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Communicable Diseases Intelligence

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Contents:

VIRUS REPORTING SCHEME: A total of 1 216 reports were processed for this period.

Eleven cases of Q fever were reported, 5 from New South Wales and 6 from Queensland. Occupational exposure data were only available for one Queensland case, a 46 year old male meatworker from Kingaroy. None of the eleven patients was involved in the Q fever vaccine field trial conducted in South Australia.

Rotavirus was isolated from the faeces of a 25 year old, HIV antibody positive male who presented with persistent diarrhoea.

Adenovirus type 2 was isolated from the faeces of a one month old male who died of Sudden Infant Death Syndrome. Adenoviruses infect epithelial cells of mucous membranes, the cornea and other organ systems. Intussusception of infancy has also been associated with adenoviruses types 1, 2, 3 and 5.

Cytomegalovirus was isolated from the urine of a 52 year old male who experienced prolonged fever of unknown origin following cardiac transplantation surgery.

EFFICACY OF HEPATITIS B VACCINE IN ABORIGINAL INFANTS

(Dr J. Sheridan - Personal Communication)

A hepatitis B vaccination program for Aboriginal and Torres Islander neonates in Queensland commenced in 1986. Follow up studies identified 235 infants in whom the third dose of hepatitis B vaccine was given at least 8 months prior to the survey. To date, serological test results available for 81 infants living in locations scattered throughout Queensland, indicate that 71 infants had seroconverted. These preliminary findings suggest a seroconversion rate of 88% indicating that hepatitis B vaccine is effective in Queensland Aboriginal and Islander infants.

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In a much smaller pilot study a seroconversion rate of 50% was found in a limited hepatitis B vaccination program conducted on 22 Aboriginal infants in several remote Central Australian communities of the Northern Territory.

It was stated (without documentary evidence) that the vaccine had been correctly transported, stored and administered, and was suggested that the results may be related to a reduced immune responsiveness in the study group.

CDI editorial comment.

It is not possible to determine from the limited data supplied whether any real differences in immune responsiveness exist between the two study groups. Despite assurances that the vaccine used in the second study was properly transported, stored and administered, no evidence has been provided describing how this was monitored.

It is essential that both the transport and storage of the vaccine be carried out within the recommended temperature range to avoid both excessive heat and freezing which can markedly reduce the immunogenicity of the product.

The route of administration is also important. Data suggest that injections given in the buttocks are frequently delivered into fatty tissue instead of into muscle. Such injections may result in a lower seroconversion rate than is expected. The anterolateral thigh is the recommended site for intramuscular injection in infants and children.

Finally, the small number of subjects in the second study renders questionable the clinical significance of the results obtained. A larger, properly controlled study would be necessary to assess accurately the seroconversion rates of infants in the community under study. The editor is aware that such a study is being planned/undertaken by the Northern Territory Department of Health.

CONFERENCE ON HEPATITIS B IMMUNISATION
(based on CDWR Vol 12-47, 22 November 1986)

The following is a summary of selected topics presented at the Conference, held on 26 September 1986 in Toronto, Ontario.

Hepatitis B Vaccine - The First 5 Years

Highlights of vaccine utilisation, including the early trials, were reviewed. Results of efficacy studies on medical staff in haemodialysis units and male homosexuals indicated protection rates of over 90%.

Failure was observed only among individuals who had just begun the immunisation process. Dialysis units in the United States are among the few settings which have infection with one type of virus, namely the ay subtype. Although vaccine is made from subtype ad, it still provides protection against ay through stimulation of antibody formation to the common a antigen. Early vaccine trials demonstrated that if infection was

prevented, so was a hepatitis B carrier state which follows in 5 to 10% of adult cases.

Antibody response is commonly described in terms of the ratio of the sample to the negative controls, the so called S/N ratio. A relatively weak antibody response would be 2.1 to 9.9 S/N units. Measuring antibody in reference to a World Health Organization standard of milli international units gives a more accurate determination of antibody response. Antibody level declines gradually over time which is similar to that generally seen following vaccination against other diseases. Vaccinees followed over 4 to 5 years show that about 10% lose detectable antibody; 15 to 20% will decline to relatively low levels of antibody of 10 S/N. The protective level of antibody has been generally accepted as 10 S/N or greater which is usually achieved by at least 90% of individuals receiving a course of 3 injections of vaccine. At 5 years post-vaccination, about 25 to 30% will have undetectable antibodies or less than 10 S/N. The crucial question is whether such persons will retain protection over the long term. Infections have been documented however, they are usually manifested only by seroconversion (core antibody-positive), without detectable antigenemia or evidence of liver inflammation. Hence, although infection can occur in these vaccinees, the resulting disease is not clinically significant. To date, more than 50 such infections have been identified in 2 separate studies. Only one individual was shown to have transient antigenemia and mild liver enzyme elevation. None developed a chronic carrier state. Therefore, even though antibody levels decline over time, vaccinees are still generally protected. If initial antibody response is low, detectable antibody may be lost in 2 to 3 years. If initial response is high, detectable levels have now been shown to be retained for 7 or 8 years and probably will persist longer. Booster doses could therefore be individualised in terms of the initial antibody response or the amount of decline in titre. A universal recommendation could be made for a dose of vaccine at 5, 7 or 10 years post-initial immunisation. The most practical and easiest solution would be to take a uniform position which would protect most people, e.g. a booster at 5 years.

Incident at St Michael's Hospital, Toronto

The events surrounding the death of a Toronto surgeon due to fulminant hepatitis B following a needlestick injury during surgery, and the impact on the medical community. Earlier attempts at vaccinating appropriate hospital staff had achieved a success rate of less than 50%. Following the death of the surgeon, a rush for vaccination occurred and over 1 000 staff members started a course of vaccine. Problems in coping with various staff groups and the media were described.

Metropolitan Toronto Hospitals:

The hepatitis B vaccine experience in Metro Toronto hospitals since the programs began in 1983 was reviewed. All teaching hospitals and most community hospitals now offer the vaccine to employees whose work places them at risk for hepatitis B virus infection. Most hospitals have provided the vaccine at no cost

to the individual as part of the commitment to the health and safety of employees. However, others have funded the vaccine through an extended health benefit program. Physicians in private practice are responsible for their own vaccine. Hospital staff in general were slow to accept the vaccine, and many choose to delay their immunisation. Following the hepatitis B death of a Toronto surgeon, however, earlier irrational resistance was replaced by irrational demand, and for a brief time the vaccine supply was exceeded by the demand. As of 1 September 1986, the course of 3 injections had been given to 7720 hospital staff including medical staff, interns/residents, and clinical clerks. Nevertheless, many health-care workers at risk including physicians remain unimmunised.

CHAIRMAN'S CONCLUDING REMARKS

This Conference attracted 250 doctors, nurses, public health, and hospital representatives from every province. It is clear that hepatitis B immunisation continues to be of major interest in Canada.

An earlier reluctance by many people to have the vaccine has gradually been replaced by a more positive acceptance. The efficacy and safety of the plasma-derived vaccine currently in use have been demonstrated over and over again . Public health funding has moved forward, but is inconsistent across the country. Immunisation within the male homosexual community remains a challenge.

All provinical health departments now provide vaccine without charge for infants born to HBsAg-carrier mothers. In addition, most provinces provide vaccine for household contacts or at least for sexual contacts of cases or carriers. Vaccine is also usually provided for high-risk patients such as haemophiliacs. Most provincial health departments provide vaccine for residents of institutions for the mentally retarded, but staff are not always included as they are frequently considered an institution responsibility. There is considerable variation in the provision of vaccine for health-care workers. Generally, no provision is made for vaccine for homosexuals, drug addicts, prostitutes, and prisoners.

Mothers/Babies/Children:

The following is a perspective on a community-based program for this target group. In 1984 and 1985 a program was put in place in Metro Toronto hospitals for the identification of infants born to HBsAg-positive mothers and for immediate immunisation. A personal immunisation record is provided for the parent. By the time of hospital discharge, follow-up mechanism for these infants is usually well established with the idea that subsequent doses will be given in the community. Immunisation is carried out either by the attending private practitioner or in a public health clinic. An information package was developed which includes a vaccine order form and an information letter for the attending physician. Some 80 orders for vaccine have been processed between January and August 1986 which is a rate of approximately 1.5 per 100 live births among City of Toronto residents.

Feed-back to practitioners and a thrust toward better professional education is being carried out through a quarterly newsletter to Toronto primary-care physicians. Public awareness is also being addressed through provision of a counselling service for newly identified carriers, and an information letter is available in 9 languages. A videotape has also been produced which is available through the Toronto Board of Education.

The Gay Community:

A review of the studies within the past 13 years identified male homosexuals as a group of particularly high risk for hepatitis B infection. Male homosexuals have an 8 to 10-fold greater chance of being exposed to hepatitis B than their heterosexual counterparts. However, there does not appear to be any evidence that they are more likely to become carriers of the virus. Attention was drawn to clinical trials which demonstrated high vaccine efficacy in homosexual populations.

Infection rates of 17% per year were seen in the unimmunised compared to only 1.4% in the immunised. Those who do not show an antigenic response to an initial course of vaccine do not generally respond to boosters. Institution of vaccination programs for the homosexual population is difficult but physicians bear a responsibility to recommend immunisation to known homosexuals in their practice.

Legal Issues:

A number of important legal issues relating to vaccine usage have been reviewed.

Negligence can be established only by proving a failure to live up to average, reasonable and prudent standards. Harm must be reasonably foreseeable as an outcome of an act for such an act to be one of negligence. Handling blood or blood products contaminated with hepatitis B in a way which could result in forseeable injury, failure to take proper measures in respect to instituting appropriate immunisation of neonates, failure to provide proper training for staff working with hepatitis B patients or residents, and failing to abide by legislated requirements could all constitute negligence. Medical staff privileges should be contingent on prudent standards such as hepatitis B vaccination for staff at special risk. Staff privileges are not a right; they are a licence to provide a standard of patient care. Therefore, immunisation can be made a bonafide occupational qualification for all clinical and laboratory staff within hospitals - with exceptions for those who are known to be at special risk of adverse reactions.

Consent to treatment is another important area of litigation in Canada. Criteria for a valid consent include pointing out the nature and purpose of a vaccine to all potential recipients. Risks and benefits should be specified along with reasonable options. The impact resulting from a refusal should be detailed. Documentation of consent should be incorporated in the person's record as per provincial law on the subject or if no requirement is so specified, details of any conversation

regarding the vaccination, plus date and signature should be recorded. For employees, such records should be retained as long as employment continues. A good record can make the case for defence should litigation arise. It can also assist in carrying out an obligation to warn, e.g. if antibody titre is dropping and a booster is needed.

Government involvement:

In 1982, a Hepatitis B Vaccine Advisory Committee was established for the purpose of prioritising use, developing methods for screening candidate vaccinees, and advising on distribution of vaccine and cost recovery. Release of the vaccine coincided with concerns about AIDS and this together with the cost recovery aspect resulted in an unexpectedly low acceptance of the vaccine. In Ontario, vaccine is now provided free for infants born to carrier mothers. This was extended early in 1986 for all close family contacts. In addition, other categories of persons are covered such as persons suffering from haemophilia and thalassemia, and medical and paramedical staff employed by the province who are at risk, e.g. ambulance attendants.

Hepatitis B Vaccine - Present and Future:

These steps taken to remove all adventitious agents from the vaccine, ie treatment with urea, pepsin and formalin, were reviewed. These will eliminate any known virus contaminant including AIDS virus. Recipients of Hepatavax - B(R) have never been shown to have a related increase in HIV antibodies.

It is recognised that there are low or non-responders to the vaccine. Some are immunodeficient or immunodepressed individuals and some may have a genetic inability to respond (about 5% of the population). Route of administration is also important.

Production of a second generation vaccine by recombinant DNA technology has been encouraged because of a lack of acceptance by some persons of a plasma-derived product, and because plasma sources for manufacture of vaccine will eventually become more limited.

The production of the second generation vaccine from yeast cells was described and evidence was reviewed showing that the vaccine is similar in immunogenicity to plasma-derived vaccine. It was licensed in late summer of 1986 by the U.S. Food and Drug Administration and marketing will probably commence in January 1987 in the United States; it is currently being reviewed by the Canadian Bureau of Biologics. It is the first recombinant vaccine licensed for use in humans. The question of possible reactions due to hypersensitivity to yeasts has arisen but the vaccine is highly purified, and bread and beer contain many more yeast contaminants than the vaccine. Hence, individuals would not likely react to the vaccine unless they had a known anaphylactic hypersensitivity reaction to bread or beer. Furthermore, anti-yeast antibodies are easily demonstrated in all individuals.

The need for a third generation vaccine is being considered to deal with the 5% of the population who will not respond to the currently licensed plasma-derived and recombinant vaccines.

Results of antibody studies conducted in New York City showed that recombinant vaccine gave comparable results to plasma-derived vaccine. Efficacy studies in newborn infants were also described showing that prevention of infection and of the chronic carrier state is also being achieved with the recombinant vaccine.

In regard to intradermal inoculation, it is known that higher immune responses are elicted by intradermal injection compared to the same antigen deposited intramuscularly. However, there are 2 problems with giving hepatitis B vaccine intradermally: 1) the vaccine in current use is alum-absorbed and will frequently cause considerable soreness, discoloration, and often nodule formation at the site of inoculation; and 2) it is very difficult to consistently give good intradermal injection and, if the vaccine is deposited subcutaneously, it will be less effective.

Regarding pregnancy and vaccination, if a susceptible pregnant woman is in a high-risk setting and will be continuing in that setting, she should be vaccinated. Otherwise, if she becomes infected, she may transmit the virus to her infant.

Perinatal transmission of hepatitis B to offspring can occur if the mother suffers acute infection near the time of delivery or if she is a chronic carrier. Transmission is not likely if the acute infection occurs early in pregnancy. The major point of transmission is probably at the time of delivery. Mothers carrying e antigen are more likely to infect their babies as this marker is indicative of high levels of virus. When carrier mothers have no e antigen or have anti-e antibody, rate of transmission to offspring is low.

Babies infected in the newborn period generally do not have clinical symptoms but they may have slight elevation of liver enzymes and become chronic carriers with risk of cirrhosis or primary liver cancer in later years. However, hepatitis B immune globulin given right after birth will provide an infant immediate protection and give time for active response to a course of vaccine. A study being conducted in New York City, San Francisco and Los Angeles shows that passive-active immunisation reduces the chronic carrier state to 10 to 12% compared to 40 or 50% in infants treated with HBIG alone. There is also a difference in the time of onset of antigen positivity. Vaccine appears to prevent the late infections which occur in infants treated with HBIG alone.

Pre-exposure prophylaxis is recommended in settings with a recognised risk of infection, e.g. health-care workers, homosexually active men, patients with requirements for clotting factor concentrates, household contacts of antigen carriers, institutionalised mentally retarded persons, haemodialysis patients, and I.V. drug abusers.

Post-exposure prophylaxis is recommended in 3 situations, i.e. passive-active immunisation in neonates, percutaneous inoculation as in a needlestick accident, and in certain categories of sexual exposure.

Screening is generally recommended for pregnant women who belong to groups known to have high carrier rates, e.g. women of Pacific-Island, Asian or Eskimo descent, those born in an endemic areas such as Haiti or Africa, and those with a history of liver disease or occupational exposure. This screening approach is based on the likelihood of discovering the greatest number of high-risk babies. It was suggested that it would probably be better to screen all pregnant women as a routine because obstetricians are unlikely to search diligently for the possibility of pregnant women patients belonging to high-risk groups. Only a test for surface antigen need be used for screening. Since it is recommended that all infants born to carrier mothers be immunised, it is unnecessary to test for other markers. Screening should be performed as close as possible to the time of delivery since there is a continuing risk of infection in some categories of women such as drug addicts or unvaccinated health-care workers.

Lack of immunogenic response following vaccination may be due to several factors including freezing of the vaccine, making it ineffective; inadequate mixing of the alum-absorbed vaccine and dilutent; and inadvertent injection into fat rather than muscle. In addition, heavy people respond less well than thin individuals, and people over 50 respond less well than those in their 20s and 30s.

The issue of declining antibody following immunisation was addressed. Follow-up studies extending over several years provide assurance that even when antibody values decline to low and undetectable levels, protection against clinical disease persists. This leads to the question of booster injections and their frequency. Formal recommendations must await the deliberations of the national advisory committees on immunisation in both the United States and Canada. The U.S. speakers agreed that a booster injection after 5 years might be a practical strategy.

While much has been accomplished in the development of hepatitis B vaccines and immunisation programs, there is more to be done. Many physicians and dentists remain unimmunised as do thousands of hospital clinical and laboratory staff. Existing programs must renew their efforts at least until immunisation becomes routine at the undergraduate level of medical, nursing, dental and laboratory technology schools.

A recombinant vaccine has been developed, and will likely be available soon. The future is bright, and awareness has never been better.

SPONTANEOUS ABORTION CAUSED BY CHLAMYDIA PSITTACI OF OVINE ORIGIN (U.K.)

(Based on CDR 86/45 - 7 November 1986)

A sheep farmer's 30 year old pregnant wife became unwell on 13 February 1986 with fever, sore throat and generalised arthralgia. This episode settled spontaneously after 2 weeks but on 13 March she became ill again with a high temperature,

arthralgia and retrosternal chest pains, made worse by coughing. Over the next 2 weeks she became jaundiced, looked thin and unwell. On 28 March she was admitted to hospital with jaundice and dehydration, a temperature of 38°C and a pulse of 100/min. On admission she was 15 weeks pregnant, her chest was clear, the abdomen was soft and the liver enlarged, palpable 2-3 finger breadths below the right costal margin and the spleen palpable 1 finger breadth.

Serological investigations on a sample of clotted blood submitted on admission demonstrated elevated complement fixation titres (CFTs) to Chlamydia psittaci, Coxiella burnetii and Influenza A. Other agents associated with atypical pneumonia (Mycoplasma pneumoniae, Influenza B and Legionella pneumophila) had negative titres. Interpretation of differential titres to these agents was made possible by the retention of an antenatal sample of blood submitted in February for rubella antibody tests. Comparison of CFTs on the two samples showed rising titres to C.psittaci, C.burnetii and Influenza A (Table).

Table

	Serum of 20.2.86 titre	Serum of 27.3.86 titre
Influenza A	< 8	192
Influenza B	< 8	< 8
C. psittaci	< 8 (IF titre < 16)++	768+ (IF titre 1024)
C. burnetii	48*	1024
Legionella	< 8	< 8
M. pneumoniae	< 8	< 8

Comments: Bi-phasic illness commencing 13.2.86 resolution and recurrence on 13.3.86.

* C. burnetii specific IgM - POSITIVE (by immunofluorescence).

\+ C. psittaci specific IgM - POSITIVE (by immunofluorescence).

++ Immunofluorescence using ovine strain of C. psittaci.

Other serological tests for hepatitis A and B and Leptospirosis were negative. The husband was found to have evidence of past infection with C. burnetii (CFT 24) and C. psittaci (CFT 16).

Other biochemical investigations included:

. liver function tests on admission showed:
  - alkaline phosphatase of 1 233 IU/L (normal 80-120 IU/L)
  - albumen 28 g/L (normal 35-50 g/L)
  - bilirubin 39 µmol/L (normal 5-17 µmol/L)
  - alanine aminotransferase of 32 IU/L (normal 40 IU/L)

. haematological profile showed:
  - blood viscosity of 1.75

- haemoglobin of 11.4 g/L
- white cell count of 7.4 x 10^6/L (81% neutrophils)
- platelets of 184 x 10^7/L (a previous platelet count taken by her general practitioner was low at 35 x 10^7/L, suggesting disseminated intravascular coagulation)

. urea and electrolytes were normal but blood gases revealed mild hypoxaemia (Po_2 71.6 mm Hg)

. chest X-ray showed consolidation in the medial segment of the right middle lobe

. an abdominal ultrasound showed that foetal movement had ceased - clinical examination suggested that the foetus was non-viable and the dead foetus was aborted spontaneously 2 days after admission.

The aborted foetus and placenta were collected and laboratory examined for evidence of C. psittaci and C. burnetii infection:

. Foetus: heart, lung, spleen, liver and brain tissues were all negative when examined using genus specific (detects C. trachomatis and C. psittaci) and species specific (C. trachomatis) monoclonal antibodies in a direct immunofluorescence test.

. Placenta: smears prepared from the placenta were examined using both monoclonal antibodies
  - the species specific monoclonal antibody (C. trachomatis) gave negative results
  - the genus specific monoclonal antibody (C. psittaci/C. trachomatis) demonstrated large numbers of elementary bodies, brightly staining cells with large intracytoplasmic and an abundance of smaller particulate bodies resembling free lipopolysaccharide antigen. It was concluded from these studies that C. psittaci was present in the placental tissue in large amounts. Further examination of extracts of the placenta in McCoy cell culture demonstrated cells containing intracytoplasmic inclusions which were stained by the genus specific monoclonal antibody but not by the species specific monoclonal antibody. This strain of C. psittaci isolated in McCoy cell cultures is undergoing DNA analysis in an attempt to demonstrate the ovine origin of the isolate.

Attempts to isolate C. burnetii from the foetus and placenta by inoculation of guinea-pigs were unrewarding.

TREATMENT

On admission the patient was treated with erythromycin and ampicillin 500 mgs four times a day. Ten days previously she had received an oral course of erythromycin from her general practitioner. After the confirmed diagnosis of C. psittaci infection (and after her spontaneous abortion) doxycycline 100 mgs once daily for 10 days was substituted for the erythromycin which had been given for 3 days. Following a second course of erythromycin her jaundice began to settle and

her temperature returned to normal three days after admission. She was discharged on doxycycline which was continued for one week. When presented to outpatient clinic for review 2 weeks later she was in good health and a full blood count, liver function tests and chest X-ray were normal.

DISCUSSION

The patient and her husband lived on a sheep farm. Both had been counselled and were fully aware of problems associated with C. psittaci of ovine origin. They owned a flock of British Friesland milking sheep which had experienced an abortion problem in January 1985. Enzootic abortion was diagnosed following the demonstration of C. psittaci inclusion bodies in modified Ziehl - Nielson stained smears from thickened placenta, and high complement fixation (CF) titres to C. psittaci in the blood of aborting ewes. Following this incident the flock was vaccinated against enzootic abortion in the autumn of 1985 and there were no abortions during the current lambing season.

After the patient's illness, blood from 13 ewes in the flock was serologically tested for chlamydia and C. burnetii. All were positive to both organisms and CF titres for C. psittaci and C. burnetii ranged from 16 to 256.

The patient presented with a bi-phasic illness which commenced on 13 February 1986:

- her first illness could be attributed to Q fever infection as a low level of antibody was present in her antenatal blood which subsequently rose to a high level. The positive C. burnetii IgM test on the first serum sample supported this conclusion. In addition, all the sheep on the farm which had been tested showed antibody to Q fever in the CFT. Seropositive sheep are known to excrete C. burnetii and this infection in sheep is not reportable in the UK.

- After resolution of her first illness (without treatment), symptoms reappeared 2 weeks later. These symptoms were associated with influenza A virus (virus was circulating in the community at the time) and C. psittaci by serological tests (Table). It is likely from the clinical presentation, but unproven, that C. psittaci was responsible for the onset of her jaundice and her abnormal chest X-ray. Furthermore disseminated intra-vascular coagulation has been previously described in human cases of C. psittaci (ovine) infection. The rises in antibody to influenza A and Q fever with a positive IgM response to Q fever is not an anamnestic reponse as other antigens tested in parallel (influenza B, legionella and M. pneumoniae) were negative. The isolation of C. psittaci from the placenta identified the aetiological agent of the spontaneous abortion and DNA analysis of this isolate has recently demonstrated its ovine origin.

The values of storing serum samples is highlighted by this case which allowed more precise serological assays to be carried out pinpointing the aetiology of the bi-phasic illness of this patient. The use of monoclonal antibodies to differentiate

rapidly between C. psittaci and C. trachomatis is also shown in this case. This report adds to the accumulating evidence that C. psittaci of ovine origin is a hazard to pregnant women in the farming community[1,2,3,4]. However, the exact mode of transmission has not been determined in this case since on close questioning the patient stated that she had no contact with either the sheep or the sheep's milk from the onset of her pregnancy. In this case at least, other mode of transmission eg. mechanical means involving the husband's boots, clothes etc., or airborne routes should be considered.

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EPIDEMIOLOGICAL FEATURES OF AUSTRALIAN ENCEPHALITIS

Australian Encephalitis (AE), formerly known as Murray Valley Encephalitis, is an acute viral encephalitis due to infection with the mosquito-borne flavivirus, Murray Valley Encephalitis virus (MVEv), and rarely with the closely related Kunjin flavivirus[1]. The disease is one of a group of arthropod-borne viral (arboviral) diseases which occur in Australia. Asymptomatic infection is very common[2], and estimates vary from 1:800 to 1:3000 cases to infections. Significant sequellae are seen in approximately 30% of cases.

MVEv was first isolated from encephalitis cases during an epidemic in south-east Australia in 1951[3,4,5]. Subsequent analyses of sera and clinical observations from earlier epidemics of 'X-disease' or 'mysterious-disease' suggested that these were also due to infection with MVEv[6].

AE occurs as infrequent but severe epidemics in south east Australia,[2] with occasional outbreaks of only a few cases. Sporadic cases have also been recorded from northern Australia[7], and two cases have been reported from Papua New Guinea[8,9]. A summary of known human cases is presented in Table 1.

During epidemics of AE in south-east Australia, cases are more common in males (61.9%) than females (38.1%). Case fatality rates are also higher in males (35.4%) than females (22.5%). The overall case fatality rate is 28.8%. Cases are more common in children under 15 years of age (47.6% of cases, case fatality rate of 38%) and in people over 50 years old (22.9% of cases, case fatality rate of 29.2%). This trend is more pronounced in males than females. The date of onset of the first case in severe epidemics is generally in the first week of January, and cases are recorded through to late April, early May. In minor outbreaks in south-east Australia, the first case is generally not recorded until mid February.

In north-west Australia, the disease is generally more benign, with no fatal cases being recorded since 1974. Severe sequellae have been recorded in a number of cases. More males than females are affected (76.5% of cases are males). The mean

age of cases in Aboriginals is less than two years whilst in Caucasians, it is approximately 30 years. Cases are generally recorded from February to May and occasionally much later (June/July) in the year.

Detailed studies on arbovirus ecology have shown that MVEv activity can be detected annually in north west Australia[14], but is less frequently detected in north eastern Australia[15]. Studies in the Murray Valley since the last epidemic in 1974 have not demonstrated the presence of MVEv. However, epidemiological data cast doubt on the theory that the virus survives in northern Australia and spreads southwards during periods of epidemic activity[16]. Current views suggest that MVEv survives in cryptic (possibly by trans-ovarial transmission) cycles throughout Australia, and that overt activity, and hence human infection, is the result of specific combinations of environmental and biological conditions.

PREDISPOSING FACTORS IN SOUTH EAST AUSTRALIA

AE activity in south-east Australia is associated with excessive summer rainfall[2] and generally accompanies severe flooding in the Murray/Darling basin. Summer rainfall in eastern Australia is directly related to the phenomenon known as the Southern Oscillation (SO)[17], the most severe manifestations of which are the 'El Nino' and its accompanying weather disruptions. The SO can be used to give some predictive indices for the occurrence of AE in south-east Australia[19]. Biological correlates with AE in south-east Australia are a) the early appearance of mosquito plagues in August/September (as opposed to late November in most years)[19]; and b) the overt breeding of water fowl, particularly herons, on a very large scale[20].

Table 1: NUMBERS OF CASES OF AUSTRALIAN ENCEPHALITIS IN ALL STATES OF AUSTRALIA SINCE 1917[a,b,c].

	NSW	VIC	SA	QLD	NT	WA	TOTALS	REFERENCE
1917	70			44			114	2,7
1918	49	13		5			67	2,7
1922				75			75	2,7
1925	10			11			21	2,7
1951	10	34	4				48	2,7,10
1956		3					3	2,7
1969						1	1	11
1971	1			1			2	2,7
1974	5	27	10	10	5	1	58	2,7
1978						8	8	7
1979						2		7
1981				2	1	8	11	7
1984						2	2	12
1986				1		1	2	13
TOTALS	145	77	14	149	6	23	413	

a) The numbers refer to clinically recognizable cases, usually confirmed by serology after 1951.
b) To June 30, 1986.
c) Two cases from New Guinea in 1956[9] and 1960[8]

DIFFERENTIAL DIAGNOSIS OF AUSTRALIAN ENCEPHALITIS

AE is not readily identified by symptoms alone. Presentation of cases is variable. The major symptoms are sudden onset, fever, nausea, headache, vomiting and non-specific dizziness; followed after 2-5 days by symptoms of meningitis and brain dysfunction[21]. Serological confirmation is required for correct diagnosis.

CDI EDITORIAL COMMENT:

Arboviral diseases are notifiable in all States and Territories of Australia. It is essential that serological confirmation is sought whenever an arboviral disease is being considered in a patient, and that the case is correctly notified to the appropriate State or local authorities.

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AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 5-1-87 to 18-1-87 BULLETIN NUMBER 87/2
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	4	1	5		2		6	1	19
0101 ADENOVIRUS TYPE 1				2	3	4		2	11
0102 ADENOVIRUS TYPE 2	1				4	1		1	7
0103 ADENOVIRUS TYPE 3				1	4	1			6
0105 ADENOVIRUS TYPE 5				1		4			5
0106 ADENOVIRUS TYPE 6						4			4
0107 ADENOVIRUS TYPE 7					3				3
0108 ADENOVIRUS TYPE 8				1		1			2
0109 ADENOVIRUS TYPE 9	1								1
0111 ADENOVIRUS TYPE 11	1								1
0113 ADENOVIRUS TYPE 13	1								1
0119 ADENOVIRUS TYPE 19						1			1
0126 ADENOVIRUS TYPE 26	1								1
0137 ADENOVIRUS TYPE 37								1	1
0199 ADENOVIRUS TYPING PENDING			1		3				4
0201 INFLUENZA A VIRUS		1				2			3
0203 INFLUENZA B VIRUS	1								1
0301 PARAINFLUENZA VIRUS TYPE 1						1			1
0302 PARAINFLUENZA VIRUS TYPE 2								1	1
0303 PARAINFLUENZA VIRUS TYPE 3	3	1	1	2	4	7	3		21
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	1					5		7	13
0500 RHINOVIRUS (ALL TYPES)	2		1	1	6	13	4		27
0600 MYCOPLASMA PNEUMONIAE	1	1	4			7		6	19
0700 ORNITHOSIS-PSITTACOSIS	1					1	5		7
0816 COXSACKIEVIRUS A16						1			1
1005 ECHOVIRUS TYPE 5				1					1
1006 ECHOVIRUS TYPE 6								5	5
1011 ECHOVIRUS TYPE 11	4	1		14	22	3			44
1022 ECHOVIRUS TYPE 22						1			1
1031 ECHOVIRUS TYPE 31						1			1
1100 POLIOVIRUS NOT TYPED			5		4				9
1102 POLIOVIRUS TYPE 2	1	2							3
1200 MUMPS VIRUS								2	2
1300 HERPES VIRUS GROUP-NOT TYPED	10		1			2	4	2	19
1301 HERPES SIMPLEX VIRUS NOT-TYPED								1	1
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	2	2	2				8	2	16
1303 VARICELLA-ZOSTER VIRUS	2		2			2	1	6	13
1306 HERPES SIMPLEX TYPE 1	14		11	19	1	19	50	32	146
1307 HERPES SIMPLEX TYPE 2	78		31	22		21	62	63	277
1399 HERPES VIRUS TYPING PENDING					3				3
1401 COXIELLA BURNETI	5						6		11
1502 PICORNA VIRUS-NOT TYPED	1	1	6	1			5	1	15
1521 MEASLES VIRUS								4	4
1522 RUBELLA VIRUS	7		5	3		4	14	4	37
1532 HEPATITIS B ANTIGEN	36	2	4	27	1	8	18	11	107
1535 HEPATITIS A ANTIBODY	3		4	8		4	2	3	24
1541 CHLAMYDIA A - C TRACHOMATIS	60		5	21	1	27	35	67	216
1556 CMV - CYTOMEGALOVIRUS	5	1	7	10	1	3	5	10	42
1563 CORONAVIRUS				2					2
1564 ROTAVIRUS	20	2	2			5		3	32
1571 ENTEROVIRUS TYPE 71 (BRCR)	1	1		1					3
1599 ENTEROVIRUS TYPING PENDING	2	1	3		4		2		12
9992 ROSS RIVER VIRUS							5	2	7
9994 SMALL VIRUS (LIKE) PARTICLE				1					1
9995 DENGUE							1		1
Total	269	17	100	138	66	153	236	237	1,216

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 5-1-87 to 18-1-87 Bulletin Number 87/2

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	Respir-atory	Enceph-alitis	Mening-itis	Para-lysis	CNS other unspec	GI	Hepa-tic	CVS	Urin-ary	Skin/ mucs memb
0101 ADENOVIRUS TYPE 1.....		7				1	1				
0102 ADENOVIRUS TYPE 2.....		5		1							
0103 ADENOVIRUS TYPE 3.....		5					1				
0105 ADENOVIRUS TYPE 5.....		4									
0106 ADENOVIRUS TYPE 6.....	1	2			1		1				
0107 ADENOVIRUS TYPE 7.....										1	
0109 ADENOVIRUS TYPE 9.....							1				
0111 ADENOVIRUS TYPE 11.....							1				
0113 ADENOVIRUS TYPE 13.....		1									
0126 ADENOVIRUS TYPE 26.....							1				
0203 INFLUENZA B VIRUS.....	1										
0301 PARAINFLUENZA VIRUS TYPE 1....		1									
0303 PARAINFLUENZA VIRUS TYPE 3....	1	20									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....		12									
0500 RHINOVIRUS (ALL TYPES).....		26		1							
0600 MYCOPLASMA PNEUMONIAE.....	3	17									
0700 ORNITHOSIS-PSITTACOSIS.....	1	4									
0816 COXSACKIEVIRUS A16.....											1
1005 ECHOVIRUS TYPE 5.....				1							
1006 ECHOVIRUS TYPE 6.....	1	1					1				
1011 ECHOVIRUS TYPE 11.....		5		26		1	3				
1022 ECHOVIRUS TYPE 22.....							1				
1031 ECHOVIRUS TYPE 31.....				1							
1102 POLIOVIRUS TYPE 2.....	1										
1200 MUMPS VIRUS.....				1							
1300 HERPES VIRUS GROUP-NOT TYPED..	1										
1301 HERPES SIMPLEX VIRUS NOT-TYPED											1
1302 EPSTEIN-BARR VIRUS (EB VIRUS).		4				1	1	2			
1303 VARICELLA-ZOSTER VIRUS.....	1					1					9
1306 HERPES SIMPLEX TYPE 1.....	2	3		1		1	1			1	87
1307 HERPES SIMPLEX TYPE 2.....	14										84
1401 COXIELLA BURNETI.....	6	2									
1502 PICORNA VIRUS-NOT TYPED.....											1
1521 MEASLES VIRUS.....		2									
1522 RUBELLA VIRUS.....	1	3						1			30
1532 HEPATITIS B ANTIGEN.....	31							68			
1535 HEPATITIS A ANTIBODY.....	5							18			
1541 CHLAMYDIA A - C.TRACHOMATIS...	22	1									
1556 CMV - CYTOMEGALOVIRUS.....	3	10				1	1	3			
1563 CORONAVIRUS.....							2				
1564 ROTAVIRUS.....					1	1	31				
1571 ENTEROVIRUS TYPE 71 (BRCR)....				2							1
9992 ROSS RIVER VIRUS.....			2								1
9994 SMALL VIRUS (LIKE) PARTICLE...							1				
9995 DENGUE.....											1
Total.....	95	135	2	34	2	7	48	92		2	216

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 5-1-87 to 18-1-87 Bulletin Number 87/2
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
0101 ADENOVIRUS TYPE 1.....								3	1	
0102 ADENOVIRUS TYPE 2.....										1
0103 ADENOVIRUS TYPE 3.....	1							1		
0105 ADENOVIRUS TYPE 5.....	1									
0107 ADENOVIRUS TYPE 7.....					1					1
0108 ADENOVIRUS TYPE 8.....	2									
0119 ADENOVIRUS TYPE 19.....	1									
0137 ADENOVIRUS TYPE 37.....	1									
0201 INFLUENZA A VIRUS.....								1	2	
0302 PARAINFLUENZA VIRUS TYPE 2....								1		
0303 PARAINFLUENZA VIRUS TYPE 3....								2		
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....									1	
0500 RHINOVIRUS (ALL TYPES).....								1		
0700 ORNITHOSIS-PSITTACOSIS.....							1	2		
1006 ECHOVIRUS TYPE 6.....	1								1	
1011 ECHOVIRUS TYPE 11.....					1		1	5	5	
1102 POLIOVIRUS TYPE 2.....						2				
1200 MUMPS VIRUS.....									1	
1300 HERPES VIRUS GROUP-NOT TYPED..		1								
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			4	3			1	6	3	
1303 VARICELLA-ZOSTER VIRUS.....									1	
1306 HERPES SIMPLEX TYPE 1.....	5	44							3	
1307 HERPES SIMPLEX TYPE 2.....		182								
1401 COXIELLA BURNETI.....								4		
1502 PICORNA VIRUS-NOT TYPED.....								1		
1521 MEASLES VIRUS.....									2	
1522 RUBELLA VIRUS.....			1		7	1	1	4	1	
1532 HEPATITIS B ANTIGEN.....									8	
1535 HEPATITIS A ANTIBODY.....									1	
1541 CHLAMYDIA A - C.TRACHOMATIS...	1	191			1				1	
1556 CMV - CYTOMEGALOVIRUS.....		3		1		8	3	5	7	
9992 ROSS RIVER VIRUS.....					4			2	1	
9995 DENGUE.....								1		
Total.....	13	421	5	4	14	11	7	39	39	2