ISSN 0725 - 3141

Communicable Diseases Intelligence

Bulletin number 88/6
Issue date: 28 March 1988

Contents:

VIRUSES, CHLAMYDIAS, COXIELLAS, RICKETTSIAS AND MYCOPLASMAS REPORTING SCHEME: A total of 1 317 reports were processed during this period.

Two cases of Q fever were reported, one from South Australia and one from Victoria. No occupational data was available for either of the patients - males aged 17 and 20 years.

Dengue fever (serotypes unknown) was diagnosed;

- in a 26 year old female visiting Western Australia who had recently spent six weeks in Thailand; and
- a 50 year old woman in the Northern Territory for whom no travel history was available.

Cytomegalovirus (CMV) was isolated from:

- bronchial washings of a 23 year old female with acute myelocytic leukaemia;
- saliva and mouth swabs of a 54 year old renal transplant patient with fever;
- leucocytes from a 28 year old male with fever and night sweats, who had recently returned from South East Asia;
- leucocytes from a 30 year old HIV positive male with CMV retinitis;
- leucocytes and urine from a 27 year old male with CMV retinitis, currently receiving DHGP treatment;

The Bulletin is compiled and distributed by the Public Health Section, Communicable Diseases Branch, Department of Community Services and Health.

- bronchial biopsy tissue from a 35 year old HIV positive male;
- saliva of two HIV infected males after 48 weeks of AZT therapy; and
- heart, kidney and liver post-mortem specimens from a 32 year old HIV positive male.

AIDS SURVEILLANCE - AUSTRALIA

To 15 March 1988, 795 cases of AIDS fulfilling the criteria of case definition have been reported to the National Health and Medical Research Unit in AIDS Epidemiology and Clinical Research. The distribution of those patients by State or Territory of notification (Table 1), by age group (Table 2), by risk category (Table 3) and by clinical presentation (Table 4) are shown below:-

TABLE 1: AIDS patients by State or Territory of Notification

STATE/ TERRITORY	CASES			DEATHS		
	Male	Female	Total	Male	Female	Total
NSW	517	20	537	279	16	295
VIC	141	3	144	57	2	59
QLD	52	4	56	34	3	37
WA	30	2	32	16	1	17
SA	16	1	17	4	1	5
NT	2	0	2	1	0	1
TAS	1	1	2	1	0	1
ACT	5	0	5	3	0	3
	764	31	795	395	23	418

TABLE 2: AIDS patients by age group

AGE (YEARS)	CASES			DEATHS		
	Male	Female	Total	Male	Female	Total
0 - 9	7	1	8	6	1	7
10 - 19	3	1	4	3	1	4
20 - 29	159	8	167	86	2	88
30 - 39	333	2	335	158	1	159
40 - 49	188	4	192	92	3	95
50 - 59	60	6	66	40	6	46
60 +	14	9	23	10	9	19
	764	31	795	395	23	418

COMMENT: Since 1 January 1988 all cases of infection with HIV have been classified according to the clinical classification produced by CDC in Atlanta. In the future, all reporting of clinical manifestations of HIV will be presented using that classification (see Table 4).

TABLE 3: AIDS patients by risk category

RISK GROUP	CASES	DEATHS
Homo-/Bi-sexual	696	350
IV drug user	4	2
Homo-/Bi-sexual IV drug user	24	13
Blood transfusion recipient	46	39
Person with haemophilia	8	5
Heterosexual transmission	8	2
Under investigation	3	2
None of the above	6	5
	795	418

TABLE 4: AIDS patients by clinical presentation

INITIAL DISEASE REPORTED	CASES	DEATHS
GROUP IV:		
B: Neurologic disease	15	9
C: Secondary infectious diseases	574	304
D: Secondary cancers	166	78
E: Other conditions	0	0
BC: Neurologic disease + infectious diseases	7	7
BD: Neurologic disease + cancers	1	0
CD: Infectious diseases + cancers	32	20
	795	418

AIDS DUE TO HIV-2 INFECTION
(based on MMWR, Vol. 37, No. 3, 29 January 1988)

The first case of AIDS caused by human immunodeficiency virus type 2 (HIV-2) in the United States was diagnosed in December 1987. The patient was a West African who had arrived in the United States in 1987. When the patient presented to a physician in the United States in December 1987 he reported:

. a 3 year history of weight loss;

. recent onset of neurological symptoms;

. no history of sexual intercourse, use of non-sterile needles, or donation of blood whilst in the United States.

Investigations showed:

. mass lesions of the brain revealed by CAT scan;

. Toxoplasma gondii in the biopsy of the lesions; and

. acid-fast bacilli in lymph node biopsy.

The diagnosis of cerebral toxoplasmosis without other underlying causes of immunodeficiency fits the Centers for Disease Control (CDC) surveillance definition for AIDS, thus laboratory evidence of infection with HIV was sought. Testing of the patient's serum revealed:

. a negative enzyme immunoassay (EIA) for antibody to HIV-1;

. an intermediate HIV-1 Western blot;

. a repeatedly reactive EIA for HIV-2 (Genetic Systems Corporation, Washington,[research test kit]);

. bands for antibodies to *gag* (p26), *pol* (p34), and *env* (gp140) proteins on Western blot for HIV-2; and

. HIV-2 DNA but not HIV-1 DNA in the patients lymphocytes by using the polymerase chain reaction technique with HIV-1-specific and HIV-2-specific DNA probes[1].

All family members and household contacts, both in the United States and abroad, are reported to be well.

MMWR Editorial Note:

This patient represents the only documented case of HIV-2 infection in the United States. HIV-2 is closely related to HIV-1 and was first reported to be associated with AIDS in 1986[2-8] in West Africa where the virus is believed to be endemic. There have also been well documented cases of HIV-2 infection among Europeans and West Africans residing in Europe[3,4,8].

The spectrum of disease and modes of transmission of HIV-2 are similiar to those of HIV-1[2-5]. Persons infected with HIV-2 present no risk to nonsexual household contacts[9].

The present case undoubtedly represents infection acquired in West Africa since illness began before the patient's arrival in the United States. The patient has had no known activities that would have exposed others in the United States to HIV-2.

Due to the reports of HIV-2 infection in West Africa and Europe, CDC and the Food and Drug Administration (FDA) initiated surveillance for HIV-2 in the United States in January 1987. To date, CDC, FDA, and collaborating investigators have screened 22,699 serum samples with an anti-HIV-2 EIA[10]. These specimens consisted of:

. 14,196 (63%) from individuals whose activities placed them at increased risk for HIV-1 infection and would, therefore, potentially be at risk for HIV-2 infection; and

. 8,503 from asymptomatic blood donors randomly selected from three areas of the United States, two of which have reported large numbers of AIDS patients;

Of these specimens:

. 35 (0.2%) were reactive by anti-HIV EIA using HIV-2 antigens but not by anti-HIV EIA using HIV-1 antigens. However, none of these EIAs could be confirmed when tested by HIV-2-specific Western blot.

. an additional 70 (0.3%) of the samples were reactive by Western blot with *gag, pol,* and *env* antigens of both HIV-1 and HIV-2 and

. all of the dually reactive specimens were from individuals whose activities placed them at increased risk for HIV-1 infection. None were from the randomly selected blood donors.

Sera from these dually reactive subjects were studied for the presence of type-specific neutralizing antibody to HIV-1 or HIV-2, antibody to synthetic peptides specific for HIV-1 or HIV-2 (Genetic Systems Corporation, Seattle, Washington [research test kit]), or HIV-1 and HIV-2 DNA by DNA amplification[1]. Sixty of the subjects were shown to be infected with HIV-1 but not HIV-2. Ten are still under investigation.

It is reassuring that HIV-2-specific tests on sera from 22,699 persons, including 8,503 randomly selected U.S. blood donors, failed to reveal HIV-2 infection. However, the occasional presence of this virus in the United States, as in Europe, should be anticipated. The anti-HIV-1 EIA tests currently used for screening all U.S. blood donors are estimated to detect 42% to 92% of HIV-2 infections[4,11]. Surveillance for HIV-2 in the United States is being continued to monitor the frequency of infection. Because the modes of transmission of HIV-1 and HIV-2 are similar, preventive measures for these related viruses are the same[12].

CDI Editorial Comment:

HIV-2 specific tests are not used for routine screening in Australia. However, since May 1987 the National Reference Laboratory (NRL) for HIV at Fairfield Hospital has tested 100 sera for HIV-2 using the Genetic Systems Incorporated EIA test. The sera tested were from either West African patients (with symptoms of HIV infection) or patients with symptoms of HIV infection, who have been found to be negative or given equivocal results in HIV-1 tests. All sera tested so far have been negative for HIV-2. Testing for HIV-2 is continuing on sera from the above groups[13].

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INFLUENZA UPDATE

Influenza A (H1N1) predominated in Asia, North America and Europe from October 1986 to the end of February 1987[11]. It was also the most common virus in some of the countries reporting influenza during the period July to September 1987. Influenza B was increasingly reported in parts of Europe in March and April 1987 and it was also the most common influenza type in Oceania, during July and August 1987[11]. Influenza A (H3N2) virus in general, contributed very little to the influenza activity reported in 1986-87, it was however isolated sporadically or in localised outbreaks, especially during the second half of the season. Influenza A (H3N2) has however become the predominant influenza isolate since October 1987 in North America[12].

Although influenza A (H1N1) and influenza B viruses were widespread and caused epidemics, they were not on the whole associated with much mortality. This was particularly the case with influenza A (H1N1) viruses which, as in previous years, were mainly diagnosed in patients under 40 years of age.

Influenza activity in countries of the region was as follows:

ASIA

Twelve Asian countries reported influenza activity in the 1986-1987 season, but none indicated severe epidemics. Virus activity was first reported in the western part of the Democratic People's Republic of Korea (Taiwan) in October 1986, peaking in early November. Most isolates were influenza A (H1N1) but a few were influenza A (H3N2). During the same month cases of influenza A (H1N1) appeared in Japan and spread throughout the country without causing large epidemics.

China, Hong Kong, the Republic of Korea, and Singapore reported very little influenza activity for the first 6 months of the 1986-1987 season. Some cases of influenza A (H1N1) were confirmed in China and Hong Kong up to the end of March. A few cases of influenza A (H3N2) were also detected in China and of influenza B in Hong Kong, the Republic of Korea and Singapore. In the latter part of the season increased influenza activity was noted in the southern part of China and Beijing. Most cases confirmed by laboratory studies were influenza A (H3N2); influenza A of the H1N1 subtype and influenza B viruses were also isolated. In Singapore, influenza B predominated from January to May, and influenza A (mainly of the H1N1 subtype) thereafter; only 3 cases of influenza A (H3N2) were diagnosed during the year.

Japan experienced outbreaks associated with influenza isolates in the southern part of the country in April and early May 1987. Malaysia reported sporadic cases of mild, influenza-like illness from May to October 1987, most cases were in adults. Influenza B was isolated in May and influenza A (H1N1) virus in June and September 1987. An increase in the incidence of acute respiratory diseases with isolates of influenza B was reported in Pune, India during July and August 1987. Isolates of influenza A (H3N2) and A (H1N1) were reported in January and June in India. Influenza B was isolated in April and May 1987 in Indonesia.

SOUTH AMERICA

Apart from a local outbreak associated with influenza B in Panama in October, almost all influenza activity reported in the Americas during the first 6 months of the season was caused by influenza A (H1N1) viruses. A few localised outbreaks in Brazil during the second half of the season were associated with influenza A (H3N2) and influenza B virus isolates[1].

OCEANIA

Influenza B predominated in both Australia and New Zealand although influenza A (H3N2) became rather common towards the end of the season.

Overall, the influenza activity increased during the 1986-1987 influenza year but it was still much lower than that detected during an epidemic year. A total of 194 cases of influenza A virus have been reported to CDI (from January 1987 to December 1987) which included 16 isolates of type A (H1N1). A total of 372 cases of influenza B were reported for the same period[2].

Influenza B predominated also during the season in New Zealand. The virus was isolated from all parts of the country during an outbreak which reached a peak in July and continued into September. In September there was a brief upsurge of cases of acute respiratory infections, associated with isolates of influenza A (H3N2).

Antigenic Composition of the Influenza strains

The viruses of the H1N1 subtype were all antigenically similiar to A/Singapore/6/86 and the H3N2 viruses were in the main similiar to A/Leningrad/360/86. Analysis of about 50 recently isolated A (H3N2) viruses from Asia, Oceania and North America indicates a spectrum of antigenic specificity with many isolates having reaction patterns intermediate between A/Leningrad/360/86 and A/Sichuan/2/87 (isolated in China in April 1987). Another reference variant A/Victoria/7/87 was isolated in Australia in April 1987. The above variants are associated with the reappearance of influenza A(H3N2) viruses after a period of quiescence during the influenza season of 1986/87. The influenza B viruses isolated were antigenically distinguishable from B/Ann Arbor/1/86 prototype.

Recommendations for 1987-88 Influenza Vaccine

The composition of the influenza vaccine for the 1988 winter season in Australia decided by the Influenza Vaccine Committee is:

- A/Singapore/6/86 (H1N1)-like strain, 15 micrograms haemagglutinin/.5ml dose;
- A/Leningrad/360/86-like strain, 15 micrograms haemagglutinin/.5ml dose; and
- B/Victoria/2/87-like strain, 15 micrograms haemagglutinin/.5ml dose.

Recommendations for the use of the inactivated influenza vaccine.

The Centers for Disease Control (CDC) recommend that inactivated influenza virus vaccine be given to high risk persons over 6 months of age and to their medical-care providers, or household contacts[3].

CDC classifies high risk groups according to observations of morbidity and mortality. They recommend that vaccination efforts are most necessary for the following two groups which are considered at high risk:[3]

- Adults and children with chronic disorders of the cardiovascular or pulmonary systems requiring regular medical follow-up or hospitalisation during the preceding year; and

- residents of nursing homes and other chronic-care facilities housing patients of any age with chronic medical conditions.

Persons at moderate risk of serious illness compared with the general population include:

- otherwise healthy individuals;

- adults and children who have required regular medical follow-up or hospitalisation during the preceding year because of chronic metabolic diseases (including diabetes mellitus), renal dysfunction, anaemia or immunosuppression; and

- children and teenagers (6 months to 18 years) of age who are receiving long-term aspirin therapy, and therefore may be at risk of developing Reye's syndrome following influenza infection[3].

The potential for transmitting influenza to high risk persons should be reduced by vaccinating:

- physicians, nurses and other personnel having extensive contact with high risk patients (eg primary-care and certain specialty physicians and staff of chronic-care facilities and intensive-care units, particularly neonatal intensive care units.

- providers of care to high risk persons in the home setting (eg visiting nurses, volunteer workers) as well as all household members[3].

Persons who should not be vaccinated

Influenza vaccine should not be given to persons who have severe allergies to eggs. This includes those who develop hives, swelling of the lips or tongue, or acute respiratory distress or collapse after eating eggs. It also includes persons who have developed evidence of occupational asthma or other allergic responses from occupational exposure to egg protein[3].

CDI Editorial Note

Laboratory studies, as well as preliminary observations during outbreaks of influenza A(H3N2) among high risk residents of

nursing homes in North America, suggest that the A/Leningrad/360/86 component of the current vaccine may not provide the optimal protection against presently circulating strains. These findings emphasize the need for health care providers to be aware of the recommendations for the use of the antiviral drug amantadine, both in the treatment and prevention of infection in susceptible persons.

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ECHOVIRUS 11 ENCEPHALITIS - A CASE REPORT

(Contributed by M.W. Giles and J.H. Andrew, Department of Microbiology, St Vincent's Hospital, Melbourne, Victoria)

History

In April 1987, a 39 year old woman with a 15 year history of Diabetes mellitus presented with a one day history of dizziness, lethargy and headache.

Initial examination:

- The patient was drowsy and unco-operative but well-orientated with a temperature of 38.9°C.
- No neck stiffness or focal neurological signs were elicited and optic fundi were considered normal;

Investigations

- Biochemical and haematological tests, including blood cultures, were unremarkable except for a blood glucose levels 22.6 mmol/l (slightly elevated)
- Lumbar puncture revealed:
  - clear and colourless CSF;
  - a slightly elevated glucose level;
  - an opening pressure of 20 cm water; and
  - normal CSF by culture and microscopy.
- A CAT scan revealed mild communicating hydrocephalus with no obvious cause.

Management

Intravenous ampicillin 1g was administered 6 hourly.

The diabetes was managed by diet and insulin administration. Seizures were controlled by anti-epileptic medication.

Progress

By day 4 there was:

- right upper motor neuron facial weakness and flaccid right hemiparesis;
- marked neck stiffness; and
- generalised tonic/clonic seizures.

On day 7:

. CSF sample contained $2x10^6$ WBCs/l and $800x10^6$ RBCs/l (this could be the result of a bloody tap) - no other abnormalities were observed;
. CSF culture was again negative; and
. a selective left carotid angiogram demonstrated no significant abnormality.

On day 12:

. a brain biopsy was taken - immunofluorescence and subsequent culture for herpes simplex virus were negative; and
. electroencephalogram results were consistent with diffuse encephalopathic process.

Outcome

By day 14 the patient was afebrile and becoming more alert with the right hemiparesis beginning to resolve. Ampicillin was discontinued on the 17th day.

The patient was discharged after 5 weeks in hospital. Some minor weakness and parasthesia of the right arm and leg remained.

Diagnosis

Diagnosis was established by isolation, after 45 days, of echovirus 11 from the brain biopsy specimen. A significant rise in antibody titre to this virus was also observed in paired sera (day 13 - less than 4; day 25 - greater than 1024). Virus isolation and serology were carried out at Fairfield Infectious Diseases Hospital.

Comment

Echovirus 11 has been described as a cause of aseptic meningitis, encephalitis and paralysis as well as less serious conditions such as acute respiratory infection, exanthemata and diarrhoea. It is believed that a large proportion of such enteroviral infections are either asymptomatic or produce an undifferentiated febrile illness. Neonates in particular, appear highly susceptible to infections with echovirus and epidemics of disease caused by echoviruses, including serotype 11 have been reported.

The CSF findings in echovirus encephalitis are similar to those seen in aseptic meningitis, where white cell counts in the range of 10-500 x 10^6/l are typically seen, with lymphocytes the predominant cell type. However, normal CSF findings are not uncommon, even in cases of severe encephalitis. The evidence linking specific viral serotypes to encephalitis is not always compelling as definitive diagnosis is often inferred by the isolation of the virus from non-neurological sites or from the detection of a rise in titre of specific antibody. In this case the pathogen was isolated from the brain biopsy specimen and diagnosis was confirmed by a significant rise in antibody titre against echovirus 11.

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A CASE OF RABIES

(Contributed by Dr P. Debuse, Royal Childrens Hospital, Dr J.L. Faoagali and Dr H. Samaratanga, Department of Pathology, Royal Brisbane Hospital, Herston Rd, Brisbane, Queensland).

On 2 July 1987, a 10 year old male was transferred to the Royal Childrens Hospital, Brisbane from a peripheral hospital where he had been admitted 12 hours previously following a 9 day history of anorexia, fever, vomiting and changes in his mental state. In the 4 days prior to his admission he developed gradually increasing weakness of his legs and arms and strabismus (squint).

The provisional diagnosis on admission was Guillan-Barre syndrome (atypical) because of the unusual neurological findings and the absence of abnormal CSF findings (no white cells or elevated protein). Within a few hours of admission he was transferred to the Intensive Care Unit where his paralysis increased and his mental state deteriorated so that on the 12th day of his illness (4/7/87) he was intubated and placed on a ventilator. A lumbar puncture taken at this time contained 50 white cells with 95% mononuclear cells.

Various treatments were instituted including acyclovir (10mg/kg/day) intravenously, and triple drug antituberculosis therapy as well as maintenance therapy.

Specimens of secretions from all sites were collected for bacteriological, viral and mycological examination. Serial serum samples were analysed for antibodies to herpes simplex virus, measles, CMV and EBV. Serum and CSF were stored at -20°C.

The vaccination and travel history taken on admission revealed a visit to the Indian sub-continent with extensive travel from February to early September 1986 followed by a 3 week visit to Singapore and Thailand. There was extensive travel in rural areas and contact with animals throughout the journey, though there was no history of an animal bite or exposure to a rabid or sick animal. None of the family had received rabies vaccination prior to the journey.

The patient died on the 23rd day of his illness and a post-mortem on the day of death revealed a heavy and congested brain but there was no evidence of coning. Inoculation of routine tissue cultures with fresh brain were negative for viruses. Sections from all areas of the brain including cerebrum, basal ganglia, cerebellum, nerve roots and spinal cord, examined by light microscopy, showed non-specific perivascular cuffing with lymphocytes and plasma cells, neuronal degeneration and neuronophagia, a lymphocytic infiltration into the meninges and scattered neuronal intracytoplasmic eosinophilic inclusions some of which were multiple (Negri bodies). These histological findings are consistent with those found in brain tissue from human rabies cases.

Subsequent consultation with the Pasteur Institute, CSIRO Australian National Animal Health Laboratory (ANAHL) and the Centers for Disease Control (CDC) Rabies Reference laboratory confirmed:

- rabies specific antibodies in high titre in antemortem serum;
- negative rabies antibodies in a CSF sample from 4/7/87;
- rabies specific immunoperoxidase and immunofluorescent positive intracytoplasmic neuronal inclusions in fixed tissue sections;
- rabies virus present in the inclusions examined by electron microscopy.

Follow up of contacts was aimed at determining those people who had been directly exposed to inoculation incidents with saliva, CSF or brain tissue. Four relatives and nine hospital staff were identified as receiving significant mucous membrane or conjunctival exposure to potential virus containing material and were subsequently vaccinated with human diploid vaccine following the CDC 1984 regime for post-exposure rabies prophylaxis.

This case of imported rabies is the first recognised in Australia since a reputed case in the mid-1800's.

CDI Editorial Comment

This case of rabies highlights a number of important issues :

- Australia and most of Pacific Oceania are rabies free yet rabies is highly endemic in most countries of South East Asia. The risks of rabies is highest in countries where dog rabies remains highly endemic particularly India, Nepal, the Phllipines, Thailand and Vietnam.
- Australians travelling overseas, particularly to South East Asia, should be warned of the risks of acquiring rabies[1]. Pre-exposure vaccination does not eliminate the need for additional therapy after a rabies exposure (it only simplifies post-exposure treatment) and is not routinely recommended;
- any animal bite or scratch that occurs in a rabies endemic country, should receive prompt local treatment. When wounds are thoroughly cleansed with copious amounts of soap and water, the risk of rabies is significantly reduced. Persons who are exposed should report to the nearest health authority as soon as practicable, to be assessed for post-exposure prophylaxis;
- rabies is almost always transmitted by bites of rabid animals which introduce the virus into wounds. Very rarely has rabies been transmitted by non-bite exposure which introduces the virus into open cuts or mucous membranes[2];

- there are no well documented instances of human - to - human transmission of rabies other than the special circumstance of corneal transplants. Contact isolation is an effective means of limiting exposure[3]; and

- rabies should be considered and excluded from cases of undiagnosed encephalitis where there is a travel history which has included animal exposure in rabies endemic countries within the past year.

Diagnosis of Rabies

Diagnostic facilities for rabies in Australia are limited, however rapid diagnosis is available from the CSIRO ANAHL in Geelong. The CDC Rabies Reference Laboratory can give confirmation 6 hours after receipt of fresh brain samples.

In a suspected case of rabies where symptoms and other clinical findings are consistent with and include an exposure history to possible rabies, specimens are taken for detection of virus. These specimens include:-

- conjunctival scraping;

- hair follicle biopsy from nape of neck; and

- CSF.

An immunofluorescence test using fluorescent-tagged antibody to stain rabies-infected cells, obtained by impression smears of corneal or skin biopsy specimens, is used. Rabies antibody levels are tested for in CSF. Complete diagnosis usually requires following the serum antibody levels and the clinical course. In most cases the antibody titre rises during the second or third week of illness.

In cases where persons had been bitten by suspected rabid animals but remain asymptomatic, rabies serology is not warranted. A complete travel and exposure history should be taken and assessed for the necessity of post-exposure prophylaxis.

In post-mortem situations where rabies is suspected, brain tissue from four sites (cerebral cortex, cerebellum, brain stem and hippocampus) are collected for:

- direct immunofluorescence test on smears of tissue (same day results);

- histopathology and immunostaining;

- homogenisation and inoculation intracerebrally into suckling mice which are held for 28 days[4].

Histologic examination of brain tissue from human rabies cases typically shows perivascular inflammation of the gray matter, various amounts of neuronal degeneration and in many cases characteristic cytoplasmic inclusion bodies (Negri bodies). Specific staining with rabies fluorescent antibodies may be needed to demonstrate the inclusions.

Post-exposure Prophylaxis

If rabies has been proven in the patient, contacts should be asked to report possible exposures and should be interviewed to determine if, in fact, an exposure occurred. The period of possible risk of transmission is not well defined but should be assumed to be highest during the first three weeks of illness and to persist to the fifth week.[3].

Persons exposed to a rabies patient's infectious fluids or tissues should immediately wash the area with soap and water. If rabies is diagnosed in a post-mortem situation, all contacts should be assessed for their risk of rabies. Exposures for which prophylaxis might be recommended include:

- bites with penetration of skin by teeth;
- exposure to patient's saliva or other potentially infectious material in direct contact with mucous membrane or broken skin (cut, scratch or abrasion)
- scalpel nicks or needle sticks if the treatment was in contact with CSF, nervous tissue, ocular tissue or internal organs[3].

Exposures for which prophylaxis is considered unnecessary include:

- exposure to potentially infectious material in direct contact with unbroken skin;
- any contact with blood, stool or unspun urine[3].

When post-exposure prophylaxis is recommended it should be administered according to National Health and Medical Research Council guidelines[5]. These guidelines must be strictly adhered to as vaccine failures have been reported when vaccine was administered into sites other than that recommended.

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AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

TOTAL VIRAL ISOLATIONS BASED ON DATE OF COLLECTION
PERIOD - FORTNIGHTLY
VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

Period 7-3-88 to 20-3-88.

1. CODE 019 - FAIRFIELD(VIC)
2. CODE 065 - STATE LAB(WA)
3. CODE 110 - IMVS(SA)
4. CODE 111 - RCH(VIC)
5. CODE 112 - ICPMR(NSW) WVH(ACT)
6. CODE 113 - PHH POW(NSW)
7. CODE 114 - RAHC(NSW)
8. CODE 115 - STATE LAB(QLD)

		019	065	110	111	112	113	114	115	TOTAL
0100	ADENOVIRUS NOT TYPED	4	7	3	3	3	6	1	5	32
0101	ADENOVIRUS TYPE 1	0	1	0	0	1	0	0	0	2
0102	ADENOVIRUS TYPE 2	0	0	4	0	0	0	1	0	5
0103	ADENOVIRUS TYPE 3	1	0	1	0	1	0	0	0	3
0104	ADENOVIRUS TYPE 4	4	0	0	0	0	0	0	0	4
0105	ADENOVIRUS TYPE 5	0	0	1	0	0	0	0	0	1
0106	ADENOVIRUS TYPE 6	0	0	3	0	0	0	0	0	3
0108	ADENOVIRUS TYPE 8	1	0	0	0	0	0	0	0	1
0110	ADENOVIRUS TYPE 10	0	0	0	0	1	0	0	0	1
0124	ADENOVIRUS TYPE 24	1	0	0	0	0	0	0	0	1
0126	ADENOVIRUS TYPE 26	1	0	0	0	0	0	0	0	1
0137	ADENOVIRUS TYPE 37	0	1	0	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	1	0	0	2	0	0	0	0	3
0201	INFLUENZA A VIRUS	0	0	1	0	0	0	0	0	1
0203	INFLUENZA B VIRUS	0	0	2	0	0	0	0	0	2
0301	PARAINFLUENZA VIRUS TYPE 1	0	0	0	3	0	0	0	0	3
0302	PARAINFLUENZA VIRUS TYPE 2	1	0	1	1	0	0	0	0	3
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	2	1	1	0	0	0	4
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	2	1	0	0	0	0	0	3
0500	RHINOVIRUS (ALL TYPES)	2	2	0	8	1	0	3	1	17
0600	MYCOPLASMA PNEUMONIAE	6	1	9	2	4	0	1	0	23
0700	ORNITHOSIS-PSITTACOSIS	3	0	2	0	2	0	0	0	7
0809	COXSACKIEVIRUS A9	3	0	0	0	0	0	0	0	3
0816	COXSACKIEVIRUS A16	3	0	0	0	0	0	0	0	3
0821	COXSACKIEVIRUS A21	1	0	0	0	0	0	0	0	1
0902	COXSACKIEVIRUS B2	0	0	1	0	0	0	0	0	1
0905	COXSACKIEVIRUS B5	3	0	0	0	1	0	0	0	4
1000	ECHOVIRUS NOT TYPED	0	1	0	0	0	0	0	0	1
1018	ECHOVIRUS TYPE 18	0	1	0	0	0	0	0	0	1
1022	ECHOVIRUS TYPE 22	0	1	0	0	0	0	0	0	1
1101	POLIOVIRUS TYPE 1	0	1	2	0	0	0	0	0	3
1102	POLIOVIRUS TYPE 2	0	1	1	0	0	0	0	0	2
1200	MUMPS VIRUS	1	0	0	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	3	1	0	35	0	0	0	39
1301	HERPES SIMPLEX VIRUS - NOT TYP	1	3	0	0	0	0	0	0	4
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	1	3	4	0	1	0	1	0	10
1303	VARICELLA-ZOSTER VIRUS	2	3	0	0	3	0	0	4	12
1306	HERPES SIMPLEX TYPE 1	44	21	11	0	83	23	0	27	209
1307	HERPES SIMPLEX TYPE 2	68	43	21	0	176	15	0	74	397
1399	HERPES VIRUS TYPING PENDING	0	0	0	2	0	0	0	0	2
1401	COXIELLA BURNETI	1	0	1	0	0	0	0	0	2
1502	PICORNIA VIRUS - NOT TYPED = E	0	1	0	0	0	3	0	14	18
1521	MEASLES VIRUS	1	0	0	0	0	0	0	0	1
1522	RUBELLA VIRUS	2	0	0	0	0	0	0	0	2
1532	HEPATITIS B ANTIGEN	24	25	12	0	144	4	0	17	226
1535	HEPATITIS A ANTIBODY	16	4	2	0	9	3	0	0	34
1541	CHLAMYDIA A - C. TRACHOMATIS	2	40	28	0	16	0	0	12	98
1556	CMV - CYTOMEGALOVIRUS	35	6	4	4	4	1	4	8	66
1564	ROTAVIRUS	1	0	0	15	5	2	3	0	26
1599	ENTEROVIRUS TYPING PENDING	0	0	0	2	0	6	1	0	9
9992	ROSS RIVER VIRUS	7	9	0	1	0	1	0	0	18
9995	DENGUE	0	2	0	0	0	0	0	0	2
TOTAL		241	182	118	44	491	64	15	162	1317

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1.

Period 7-3-88 to 20-3-88.

1. CODE 00, 99 ..... - NO ILL OR DATA
2. CODE 01, 02, 11, 12 - RESPIRATORY
3. CODE E3 ..... - ENCEPHALITIS
4. CODE M3 ..... - MENINGITIS
5. CODE 04 ..... - PARALYSIS
6. CODE 05, 13 ..... - CNS OTHER UNSPEC
7. CODE 07, 49 - GASTRO INTESTINAL
8. CODE 17, 47 - HEPATIC
9. CODE 19 ... - CVS
10. CODE 89 ... - URINARY TRA CT
11. CODE 06 ... - SKIN MUCOUS

		1	2	4	5	6	7	8	9	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	0	7	0	0	0	18	0	0	0	0	25
0101	ADENOVIRUS TYPE 1	0	1	0	0	0	1	0	0	0	0	2
0102	ADENOVIRUS TYPE 2	1	3	0	1	0	0	0	0	0	0	5
0103	ADENOVIRUS TYPE 3	0	1	0	0	0	1	0	0	0	0	2
0105	ADENOVIRUS TYPE 5	0	1	0	0	0	0	0	0	0	0	1
0106	ADENOVIRUS TYPE 6	0	3	0	0	0	0	0	0	0	0	3
0126	ADENOVIRUS TYPE 26	0	0	0	0	0	1	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	2	0	0	0	0	0	0	0	0	2
0201	INFLUENZA A VIRUS	0	1	0	0	0	0	0	0	0	0	1
0203	INFLUENZA B VIRUS	0	1	0	0	0	0	0	0	0	0	1
0301	PARAINFLUENZA VIRUS TYPE 1	0	3	0	0	0	0	0	0	0	0	3
0302	PARAINFLUENZA VIRUS TYPE 2	0	3	0	0	0	0	0	0	0	0	3
0303	PARAINFLUENZA VIRUS TYPE 3	0	3	0	0	0	0	0	1	0	0	4
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	3	0	0	0	0	0	0	0	0	3
0500	RHINOVIRUS (ALL TYPES)	0	14	0	0	1	0	0	0	0	0	15
0600	MYCOPLASMA PNEUMONIAE	1	20	0	0	0	0	0	0	0	1	22
0700	ORNITHOSIS-PSITTACOSIS	1	4	0	0	0	0	0	0	0	0	5
0809	COXSACKIEVIRUS A9	0	0	3	0	0	0	0	0	0	0	3
0816	COXSACKIEVIRUS A16	0	0	0	0	0	0	0	0	0	3	3
0902	COXSACKIEVIRUS B2	0	0	0	0	0	0	0	0	0	1	1
0905	COXSACKIEVIRUS B5	0	0	2	0	0	0	0	0	0	1	3
1000	ECHOVIRUS NOT TYPED	0	0	0	0	0	1	0	0	0	0	1
1018	ECHOVIRUS TYPE 18	1	0	0	0	0	0	0	0	0	0	1
1022	ECHOVIRUS TYPE 22	0	0	0	0	0	1	0	0	0	0	1
1101	POLIOVIRUS TYPE 1	0	0	0	0	0	3	0	0	0	0	3
1102	POLIOVIRUS TYPE 2	0	1	0	0	0	1	0	0	0	0	2
1200	MUMPS VIRUS	1	0	0	0	0	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	4	2	1	0	0	0	0	0	0	20	27
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	0	0	0	0	0	0	0	0	2	2
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	3	0	0	0	0	0	0	0	0	0	3
1303	VARICELLA-ZOSTER VIRUS	2	0	0	0	0	0	0	0	0	10	12
1306	HERPES SIMPLEX TYPE 1	21	7	0	0	0	0	0	0	0	90	118
1307	HERPES SIMPLEX TYPE 2	41	1	0	1	0	0	0	0	0	82	125
1399	HERPES VIRUS TYPING PENDING	0	1	0	0	0	0	0	0	0	1	2
1401	COXIELLA BURNETI	1	0	0	0	0	0	0	0	0	0	1
1502	PICORNIA VIRUS - NOT TYPED = E	0	0	0	0	3	9	0	0	0	6	18
1521	MEASLES VIRUS	0	0	0	0	0	0	0	0	0	1	1
1522	RUBELLA VIRUS	1	0	0	0	0	0	0	0	0	1	2
1532	HEPATITIS B ANTIGEN	86	0	0	0	0	0	117	0	1	0	204
1535	HEPATITIS A ANTIBODY	0	0	0	0	0	0	32	0	0	0	32
1541	CHLAMYDIA A - C. TRACHOMATIS	7	0	0	0	0	0	0	0	0	0	7
1556	CMV - CYTOMEGALOVIRUS	5	17	1	0	0	0	2	0	6	0	31
1564	ROTAVIRUS	0	0	0	0	0	25	0	0	0	0	25
1599	ENTEROVIRUS TYPING PENDING	0	1	1	0	0	5	0	0	0	0	7
9992	ROSS RIVER VIRUS	3	0	0	0	0	0	0	0	0	4	7
TOTAL		179	100	8	2	4	66	151	1	7	223	741

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2.

Period 7-3-88 to 20-3-88

12. CODE 10 - EYE
13. CODE 59 - GENITAL
14. CODE 39 - ENDOCRINE/SALIVARY GL.
15. CODE 38 - RETICULO-ENDOTHELIAL
16. CODE 29 - MUSCLE/JOINT
17. CODE 69 - CONGENTIAL
18. CODE P8 - PUO
19. CODE G8 - FEVER/MALAISE
20. CODE 09 - OTHER
21. CODE A1 - SIDS

		12	13	14	15	16	17	18	19	20	21	TOTAL
0100	ADENOVIRUS NOT TYPED	2	0	1	0	0	0	1	1	1	1	7
0103	ADENOVIRUS TYPE 3	1	0	0	0	0	0	0	0	0	0	1
0104	ADENOVIRUS TYPE 4	4	0	0	0	0	0	0	0	0	0	4
0108	ADENOVIRUS TYPE 8	1	0	0	0	0	0	0	0	0	0	1
0110	ADENOVIRUS TYPE 10	1	0	0	0	0	0	0	0	0	0	1
0124	ADENOVIRUS TYPE 24	0	0	0	0	0	0	0	1	0	0	1
0137	ADENOVIRUS TYPE 37	1	0	0	0	0	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	0	0	0	0	0	0	1	0	0	1
0203	INFLUENZA B VIRUS	0	0	0	0	1	0	0	0	0	0	1
0500	RHINOVIRUS (ALL TYPES)	0	0	0	0	0	0	0	1	1	0	2
0600	MYCOPLASMA PNEUMONIAE	0	0	0	0	0	0	0	0	1	0	1
0700	ORNITHOSIS-PSITTACOSIS	0	0	0	0	0	0	0	0	2	0	2
0821	COXSACKIEVIRUS A21	0	0	0	0	0	0	0	0	1	0	1
0905	COXSACKIEVIRUS B5	0	0	0	0	0	0	0	1	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	7	0	0	0	0	0	0	5	0	12
1301	HERPES SIMPLEX VIRUS - NOT TYP	1	1	0	0	0	0	0	0	0	0	2
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	0	2	0	0	1	1	3	0	7
1306	HERPES SIMPLEX TYPE 1	4	76	0	0	0	0	0	0	11	0	91
1307	HERPES SIMPLEX TYPE 2	0	270	0	0	0	0	0	1	1	0	272
1401	COXIELLA BURNETI	0	0	0	0	0	0	0	1	0	0	1
1532	HEPATITIS B ANTIGEN	0	1	0	0	1	0	0	0	20	0	22
1535	HEPATITIS A ANTIBODY	0	0	0	0	0	0	0	0	2	0	2
1541	CHLAMYDIA A - C. TRACHOMATIS	2	89	0	0	0	0	0	0	0	0	91
1556	CMV - CYTOMEGALOVIRUS	4	1	1	1	0	2	2	4	20	0	35
1564	ROTAVIRUS	0	0	0	0	0	0	0	0	0	1	1
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	0	0	1	0	1	2
9992	ROSS RIVER VIRUS	0	0	1	0	8	0	1	0	1	0	11
9995	DENGUE	0	0	0	1	0	0	0	1	0	0	2
TOTAL		21	445	3	4	10	2	5	14	69	3	576

CDI 88/6

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

Period 10 - 6 September 1987 to 3 October 1987.

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	4						5	42
Ankylostomiasis				3			NN		3	40
Anthrax									-	1
Arbovirus infection	1	1	18		3				23	1 118
Brucellosis									-	11
Campylobacter infections	85		NN	105	22	NN	4		216	* 2 498
Chancroid				NN					-	5
Cholera									-	2
Congenital rubella syndrome			NN			NN		NN	-	-
Diphtheria									-	25
Donovanosis				NN	1		5		6	84
Giardiasis	30		NN	72	23	NN	NN	NN	125	1 176
Genital herpes	52	1	29	32	NN	NN	2	3	119	1 508
Gonococcal ophthalmia neonatorum		NN			NN	NN		NN	-	257
Gonorrhoea	65	2	43	26	65	3	44		248	* 3 880
Hepatitis A (infectious)	15	1	13	2	11	1	10		53	* 551
Hepatitis B (serum)	36	13	56	1	42	4	5	3	160	* 1 392
Hepatitis — unspecified	3			1	NN	NN			4	127
Hydatid disease									-	14
Lassa fever			NN			NN		NN	-	-
Legionnaires disease	2		NN			NN		NN	2	82
Leprosy							5		5	22
Leptospirosis			6						6	114
Lymphogranuloma venereum				NN	NN	NN		NN	-	-
Marburg disease			NN			NN		NN	-	-
Malaria	9	7	32	1	3		2	2	56	508
									-	-
Meningococcal infections	2	2			4	NN			8	65

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	157		1	35	NN	NN	NN	NN	193	4 097
Ornithosis									-	8
Pertussis (whooping cough)	2	1	NN	6	5	NN	1	NN	15	244
Plague									-	-
Poliomyelitis									-	-
Q. fever	7		6	1	1				15	* 305
Rabies				NN		NN		NN	-	-
Salmonella infections	45	9	26	16	26	4	11		137	2 148
Shigella infections	5	2	4	3	9		14		37	485
Smallpox									-	-
Syphilis	24	1	15	6	15		81	1	143	1 675
Tetanus									-	5
Trachoma		NN		5	3	NN	NN		8	192
Tuberculosis (all forms)	24	26	26	7	9		6	2	100	* 822
Typhoid fever	3	1		1					5	42
Typhus (all forms)									-	6
Vibrio parahaemolyticus infections			NN			NN		NN	-	3
Yellow fever									-	-
Yersinia infections	9		NN			NN		NN	9	90

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.)

* Adjustments to the Cumulative Total since last report:

Campylobacter inf.	+32	South Australia
Gonorrhoea	+ 6	South Australia
Hepatitis A	+ 7	South Australia
Hepatitis B	+ 5	South Australia
Q fever	+ 1	South Australia
Tuberculosis	+ 2	South Australia