents have an intermediate likelihood of spontaneously resolving their infection. If the HBsAg remains positive for 6 months after the onset of the illness or is documented to be positive on two occasions 6 months apart or is positive on one occasion but in the absence of an IgM anti-HBc then the individual almost certainly has a chronic HBV infection.

The second phase of the illness is referred to as the chronic hepatitis phase. During this phase, which often lasts 7–10 years in North American/European adults, the disease is active with originally symptomatic patients continuing to have symptoms (albeit less severe) and liver enzyme abnormalities tending to be abnormal in a hepatocellular injury pattern (serum ALT levels disproportionately elevated compared with serum alkaline phosphatase levels). The HBeAg is often positive indicating active viral replication. Eventually, symptoms resolve, liver enzymes return to normal or near-normal values and the patient seroconverts from HBeAg to anti-HBe positive, indicating entry into the third and final phase of the infection, the "inactive disease" state.

The inactive disease state tends to last from years to decades before the HBsAg spontaneously seroconverts from positive to negative and anti-HBs appears marking resolution of the infection. During this phase, patients are largely asymptomatic, have normal or near-normal liver enzyme levels, are HBeAg negative but anti-HBe positive, have low or undetectable levels of HBV-DNA in the blood and if biopsied, have little evidence of active inflammation.

The major complications of the hepatitis B carrier state—cirrhosis and hepatocellular carcinoma—tend to occur in approximately 30% and 15% of cases respectively [26,27]. When cirrhosis does develop, it is often present at the end of the chronic hepatitis phase, whereas hepatocellular carcinoma tends to be diagnosed during the late chronic hepatitis or inactive disease phase of the infection.

Because the majority of acute HBV infections resolve spontaneously and viral replication is already limited in those with inactive disease, treatment is confined to the chronic hepatitis phase of HBV infections. The only licensed treatments presently available are interferon, lamivudine and adefovir. Response rates to these agents in terms of converting an actively replicating virus (HBeAg positive) to a nonreplicating state (HBeAg negative and anti-HBe positive) are approximately 30–40%, but lower in those who acquired their infections earlier in life [28–30]. Response rates are highest in patients with low baseline levels of viral replication and those with biochemical or histologic evidence of active hepatic inflammation [30].

Although maternal–infant transmission is the most common route of HBV infections in the world at the present time, in industrialized nations, parenteral drug abuse and needle stick exposures represent significant high-risk activities [31]. Needle stick exposures involving blood from an individual with high levels of viral replication (HBeAg positive or high HBV-DNA levels) tend to result in HBV infections occurring in approximately 60% of cases, whereas when the infection in the source is not actively replicating (HBeAg negative or low HBV-DNA levels), the figure falls to approximately 30% [32]. In addition to the size of the inoculum, features of the needle itself, hollow or solid, appear to be important factors influencing the risk of viral transmission [33,34].

The prevalence of HBV infection among health care workers is 3–5-fold higher than that of the general population with surgeons (particularly orthopedic surgeons and gynecologists) and dentists having the highest reported rates [35,36]. The annual rate of HBV infections occurring in health care workers ranges between 0.5 and 5% compared with an annual rate of 0.1% in the general population [37]. The risk of transmission from patients to health care workers is related to several factors including the degree of exposure to patient blood or body fluids, blood-contaminated sharps and the duration of employment [38]. Thus, those at highest risk of infection are surgeons and surgical house officers, laboratory technicians, blood bank workers, assistants in surgery, pathologists, and anesthetists. An additional risk factor for health care workers is the underlying prevalence of HBV in the patient population with higher risks in urban hospitals and tertiary care centers than those in rural and primary care hospitals [39].

Over the past 25 years, there have been approximately 50 reports of HBV transmission from health care workers to patients in the United States and Europe and 400 worldwide [40]. A large majority of these cases involved either surgeons or dentists. The number of patients infected by the health care worker ranged from 1 to 55. Although HBV transmission from dentists to patients have decreased in recent years, the frequency of transmission from surgeons to patients remains unaltered [40]. Between 1984 and 1993, 10 clusters associated with HBV-infected surgeons were documented in England with a rate of transmission to patients of 0.3–9% [41]. In several investigations, the two major risks of transmission identified were the presence of HBeAg and/or high levels of HBV-DNA in the health care worker and the degree of invasiveness of the procedure performed (dental extraction > dental prophylaxis, hysterectomy > curettage > cardiothoracic surgery > other surgery) [42]. Other minor factors included: glove failure, skin lesions, long duration of the operation and the use of blood products. While in the majority of cases there were no identifiable causes for the surgical outbreaks, the most likely explanations were thought to be related to needle sticks incurred while suturing and inadvertent cuts through gloves while securing knots [43]. For these reasons the following are thought to decrease the risk of intraoperative transmission: use of double latex gloves, changed hourly, enclosed hood and face-masks and operative isolator with umbilical-cord aspirator, knee-length impermeable gowns, a combination of shoe-covers with coverage to the knee and disposable drapes. Inadvertent pricks or cuts should be bled and washed immediately with iodine, soap and water. The injured person should then be considered for immunoprophylaxis [44].

Both passive and active immunity against HBV are available. The former consists of high titer anti-HBs referred to as hepatitis B immunoglobulin (HBIG). As with ISG for HAV, HBIG for HBV provides prompt protection and should be given as soon as possible to susceptible individuals following viral exposure. Generally, when the exposure was of a nonparenteral form (sexual contact, maternal–infant, etc.) HBIG is considered effective for a period of approximately 2 weeks post-exposure [45]. On the other hand, when the exposure was parenteral (needle stick injury or shared drugs) HBIG efficacy is limited to only 1 week post-exposure [45]. The duration of protection provided by HBIG is approximately 3–4 months.

Active immunity against HBV (the HBV vaccine) can be acquired through a series of three vaccinations with recombinant HBV vaccines. Approximately one-third of individuals respond to the first dose of the vaccine (time 0), two-thirds following dose no. 2 at 1 month and 95% following dose no. 3 at 6 months [46]. Males and individuals over the age of 40 tend to respond less well to the vaccine than females and younger individuals [47]. For maximum effect, the HBV vaccine should be administered intramuscularly. As a result, injections in the deltoid rather than hip (where inadvertent injection into adipose tissue is more likely to occur) are suggested. Individuals at high risk of HBV exposure such as health care workers should have their response to the vaccine documented by testing for anti-HBs approximately 2–4 weeks after the final vaccination innoculation. Antibody levels >50 IU and perhaps >10 IU should be considered protective and indicative of long-term protection despite weakening

or loss of antibody titers over time. This long-term protection in the absence of appreciable antibody titres relates to the amnestic response of vaccine responders to subsequent viral exposures [48]. Thus, booster doses of the vaccine at 5 years or thereafter are not presently advocated. Nonresponders to a complete series of HBV vaccinations should be reassessed to ensure that they are not carriers of the virus (HBsAg positive) and if negative, revaccinated with a second series of either recombinant or the original plasma-derived vaccine (if available). The response rate to a second vaccination series is approximately 10–20% [49].

Given the safety and efficacy of the vaccine, all healthy care workers, as well as other high risk individuals, are strongly encouraged to undergo vaccination. It is in the best interest of the health care worker exposed to HBV to present immediately for assessment so that appropriate post-exposure prophylaxis can be administered if needed (see Table II). If not previously measured, anti-HBs titers should be determined, even in those having received a full course of HBV vaccine. Individuals who have developed anti-HBs (and anti-HBc) as a result of natural infections or anti-HBs as a result of immunization in the past can be assumed to be immune and require no further treatment. Individuals who have never been immunized, those in whom a response to the vaccine has not been documented or those who have been documented to be nonresponders should receive HBIG as soon as possible and again 1 month later. The former group (never immunized) should be offered the first of the vaccination series at the same time but at a different injection site. The remainder of the vaccination series should proceed accordingly. For those in whom an anti-HBs response was not documented following previous vaccination, approximately 6 months after the second HBIG administration (when passively administered anti-HBs has cleared), the individual should receive a single injection of HBV vaccine. If the anti-HBs titre is high (>50 IU) 1–2 weeks thereafter, it can be assumed that the individual had responded to the previous vaccine series and no further vaccination is required. If the anti-HBs is undetectable or only present in low titer (<10 IU) the full course of the vaccination should be completed.

Table II. Post-exposure prophylaxis for hepatitis B

Status	HBV vaccine	HBIG
Vaccinated; adequate titers of anti-HBs documented in the past	Not required	Not required
Vaccinated; adequate titers of anti-HBs not previously documented	Booster, recheck in 1 month	One dose
Unimmunized	Full course (day 0, 1 and 6 months)	Two doses; day 0 and 1 month post exposure

Based on: CDC recommendations of the Immunization Practical Advisory Committee: Post operative prophylaxis of HBV. *Ann Intern Med* 1984;**101**:351–4.

Antiviral therapy for health care workers who seroconvert from HBsAg negative to positive as a result of a recent exposure should be withheld until the individual has been documented to be HBsAg positive on two occasions a minimum of 6 months apart (i.e. demonstrated chronicity). Thereafter, considerations regarding treatment such as indications, contraindications, choice of antiviral agents, duration of therapy, etc. are similar to those for other chronic HBV carriers [50]. Antiviral therapy may also be of value in keeping serum HBV-DNA levels low or undetectable as a means of limiting the risk of transmission from health care workers to others [51].

Finally, concerns regarding documentation of the health care worker's HBV status and their susceptibility to HBV infection can be alleviated somewhat by anti-HBc testing in that a positive anti-HBc result indicates that the individual has already been exposed to the hepatitis B virus (whether a carrier or immune can subsequently be determined by their family physician) and would not benefit from either passive or active immunization.

Hepatitis C

The hepatitis C virus is a 60 nanometre, single-stranded RNA virus [52]. The diagnosis of HCV infection is most often based on a positive anti-HCV in a patient with a history of viral exposure (previous blood transfusions or intravenous drug use) and elevated liver enzyme tests. Many of the problems initially associated with anti-HCV testing have since been resolved. Specifically, third generation testing (which incorporates a wider spectrum of viral antigens in the assay) has largely eliminated the high frequency of false-positive results, particularly in patients with autoimmune disorders and hypergammaglobulinemia [53]. Moreover, the delay in the appearance of anti-HCV with acute infections (12–16 weeks) can now be circumvented by testing for HCV-RNA, which is often present within 2 weeks of infection [54]. HCV-RNA testing also helps to distinguish the small number of patients ($<20\%$) who resolve their acute HCV infections and normalize their liver enzyme abnormalities but remain anti-HCV positive. Finally, third and fourth generation anti-HCV and HCV-RNA testing can be used to establish the diagnosis in immunocompromised individuals who do not develop or develop only limited anti-HCV responses [54].

Unlike HBV infections, HCV has a high (60–80%) propensity to progress to chronic liver disease [55,56]. As with HAV and HBV, the majority of infections are subclinical and when symptoms do appear they tend to be relatively nonspecific [56]. Fulminant hepatitis appears to be uncommon in North American forms of the disease [57]. The percentage of patients

progressing to cirrhosis and/or hepatocellular carcinoma remains to be determined. Retrospective follow-up of patients thought to be infected 20–30 years earlier suggests that 20–30% of HCV infections will progress to cirrhosis during that time period [56]. These findings have led some investigators to suggest that the risk of cirrhosis increases by 10% per decade. Similarly, the risk of hepatocellular carcinoma remains to be determined but appears to be as high as in patients with hepatitis B infections [58]. To date, hepatocellular carcinoma developing in HCV-infected patients without cirrhosis is considered to be extremely rare [59].

Until the early 1990s blood transfusions represented the major source of HCV infections in the developed world [60]. Following initiation of donor screening, parenteral drug abuse has emerged as the most common source of HCV infections [61]. The rates of transmission through other activities such as sexual promiscuity, homosexual contact, tattoos, ear and body piercing, etc., appear to be relatively uncommon but may serve as surrogate markers of higher risk activities [61,62]. Maternal–infant transmission of HCV occurs in approximately 5% of births, but more often (10–15%) when the mother is immunocompromised or in the acute stage of the infection (initial 6 months) at the time of delivery [63]. Between 10% and 40% of patients have no identifiable risk factor on careful, physician to patient interviews [61,64,65].

The risk of health care workers acquiring HCV infections from their patients has not been defined but appears to be relatively low. Seroepidemiologic surveys of hospital staff have revealed anti-HCV prevalence rates similar to those of the general population (1–1.4%) [66]. The prevalence of HCV infections among patients is variable and site-dependent [67–69]. The average risk of HCV infection occurring after needle stick injury involving HCV-infected blood has ranged between approximately 2.5% and 10%, which is significantly lower than the rates associated with HBV exposure but approximately 10-fold greater than that associated with HIV exposures (0.3%) [66,70]. Thus, it appears that health care workers have a significantly lower occupational risk of acquiring HCV infections when compared with HBV infections but higher than that for HIV infections.

When nosocomial transmission of HCV does occur, it is often from health care workers to patients and involves patients undergoing surgical procedures [71,72]. Factors that appear to facilitate transmission include needle stick injuries, inapparent glove perforations related to securing sutures and perhaps active liver disease in the source with high levels of viremia [72,73]. Interventions that are thought to limit the risk of transmission include: selection of less invasive surgical approaches (e.g. laparoscopic procedures), alternatives to needles and sharps (e.g. tapes, glues, staples), sharps with injury-preventive features (e.g. blunted suture needles), instruments rather than hands for retraction, avoiding the simultaneous presence of hands from two or more surgeons in the operative field and using a neutral zone for passing sharps rather than from hand to hand [74].

Presently, recombinant or pegylated interferon alone or in combination with ribavirin is the only approved therapy for chronic hepatitis C infections. In unselected cases, approximately 55% of patients will respond to 6–12 months of therapy with normalization of liver enzyme abnormalities and loss of HCV-RNA [75–78]. With selection of patients (those infected with genotypes 2 or 3, low viral load, early disease and limited hepatic inflammation) long-term response rates to interferon treatment are improved [79].

Passive and active immunoprophylaxis against HCV have yet to be developed, leaving standard infection control measures, a high degree of vigilance, proper surgical techniques, and screening of blood donors as the only effective means of limiting the spread of this virus in the hospital setting [80]. When such efforts fail and a clear exposure to HCV-contaminated blood or fluids has occurred, the individual should be screened for underlying HCV infection (baseline anti-HCV) and tested for HCV-RNA by PCR at 1, 3 and 6 months thereafter. If HCV-RNA is detected and remains positive for three months, antiviral therapy with standard recombinant or perhaps, pegylated interferon plus ribavirin (trials presently underway) for 4–6 months should be undertaken unless contraindications to one or both of these agents exist [81,82].

Hepatitis D

The hepatitis D agent is a single-stranded RNA viroid (incomplete virus) which requires the replicative capacity of HBV to infect and replicate in humans [83]. Thus, only patients simultaneously co-infected with HBV and HDV or those with pre-existing chronic HBV infections who are subsequently exposed to HDV can acquire HDV infections. The disease is most often diagnosed by the presence of high titered antibodies to HDV (anti-HDV) in a patient with serologic evidence of HBV infection (HBsAg or IgM anti-HBc positive) [84]. Often, the HDV patient has a particularly severe form of hepatitis which more rapidly progresses to cirrhosis yet has little evidence of active HBV replication (HBeAg negative). HDV also appears to be responsible for 2–20% of previously considered fulminant hepatitis B infections [85].

HDV infections are most often spread via parenteral transmission [86]. Sexual or intimate contact and maternal–infant transmission are less common and largely confined to certain areas of the world [87].

There are no reported cases in the English literature of HDV transmission occurring from patients to health care workers or vice versa.

Treatment for HDV infection is as outlined for HBV infections in that eradication of the latter will result in clearance of the former.

While passive and active immunoprophylaxis against HBV in HBV-susceptible individuals will also protect against HDV infection, there is no effective HDV immunoprophylaxis for the established HBV carrier who remains at risk of acquiring HDV infection.

Hepatitis E

The hepatitis E virus is a single-stranded RNA virus belonging to the Calici virus family [88]. A positive anti-HEV test is used to establish the diagnosis [89]. Like HAV, HEV does not cause chronic liver disease and is spread by fecal/oral transmission [90–92]. Unlike HAV and other hepatatropic viruses, the mortality of acute HEV infections in pregnant women is high (15–20%) [93]. The reason(s) for this finding has yet to be determined.

Regarding nosocomial transmission, there is a single case report of a pregnant woman returning from India to South Africa with fulminant hepatitis where the HEV was subsequently transmitted to a nurse (confirmed serologically) and presumed to have also been transmitted to an additional nurse and physician (not serologically confirmed). The mode of transmission in this case (or cases) was thought to have been related to the staff members having assisted the patient from a bedpan and/or having transferred her to the operating room for repair of vaginal lacerations [94]. There have been no case reports of health care workers transmitting HEV to their patients.

Because acute HEV infections resolve spontaneously and do not progress to chronic hepatitis, antiviral therapy is less urgent. However, patients with fulminant hepatic failure secondary to HEV infections should be considered for liver transplantation.

Passive and active immunoprophylaxis have yet to be developed for HEV infections.

Hepatitis G

Recently, a new RNA virus (and closely related variants) was discovered by independent groups of investigators [95–97]. This agent is referred to as the hepatitis G virus or HGV. One of the groups responsible for its discovery employed sera from a surgeon (initials GB) who had developed jaundice (attributed to non-A, non-B hepatitis but was subsequently tested and found to be negative for HCV-RNA) as the essential reagent in their assay. Although there is some homology between HGV and HCV it is not sufficient to warrant considering HGV a new HCV genotype. Presently, the diagnosis of HGV infection is established by documenting the presence of HGV-RNA in the serum [98].

Little is known of the natural history of HGV infections [99]. Data suggest that infection with this virus is associated with but not responsible for as many as 20% of previously undiagnosed fulminant, acute and chronic hepatitis as well as "cryptogenic cirrhosis" cases [100]. Concerns regarding nosocomial transmission of HGV have largely been limited to the dialysis setting [101–103]. However, postoperative HGV infection has also been described [104]. Fortunately, such cases are infrequently associated with biochemical evidence of hepatitis.

The high prevalence of this virus in the general population (estimated to be approximately 1--2%) suggests that the route of transmission may extend beyond blood transfusions and intravenous drug usage to include sexual and perhaps maternal–infant and other forms of nonparenteral transmission [105]. The risk of transmission from patients to health care workers and vice versa beyond the setting of hemodialysis units remains to be determined.

Conclusion

Hepatitis viruses can be transmitted from patients to health care workers and vice versa. While the majority of these infections involve HBV and HCV, other hepatotropic viruses as well as viruses that are non-hepatropic such as HIV (for which effective post-exposure prophylaxis is available) should also be considered. Needle stick injuries and suturing are the most common routes of transmission within the hospital setting. Environmentally mediated infections of hepatotropic viruses (such as blood contamination of countertops and equipment dials or switches, etc.) are rare, with only HBV having a sufficient viral load in the blood and resistance to physical insults to render it a risk for such forms of transmission. Although therapeutic and preventive measures have improved significantly in recent years, viral hepatitis remains a common and dangerous infection to surgeons. Until new, more effective interventions are developed, a greater awareness of the viruses, the diseases they cause and how to minimize the risks of their transmission remain the best defense for health care workers, other high-risk groups and patients.

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