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Communicable Diseases Intelligence

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Contents:

CHOLERA SURVEILLANCE - QUEENSLAND

Vibrio cholerae O- group 1, biotype El Tor, serotype Ogawa, has been isolated from faecal specimens of a 20 month old boy from Mt. Morgan, approximately 40 Km south of Rockhampton. The child was admitted to hospital on 29 January 1983, with severe vomiting and diarrhoea. The patient's three year old sister also had episodes of diarrhoea, but all cultures from her and other family members were negative for the pathogen. All water supplies tested to date have been negative, and investigations for the vehicle of infection are continuing.

Contaminated lettuce has been implicated as the source of infection for the earlier indigenous cholera case (V. cholerae O-1, biotype El Tor, serotype Inaba) in a 32 year old male from Rockhampton (CDI 83/2). The locally grown lettuce had been irrigated with creek water.

VIRUS REPORTING SCHEME - A total of 1110 reports were received this period. The low summer rainfall and the entrenched drought conditions prevalent in Australia at present have severely aborted transmission of all arbovirus infections. Six reports of Ross River virus infection have been received to date this year, compared with 77 dengue reports and 34 Ross River virus reports received in the same periods of 1982. No seroconversions have yet been detected among the sentinel chicken flocks established in the Murray Region, Victoria (see CDI 82/24).

- The Royal Childrens Hospital, Melbourne isolated measles virus from nasal aspirate and lung biopsy of a three year old girl with pneumonia. Involvement of the respiratory tract is the most common complication of measles and may either be due to direct viral invasion or to bacterial superinfection.

- Seroconversion against Chlamydia trachomatis group antigen was reported by Fairfield Hospital, Melbourne, in a 28 year old male with urethritis. Penicillinase-producing N. gonorrhoeae was also cultured from the patient who acquired his infection in the Philippines.

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CREUTZFELDT-JAKOB DISEASE

Creutzfeldt-Jakob disease (CJD) is a rapidly progressive, fatal disease of the central nervous system. It is one of the spongiform encephalopathies caused by a group of unconventional viruses that cause kuru in man, scrapie in sheep and goats, and transmissible mink encephalopathy[1]. Subacute sclerosing panencephalitis and progressive multifocal leucoencephalopathy caused by measles and papovaviruses respectively, are diseases with similar clinical features following infection with conventional viruses.

CJD is found worldwide. Incidence rates range from 0.09 to 31.3 per million per year, with an average of approximately one case per million population per year[2]. Most reported cases have occurred sporadically, with only about 15% of cases being familial suggestive of autosomal dominant determinants[2]. Spatiotemporal clusterings have also been reported in England[3] and Czechoslovakia[4] wherein familial CJD genes controlling susceptibility to the disease may be shared with relatives, leading to an increase in some populations by drift, founder effect or inbreeding, with the virus being acquired by contact or vertical transmission or perhaps activated by genetically determined mechanisms. One Israeli study reported a 30-fold higher incidence of CJD in Jews of Libyan origin than in Jews of European origin[5].

The illness usually presents between the ages of 40 and 60 years, with the sexes being affected equally . The classical presentation is a subacute, rapidly progressive dementia, invariably associated with myoclonus and often accompanied by florid psychiatric symptoms including visual and auditory hallucinations[6]. The patient becomes apathetic and confused with a poor memory and is dysphasic. Dysarthria, bulbar or pseudobulbar palsy then appear, and dysphagia is common. Some patients present with cortical blindness, while others develop it in the course of the disease. Ataxia may appear early as evidence of cerebellar damage. The disease progresses towards eventual stupor and coma, usually with continued myoclonus, and death is invariable, generally within a year. The duration of survival depends largely on the quality of the nursing care. On occasions the disease may pursue a more chronic course without myoclonus, but accompanied by muscle atrophy and fasciculations. This resemblance to motor neurone disease may cause confusion with amyotrophic lateral sclerosis, Parkinson's disease, or Alzheimer's disease[7,8].

Also from clincopathological reports collated by an informal worldwide registry of CJD patients held at the National Institute of Neurological and Communicative Disorders and Stroke (NINCDS), Bethesda, USA, a subset of about 20 cases of CJD has been identified that clinically and pathologically are very similar to kuru[9]. Kuru, confined to the Fore people of Papua New Guinea, has now almost disappeared following the elimination of cannibalism (mortality under 20 in 1981[10]). The patients had an insiduous onset characteristic of kuru, with cerebellar ataxia, tremors, wasting lassitude, and death by terminal inanition. Pathologically, there was less spongiform encephalopathy and a different distribution of lesions within the brain compared with classical CJD disease.

Pre-mortem confirmation of the clinical CJD diagnosis is not possible because no immunological response is detectable and traditional virological methods have not identified the aetiological agent. Electroencephalograms show only non-specific changes, but later there may be high-voltage, bilaterally synchronous polyphasic discharges which become periodic at the rate of one or two a second. Confirmation may be achieved by microscopic examination of cerebral biopsy material which shows neuronal loss, intense gliosis and a fine-meshed vacuolation suggesting minute accumulations of fluid, and due to vacuoles within the cytoplasm of astrocytes and neurones. The degree of change depends on the stage of the disease.

Except for the spontaneous remission reported in one patient[11], the natural course of CJD has been progressive deterioration and death. One report has described repeated suppression of pathologically-proven CJD in a patient for more than six months with adenine arabinoside[12]. However, the early stage of the disease in the patient and evidence of an intact immune system may have accounted for the favourable palliative response, since drug treatment of natural and experimental CJD has yielded equivocal results for other antiviral agents including cytosine arabinoside[13], amantadine[14], idoxuridine[15] and interferon[16].

The exact mode of acquisition of CJD in humans is unknown. Person-to-person transmission has been documented in a corneal graft recipient[17], in two epileptics on whom contaminated stereotactic electrodes had been used[18], and a possible case in a neurosurgeon[19]. Transmission studies in non-human primates serve as the major source of information on the infectivity and transmission of CJD[20]. Cerebrospinal fluid (CSF), brain and spinal cord tissue from patients or animals with CJD frequently demonstrate the capacity to produce infection when inoculated into animals; liver, kidney, lung and lymph node tissue less regularly display such activity. Blood, specifically leucocytes, has been found to be infective during transmission studies with guinea pigs, although viraemia has not yet been documented in humans. Skin, muscle, external secretion, urine and faeces have not been found to be infective. Since CJD virus has yet to be isolated from body surfaces, secretions or excretions, and only irregularly in non-neural peripheral tissues from patients, there is no evidence that people in closest contact with CJD patients (e.g. wives, friends, employee contacts, members of the medical or nursing professions or paramedical personnel) are at greater risk of contracting CJD infection than the general population[20,21]. However, workers most exposed to infected tissues from CJD patients (neurologists, neurosurgeons, neuropathologists, pathologists, research scientists and laboratory personnel) should be aware of possible transmission following tissue penetration[22,23]. Although CJD patients do not need to be placed in special isolation rooms, special care must be taken when handling blood, CSF and tissues (particularly brain tissue). This includes the use of gloves and disposable gowns in routine nursing procedures such as handling blood-stained dressings or cleaning bed sores, and the needle precautions in use for hepatitis B patients. If percutaneous exposure to blood, CSF or tissue does occur, it has been recommended that the wound should be irrigated immediately with 0.5% sodium hypochlorite[24]. Specimens sent to the laboratory from CJD patients should be labelled as

"biohazards" to facilitate appropriate handling of such materials. Organs and tissues of CJD patients should not be used for transplantations[24]. Potentially contaminated materials should be sterilized before being cleaned or reprocessed. Reusable and disposable equipment that has been in direct contact with patient's blood or tissues should be immersed in 0.5% sodium hypochlorite for 1-2 hours or autoclaved at 121°C (15 psi) for 1 hour[25]. Instruments should not be washed until after initial autoclaving at which time they can be discarded, cleaned or re-sterilized. Surfaces should be disinfected with 0.5% sodium hypochlorite before routine cleaning.

The CJD agent has many of the biological properties of a virus, but is "unconventional" in its high degree of physicochemical stability, its small molecular weight and apparent lack of specific antigenicity. As a result many hypotheses have been proposed for the structure of the spongiform encephalopathic agents including a novel proteinacious infectious particle termed a prion[26]. Such particles may either contain an oligonucleotide that acts as a regulatory element, or may even be devoid of nucleic acid, so that the prion protein acts as its own template, thus contradicting the "central dogma" of molecular biology[27]. A less heretical model consists of a small specific nucleic acid that is replicated (using host enzymes) but not translated, and which interacts with the host in strain-specific ways to produce disease. The agent-associated protein is host-derived and "sticky", so that the nucleoprotein does not have a specific antigenicity, and is difficult to purify from host components[28].

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BOTULISM AND COMMERCIAL POT PIE - CALIFORNIA

(Based on California Morbidity (1982). Number 44)

On 3 August 1982, a 56 year old woman residing in Los Angeles County, California, developed diplopia, weakness, difficulty breathing and chest pain. She had respiratory arrest on admission to the hospital but was intubated, resuscitated, and placed in intensive care. Examination showed complete bilateral ptosis, ophthalmoplegia, facial muscle weakness and areflexia. Cerebrospinal fluid was normal except for increased glucose; Tensilon test was negative. She had a history of seizure disorder, diabetes mellitus and organic brain syndrome. It was thought that her subsequent fever was due to pneumonia secondary to aspiation, and that botulism was suspected as the underlying cause of her illness.

The patient lived with her husband and grown son who both prepared meals for her and attempted a strict diet in consideration of her diabetes. When asked about the patient's food history before onset of illness, the husband and son named no likely suspects for botulism. No home-preserved foods had been served, and, with one exception, she had not eaten other foods that were not freshly prepared for her or were not also consumed by her husband and son. The exception was commercial beef pot pie, which was accidently mishandled, then consumed by the patient one day before illness began.

The son had prepared the pot pie for an earlier evening meal. The frozen pie was baked in an oven for 40-45 minutes. As he was about to serve it to his mother, his father came home with some freshly cooked hamburgers just purchased at a take-out restaurant. The pot pie was put aside on an unrefrigerated shelf. Two and one-half days later, the son came home and found his mother had just consumed this pot pie without reheating it. An uneaten portion of the pot pie, still in its metal plate, was retrieved by the family members. Type A botulism toxin was found in this pie by mouse-inoculation test, and type A toxin was demonstrated in the patient's serum.

The Editor commented that this was the third case of botulism associated with commercial pot pies reported from California; and one other incident (involving two clinically diagnosed patients) was reported from Minnesota in 1960. Mishandling of the pot pies occurred in three of these episodes, and mishandling was also suspected in the fourth. The known mishandling consisted of leaving the baked pot pie in the oven with the pilot light on, thereby maintaining "incubator" temperatures overnight. The pies were then eaten with no (or insufficient) reheating to destroy toxin. Or, as in the present case, the baked pie sat out at room temperature for over two days during hot weather-conditions that also could simulate an incubator.

In these situations, it is suspected that the original baking killed competing organisms in the pies and eliminated much of the oxygen. The heat-resistant, anaerobic Clostridium botulinum, which was evidently present and can be found in many fesh, frozen and other food products, was then presumably able to germinate and produce toxin under the crust during storage at warm, incubator-like temperatures. Products such as pot pies should be kept frozen before heating and ideally should be served hot after the first cooking. If any such products are to be saved, it should be quickly refrigerated, then reheated to hot temperatures. This would minimise any risk of botulinal poisoning.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 20/1/83 - 2/2/83 BULLETIN NUMBER . 83/3

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	1		1	1	1	3	5		12
0101 ADENOVIRUS TYPE 1					9	2			11
0102 ADENOVIRUS TYPE 2	3			1	8	2			14
0105 ADENOVIRUS TYPE 5	1				2				3
0109 ADENOVIRUS TYPE 9								1	1
0111 ADENOVIRUS TYPE 11				1					1
0119 ADENOVIRUS TYPE 19				2				5	7
0199 ADENOVIRUS TYPING PENDING			4		5	6			15
0201 INFLUENZA A VIRUS	2		3			1	2	1	9
0203 INFLUENZA B VIRUS			3					3	6
0301 PARAINFLUENZA VIRUS TYPE 1	2						1		3
0303 PARAINFLUENZA VIRUS TYPE 3		2		2	5	6	4	5	24
0399 PARAINFLUENZA VIRUS TYPING PENDING.						1	1		2
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)						1	1	3	5
0500 RHINOVIRUS (ALL TYPES)	1		1	4	13		3		22
0600 MYCOPLASMA PNEUMONIAE	21	1	2	2	1	1	6	15	49
0700 ORNITHOSIS-PSITTACOSIS	2								2
0809 COXSACKIEVIRUS A9							2		2
0816 COXSACKIEVIRUS A16	1			2					3
0899 COXSACKIEVIRUS GROUP A TYPING PENDING							2		2
0903 COXSACKIEVIRUS B3			5		1			1	7
0904 COXSACKIEVIRUS B4								1	1
0905 COXSACKIEVIRUS B5								2	2
1011 ECHOVIRUS TYPE 11	7	2	21	5	5	2	11	6	59
1012 ECHOVIRUS TYPE 12	1								1
1022 ECHOVIRUS TYPE 22	1							1	2
1024 ECHOVIRUS TYPE 24				1					1
1025 ECHOVIRUS TYPE 25							1		1
1030 ECHOVIRUS TYPE 30	2							1	3
1099 ECHOVIRUS TYPING PENDING							1		1
1101 POLIOVIRUS TYPE 1						1			1
1102 POLIOVIRUS TYPE 2						1			1
1104 POLIOVIRUS-VACCINAL STRAIN					2				2
1200 MUMPS VIRUS	4		1	1	1	1	1		9
1300 HERPES VIRUS GROUP-NOT TYPED	27					5			32
1301 HERPES SIMPLEX VIRUS NOT-TYPED		3		4				58	65
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	4		1					3	8
1303 VARICELLA-ZOSTER VIRUS				1		1		2	4
1306 HERPES SIMPLEX TYPE 1	6		10	27		27	21		91
1307 HERPES SIMPLEX TYPE 2	88		13	58		32	48		239
1399 HERPES VIRUS TYPING PENDING			19		6	4			29
1401 COXIELLA BURNETI	3					1	7		11
1502 PICORNA VIRUS-NOT TYPED	7		9						16
1514 MOLLUSCUM CONTAGIOSUM								1	1
1521 MEASLES VIRUS				4	3				7
1522 RUBELLA VIRUS	7			10	1		4	1	23
1532 HEPATITIS B ANTIGEN	18		11	24		14	8	10	85
1535 HEPATITIS A ANTIBODY	3		1	8		6	3	19	40
1541 CHLAMYDIA A - C TRACHOMATIS	11		6	1		6		73	97
1556 CMV - CYTOMEGALOVIRUS	7	1	3	11	5	1	1	1	30
1564 ROTAVIRUS	1	7	10	3		2			23
1571 ENTEROVIRUS TYPE 71 (BRCR)				2					2
1599 ENTEROVIRUS TYPING PENDING		1	10		6		1		18
ROSS RIVER VIRUS								2	2
SMALL VIRUS (LIKE) PARTICLE	3								3
Total	234	17	134	175	74	127	134	215	1,110

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 20/1/83 to 2/2/83 .... 83/3

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.....		5					3				1
0102 ADENOVIRUS TYPE 2.....	1	1					9				
0105 ADENOVIRUS TYPE 5.....							2				
0201 INFLUENZA A VIRUS.....	1	5		1		1					
0203 INFLUENZA B VIRUS.....		1		1			1				
0301 PARAINFLUENZA VIRUS TYPE 1....		3									
0303 PARAINFLUENZA VIRUS TYPE 3....		22		1				1			
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....		3							1		
0500 RHINOVIRUS (ALL TYPES).....	1	18					1				
0600 MYCOPLASMA PNEUMONIAE.....	7	38						1			
0700 ORNITHOSIS-PSITTACOSIS.....	1				1						
0809 COXSACKIEVIRUS A9.....	2										
0816 COXSACKIEVIRUS A16.....		1									2
0903 COXSACKIEVIRUS B3.....		1		5							
0904 COXSACKIEVIRUS B4.....				1							
0905 COXSACKIEVIRUS B5.....				2							
1011 ECHOVIRUS TYPE 11.....	1	4		26		1	12	2	1		1
1012 ECHOVIRUS TYPE 12.....		1									
1022 ECHOVIRUS TYPE 22.....							1				
1024 ECHOVIRUS TYPE 24.....				1							
1025 ECHOVIRUS TYPE 25.....	1										
1030 ECHOVIRUS TYPE 30.....				2							
1101 POLIOVIRUS TYPE 1.....							1				
1102 POLIOVIRUS TYPE 2.....							1				
1104 POLIOVIRUS-VACCINAL STRAIN....		2									
1200 MUMPS VIRUS.....	2		1								1
1301 HERPES SIMPLEX VIRUS NOT-TYPED	1	1	1	2		1					38
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	1							1		1	
1303 VARICELLA-ZOSTER VIRUS.....											2
1306 HERPES SIMPLEX TYPE 1.....	4	9						1		3	43
1307 HERPES SIMPLEX TYPE 2.....	2										10
1401 COXIELLA BURNETI.....	2	1									1
1514 MOLLUSCUM CONTAGIOSUM.....											1
1521 MEASLES VIRUS.....	1		1								6
1522 RUBELLA VIRUS.....	3										18
1532 HEPATITIS B ANTIGEN.....	32							41			
1535 HEPATITIS A ANTIBODY.....	13	1						22			
1556 CMV - CYTOMEGALOVIRUS.....	4	9				1		2		5	
1564 ROTAVIRUS.....				2			20				
1571 ENTEROVIRUS TYPE 71 (BRCR)....											2
9992 ROSS RIVER VIRUS.....											1
9994 SMALL VIRUS (LIKE) PARTICLE...							3				
Total.....	80	126	3	44	1	4	54	71	2	9	127

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 20/1/83 to 2/2/83 ... 83/3
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.....								1		1
0102 ADENOVIRUS TYPE 2.....							1	2		
0105 ADENOVIRUS TYPE 5.....								1		
0109 ADENOVIRUS TYPE 9.....	1									
0119 ADENOVIRUS TYPE 19.....	4	3								
0201 INFLUENZA A VIRUS.....								2		
0203 INFLUENZA B VIRUS.....								4		
0303 PARAINFLUENZA VIRUS TYPE 3....				1		1		2		
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....					1			1		
0500 RHINOVIRUS (ALL TYPES).....								1		1
0600 MYCOPLASMA PNEUMONIAE.....				1	1		1	7		
0903 COXSACKIEVIRUS B3.....								2		
1011 ECHOVIRUS TYPE 11.....						1		8		1
1022 ECHOVIRUS TYPE 22.....							1			
1030 ECHOVIRUS TYPE 30.....								1		
1200 MUMPS VIRUS.....			3					1	1	
1301 HERPES SIMPLEX VIRUS NOT-TYPED		21					1			
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			3	2						
1303 VARICELLA-ZOSTER VIRUS.....				1			1			
1306 HERPES SIMPLEX TYPE 1.....	3	27						3	1	
1307 HERPES SIMPLEX TYPE 2.....		222	1							
1401 COXIELLA BURNETI.....			1					8		
1522 RUBELLA VIRUS.....				1	1			4		
1532 HEPATITIS B ANTIGEN.....									8	
1535 HEPATITIS A ANTIBODY.....								1	2	
1541 CHLAMYDIA A - C.TRACHOMATIS...		97								
1556 CMV - CYTOMEGALOVIRUS.....		1				3		7	3	
1564 ROTAVIRUS.....	1									
9992 ROSS RIVER VIRUS.....					1			1		
Total.....	9	371	8	6	4	5	5	57	15	3