Review Article

Viral hepatitis and the anaesthetist

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Viral hepatitis is a constant hazard to all operating room personnel. The anaesthetist should avoid contact with patients' blood and saliva as much as is possible.

Hepatitis A (HAV) is spread mainly by faecal/oral contact. Carriers are almost non-existent in this disease and the main importance to the anaesthetist is that he may contact a patient who is acutely infected or one who is incubating HAV. Diagnosis of postoperative hepatic dysfunction may then be a problem. Prophylaxis with Gamma globulin is also stressed.

Hepatitis B (HBV) and Non-A Non-B hepatitis (NANB) have a high incidence of carriage, and are spread mainly by blood contact. The groups of patients whom the anaesthetist should especially be aware of are reviewed, as is prophylaxis using Hepatitis B Immune Globulin and the recently introduced Hepatitis B vaccine. NANB continues to be a diagnostic problem, its diagnosis being mainly by exclusion of other causes of viral hepatitis. It appears to be responsible for more than 90 per cent of cases of posttransfusion hepatitis and more than one virus may be involved.

Key words

LIVER: viral hepatitis, hepatitis A, hepatitis B, hepatitis Non-A Non-B; OCCUPATIONAL HAZARDS: hepatitis.

Viral hepatitis is a disease which causes an enormous amount of morbidity and mortality.¹ There are several types with which the anaesthetist should be familiar, some of which represent occupational hazards while others are part of the differential diagnosis in the patient who develops hepatitis postoperatively.

Viral hepatitis is usually caused by Hepatitis A virus (HAV), Hepatitis B virus (HBV), Non-A Non-B Hepatitis viruses (NANB), Cytomegalovirus (CMV) or Epstein-Barr Virus (EBV) and less commonly found associated with other viruses (Herpes simplex virus, yellow fever virus, etc.). The first three of these are of special importance to the anaesthetist and shall be discussed. The purpose of this review is to examine current knowledge with regard to viral hepatitis as it may affect the anaesthetist. Treatment of acute hepatitis, which is mainly symptomatic, will not be discussed, but prophylaxis will be stressed. The reader is also referred to a recent review on "The liver and anaesthesia."²

HAV

Hepatitis A infection is predominantly found in children but accounts for 20 per cent of cases of viral hepatitis in adults in the U.S.A. and other industrialized countries.³ The disease can occur in epidemic or in sporadic form following an incubation period of fifteen to forty-five days.

The virus is spread typically by the faecal/oral route and about 50 per cent of infections may be asymptomatic. The chronic carrier state is non-existent and chronic hepatitis with cirrhosis is very rare.⁴

The disease is usually brief and self-limiting but its importance lies in the fact that the patient may be incubating it at the time of the anaesthetic and that anaesthetic agents, e.g., halothane, may be suspected of contributing to the liver injury. Faeces

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FIGURE 1 Clinical, serological and biochemical course of typical type A viral hepatitis.

from infected patients is most infectious during the incubation period before acute symptoms appear. Acute exposure of the anaesthetist to the patient with acute hepatitis A should result in him/her being given a single dose of gamma globulin, within two weeks of exposure. This provides a good degree of protection, which lasts for approximately three months.

The diagnosis of hepatitis A is made by a history of contact with the disease, either via another case or contaminated food or water. HAV appears to be rarely if ever transmitted by blood transfusion.⁵ Laboratory diagnosis can be made by examining the patient's serum for HAV specific IgM antibodies which appear in the acute phase and persist for a few months thereafter.⁶ The presence of antibody to HAV in the IgG fraction indicates past infection and immunity and this remains for life. Stool anti HAV IgA parallels the serum IgG response and is not contributory for early diagnosis. Figure 1 illustrates the clinical, serological and biochemical course of typical type A hepatitis. Because patients with clinical symptoms (jaundice, dark urine, etc.) usually no longer have virus in their faeces, it is not usually productive to examine stool for virus.

HBV

Since the discovery of Hepatitis B surface Antigen (HBsAg, previously called Australia Antigen) by Blumberg in 1964⁷ and demonstration of its association with Hepatitis B in 1967,⁸ several specific markers of HBV infection have been described.⁹

The anaesthetist should appreciate that there are

at least three immunologically distinct antigen-antibody systems associated with Hepatitis B virus. The mature virus, called the Dane particle consists of an inner core and outer shell. HBsAg makes up the outer shell and core antigen (HBcAg) the inner core. HBsAg may appear as spherical particles 22 nm in diameter or long filamentous rods. HBsAg is found in large amounts in the blood of acutely infected patients and chronic carriers. The usual sequence of antigenic events that occur with this infection is illustrated in Figure 2. After a long incubation period of about three months the acutely infected patient will be antigenemic (HBsAg positive) and this eventually disappears to be replaced by antibody to surface antigen (anti HBs). There is a tendency toward shorter incubation periods with large viral dosage¹⁰ and long incubation periods with small inocula.¹¹

The onset of acute Hepatitis B, unlike Hepatitis A, is often insidious with symptoms such as malaise, joint pains, fever and skin rash (10–20 per cent of cases). Anti HBc appears early in the infection and in most persons will remain for life. An antigen found clearly associated with the core, Hepatitis Be antigen (HBeAg) can usually be measured during HBsAg presence and appears to be an indication of infectivity.¹² This antigen is replaced by antibody (anti HBe). During the HBsAg positive stage an enzyme responsible for virus replication (DNA polymerase) can also be measured. Type B hepatitis, unlike hepatitis A, shows a tendency to progress to the chronic carrier state in five to ten per cent cases. This is more likely when

FIGURE 2 Serological markers following onset of an acute viral hepatitis B infection.

prolonged low-grade symptoms with mildly abnormal liver function tests are seen and in subclinical infection.^{13,14} The profile of a chronic carrier would include both HBsAg and anti HBc. The persistence of HBeAg in a chronic carrier may be a predictor of future problems (chronic liver disease, hepatocellular carcinoma).

It is important to note that since the introduction of routine testing of donor blood by a sensitive radioimmunoassay for HBsAg, the incidence of post-transfusion hepatitis B infection has markedly decreased, but has not been completely eliminated. It is now recognized that persistent and, in particular high titres of anti HBc, in the absence of detectable HBsAg and anti HBs (Figure 2; 12–14 weeks) may be an indicator of continued viral replication.^{15,16} This may explain the small number (five to ten per cent) of cases of hepatitis B which still occur after transfusion with supposedly HBsAg negative blood.

Approximately ten per cent of acutely infected patients become chronic carriers and this may be much higher when infection takes place from mother to newborn.

It has been estimated that there are probably between 150 and 200 million carriers of hepatitis B in the world today.¹⁷ Many of these individuals are totally asymptomatic and have normal liver function tests, findings which are good presumptive evidence that there is no significant liver disease.¹⁸

A carrier is defined by WHO (World Health Organization) as a person with a HBsAg positive serum for more than three months.¹⁹ The percentage of carriers in a population varies tremendously from country to country and indeed even between areas within a country. Serological surveys on selected groups in different geographical regions of the world have shown that the prevalence of HBsAg in apparently healthy individuals in North America, Western Europe, Australia and New Zealand is low (0.1–0.6) per cent of the population, with intermediate rates in the Mediterranean countries, Eastern Europe and the Middle East, whereas in tropical Africa, Southeast Asia and the Far East as many as 5 to 20 per cent of the "healthy" population may carry this antigen.^{20,21} These carriers are all capable of infecting persons who come into contact with their blood or secretions. Body fluids other than blood, e.g., saliva, mucus, urine, may periodically contain significant concentrations of antigen.^{22,23} Anaes-

FIGURE 3 Possible outcomes in hepatitis B viral infection.

thetists and surgeons therefore, together with other operating room personnel are exposed to this risk.

The incidence of asymptomatic carriers of hepatitis B antigen in North America has been estimated at 0.1 to 0.6 per cent of the population. Therefore, a full-time anaesthetist in North America who administers 1,200 anaesthetics per year (a conservative estimate) may be expected to have to deal with one to six carriers per year, most of whom would be asymptomatic. A long list of patients who should be treated with suspicion and tested for HBsAg preoperatively might be constructed. These might include patients on haemodialysis, those recently from high risk countries, those who have received multiple blood or blood product transfusions, patients with leukaemia or reticuloses, drug addicts, prostitutes and homosexuals, and inmates of mental institutions, prisons etc.

Carstens, Macnab and Kew²⁴ found, in a study of 95 South African anaesthetists that 17.9 per cent were positive for anti HBs compared with five per cent of the normal population. We have found a similar prevalence of anti HBs in a Canadian population of anaesthetists.²⁵

It has been suggested that some health care workers who are repeatedly exposed to small amounts of blood may possibly develop immunity (anti HBs positive) without being clinically infected, so that they remain anti HBc negative.²⁶ Anaesthetists may be in this group.

The possible sequelae of HBV infection are shown in Figure 3. As shown, there are many possible outcomes, the most favourable being recovery with reversion to HBsAg negative, and the unfavourable ones being chronic hepatitis, cirrhosis, carriage or development of hepatoma.

An estimated 200,000 persons, primarily young adults, are infected in the U.S.A. each year. One-quarter of them become jaundiced. More than 10,000 patients are hospitalized each year with hepatitis B, and an average of 250 die of fulminant disease. Between six and ten per cent of young adults with HBV infection become carriers. The U.S. currently contains an estimated pool of 400,000–800,000 carriers. Chronic active hepatitis develops in over 25 per cent of carriers (100,000–200,000) and often progresses to cirrhosis. It has been estimated that 4,000 persons die from hepatitis B-related cirrhosis each year in the U.S.A. and that more than 800 die from hepatitis B-related liver cancer.^{27–29}

Approximately 1,000 cases of hepatitis B were reported annually in 1979, 1980, and 1981 in Canada,³⁰ but it appears likely that many more may not have been reported when one considers the incidence rate differences between Canada and the U.S.A.

Prevention of HBV

1. A "high risk" sticker should be placed on charts and specimens of all suspected patients until proven negative.

2. Routine testing of all high-risk cases preoperatively. It is not feasible to routinely test all surgical patients preoperatively, due to expense.

The anaesthetist must therefore appreciate that he will almost certainly come into contact with asymptomatic carriers in the operating room. He must play an active role in insisting on testing patients who are in the suspect groups preoperatively, be prepared to deal with patients in the operating room who are known carriers of hepatitis B, and appreciate the possible outcomes if he contacts the disease.

3. Accidental skin puncture in the operating room by a needle or scalpel which has previously entered the patient or contamination of a wound or mucous membranes with blood or secretions from a patient should result in the following actions:

A specimen of blood should be taken from the patient and the anaesthetist and sent to the laboratory. If the patient is HBsAg negative, then no further action is required. If the patient is HBsAg positive, and the anaesthetist has a high titre of anti HBs (> 10 RIA units) it is recommended that nothing further be done since this will give him a high degree of protection.³¹ It has been shown that

previously infected individuals with residual anti HBs show a dramatic (secondary or anamnestic) anti-HBs response one to two weeks after re-exposure.⁹ If, however, the anaesthetist has a low anti HBs titre or no antibody it is advisable for him to receive Hepatitis B Immune globulin (HBIG). This must be done within 48 hours to achieve success (0.06 ml·kg⁻¹ intramuscularly) followed by a similar dose one month later. Gamma globulin is not recommended since its anti HBs content is unpredictable.

Anaesthetists who routinely give injections and instrument the oral cavity are at obvious risk of accidental needle puncture and contamination by saliva. However, it has been suggested that medical personnel very rarely give a history of exposure when they develop acute hepatitis B, and so it is possible that HBV infection may be acquired by contact with infected blood without specific inoculating injury.³²

4. When a known carrier (HBsAg positive) is operated on, steps must be taken to prevent contact with the patient's blood or secretions. Contact must therefore be reduced by barrier methods. Heating to 60° C and the use of Chlorox or formalin will kill the virus. Waterson³³ recommends that all staff should wear gowns, gloves and plastic overshoes, and the use of disposable linen, and the soaking of all instruments after use in either hypochlorite-detergent or formalin for at least three hours before they are cleaned or autoclaved. All specimens should be sent to the laboratory bearing a "high risk" label. In addition, needle punctures should be avoided as far as possible. After the operation, the floors, furniture and any visibly soiled areas on the walls should be washed down with hypochlorite-detergent.

The anaesthetic machine should be washed down with hypochlorite-detergent solution. If a respirator has been used, it should be decontaminated with formalin gas or ethylene oxide.

5. A vaccine is now available which induces active immunity against hepatitis B. The vaccine is prepared from the components of the hepatitis B viral coatings which are produced in chronic carriers, and does not contain the infectious viral nucleic acid. Elaborate procedures are carried out during preparation of the vaccine to eliminate any infectious material.³⁴ Primary adult vaccination consists of three intramuscular doses of 1 ml of

vaccine ($20 \mu\text{g}\cdot\text{ml}^{-1}$ HBsAg protein each). Second and third doses are given one and six months respectively after the first. After the third dose greater than 90 per cent of vaccinated individuals develop significant protective antibodies.

The HBV vaccine cannot modify or eliminate the chronic carrier state, and it is not harmful if given to carriers or people who are already immune due to past infection.³⁵ It is cost beneficial to screen populations at risk for the presence of anti-HBs when the expected antibody prevalence rate is high enough to save sufficient vaccine compared to the cost of antibody testing to identify the persons with naturally acquired antibody.

NANB

Since the discovery of HBsAg and its association with viral hepatitis B, widespread screening of blood donors by sensitive tests revealed that only five to ten per cent of cases of post-transfusion hepatitis (PTH) were due to HBV. It was also found that virtually no cases of PTH were due to HAV, by testing for anti HAV IgM.³⁶ Overall, the transmission of NANB appears similar to that of type B hepatitis but precise patterns of spread are not yet clear. It appears that carriers are even more common in NANB hepatitis than in hepatitis B. Reports from India³⁷ indicate that some NANB hepatitis may occur in epidemic form. It appears that the incubation period may be 2–26 weeks, with the majority of cases occurring between five and ten weeks after transfusion. Onset is insidious and often without jaundice, less than 25 per cent of patients developing overt icterus.³⁸

Diagnosis depends on the presence of elevated serum transaminases with clinical exclusion of non-viral causes of hepatocellular injury (e.g., congestive heart failure, drugs) and on the serological exclusion of other known hepatic viruses (HAV, HBV, CMV and EBV). A frequent characteristic is a tendency for serum transaminases to fluctuate drastically.³⁸ The diagnosis of NANB hepatitis is therefore essentially by exclusion. From a practical point of view laboratory exclusion of HBV or HAV infection is sufficient to assume NANB infection in a particular patient in an operating room.

Since carriers are common, the anaesthetist must be careful with patients who are in the high risk groups, but who have been proved negative for

HBsAg by testing since these individuals may still be carriers of NANB hepatitis. Donor blood, even though negative for HBsAg by sensitive tests should be treated with respect since it could still contain NANB hepatitis viruses. Virus-like particles have been demonstrated in the blood of apparently healthy blood donors capable of inducing NANB hepatitis in humans and chimpanzees.^{39–41}

Since NANB is primarily diagnosed by exclusion, it is uncertain how many NANB viruses are involved, but reports suggest that more than one agent is implicated.^{42–44}

It has been estimated that approximately 230,000 new cases of NANB hepatitis related to transfusion can be expected to occur annually in the U.S.A.,⁴⁵ but it is a worldwide problem.⁴⁶

Chronic hepatitis is a frequent sequel of NANB occurring after transfusion, with abnormal liver function tests persisting for more than a year in 10–60 per cent of patients.^{47,48} NANB not associated with blood transfusion appears to have a different outcome, with infrequent occurrence of chronic hepatitis.⁴⁷ This may possibly reflect different infectious agents or different infecting doses.⁴⁷ So far, no cases of hepatoma specifically associated with chronic NANB infection have been described, but fulminant hepatitis, chronic active hepatitis and cirrhosis have been reported.⁴⁸

NANB is thus very difficult to prevent, but because of its similarity to HBV the following measures may help in reducing the number of cases:

1. Avoidance of commercial blood donors who may have a higher carrier rate than the volunteer group.
2. Not giving unnecessary blood or blood product transfusions.
3. Careful handling of donor blood, as well as avoiding contact with patient's blood and oral secretions. Frequent handwashing if the hands become contaminated.
4. If a patient who is diagnosed as having NANB hepatitis requires an operation, all the precautions described above for HBV infection are required in the operating room.

Summary

Hepatitis A does not represent any great problem to the anaesthetist. Now that accurate tests are available for the diagnosis in the acute stage, its presence or absence can be determined.

With the advent of sensitive tests for HBsAg, hepatitis B can now be readily diagnosed. However, the chronic carrier patient must still be treated with great respect. The problem is that unless we always have an index of suspicion we may not even be aware of the patient-carrier. No doubt the majority of carriers who come to surgery pass through hospital undetected. Thus, the anaesthetist must always be careful in handling patients' blood and oral secretions and frequently wash his hands. If skin penetration or mucous membrane contamination occurs, he should take the precautions suggested. Anaesthetists who are HBsAg and anti HBs negative should consider whether they desire vaccination in order to attain active immunity. This is a personal decision.

If the anaesthetist is discovered to be a chronic carrier of HBV, he would have to exercise great care in dealing with patients. Recent data suggests that persistence of DNA polymerase activity and high levels of HBeAg may indicate infective potential in chronic HBV carriers.¹²

NANB hepatitis is still a problem in that it is diagnosed by excluding other known causes of hepatitis. Essentially, as in hepatitis B, carriers are common and so the anaesthetist has to be careful in handling all routine cases. Evidence suggests that NANB may comprise more than one virus.⁴²⁻⁴⁴

Viral hepatitis now constitutes the main hazard of transfusion of human blood and certain blood products.⁴⁹ Average-risk materials are fresh blood, and single donor plasma, while high-risk products are pooled plasma, fibrinogen, Antihæmophilic Globulin (Factor VIII), Christmas Factor (Factor IX), platelet concentrate and cryoprecipitate. As stated, 90-95 per cent of cases of post-transfusion hepatitis are due to NANB hepatitis. Care and vigilance must be exercised in the operating room at all times in order to avoid the hazard of viral hepatitis.

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Résumé

L'hépatite virale constitue une menace constante pour le personnel d'une salle d'opération. L'anesthésiologiste, en raison de ses fonctions mêmes, ne peut éliminer complètement tout contact avec le sang ou la salive des malades et il doit être très prudent à ce sujet et viser à réduire ses contacts autant que possible.

L'hépatite A (HAV) est disséminée principalement par contact fécal-oral. Les porteurs sont à toutes fins utiles inexistants et le risque pour l'anesthésiologiste est celui d'un contact direct avec un patient qui est manifestement affecté ou en phase d'incubation durant son anesthésie. Chez ce dernier type de malades, la HAV peut présenter en post-opératoire des problèmes diagnostiques de taille. Des mesures prophylactiques à la gamma globuline sont recommandées.

L'hépatite B (HBV) et l'hépatite Non A Non B (NANB) sont très fréquemment disséminées par porteurs sains dont le sang constitue le principal contaminant. Certaines classes de malades doivent demeurer suspectes à l'anesthésiste vigilant par exemple: les malades en hémodialyse, les malades originaires de pays à prévalence élevée, ceux qui ont reçu des transfusions multiples ou des produits du sang, les patients leucémiques ou souffrant d'autres réticuloses, le morphinomane, les prostituées, les homosexuels et les internés en institution psychiatrique ou pénitentiaire. Ici encore, des mesures préventives par l'emploi d'immuno-globuline HB et le vaccin anti-hépatite B sont recommandés. L'hépatite NANB demeure un problème diagnostique, celui-ci n'étant posé que par l'élimination des autres causes d'hépatite virale. Il semble que 90 pour cent des hépatites post-transfusionnelles soient du type NANB et on pense que plus d'un virus seraient impliqués dans son étiologie.