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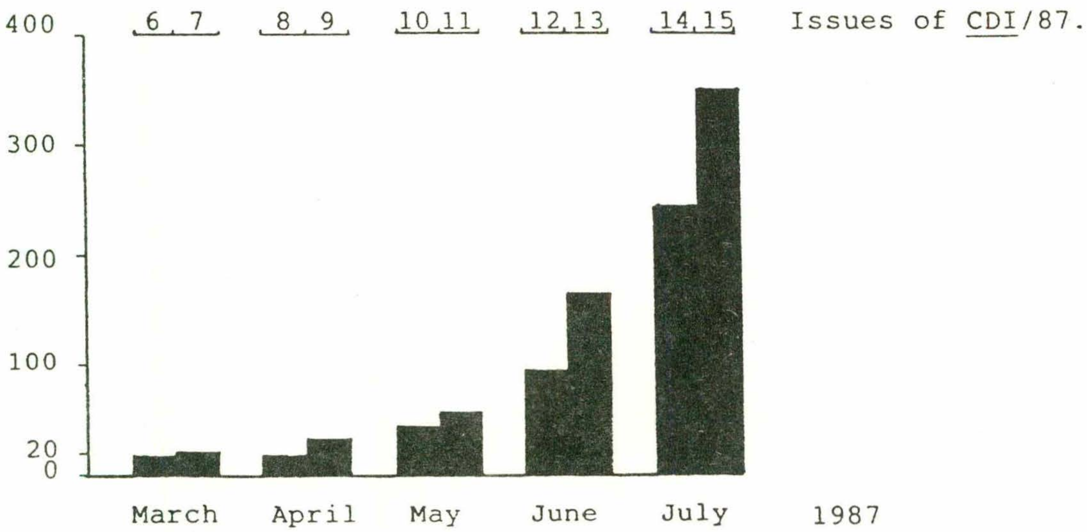
Contents:

VIRUS REPORTING SCHEME: A total of 1501 reports were processed for this period.

Eleven cases of Q fever were reported, 2 from South Australia and 9 from New South Wales. No occupational exposure data was available for the reported cases. However none of the eleven patients was involved in the Q fever vaccine field trial conducted in South Australia.

The expected increase in respiratory syncytial virus (RSV) activity has occurred since the beginning of the winter months (Figure). The usual demographic trend of infection in children under 5 years of age has been seen with this group accounting for eighty percent of the reported cases.

FIGURE: RSV ACTIVITY FOR THE PAST QUARTER



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Contributions are solicited, and do not preclude later publication elsewhere.

Figures given may be subject to revision.

Influenza A virus subtype H3N2 has recently been isolated from the nasal aspirate of two patients:-

. a 26 year old male with severe respiratory tract infection,
. a 10 month old male who required hospitalisation with fever, irritability, a mild cough and a right periorbital cellulitis. Viral isolate typing by the Commonwealth Serum Laboratories showed that the strain resembled A/Leningrad/360/86. Further antigenic analyses are in progress.

Herpesvirus was isolated at autopsy from the skin lesions of an 11 year old male with disseminated varicella infection.

Cytomegalovirus was isolated from the bronchial washings of a 43 year old male who developed pneumonia and septicaemia following recent bone marrow transplantation.

UPDATE ON HEPATITIS B PREVENTION
(Based on MMWR Vol. 36/No. 23, 19 June 1987)

Recommendations of the Immunisation Practices Advisory Committee

Introduction

Hepatitis B virus infection is a major cause of acute and chronic hepatitis, cirrhosis, and primary hepatocellular carcinoma in the United States and world wide. Since 1982, a safe and effective hepatitis B (Hep B) vaccine manufactured from human plasma has been available in the United States. This vaccine has been recommended as pre-exposure prophylaxis for persons at high or moderate risk of acquiring Hep B virus infection(1). In addition, the combination of Hep B vaccine and Hep B immunoglobulin has been recommended for post-exposure prophylaxis in susceptible persons who have perinatal or needle-stick exposure to known Hep B virus-positive persons or their blood.

This statement provides an update on Hep B vaccine usage and on its impact on disease incidence in the 5 years following its licensure. In addition, it provides both recommendations for using a new Hep B vaccine produced in yeast by recombinant DNA technology and an assessment of the need for Hep B vaccine booster doses for persons who have received the initial three-dose regimen. Basic recommendations on pre-exposure and post-exposure usage of Hep B vaccine and on pre-vaccination serologic testing for susceptibility to Hep B are unchanged. Previous recommendations should be consulted for a complete discussion of the usage of Hep B vaccine(1).

PLASMA-DERIVED HEP B VACCINE

Patterns of usage to date

Since the plasma-derived Hep B vaccine became available in June 1982, 4 400 000 doses have been distributed in the United States, and an estimated 1 400 000 persons have completed the three-dose series (Merck Sharp & Dohme, unpublished data). During this 5-year period, vaccination programs and overall vaccine usage have focused primarily on three risk groups:

- persons who work in health care professions and have exposure to blood,
- staff and clients of institutions for the developmentally disabled,
- staff and patients in haemodialysis unit.

Although no precise figures are available, it is estimated that more than 85% of distributed vaccine has been used for these groups.

Development of vaccination programs for health care workers has progressed steadily since vaccine licensure. Several surveys of hospitals in 1985 showed that between 49% and 68% of hospitals had established Hep B vaccination programs and that the number has increased steadily each year. Large hospitals (> 500 beds) were most likely to establish programs (90%). However, by June 1985, 60% of hospitals with fewer than 100 beds had also begun vaccination programs. In 75% of the programs, vaccination was recommended for high risk health care workers (as defined by the hospital), and, in 77%, the hospital paid for these vaccinations. In addition, 70% of states had established programs for vaccinating health care workers under state jurisdiction.

In spite of these programs, the actual use of vaccine in high risk health care professions has been modest. One statewide survey showed that, in hospitals with Hep B vaccine programs, only 36% of persons at high risk had actually received vaccine. In one survey in three large cities, only 24% of physicians had received the vaccine. National surveys have shown higher rates of vaccination among dentists (44% in early 1986) and haemodialysis staff (an estimated 44% in 1985); however, even these rates fall well short of optional coverage.

Development of vaccination programs has also progressed for several other groups at high risk of Hep B virus infection. By mid-1985, 94% of states had established vaccination programs for the developmentally disabled in institutions under state jurisdiction, and 75% had programs for staff of such facilities. By 1986, an estimated 27% of the developmentally disabled had received Hep B vaccine (Merck Sharp & Dohme). In addition, wide-scale programs directed at vaccinating all susceptible persons were established in 1981 for Alaskan natives and in 1985 for the population of American Samoa.

Nevertheless, there has been little progress in developing vaccination programs for other major risk groups, including parenteral drug abusers, homosexual men, and heterosexually active persons with multiple sexual partners. Few states have established programs for offering vaccine to any of these groups, and private usage of vaccine among these groups is believed to be limited.

Impact on disease incidence

The incidence of reported Hep B has increased steadily over the last decade. Hepatitis B is now the most commonly reported type of hepatitis in the United States. In 1987, 15 000 cases of clinical hepatitis B were reported to the Centers for Disease Control (CDC), Atlanta, for an incidence rate of 6.9/100 000 population. At that time, CDC estimated that there were actually 200 000 persons with Hep B virus infection and that 50 000 of these had clinically confirmed cases with jaundice. The incidence rate of reported disease increased 33%, to 9.2/100 000 population in 1981, the year prior to vaccine availability. It continued to increase during the initial 4 years of vaccine availability, reaching a rate of 11.5/100 000 in 1985(2). Based on a comparison with the overall infection rate estimated in 1978, the incidence of Hep B virus infection in the United States is now estimated at over 300 000 cases per year.

The apparent lack of impact of Hep B vaccine on the incidence of Hep B is attributable to several factors:

- the majority of acute hepatitis B cases now occur in three groups: homosexual men, parenteral drug abusers, and persons acquiring disease through heterosexual exposure(3); none of these groups is being reached effectively by current Hep B vaccine programs,
- in contrast, fewer than 10% of cases occur in health care workers, the institutionalised developmentally disabled, and other groups currently accounting for the bulk of vaccine usage,
- up to 30% of patients deny any of the recognised risk factors, even after careful questioning. No effective strategy has been devised to prevent disease among this group, although some are probably undisclosed members of the three major risk groups.

A reduction in the incidence of hepatitis B can be expected only if significant proportions of persons at high risk receive the vaccine. Increased efforts are needed to develop programs to vaccinate persons in all high-risk groups and to increase compliance among those who are susceptible in areas where programs are established. To have any effect on the incidence of hepatitis B, use of Hep B vaccine in the United States must extend beyond the current groups of recipients.

NEW RECOMBINANT DNA HEP B VACCINE

Formulation

In July 1986, a new, genetically engineered Hep B vaccine (Recombivax HB ®; Merck Sharp & Dohme) was licensed by the US Food and Drug Administration. This vaccine, as formulated, has an immunogenicity comparable to that of the currently available plasma-derived vaccine (Heptavax B ®; Merck Sharp & Dohme). The two vaccines are also comparably effective when given with Hep B immunoglobulin to proven perinatal Hep B virus transmission. The new vaccine provides an alternative to the plasma-derived Hep B vaccine for almost all groups at risk of Hep B virus infection.

The recombinant vaccine is produced by Saccharomyces cerevisiae (common baker's yeast) into a plasmid containing the gene for the Hep B surface antigen (HBsAg) subtype adw has been inserted(4). HBsAg is harvested by lysing the yeast cells and is separated from yeast components by hydrophobic interaction and size- exclusion chromatography. The purified HBsAg protein undergoes sterile filtration and treatment with formalin prior to packaging. The vaccine is packaged to contain 10µg HBsAg protein per mL, adsorbed with 0.5mg/mL aluminium hydroxide; a 1:20 000 concentration of thiomersal is added as a preservative.

The recombinant HBsAg takes the form of 17-25nm spherical particles, similar in appearance to human plasma-derived HBsAg. The recombinant particles differ in that the HBsAg is not glycosylated, whereas up to 25% of plasma-derived HBsAg is glycosylated. The vaccine contains more than 95% HBsAg protein. Yeast derived protein can constitute up to 4% of the final product, but no yeast DNA is detectable in the vaccine.

Immunogenicity and Efficacy

The immunogenicity of the recombinant Hep B vaccine is comparable to that of the plasma-derived product[5]. When given in a three-dose series (10µg per dose), recombinant Hep B vaccine induces protective antibodies (anti-HBs > 10 (mIU)/mL, approximately equal to 10 sample ratio units by radioimmunoassay or positive by ELISA) in over 95% of healthy adults 20-39 years of age. Studies comparing antibody responses of healthy adults show equal rates of seroconversion following the three doses of either the recombinant vaccine (10µg per dose) or the plasma-derived vaccine (20µg per dose). However the geometric mean titre (GMT) of antibodies developed by recipients of the recombinant vaccine have ranged from equal to 30% as high as those developed by recipients of the plasma-derived vaccine. The recombinant vaccine, like the plasma-derived vaccine, produces a somewhat lower antibody response in older adults than in younger adults[5].

In studies using three 5µg doses of recombinant vaccine for children over 12 years of age, over 99% of the recipients have developed protective levels of antibodies. Haemodialysis patients develop a poorer response to the recombinant vaccine than do healthy adults. For example, in one study using three 40-µg doses of recombinant Hep B vaccine, only 64% of vaccine recipients developed protective levels of antibodies.

The recombinant Hep B vaccine has been shown to prevent Hep B virus infection of vaccinated chimpanzees challenged intravenously with Hep B virus of either adw or ayr subtypes. In studies of infants born to HBsAg and HBeAg positive mothers, the combination of Hep B immunoglobulin (0.5 mL at birth) and recombinant Hep B vaccine (5µg in each of three doses) protected 94% of infants from developing the chronic carrier state, an efficacy equalling that of Hep B immunoglobulin plus plasma-derived Hep B vaccine[6]. The simultaneous administration of Hep B immunoglobulin did not interfere with induction of anti-HBs antibody response by the recombinant Hep B vaccine.

There have been no large-scale efficacy trials of recombinant vaccine in adults. Neverless, the immunogenicity studies, the challenge studies using chimpanzees, and the efficacy trials of the Hep B vaccine and Hep B immunoglobulin in infants born to mothers who are carriers of Hep B virus, strongly suggest that the efficacy of recombinant Hep B vaccine in adult is comparable to that of the plasma-derived product.

Safety

Because only the portion of the Hep B virus genome that codes for the surface coat of the virus (HBsAg) is present in the recombinant yeast cells, no potentially infectious viral DNA or complete viral particles can be produced. No human or animal plasma or other blood derivative is used in the preparation of recombinant Hep B vaccine.

During prelicensure trials, approximately 4 500 persons received at least one dose, and 2 700 persons completed the vaccine series(5). Reported side effects were similar in extent and variety to those following administration of the plasma-derived vaccine:

. 17% of those vaccinated experienced soreness at the injection site, and
. 15% experienced mild systemic symptoms (fever, headache, fatigue, and nausea).

To date, no severe side effects have been observed, nor have significant allergic reactions been reported. Although yeast-derived proteins may constitute up to 4% of the protein in the vaccine, no adverse reactions that could be related to changes in titres of antibodies to yeast-derived antigens occurred during clinical trials.

Early concerns about safety of plasma-derived Hep B vaccine, especially the concern that infectious agents such as human immunodeficiency virus (HIV) present in donor plasma pool might contaminate the final product, have proven to be unfounded(7). There are no data to indicate that the recombinant vaccine is potentially or actually safer that the currently licensed plasma-derived product.

Dosage and Schedule

The recombinant Hep B vaccine is given in a series of three doses over a 6-month period. The second dose is administered one month after the first, and the third dose, five months after the second:

. for normal adults and children older than 10 years of age, the recommended dose is 10µg (1mL) intramuscularly in each of the three inoculations,
. children less than 11 years of age should receive a 5-µg dose (0.5mL) by the same schedule,
. neonates of mothers who are carriers of HBsAg should receive the three-dose series (5 µg per dose) by the same schedule; however, the first dose, which is given at birth, should be combined with a single dose of Hep B immunoglobulin (0.5mL) given intramuscularly at another site.

The recommended dose of recombinant Hep B vaccine for haemodialysis patients or other immunosuppressed persons is 40µg, which is identical to the dose of plasma-derived vaccine recommended for these groups. A specially formulated preparation (40µg HBsAg protein/mL adsorbed with 0.5mg aluminium hydroxide) is being developed for these patients. At present, it is not advisable to administer the standard formulation of recombinant Hep B vaccine to these patients because this would require a large volume (4.0 mL), which is inconvenient for injection in the deltoid muscle, and would contain more aluminium hydroxide (2.0mg) than currently recommended as an adjuvant in vaccines (1.25mg per dose). Only plasma-derived vaccine should be used for these patients.

As with plasma-derived vaccine, recombinant Hep B vaccine should only be given to older children and adults in the deltoid muscle and to neonates or infants in the anterolateral thigh muscle. The vaccine should be stored at 2°C (36°F to 6°C 43°F) and should not be frozen; freezing destroys the potency of this vaccine.

The response to vaccination by the standard schedule using one or two doses of plasma-derived vaccine followed by the remaining doses of combinant vaccine has not been studied. However, because the immunogenicities of the two vaccines are similar, it is likely that the response will be comparable to that induced by three doses of either vaccine alone. The response to revaccination with the recombinant vaccine following non-response to an initial series of plasma vaccine has not been evaluated.

Indications for use

The indications for use of the recombinant Hep B vaccine are identical to those for the plasma-derived product, except that the present formulation of the recombinant Hep B vaccine should not be used for haemodialysis patients or other immunosuppressed persons (Table 1)[1].

TABLE 1: PERSONS FOR WHOM HEP B VACCINE IS RECOMMENDED OR SHOULD BE CONSIDERED[1].

PRE-EXPOSURE

Persons for whom vaccine is recommended

- Health care workers having blood or needle-stick exposures
- Clients and staff of institutions for the developmentally disabled
- Haemodialysis patients
- Homosexually active men
- Users of illicit injectable drugs
- Recipients of certain blood products
- Household members and sexual contacts of Hep B virus carriers
- Special high-risk populations

Persons for whom vaccine should be considered

- Inmates of long-term correctional facilities
- Heterosexually active persons with multiple sexual partners
- International travellers to Hep B virus endemic areas

Post-Exposure

- Infants born to Hep B virus positive mothers
- Health care workers having needle-stick exposures to human blood

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For other groups, including persons with Down's syndrome, there are no data indicating that the recombinant Hep B vaccine is either superior or inferior to the plasma derived Hep B vaccine for any pre-exposure or post-exposure indication.

Precautions

The recombinant Hep B vaccine contains only non-infectious HBsAg particles; therefore, vaccination of a pregnant woman should entail no risk to either the woman or foetus. Furthermore, Hep B virus infection in a pregnant woman can result in severe disease for the mother and chronic infection of the new born. Pregnancy should not be considered a contraindication for women in high-risk groups who are eligible to receive this vaccine.

NEED FOR VACCINE BOOSTER DOSES

Long-term protection by plasma-derived Hep B vaccine. In short-term efficacy studies, the plasma-derived Hep B vaccine provided protection against Hep B virus infection for 85%-95% of vaccine recipients, including virtually all those who developed adequate levels of antibodies[8, 9]. A recent evaluation of the long-term protection afforded by this vaccine (longer than 5 years provides a basis for recommendations concerning the need for booster doses in previously vaccinated persons[10].

Currently available data indicate that the vaccine-induced antibody levels decline significantly[10]. Antibody may decrease to low levels for 30%-40% of vaccinated adults who initially develop adequate levels of antibody during the 5 years after vaccination, and it may become undetectable in 10%-15% of them. The duration of antibody persistence is directly related to the peak level achieved after the third dose of vaccine[11]. The longer persistence of detectable levels of antibody observed in children and young adults (less than 20 years of age) is consistent with the higher peak response in these age groups.

Studies of the licensed plasma derived Hep B vaccine in adults have demonstrated that, in spite of declining levels of antibody, protection against clinical (or viraemic) Hep B virus infection persists for more than 5 years[10]. Although the risks of Hep B virus infection appear to increase as antibody levels become low or undetectable, the resultant infections are almost always innocuous and do not cause detectable viraemia, liver inflammation, or clinical illness. These infections are detected by serologic evidence of an increase of anti-HBs levels associated with the appearance of antibody to Hep B core antigen (Anti-HBc). To date, only one transient viraemic infection has been recognised in a vaccine responder within 72 months after vaccination. This infection produced mild alanine aminotransferase (ALT) elevation, but no clinical illness[10]. Thus, among adults who have responded to the vaccine, protection against clinically significant Hep B virus infection appears to outlast the presence of detectable anti-HBs and can persist for two or more years among vaccine recipients whose antibodies have declined to low or undetectable levels.

For infants born to mothers who are carriers of hep B virus, there are insufficient data to assess duration of antibody persistence and protection against clinically significant Hep B virus infection with the US plasma-derived vaccine. One study, in a developing country (Senegal) and using a different plasma-derived Hep B vaccine, has demonstrated that protection against viraemic Hep B virus infection can decline within 6 months and 2 years of age[12]. Firm data on the duration of protection among infants receiving the vaccines licensed in the United States will be necessary before recommendations on booster doses can be made for this group.

Post-vaccination testing of response to vaccine

When properly administered, Hep B vaccine produces anti-HBs in more than 90% of healthy persons. Testing for immunity following vaccination has been recommended only for persons in whom suboptimal response to vaccine is anticipated, including persons who received vaccine in the buttock or persons, such as haemodialysis patients, whose subsequent management depends on knowing their immune status[1]. Revaccination, which has produced adequate antibody in only 30%-50% of persons who have not responded to primary vaccination in the deltoid, is not routinely recommended[1, 10].

Vaccine program coordinators in hospitals may decide to test vaccine recipients serologically to assess their antibody responses, even though such post-vaccination testing is not routinely recommended. Persons electing to perform post-vaccination testing should be aware of potential difficulties in interpreting the results. Serologic testing within 6 months of completing the primary series will differentiate persons who respond to vaccine from those who fail to respond. However, the results of testing undertaken more than 6 months after completion of the primary series are more difficult to interpret. A vaccine recipient who is negative for anti-HBs between 1 and 5 years after vaccination can be:

1) a primary non-responder who remained susceptible to Hep B, or
2) a vaccine responder whose antibody levels have decreased below detectability but who is still protected against clinical Hep B virus disease[10].

There is no need for routine anti-HBs testing 1 to 5 years after vaccination unless there has been a decision to provide booster doses for persons who are anti-HBs negative. This strategy is medically acceptable, but costly, and will prevent few additional cases of disease because of the excellent long-term protection already provided by the primary series of vaccine.

Recommendations for booster doses

- For adults and children with normal immune status, the antibody response to properly administered vaccine is excellent, and protection lasts for at least 5 years. Booster doses of vaccine are not routinely recommended, nor is routine serologic testing to assess antibody levels in vaccine recipients necessary during this period. The possible need for booster doses after longer intervals will be assessed as additional information becomes available.

- For haemodialysis patients, in whom vaccine-induced protection is less complete and may persist only as long as antibody levels remain above 10m IU/mL, the need for booster doses should be assessed by semi-annual antibody testing[13]. Booster doses should be given when antibody levels decline below 10m IU/mL.

Post-exposure prophylaxis of persons exposed to HBsAg positive needle-sticks

In vaccinated persons who experience percutaneous or needle exposure to HBsAg-positive blood, serologic testing to assess immune status is recommended unless testing within the previous 12 months has indicated adequate levels of antibody. If the exposed person is tested and found to have an inadequate antibody level, treatment with Hep B immunoglobulin and/or a booster dose of vaccine is indicated, depending on whether vaccination has been completed and whether the person is known to have previously responded to Hep B vaccine. Detailed recommendations on prophylaxis in this situation are provided in the previous recommendations for Hep B vaccine[1].

Dosage

When indicated, Hep B vaccine recipients can be given booster doses of either plasma derived or recombinant Hep B vaccine. Booster doses of either vaccine induce prompt anamnestic responses in over 90% of persons who initially respond to vaccine but subsequently lose detectable antibody[14, 15]. The booster dose for:

- normal adults is 20μg of plasma-derived vaccine or 10μg of recombinant vaccine,
- neonates and children under 10 years of age is 10μg of plasma-derived vaccine or 5μg of recombinant vaccine,
- haemodialysis patients, a dose of 40μg of plasma-derived vaccine is recommended; a formulation of recombinant Hep B vaccine is not yet available for this group.

Vaccine should be given in the deltoid muscle. Buttock injection does not induce adequate levels of antibody.

Precautions

Reported adverse effects following booster doses have been limited to soreness at the injection site. Data are not available on the safety of the vaccine for the developing foetus, but there should be no risk because both plasma-derived and recombinant Hep B vaccines are inactivated and do not contain live virus particles. Booster doses need not be withheld from pregnant women who are at ongoing risk of Hep B virus infection.

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HEPATITIS B PREVENTION - RECOMMENDATIONS FOR AUSTRALIA

DESCRIPTION OF PRODUCTS

Hepatitis B vaccine

The hepatitis B vaccine licensed for use in Australia (H-B-Vax, Merck Sharp and Dohme) is an inactivated vaccine consisting of 22nm HBsAg subviral particles purified from the plasma of human carriers and inactivated by treatment with 8M urea, pepsin and 1:4000 formalin. It is a sterile suspension for intramuscular injection. Each 1.0mL dose of vaccine contains 20µg of hepatitis B surface antigen formulated in an alum adjuvant, and thiomersal (mercury derivative) 1:20 000 added as a preservative.

H-B-Vax is a white slightly opalescent liquid. Any visible physical change from an amorphous flock to a granular precipitate may indicate incorrect storage conditions and consequent reduction in vaccine immunogenicity, rendering the vaccine unsuitable for use.

Store unopened and opened vials at 2-8°C (35.6-46.4°F). Unused vaccine should be discarded at the end of vaccination session. **Freezing destroys the potency of the vaccine.**

the normal adult immunisation regime consists of 3 x 1.0 mL (20µg) doses given intramuscularly into the deltoid region; use of the gluteal site may result in reduced seroconversion rates:

1st dose - at elected date
2nd dose - one month later
3rd dose - six months after the first dose[1].

A full course of H-B-Vax induces protective levels of neutralising antibody against hepatitis B virus in approximately 95 per cent of individuals older than six months of age. The frequency of seroconversion increases progressively from approximately 35 per cent after the first injection to over 95 per cent after the third injection. There is evidence of immunity (neutralising antibody) in most vaccinated subjects after administration of two doses of the 3-dose vaccine regimen. However, the third dose is necessary for both a high percentage of responses and a long-term protective effect.[2]

In dialysis patients and other immunocompromised individuals, twice the above dose (2 x 20µg dose given in different sites) at each date is recommended, although no formal large scale comparison of the two regimes in such patients are available. In this group, seroconversion rates of only 60-70 per cent have been reported.

For children under the age of 10 years, three similarly spaced doses of 0.5 mL (10μg) are sufficient. In neonates, seroconversion rates of 86-100 per cent have been described with different dosage regimens. These rates have been shown not to be affected by concurrent administration of hepatitis B specific immunoglobulin.

Early data suggested that immunity should last approximately five years in individuals who had received all three doses, after which time a booster dose of vaccine might be necessary. In a more recent study, anti-HBs declined to levels considered non-protective, within three years in those individuals who showed a weak initial response. Until more experience is available, no firm recommendation can be made other than that booster revaccination should be considered after 3-5 years.(3)

Serological confirmation of post-vaccination immunity:

Vaccine recipients should be tested for seroconversion for anti-HBs positivity 1-3 months after the third vaccination dose, both to allow ongoing monitoring of vaccine potency in different settings and to allow further management of non-responders. Non-responders to a primary course of vaccination should be offered a fourth dose of vaccine. If adequate anti-HBs levels are not reached following a fourth dose, no assurance can be given about immunity.

Hepatitis B immunoglobulin

Hepatitis B immunoglobulin (HBIG), is prepared from plasmas that have been selected on the basis that they contain high levels of antibody to HBsAg. It is a sterile 16 per cent w/v solution of the IgG fraction from such plasma, prepared by the Cohn cold ethanol fractionation process. Thiomersal 0.01 per cent w/v is added as a preservative. Hepatitis B immunoglobulin is available in containers of 100 IU and 400 IU, the volume of which will vary according to the particular batch. It should be stored, protected from light at 2° to 8°C. **DO NOT FREEZE.**

As stocks of HBIG are strictly limited, issue will be restricted to patients who fulfil clearly defined criteria (see Recommendations for Use: Post-exposure Prophylaxis). Requests should be directed to the relevant State Director of the Red Cross Blood Transfusion Service.

RECOMMENDATIONS FOR USE

Pre-exposure vaccination

Active vaccination against hepatitis B is indicated in persons who have been demonstrated or judged to be at substantial risk of hepatitis B infection, and are shown to be susceptible to infection. In any particular setting, available local data (including if possible results from serological surveys) should be used to make an informed decision about the actual risk of infection.

Health care workers

Risks of hepatitis B infection in this group have been shown to be lower in some Australian centres than in comparable USA workers. Risk to the individual depends on (i) HBsAg carrier rate among patient population served, (ii) degree of blood (not patient) exposure, (iii) thoroughness with which hygienic measures to avoid blood exposure are practised. It is anticipated that the risk involved may differ considerably from setting to setting in different parts of Australia.

The above category includes dentists, staff of institutions for the mentally retarded, morgue attendants, prison wardens, acupuncturists, etc. in some of which groups vaccination may be indicated.

Haemodialysis patients

Numerous studies have established the high risk of hepatitis B virus transmission in haemodialysis units. While studies have demonstrated control and eventual elimination of infection by appropriate containment and screening measures, vaccination is recommended for susceptible patients (see above for dosage).

Recipients of certain blood products

Although screening of all blood donors for HBsAg has decreased the incidence of transfusion related hepatitis B virus infection, patients with clotting disorders who receive factor VIII or IX concentrates have an elevated risk of hepatitis B virus infection. Vaccination is recommended for these persons and should be initiated at the time their specific clotting disorder is identified. Screening is recommended for patients who have already received multiple infusions of these products.

Inmates of institutions for the mentally retarded

Transmission of hepatitis B virus in such institutions is facilitated by recognised blood exposure and by biting and contact with skin lesions, saliva, and other infective secretions. Hepatitis B virus markers have been detected at high prevalence in some (but not all) such institutions. Vaccination of susceptible inmates should be considered in those institutions where hepatitis B virus is prevalent.

Homosexually active males

Susceptible homosexually active males should be vaccinated regardless of their age or duration of their homosexual practices. It is important to vaccinate persons as soon as possible after their homosexual activity begins. Homosexually active females do not appear to be at increased risk of sexually transmitted hepatitis B virus infection.

Illicit injectable drug users

All users of illicit injectable drugs who are susceptible to hepatitis B virus should be vaccinated as early as possible after their drug use begins.

Household and sexual contacts of hepatitis B virus carriers

Household contacts of hepatitis B virus carriers are at some risk of hepatitis B virus infection. Sexual contacts appear to be at greatest risk. Vaccination of susceptible household contacts of carriers is recommended.

Other contacts of hepatitis B virus carriers

Persons in contact with carriers at schools, offices, or similar places are at minimal risk of contracting hepatitis B virus, and vaccine is not routinely recommended for them. However, classroom contacts of deinstitutionalised mentally retarded hepatitis B virus carriers who behave aggressively or have special medical problems that increase the risk of exposure to their blood or serous secretions may be at risk. In such situations, vaccine may be offered to classroom contacts.

Special high-risk populations

Some Australian populations, such as Aboriginals and immigrants and refugees from areas with highly endemic disease (particularly eastern Asia and sub-Saharan Africa) have high hepatitis B virus infection rates and deserve special attention. Depending on specific epidemiological and public health considerations, more extensive vaccination programs may be warranted.

Inmates of long-term correctional facilities

The prison environment may provide a favourable setting for the transmission of hepatitis B virus because of the frequent use of illicit injectable drugs and homosexual practices. In such institutions, prison officials may elect to undertake screening and vaccination programs.

Post-exposure prophylaxis

Infants born to HBsAg positive mothers[4]

Administration of 0.5 mL hepatitis B immunoglobulin to an infant of an HBsAg, HBeAg-positive mother soon after birth and repeated at three months and six months reduces the probability of chronic infection from about 90 per cent to about 25 per cent (efficacy about 75 per cent). The concurrent use of hepatitis B vaccine and various combinations of hepatitis B immunoglobulin increases the efficacy to close to 90 per cent. Since approximately 5 per cent of perinatal infection may occur in utero, it appears likely that no form of postnatal prophylaxis will be 100 per cent effective in this circumstance.

The use of hepatitis B vaccine in combination with hepatitis B immunoglobulin in the perinatal setting has the advantages of increasing efficacy, eliminating the need for the second and third doses of hepatitis B immunoglobulin, and providing long-term immunity to those who are not infected during the perinatal period.

Maternal screening:

Since efficacy of the above regime depends on administering hepatitis B immunoglobulin on the day of birth, it is vital that HBsAg-positive mothers be identified before delivery. The decision whether to adopt routine ante-natal screening of all pregnant women, or only those in identified high risk groups, will depend on the carrier prevalence within the population served.

Management of HBsAg-positive mothers and their newborns:

Hepatitis B immunoglobulin (0.5mL) should be administered intramuscularly after physiological stabilisation of the infant and preferably within 12 hours of birth. Hepatitis B immunoglobulin efficacy decreases markedly if treatment is delayed beyond 48 hours. Hepatitis B vaccine should be administered intramuscularly in three doses of 0.5mL of vaccine (10μg) each. The first dose should be given within seven days of birth and may be given concurrently with hepatitis B immunoglobulin but at a separate site. The second and third doses should be given one month and six months, respectively, after the first (see table). HBsAg testing at six months may be done for counselling purposes, since HBsAg-positivity at six months indicates a therapeutic failure, and the third vaccine dose need not be given if HBsAg-positivity is found. Testing for HBsAg and anti-HBs is recommended at 12-15 months to monitor the final success or failure of therapy. If HBsAg is found, it is likely the child is a chronic carrier. If HBsAg is not detectable, and anti-HBs is present, the child has been protected. Hepatitis B vaccine is an inactivated product, and it is presumed that it will not interfere with other simultaneously administered childhood vaccines. Hepatitis B immunoglobulin administered at birth should not interfere with oral poliomyelitis and diphtheria-tetanus-pertussis vaccines administered at about two months of age.

Acute exposure to blood containing HBsAg[5]

As soon as possible, following true percutaneous (eg needlestick), ocular, or mucous membrane exposure to blood or potentially blood contaminated secretions, the source of the blood should be tested for HBsAg and a blood sample taken from the recipient for subsequent testing. If the source is HBsAg negative, no further action is required, unless there is reason to suspect non-A, non-B hepatitis when normal immune globulin prophylaxis may be considered. If the source is HBsAg-positive, or cannot be identified and tested, a single dose of hepatitis B immunoglobulin (0.06 mL/kg or 5.0 mL for adults) should be given as soon as possible after exposure and within 24 hours if possible. Hepatitis B vaccine 1mL (20μg) should be given intramuscularly at a separate site as soon as possible, but within seven days of exposure, with the second and third doses given one month and six months respectively, after the first (see table). For persons who choose not to receive hepatitis B vaccine, the previously recommended two-dose hepatitis B immunoglobulin regimen may be used (see table).

Hepatitis B virus post-exposure recommendations

	Hepatitis B immunoglobulin		Vaccine	
Exposure	Dose	Recommended timing	Dose	Recommended timing
Perinatal	100 IU IM	within 12 hrs of birth	0.5 mL (10ug)IM	within 7 days*; repeat at 1 and 6 months
Percutaneous	400 IU IM	single dose within 24 hrs	1.0 mL (20ug)IM#	within 7 days*; repeat at 1 and 6 months
		OR**		
	400 IU IM	within 24 hrs; repeat at 1 month	-	-
Sexual	400 IU IM	within 14 days of sexual contact	***	-

* The first dose can be given the same time as the hepatitis B immunoglobulin dose but at a separate site.

\# For persons under 10 years of age, use 0.5 mL (10µg).

** For those who choose not to receive hepatitis B vaccine.

*** Vaccine is recommended for homosexually active males and for regular sexual contacts of chronic hepatitis B virus carriers.

Sexual contacts of persons with hepatitis B virus infection

Sexual contacts of persons with acute hepatitis B infection are at increased risk of acquiring hepatitis B infection. Data examining the protective efficacy of hepatitis B immunoglobulin in this situation are limited. The period after sexual exposure during which hepatitis B immunoglobulin may be effective is unknown, but extrapolation from other settings makes it unlikely that this period would exceed fourteen days. The value of hepatitis B vaccine alone in this setting is unknown, and vaccine is not routinely recommended for exposure to acute cases.

A single dose of hepatitis B immunoglobulin is recommended for susceptible individuals who have had sexual contact with a HBsAg-positive person if hepatitis B immunoglobulin can be given within 14 days of sexual contact (see table). In exposures between heterosexuals, a second hepatitis B immunoglobulin dose should be given if the index patient remains HBsAg-positive three months after detection. If the index patient is a known hepatitis B virus carrier or remains HBsAg-positive for six months, hepatitis B vaccine should be offered to regular sexual contacts. For exposures among homosexual men, the hepatitis B vaccine series should be initiated at the time hepatitis B immunoglobulin is given following a sexual exposure, since hepatitis B vaccine is recommended for all susceptible homosexual men. Additional doses of hepatitis B immunoglobulin are unnecessary if vaccine is given.

SIDE EFFECTS AND ADVERSE REACTIONS

Hepatitis B vaccine

Reported side effects with hepatitis B vaccine have been transient and generally insignificant. These include sore arm (9-15.8 per cent), fever - usually low grade (2.6 per cent), nausea, dizziness, malaise, myalgia and arthalgia. With the exception of a sore arm, side effects were seen in a similar proportion of vaccine and placebo recipients.

Since hepatitis B vaccine is prepared from human plasma, it has been suggested that vaccination might provoke auto immune reactions in recipients. In one study of 20 recipients, vaccination was unrelated to the presence or development of organ-specific or non-organ specific autoantibodies.[6]

Human plasma used as the source of hepatitis B vaccine may be contaminated with a variety of infective agents, inc ng hepatitis B virus itself, non-A non-B hepatitis virus, human immunodeficiency virus (HIV) and (rarely) other agents if present in the viraemic phase of an infection. However, the three different inactivation steps used in vaccine manufacture have been shown to be effective in inactivating viruses from every known group, and each vaccine lot is extensively tested by chimpanzee inoculation as well as routine methods prior to release. In addition, extensive clinical use of hepatitis B vaccine over the past five years has confirmed the lack of transmission of hepatitis B or other infective agents to recipients. In particular, no clinical cases of AIDS, and no seroconversion to HIV positivity have been detected in recipients that might be attributable to vaccination. Continued monitoring of vaccine experience is required to extend these observations. However, all current data indicate that the risks of hepatitis B infection in those judged to be at significant exposure far outweighs the theoretical (and at present undemonstrated) possibility of transmission of AIDS (or other infections).

Hepatitis B immunoglobulin

Hepatitis B immunoglobulin, when properly prepared, does not transmit hepatitis B or non-A, non-B hepatitis. Although some donors for preparation of HBIG may be positive for antibody to HIV, there is no current evidence incriminating hepatitis B immunoglobulin as a vehicle for transmission of AIDS and this possibility is considered remote bearing in mind its mode of preparation and composition. However, further data should be obtained.

PRECAUTIONS AND CONTRAINDICATIONS

Hepatitis B vaccine

Use in pregnancy

Data are not available on the safety of the vaccine for the developing fetus, but because it contains only non-infectious HBsAg particles, the risk to the fetus from the vaccine should be negligible. In contrast, hepatitis B virus infection in a pregnant woman may result in severe disease for the mother and chronic infection for the newborn. Pregnancy should not be considered a contraindication to the use of this vaccine for persons who are otherwise eligible.

Effect of vaccination on carriers

The vaccine produces neither therapeutic nor adverse effects in hepatitis B virus carriers.

Vaccination of immune persons

Vaccination of individuals who possess antibodies against hepatitis B virus from a previous infection is not necessary but will not cause adverse effects. Such individuals will have a post-vaccination increase in their levels of antibody to HBsAg (anti-HBs). Passively acquired antibody, whether from hepatitis B immunoglobulin administration or from the transplacental route will not interfere with active immunisation.

Prevaccination serological screening for susceptibility

Prior screening of potential recipients for pre-existing immunity is recommended in order to extend current prevalence data on hepatitis B exposure, and to conserve vaccine supplies and achieve cost savings if a significant proportion of the population to be vaccinated has already been infected.

For routine screening, only one antibody tests, either anti-HBc or anti-HBs, need be used. Anti-HBc will identify all previously infected persons, both carriers and those who are not carriers, but will not discriminate between members of the two groups. Antibody to HBsAg will identify those previously infected except for carriers. For groups expected to have carrier rates of less than two percent, such as health care workers, neither test has a particular advantage. For groups with higher carrier rates, anti-HBc may be preferred to avoid unnecessary vaccination of carriers.

Hepatitis B immunoglobulin

Passively acquired antibody can interfere with the response to live, attenuated virus vaccines. Therefore, administration of such vaccines, eg poliomyelitis or measles, should be deferred until approximately three months after passive immunisation. By the same token,immunoglobulins should not be administered for at least two weeks after a vaccine has been given.

Pregnancy is not a contraindication to administration of hepatitis B immunoglobulin.

CDI Editorial Note:

The Australian Drug Evaluation Committee (ADEC) of the Commonwealth Department of Health has approved the application by Merck Sharp & Dohme (MSD) to market the new genetically engineered hepatitis B vaccine (Recombivax HB ®) in Australia.

Details on the formulation, the immunogenicity and efficacy, the safety, the dosage and schedule of vaccination, the indications for use including precautions for this new recombinant DNA hepatitis B vaccine have been fully discussed in the previous article on 'Update on Hepatitis B prevention - ACIP-USA.'

The release of this new MSD recombinant DNA hepatitis B vaccine in Australia is approved subject to the development of suitable product information and the resolution of certain chemistry and quality control matters.

The CDI has noted that the editorial in JAMA[8] which considers whether or not to switch from the plasma-derived to the recombinant product, raises some questions about the newer vaccine. The editorial commented that since the rDNA vaccine is nonglycosylated and has a different lipid composition and reduced antigenicity when compared with plasma-derived HBsAg, the decision by the manufacturer (Merck & Co) to recommend a lower dose may be ill-advised. The adoption of a 10µg dose for the rDNA vaccine may lower the seroconversion rate and protective antibody level against infection; the latter necessitates frequent booster doses to maintain immunity.

The editorial concluded that until this problem is resolved, physicians and health care providers should not hesitate to recommend the plasma-derived vaccine, not only because of its proven efficacy and safety, but also because its superior immunogenicity will permit a longer interval between booster doses.

However, experts speaking at a press conference following the 1987 international symposium on viral hepatitis and liver disease in London disagreed with the editorial comments. They encouraged the use of rDNA vaccine in view of the importance of immunising high-risk groups against hepatitis B. They believed that the rDNA vaccine would be more acceptable than the plasma-derived product and stressed that its use should not be discouraged.

Meanwhile Smith Kline-RIT which also markets a rDNA hepatitis B vaccine, but one containing 20µg antigen, Engerix-B, status that it has resisted the trend to use lower doses in order to lower prices and maintains that hepatitis B vaccine - regardless of source - should contain 20µg of the antigen. The company comments that efficacy in clinical trials should not be the only factor in determining dosage since in the real world, factors such as interruption in the cold chain of distribution and reduced responses to vaccination due to old age or concomitant infections can reduce efficacy. Hence, in these commonplace circumstances, the use of the 20µg dose becomes preferable.[9]

While Merck & Co announced recently that its rDNA vaccine has been approved in the United States, Canada, West Germany, Switzerland, the Netherlands, and Singapore, Smith Kline-RIT claims that Engerix-B is now available in over 20 countries including Belgium, West Germany, Switzerland, Spain, most countries in Asia and many in Latin America and Africa.

REFERENCES

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2. Hepatology (1981) 1:377-85
3. Lancet (1984) ii:458
4. Lancet (1983) ii:1099-1102
5. Arch Intern Med (1984) 144:81-5
6. Lancet (1979) i:721
7. MMWR (1984) 33:685-7.
8. JAMA (1987) May 16 :2612
9. Scrip (1987) 1213:29

JAPANESE ENCEPHALITIS (JE) VACCINE - NOW AVAILABLE

The Commonwealth Department of Health is making available:

. JAPANESE ENCEPHALITIS VACCINE

The product is
- BIKEN (Japan)
- lyophilised, inactivated Japanese encephalitis virus "Nakayama - NIH strain" of mouse brain origin with diluent
- available as single dose vial containing dry pellet of lyophilised, inactivated JE virus
- stored between 2° C and 8° C prior to reconstitution. Exposure of vaccine to direct sunlight should be avoided
- reconstituted by introducing 1.3mL of sterile distilled water as diluent into the vial. Withdraw air (nitrogen) into the syringe before drawing needle from vial. Shake the vial thoroughly. The reconstituted vaccine should be used as soon as possible, without storage.

DO NOT FREEZE RECONSTITUTED VACCINE

- administered by subcutaneous injection according to
  . the following dosage:
    Adults and children over 3 years of age: 1mL of reconstituted vaccine

    Children under 3 years of age: 0.5mL of reconstituted vaccine (discard remaining vaccine)

  . the following time interval between doses:
    Dose 1 and dose 2 given at intervals of 7-14 days

    Dose 3 given one month after initial dose

. Booster schedule:

Single dose immunisation of 1mL every year or

at least within four years to maintain immunity

- Contra-indicated in persons suffering from:
  - . severe febrile illness or other active infection
  - . diseases of the heart, kidney or liver
  - . diabetes or other hormonal dysfunction
  - . malnutrition or other deficient nutritional status
  - . leukaemia, lymphomas and other generalised malignancies
  - . hypersensitivity

- reported to produce the following side effects:
  - . local reactions showing varying degrees of induration, redness and tenderness
  - . systemic reactions such as headache and fever

. ON AN INDIVIDUAL PATIENT USE BASIS REQUIRING:

- the treating medical practitioner to contact

  Dr Ian Cook
  Commonwealth Department of Health, Canberra
  Phone (062) 89 1555

  with details of the patient and proposed travel

- both the treating medical practitioner and the patient to accept responsibility for treatment by signing a consent form which is to be returned to the Department before approval to issue the vaccine can be effected,

- the patient to be responsible for the cost of the vaccine and the cost of immunisation by the treating medical practitioner.

N.B. this product has not been evaluated by the Australian Drug Evaluation Committee, however the vaccine has been tested for safety by the National Biological Standards Laboratory (Virology) before release.

. FOR THE FOLLOWING GROUPS OF AUSTRALIAN TRAVELLERS:

- persons who will be resident in endemic areas for more than 12 months.

- travellers who visit rural areas during epidemics.

. TO ENDEMIC AREAS OF ASIA AND THE FAR EAST

- lowlands of Nepal, Bangladesh, India (in particular West Bengal, Uttar Pradesh, Andra Pradesh, DiBiher, Kamataka and Assam)

- Burma, Indonesia, Malaysia, Laos, Philippines, Singapore, Sri Lanka, Thailand, Democratic Kampuchea and Vietnam

- China, Taiwan, Hong Kong, Korea and Japan

- USSR (Eastern Province)

HERPETIC PELVIPERITONITIS: A NEW ENTITY (CANADA)
(A Case Report based on CDWR Vol 13/10, 14 March 1987)

A 25 year old woman was referred to a Montreal Hospital for evaluation of salpingitis. She had a history of lower abdominal pain beginning three months earlier which has ceased spontaneously. Three days before her presentation to the hospital, these symptoms reoccurred, localised in the left subumbilical region and the hypogastrium, in the form of a constant, regular but bearable sensation of heaviness, and accompanied by nausea. She stated that she had had a steady sexual partner for several years, although she had had sexual relations with a new asymptomatic young man a week before her hospitalisation. In the past 3 years she had used a diaphragm for contraception.

Medical history: Over a period of 3 to 6 months, she had had recurring sinusitis with tonsilitis and conjunctivitis, greenish sputum for a month, with progressive asthenia. She had also experienced an involuntary weight loss of 4.5 kg over the past month. The last three menstrual cycles had been characterised by progressive dysmenorrhoea. Two weeks before admission, a pregnancy had been interrupted by provoked abortion, and menstruation and started agains five days before her hospital presentation.

Clinical examination revealed the following:-

- a productive cough with post-prandial epigastric burning accompanied by anorexia, vesicorectal tenesmus and diarrhoea,
- a temperature of 38°C with tacycardia,
- pharyngeal engorgement and anterior cervical and retro-occipital adenopathy.

Gynaecological examination revealed a yellowish-brown leukorrhoea with vaginal and cervical erythemia. Mobilisation of the uterine cervix provoked exquisite adnexal pain, particularly on the left side. Pelvic echography showed right adnexal thickening, with hypertrophy and presence of liquid-filled lacunae on the left. Emergency exploratory laparoscopy demonstrated a distension of the adnexae with congestion of the pelvic organs, in particular, an inflammation of the ovarian fossae indicative of bilateral periovaritis. This was complicated by peritonitis with fluid accumulation in Douglas' cul-de-sac.

On the assumption that this was a bacterial pelviperitonitis, therapy was initiated with:

- sulbactam, 1g intravenously every 6 hours, and
- ampicillin, 2g intravenously every 6 hours.

The patient remained febrile and diarrhoeic for the first 6 days of hospitalisation.

At admission, cultures of vaginal and cervical secretions were taken for

- Neisseria gonorrhoeae,
- Chlamydia trachomatis,
- genital mycoplasmas,
- herpes simplex virus, and
- various other pathogens.

Specimens were also cultured from tubal biopsy material and the fluid in Douglas' cul-de-sac for the above pathogens, as well as for several other aerobic and anaerobic organisms.

On the sixth day of hospitalisation, herpes simplex virus type 2 (HSV-2) was isolated from the culture from:-

- the cervix,
- the left fallopian tube,
- the cul-de-sac fluid.

At that time, para-vulvar vesicular lesions erupted and culture of these also yielded HSV-2; a control culture from the cervix was also positive for HSV-2.

Endocervical cytology done at the same time revealed atypical cells associated with a viral infection of the genital herpes type.

The patient was then treated with acyclovir, intravenously and topically (peri-vulvar). In four days she was asymptomatic and returned home with a 10-day prescription for oral acyclovir antiviral treatment. Subsequent cervical and vaginal smears still indicated the presence of genital mycoplasmas and haemolytic streptococci (Lancefield Classification B). The other microbiological analyses remained negative. Ten days of tetracycline treatment resolved the genital mycoplasma condition. Post-therapeutic cultures for HSV and other organisms done during follow-up examinations remained negative.

CDWR Editorial comment:

The above case appears to be the first documented report of salpingitis and pelvic peritonitis caused by herpes simplex virus(1). While no prior history of genital herpes was reported, the patient may have had an asymptomatic cervical herpes simplex infection at the time of therapeutic abortion. Consequently, instrumentation may have facilitated spread to the upper genital tract, Swedish studies indicate that 0.5% of legal abortions are complicated by salpingitis(2). This case emphasises the importance of evaluating women for all potential genital pathogens prior to therapeutic abortion.

REFERENCES

1. Salpingitis - Sexually Transmitted Disease (1984):449-474
2. Am J Obstet Gynecol (1980) 138:880-892

NOTICE TO READERS:

- In response to the second last paragraph of our article on 'OPV - associated poliomyelitis - a review - Australia' in CDI 87/13, page 12, the National Biological Standards Laboratory (NBSL) - Virology has stated that NBSL has, as part of its routine quality control procedures, regularly monitored neurovirulence data of each batch of Sabin OPV used in Australia.

- Further to the CDI editorial comment on mefloquine in CDI 87/4, page 2, readers are now informed that the Drug Evaluation Branch of the Therapeutics Division has recently:-

  - approved the application by Roche Products Pty Limited to extend the indications for use of Lariam, mefloquine tablets 250mg to include prophylactic treatment of malaria, and

  - granted Roche Products Pty Ltd general marketing approval of Lariam, mefloquine tablets 250mg for use in the prophylactic treatment of malaria as in the product information approved by the Branch.

  Request has also been made for copies of the final version of the Product Information on mefloquine, annotated with the approval date. Roche Products Pty Ltd is yet to supply the Drug Evaluation Branch with the requested documents.

  The CDI will keep readers posted on the availability of this product. A full article on mefloquine will also be featured in the CDI once the final version of the complete product information can be obtained.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 13-7-87 to 26-7-87 BULLETIN NUMBER 87/15

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPMR (NSW)/ WVH (ACT)	RAHC (NSW)	PHH/ POW (NSW)	FAIR-FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
0100 ADENOVIRUS NOT TYPED..............	5		13		4	1	9	1	33
0101 ADENOVIRUS TYPE 1.................				1					1
0102 ADENOVIRUS TYPE 2.................	2				7	3		1	13
0103 ADENOVIRUS TYPE 3.................		1		1					2
0104 ADENOVIRUS TYPE 4.................				1					1
0105 ADENOVIRUS TYPE 5.................								2	2
0108 ADENOVIRUS TYPE 8.................						1			1
0199 ADENOVIRUS TYPING PENDING..........			3		5			1	9
0201 INFLUENZA A VIRUS.................	5								5
0202 INFLUENZA A VIRUS SUBTYPE H3N2.....						1			1
0203 INFLUENZA B VIRUS.................			1	2		1			4
0301 PARAINFLUENZA VIRUS TYPE 1.........					2	2	1		5
0302 PARAINFLUENZA VIRUS TYPE 2.........				1	1			2	4
0303 PARAINFLUENZA VIRUS TYPE 3.........	3	1		4	10	4	5	1	28
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)...	37	24	11	3	50	97	60	60	342
0500 RHINOVIRUS (ALL TYPES)............				5	7	3	5	3	23
0600 MYCOPLASMA PNEUMONIAE..............	10		13		2			3	28
0901 COXSACKIEVIRUS B1.................								1	1
0903 COXSACKIEVIRUS B3.................	3			1	12				16
0905 COXSACKIEVIRUS B5.................	1								1
1000 ECHOVIRUS NOT TYPED...............						1			1
1003 ECHOVIRUS TYPE 3..................		1							1
1011 ECHOVIRUS TYPE 11.................	2			4					6
1018 ECHOVIRUS TYPE 18.................	1	1			2				4
1022 ECHOVIRUS TYPE 22.................						1			1
1100 POLIOVIRUS NOT TYPED..............			4		4				8
1101 POLIOVIRUS TYPE 1.................		1						1	2
1102 POLIOVIRUS TYPE 2.................	1					1			2
1103 POLIOVIRUS TYPE 3.................						1			1
1104 POLIOVIRUS-VACCINAL STRAIN.........						1			1
1200 MUMPS VIRUS.......................	3			1					4
1300 HERPES VIRUS GROUP-NOT TYPED.......	23						1	1	25
1301 HERPES SIMPLEX VIRUS NOT-TYPED.....		2						3	5
1302 EPSTEIN-BARR VIRUS (EB VIRUS)......	14							9	23
1303 VARICELLA-ZOSTER VIRUS............	1		7			1	1	3	13
1306 HERPES SIMPLEX TYPE 1.............	14		13	27		5	36	14	109
1307 HERPES SIMPLEX TYPE 2.............	61		28	71		12	59	30	261
1399 HERPES VIRUS TYPING PENDING........					4				4
1401 COXIELLA BURNETI..................	8					2			10
1502 PICORNA VIRUS-NOT TYPED...........	7		8				8	3	26
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS)............................								1	1
1522 RUBELLA VIRUS.....................	3			6		2			11
1532 HEPATITIS B ANTIGEN...............	19		8	45		15	36	16	139
1535 HEPATITIS A ANTIBODY..............	1		2	9		10	2	2	26
1541 CHLAMYDIA A - C TRACHOMATIS........	4	1	10	9		19	22	34	99
1543 CHLAMYDIA A - LGV TYPE............							12		12
1556 CMV - CYTOMEGALOVIRUS.............	1		4	23	10	8	3	12	61
1563 CORONAVIRUS.......................	1								1
1564 ROTAVIRUS.........................	15	2	7	1	16	32	12	15	100
1599 ENTEROVIRUS TYPING PENDING.........		2	10		9				21
9992 ROSS RIVER VIRUS	1						1	1	3
Total................................	246	36	142	215	145	224	273	220	1,501

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 13-7-87 to 26-7-87 BULLETIN NO. 87/15
Viral Identifications by Clinical Information Table 1.
Code 00,99 -No ill or data; 01,02,11,12 -Respiratory; E3 -Encephalitis; M3 -Meningitis; 04 -Paralysis; 05,13 -CNS other unspec.; 07,49 -GI; 17,47 -Hepatic; 19 -CVS; 89 -Urinary; 06 -Skin/mucous.

VIRUS OR VIRAL ANTIGEN	No-ill or data	Respiratory	Encephalitis	Mening-itis	Paralysis	CNS other unspec	GI	Hepa -tic	CVS	Urin -ary	Skin/ mucs memb
0100 ADENOVIRUS NOT TYPED..........	2						1				
0101 ADENOVIRUS TYPE 1............		2									
0102 ADENOVIRUS TYPE 2............		10					3				
0103 ADENOVIRUS TYPE 3............		2									
0105 ADENOVIRUS TYPE 5............	1	1									
0201 INFLUENZA A VIRUS............		5									
0202 INFLUENZA A VIRUS SUBTYPE H3N2		1									
0203 INFLUENZA B VIRUS............		4									
0301 PARAINFLUENZA VIRUS TYPE 1....		5									
0302 PARAINFLUENZA VIRUS TYPE 2....		4									
0303 PARAINFLUENZA VIRUS TYPE 3....	3	26									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)............................	21	318					3				
0600 MYCOPLASMA PNEUMONIAE.........	4	12					1				1
0903 COXSACKIEVIRUS B3............		10		2			2				
0905 COXSACKIEVIRUS B5............							1				
1011 ECHOVIRUS TYPE 11............	1	1		1			1				
1018 ECHOVIRUS TYPE 18............	1		1	1			1				
1022 ECHOVIRUS TYPE 22............		1									
1101 POLIOVIRUS TYPE 1............							1				
1102 POLIOVIRUS TYPE 2............		1					1				
1103 POLIOVIRUS TYPE 3............		1									
1200 MUMPS VIRUS..................				1							1
1301 HERPES SIMPLEX VIRUS NOT-TYPED	1	1					1				1
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	6						1	2			1
1303 VARICELLA-ZOSTER VIRUS........	3		1	1		1					4
1306 HERPES SIMPLEX TYPE 1.........	11	8								1	51
1307 HERPES SIMPLEX TYPE 2.........	10									1	61
1401 COXIELLA BURNETI.............	2					1					
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS)......................											[illegible]
1522 RUBELLA VIRUS................											1[illegible]
1532 HEPATITIS B ANTIGEN..........	41							93			
1535 HEPATITIS A ANTIBODY..........	4							13			
1541 CHLAMYDIA A - C.TRACHOMATIS...	1										
1543 CHLAMYDIA A - LGV TYPE........	12										
1556 CMV - CYTOMEGALOVIRUS.........	12	19				1	2	1	1	5	
1563 CORONAVIRUS..................							1				
1564 ROTAVIRUS....................	6						94				
9992 ROSS RIVER VIRUS.............	1										
Total............................	143	432	2	6		3	114	109	1	7	131

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 13-7-87 to 26-7-87 BULLETIN NO. 87/15
Viral Identifications by Clinical Information Table 2.
Code 10 -Eye; 59 -Genital; 39 -Endo/sal gland;
38 -RES; 29 -Muscle/joint; 69 -Congenital; P8 -PUO;
G8 -Fever/malaise; 09 -Other; A1 -SIDS ...

VIRUS OR VIRAL ANTIGEN	Eye	Genital	Endo/sal gland	RES	Muscle/joint	Congenital	PUO	Fever/malaise	Other	SIDS
0100 ADENOVIRUS NOT TYPED..........								2		
0102 ADENOVIRUS TYPE 2............								1		
0104 ADENOVIRUS TYPE 4............	1									
0108 ADENOVIRUS TYPE 8............	1									
0199 ADENOVIRUS TYPING PENDING.....	1									
0203 INFLUENZA B VIRUS............								2		
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)............................					1		1	2	2	1
0600 MYCOPLASMA PNEUMONIAE.........			2				1	3	5	
0901 COXSACKIEVIRUS B1............								1		
0903 COXSACKIEVIRUS B3............						1				1
1003 ECHOVIRUS TYPE 3.............									1	
1011 ECHOVIRUS TYPE 11............								3		
1018 ECHOVIRUS TYPE 18............								1		
1101 POLIOVIRUS TYPE 1............								1		1
1104 POLIOVIRUS-VACCINAL STRAIN....									1	
1200 MUMPS VIRUS..................			2					1		
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			3	1				7	3	
1303 VARICELLA-ZOSTER VIRUS........									5	
1306 HERPES SIMPLEX TYPE 1.........	3	34					1	1	1	
1307 HERPES SIMPLEX TYPE 2.........	1	190								
1401 COXIELLA BURNETI.............					1		2	3	1	
1522 RUBELLA VIRUS................									1	
1532 HEPATITIS B ANTIGEN...........					1				6	
1535 HEPATITIS A ANTIBODY..........									9	
1541 CHLAMYDIA A - C.TRACHOMATIS...	1	94						1	2	
1556 CMV - CYTOMEGALOVIRUS.........	2	1	1	1	1	3		3	13	
9992 ROSS RIVER VIRUS.............					2					
Total............................	10	319	8	2	6	4	5	32	50	3

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

Period 3 - 22 February 1987 to 21 March 1987

Bulletin...87/15...

Disease	N.S.W.	VIC.	QLD.	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	Cumulative Total to Date for Year
Amoebiasis	1		1	2				2	6	14
Ankylostomiasis				1	2		NN		3	9
Anthrax									-	1
Arbovirus infection	10	1	111		9				131	194
Brucellosis	1		1						2	6
Campylobacter infections	121		NN	81	18	NN	8	1	229	784
Chancroid				NN	1				1	3
Cholera			1						1	1
Congenital rubella syndrome			NN			NN		NN	-	-
Diphtheria							1		1	5
Donovanosis			2	NN	4				6	21
Giardiasis	26		NN	88	20	NN	NN	NN	134	362
Genital herpes	101		22	23	NN	NN	1	NN	147	527
Gonococcal ophthalmia neonatorum		NN			NN	NN		NN	-	1
Gonorrhoea	75		156	67	103	1	64	2	468	1 421
Hepatitis A (infectious)	11	9	6	7	4		1		38	172
Hepatitis B (serum)	25	36	11	10	39		2		123	365
Hepatitis – unspecified	6		28	4	NN	NN			38	55
Hydatid disease									-	1
Lassa fever			NN			NN		NN	-	-
Legionnaires disease			NN			NN		NN	-	2
Leprosy	1		1		1			1	4	5
Leptospirosis	4	2	11		1	2			20	46
Lymphogranuloma venereum				NN	NN	NN		NN	-	-
Marburg disease			NN			NN		NN	-	-
Malaria	16	10	33	1	1	1	3	6	71	160
									-	-
Meningococcal infections	1				1	NN			2	17

Disease	N.S.W.	VIC.	QLD.	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	Cumulative Total to Date for Year
Non-specific urethritis	345		12	71	NN	NN	NN	NN	428	962
Ornithosis				1					1	2
Pertussis (whooping cough)	6	4	NN	4	10	NN	9	NN	33	136
Plague									–	–
Poliomyelitis									–	–
Q. fever	28		20	2					50	101
Rabies				NN		NN		NN	–	–
Salmonella infections	85	9	94	23	23	28	33	2	297	744
Shigella infections	5	3	12	8	23		18		69	157
Smallpox									–	–
Syphilis	26		23	13	18		81	1	162	436
Tetanus									–	1
Trachoma		NN	4		2	NN	NN		6	16
Tuberculosis (all forms)	36	21	6	7	9			3	82	207
Typhoid fever	1	1							2	18
Typhus (all forms)									–	–
Vibrio parahaemolyticus infections			NN			NN		NN	–	1
Yellow fever									–	–
Yersinia infections	6		NN	1		NN		NN	7	22

NN - Not Notifiable

(Note: Data collected under the Notifiable Diseases Returns may bear little or no correlation to that collected under the CDI laboratory scheme. Whilst the latter is a sampling program, the Notifiable Diseases data is dependent upon voluntary reporting by medical practitioners etc.)