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Communicable Diseases Intelligence

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Contents:

VIRUS REPORTING SCHEME - A total of 1137 reports were received this period. The current outbreak of echovirus type 11 infections, recognised initially in Western Australia in July 1982 (CDI 82/22), continued with 62 cases reported by seven laboratories. When stated, the age distribution of these cases was 15 cases ($\leq$ 1 month); 8 (1-6 months); 5 (7-12 months); 8 (1-4 years); 6 (5-14 years) and 13 ($\geq$ 15 years). As in previous reporting periods, meningitis was the most common clinical feature (38 cases). Outbreaks were reported in a children's reception centre, Melbourne, and in a neonatal ward, St George Hospital, Sydney.

- Enterovirus infections are common, and the great number of enterovirus types make routine serological investigations impracticable and of limited diagnostic value unless an enterovirus is isolated from the faeces, pharynx or CSF. Neutralising antibodies are present in a patient's serum for many years after infection, but complement-fixing antibodies often disappear after a few months. The Institute of Clinical Pathology and Medical Research, Sydney, now use a commercial picornavirus group antigen. The virus types are grown in Vero cells and include coxsackievirus types A9, B1-6 and echovirus types 4, 6, 9, 14, 24, and 30. Controls comprise uninfected Vero tissue culture antigen and negative sera. At present, diagnoses using this group antigen will be coded as 1599 (enterovirus type pending) until the reagent has been adopted by other laboratories, and a new virus code designated accordingly.

- Less common presentations reported this period included the detection of CF antibody against varicella zoster in a 20 year old male with encephalitis, and CF antibody against parainfluenza virus type 3 in a patient with Guillain-Barré syndrome.

- The isolation of the Cryptococcus species from sputum and rib cage abscess of a 57 year old night watchman reported by Fairfield Hospital, Melbourne, in CDI 82/22, has since been identified as C. neoformans and not C. albidus as stated previously.

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INDIGENOUS CHOLERA - QUEENSLAND

On 16 January 1983, Vibrio cholerae O-group 1, biotype El Tor, serotype Inaba was isolated from the stools of a 32 year old male resident of Rockhampton. The patient had not travelled outside the Rockhampton area because of a long-standing, underlying illness. He became ill on 12 January, and was admitted to Rockhampton Base Hospital two days later with a diminished level of consciousness, vomiting and diarrhoea. As a precautionary measure, tetracycline was prescribed for household and close contacts of the patient, although all stool cultures submitted from them were negative for the organism. Since water supplies tested to date have been negative, investigations are also being directed toward the possibility of an asymptomatic V. cholerae carrier among the patient's past visitors.

Editorial Comment

Organisms causing epidemic cholera belong to the V. cholerae 01 toxigenic serogroup. Within the 01 serogroup are the two biotypes cholerae and El Tor, and at least three serotypes, namely, Inaba, Ogawa and Hikojima. Two serotyping schemes for V. cholerae non-01 are in use; one developed by Shimada and Sakazaki [1] (83 serotypes) and the other by Smith [2] (72 serotypes).

Since 1977, three incidents of locally-acquired, clinical cholera have been reported in Australia [3, 4, 5]. Both of the severe cases in the first and third incidents had an existing gastric condition, and epidemiological and clinical studies have shown that gastric acidity is a major factor in host resistance. Subsequent to the index case, V. cholerae serogroups 01 and non-01 have been isolated continually from river water, estuaries, surface waters and sewerage treatment plants along the east coast of Australia [3, 6] (personal communication, P.M. Desmarchelier, Commonwealth Institute of Health, Sydney). Both Ogawa and Inaba serotypes have been isolated, although the Inaba serotype is predominant in Queensland rivers, and most of the strains have been shown to elaborate cholera enterotoxin using the Y-1 adrenal cell assay. The Ogawa serotype has been isolated in Queensland, but it is the only serotype isolated to date in New South Wales waters. None of the Ogawa strains submitted to, or isolated by, the Commonwealth Institute of Health have been shown to be toxigenic. On the other hand, a small percentage of the environmental non-01 serotype isolates have been cholera toxin producers.

The ecology of these autochthonous pathogenic vibrios, and the public health signficance of their presence in estuaries and freshwater bi-valves [7], are poorly understood at present. Studies have confirmed that strains isolated from clinical cases and from the environment possess phenotypic similarities [8] and extensive DNA-DNA homology [9] to reference strains of V. cholerae. Accordingly, the potency and specificity of the 01 antiserum used for agglutination studies is critical in the recognition of the true cholera vibrio [10]. It has been postulated that some of the environmental isolates are mutants of the clinical isolates, and that serotypic and toxigenic characteristics may be gained and lost as organisms move between the intestine of man and an aquatic environment. Thus organisms in the environment that appear to be non-toxigenic and non-01 could revert to become virulent V. cholerae 01 under certain circumstances. The demonstration of transposon-

facilitated recombination in V. cholerae provides a mechanism for the transfer of genetic material, and consequent gain or loss of toxigenicity, in response to ecological pressure (11). Alternatively, although the V. cholerae strains that circulate among humans are in general separate from those that form the permanent V. cholerae flora of certain saline waters, the two sets of strains are overlapping populations, and the human gut, and certain aquatic environments, may select for toxigenic strains.

In 1981, 36,840 cases of cholera were reported in 34 countries (12), but cholera vaccination is not necessary for the ordinary tourist. The complete primary series is suggested only for special high-risk groups that work and live in highly endemic areas under less than adequate sanitary conditions and those persons with compromised gastric defence mechanisms (13,14). Whenever a certificate of cholera immunisation is required from other travellers, a single dose of vaccine is sufficient to satisfy International Health Regulations. In contrast to clinical cholera disease which gives rise to long-lasting immunity, the whole cell injectable cholera vaccines give only partial immunity for less than six months. However, four oral cholera vaccines are now being developed (15); a live, chemically-mutated strain (Texas Star) that does not elaborate the enzymatically active A subunit of the toxin molecule; a recombinant strain in which the toxin A subunit genes have been deleted; a combination of the purified B subunit of cholera toxin and the standard killed whole cell vaccine (both orally); and a large nontoxic aggregate of the cholera toxin that has been inactivated by heating to 65°C.

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PROPHYLAXIS OF CONTACTS OF CASES OF HAEMOPHILUS INFLUENZAE TYPE B DISEASE

(Based on MMWR (1982) 31:672; and Alberta Epidemiologic Notes and Reports (1982) 6: 329)

Recent studies in the USA have shown an increased risk of disease among close contacts of persons with invasive Haemophilus influenzae type b (HIB) infection (i.e. meningitis, epiglottitis, pneumonia, cellulitis and bacteraemia), suggesting a need to consider chemoprophylaxis for prevention of secondary cases.

Six studies have estimated the risk of disease among household contacts of cases in the month following onset of disease in the index case[1-6]. Attack rates varied substantially with age; the rate was 3.8% among children under two years of age, 1.5% among children two to three years of age, 0.1% among children four to five years of age, and 0% among contacts over the age of six years. The overall attack rate was 0.3%, representing approximately a 600-fold increase in risk, compared with the risk in the population at large. Whether increased risk of disease occurs in day-care centre contacts of children with invasive HIB disease has not been resolved. Also it is not known whether the risk of secondary cases is different for persons in contact with a case with meningitis than for those in contact with cases with epiglottitis or other invasive HIB diseases. At this time, all index cases with invasive HIB disease are considered to increase the risk for contacts.

Initial studies focused on usefulness of various antimicrobial agents to eliminate nasopharyngeal carriage of HIB. Ampicillin, trimethoprim-sulphamethoxazole, erythromycin-sulphisoxazole and cefaclor were shown to eliminate carriage in fewer than 70% of culture-positive contacts. Also, in persons with HIB disease, pharyngeal carriage of the organism has been shown to persist following intravenous therapy with chloramphenicol or ampicillin.

However, rifampin in a dosage of 20 mg/kg per dose (halved for neonates) once daily for four days (maximum dose 600 mg) eradicated carriage in 90-100% of contacts treated. A large multi-centre, randomised, placebo-controlled trial of the efficacy of rifampin in preventing disease in household and day-care contacts has been reported recently [6]. Four secondary cases occurred among the 800 placebo-treated contacts compared with no cases among the 1166 rifampin-treated contacts ($p=0.03$). However, anecdotal reports of the failure of rifampin to prevent secondary cases have occurred. The addition of rifampin to apple sauce is acceptable to patients, and results in peak serum and salivary concentrations that are not significantly different from those obtained with a specially prepared suspension.[7] Side effects of rifampin in the 20 mg/kg dosage in the multi-centre trial occurred in 20% of recipients, compared with 11% of placebo recipients. These included nausea, vomiting, diarrhoea, headache and dizziness, but the rate was similar to the 24% rate of adverse effects in recipients of rifampin at a dosage of 10 mg/kg. No serious adverse reactions occurred. Orange discolouration of urine was noted in 84% of rifampin recipients. Rifampin usage may also cause discolouration of soft contact lenses or ineffectiveness of oral contraceptives.

Concern has been raised about the possibility of developing rifampin-resistant H. influenzae isolates. Although an occasional rifampin-resistant strain has been reported, none of the isolates from index patients or contacts was rifampin-resistant in the multi-centre chemoprophylaxis trial. Monitoring strains causing invasive disease for the development of rifampin resistance will be important for assessing the continued usefulness of rifampin as a chemoprophylactic agent.

In view of the increased risk of disease in household contacts less than four years of age and the efficacy of rifampin in eliminating carriage of H. influenzae organisms and preventing secondary cases of disease, it is recommended that:

- Contacts who develop symptoms suggestive of HIB disease, such as fever or headache, should be evaluated promptly by a physician.

- In any household in which a case of invasive HIB disease has occurred and in which another child less than four years of age resides, all members of the household, including adults, should receive rifampin in a dosage of 20 mg/kg per dose once daily (maximum dose 600 mg/day) for four days; the dose for neonates (> one month) is 10 mg/kg once daily for four days.

- In day-care centre classromms in which a case of H. influenzae disease has occurred and in which children less than four years of age are present, all parents should be notified (preferably in writing) regarding occurrence of a case, and the possibility of increased risk to their children. The symptoms to look for, the usefulness of rifampin chemoprophylaxis, and the need for prompt medical evaluation if symptoms occur should be stated. All students and staff in the classroom should be considered for chemoprophylaxis according to the above regimen. However, it should be noted that the data on risk of secondary spread and efficacy of chemoprophylaxis in day-care centres are less complete than for household contacts.

- Chemoprophylaxis should be instituted as rapidly as possible following onset of disease in the index case. If more than seven days have passed since the last contact with the index case, chemoprophylaxis is probably not indicated.

- The index case should be treated with the same rifampin regimen before discharge from hospital.

- Nasopharyngeal carriage studies should not be employed as a guide for chemoprophylaxis because of the lack of correlation of carriage with risk of disease and because the time required to complete such studies would delay implementation of chemoprophylaxis.

- Rifampin should not be used in pregnant women because it is teratogenic in laboratory animals.

Since rifampin is not 100% effective in eradicating the carrier state, close surveillance is a necessary part of management for several months following the contact. Parents and day-care centre staff should be instructed to report any acute febrile illnesses to the physician immediately. Febrile illness in a high risk contact is an indication for careful evaluation and consideration of blood cultures, lumbar puncture and intravenous antibiotics appropriate for the therapy of HIB disease until diagnosis is confirmed or excluded by culture results.

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HAEMORRHAGIC SHOCK AND ENCEPHALOPATHY - UK

(Based on CDR (1982) 82/51:)

Seven infants (aged three to seven months) were admitted to the Hospital for Sick Children, London, during 1982 with a fulminant, and usually fatal illness, characterised by severe shock, haemorrhage and encephalopathy. All had an abrupt onset of convulsions, high fever, watery diarrhoea and profound shock, and very large volumes of colloid or blood were required to restore the circulatory volume. Hypernatraemia and acidosis were always present on admission, suggesting pooling of fluid in the gut. All had severe disseminated intravascular coagulation, thrombocytopenia and bleeding. Renal and hepatic function were markedly deranged, but plasma ammonia was always normal. Intensive support resulted in improvement, but gross neurological disorder persisted. Five children died, and the two survivors have severe neurological handicap.

The disease affecting these children is distinct from previously recognised syndromes, but has features in common with the viral haemorrhagic fevers and toxin induced shock. Extensive bacteriological and virological investigations have failed to identify a specific cause. The Editor would be interested in receiving information on any Australian cases of this new syndrome with a high mortality affecting young children.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

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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	3		2		1	3	3		12
0101 ADENOVIRUS TYPE 1	1			1	2	5			9
0102 ADENOVIRUS TYPE 2	1				4				5
0103 ADENOVIRUS TYPE 3						2			2
0105 ADENOVIRUS TYPE 5	2	1			1	4			8
0107 ADENOVIRUS TYPE 7	1			1				1	3
0108 ADENOVIRUS TYPE 8								1	1
0119 ADENOVIRUS TYPE 19				1				4	5
0131 ADENOVIRUS TYPE 31						1			1
0199 ADENOVIRUS TYPING PENDING					6	9			15
0201 INFLUENZA A VIRUS	1			1			10	4	16
0203 INFLUENZA B VIRUS			2					3	5
0301 PARAINFLUENZA VIRUS TYPE 1						2			2
0302 PARAINFLUENZA VIRUS TYPE 2								1	1
0303 PARAINFLUENZA VIRUS TYPE 3	1	1		1	9	3	2	5	22
0399 PARAINFLUENZA VIRUS TYPING PENDING						2	1		3
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	1					1	4	4	10
0500 RHINOVIRUS (ALL TYPES)					5	3	2		10
0600 MYCOPLASMA PNEUMONIAE	12	1	4	5		2	19	13	56
0700 ORNITHOSIS-PSITTACOSIS				2					2
0809 COXSACKIEVIRUS A9	1								1
0816 COXSACKIEVIRUS A16				1					1
0899 COXSACKIEVIRUS GROUP A TYPING PENDING							1		1
0903 COXSACKIEVIRUS B3	1	*	1				1		3
0905 COXSACKIEVIRUS B5								2	2
1000 ECHOVIRUS NOT TYPED							1		1
1003 ECHOVIRUS TYPE 3								1	1
1011 ECHOVIRUS TYPE 11	8	4	7	6		3	28	6	62
1022 ECHOVIRUS TYPE 22	1						1	5	7
1024 ECHOVIRUS TYPE 24								1	1
1025 ECHOVIRUS TYPE 25							1		1
1030 ECHOVIRUS TYPE 30	1								1
1101 POLIOVIRUS TYPE 1						2	1		3
1104 POLIOVIRUS-VACCINAL STRAIN						1			1
1200 MUMPS VIRUS	5	2	4	1		2		2	16
1300 HERPES VIRUS GROUP-NOT TYPED	22		1	4		5			32
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1		10			2	61	74
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	5	1	2			3		1	12
1303 VARICELLA-ZOSTER VIRUS	2		1	2		3	3	1	12
1306 HERPES SIMPLEX TYPE 1	10			22			24		56
1307 HERPES SIMPLEX TYPE 2	90			31			69		190
1399 HERPES VIRUS TYPING PENDING			12		1	41			54
1401 COXIELLA BURNETI	3						8		11
1502 PICORNA VIRUS-NOT TYPED	3		4					1	8
1514 MOLLUSCUM CONTAGIOSUM						1		1	2
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS)						1			1
1521 MEASLES VIRUS			2	5	1	1		1	10
1522 RUBELLA VIRUS	4		2		3	1	7	1	18
1532 HEPATITIS B ANTIGEN	22		8	39		42	9	10	130
1535 HEPATITIS A ANTIBODY	1		1	8		15	7	5	37
1541 CHLAMYDIA A - C TRACHOMATIS	5							106	111
1556 CMV - CYTOMEGALOVIRUS	4		1	16	5	2	1	2	31
1562 REOVIRUS (ALL TYPES)						1		1	2
1564 ROTAVIRUS	1		14	2	2			2	21
1571 ENTEROVIRUS TYPE 71 (BRCR)				1					1
1599 ENTEROVIRUS TYPING PENDING		4	10		8	1	5		28
ARBO. GROUP A.(UNSPECIFIED)				1					1
ROSS RIVER VIRUS								1	1
SMALL VIRUS (LIKE) PARTICLE	1			1		2			4
Total	213	15	78	162	48	164	210	247	1,137

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

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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
0100 ADENOVIRUS NOT TYPED.....	1										
0101 ADENOVIRUS TYPE 1.....		3					1				
0102 ADENOVIRUS TYPE 2.....		2					1				
0103 ADENOVIRUS TYPE 3.....	1						1				
0105 ADENOVIRUS TYPE 5.....		2	1				3				
0107 ADENOVIRUS TYPE 7.....		2									
0131 ADENOVIRUS TYPE 31.....							1				
0201 INFLUENZA A VIRUS.....		10		1							
0203 INFLUENZA B VIRUS.....	1	2									
0301 PARAINFLUENZA VIRUS TYPE 1....		2									
0303 PARAINFLUENZA VIRUS TYPE 3....	1	18				1			1		1
0399 PARAINFLUENZA VIRUS TYPING PENDING.....		1									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....		10									
0500 RHINOVIRUS (ALL TYPES).....	1	7									
0600 MYCOPLASMA PNEUMONIAE.....	4	44									
0700 ORNITHOSIS-PSITTACOSIS.....		2									
0809 COXSACKIEVIRUS A9.....				1							
0816 COXSACKIEVIRUS A16.....											1
0903 COXSACKIEVIRUS B3.....	1			2							
0905 COXSACKIEVIRUS B5.....				1							
1003 ECHOVIRUS TYPE 3.....	1										
1011 ECHOVIRUS TYPE 11.....	10	5	1	38		2	1				
1022 ECHOVIRUS TYPE 22.....		4					1				
1024 ECHOVIRUS TYPE 24.....	1										
1025 ECHOVIRUS TYPE 25.....		1									
1030 ECHOVIRUS TYPE 30.....			1								
1101 POLIOVIRUS TYPE 1.....	1						2				
1200 MUMPS VIRUS.....	3	1		4							2
1301 HERPES SIMPLEX VIRUS NOT-TYPED	4				2					1	49
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	4	1						1		1	
1303 VARICELLA-ZOSTER VIRUS.....			2	2							8
1306 HERPES SIMPLEX TYPE 1.....	6	2	1				1				16
1307 HERPES SIMPLEX TYPE 2.....	4										13
1401 COXIELLA BURNETI.....	2	1									
1514 MOLLUSCUM CONTAGIOSUM.....											2
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS).....											1
1521 MEASLES VIRUS.....											10
1522 RUBELLA VIRUS.....	1	1									15
1532 HEPATITIS B ANTIGEN.....	59							51			
1535 HEPATITIS A ANTIBODY.....	7							30			
1556 CMV - CYTOMEGALOVIRUS.....	6	7	1		1	1				9	
1562 REOVIRUS (ALL TYPES).....						1	1				
1564 ROTAVIRUS.....							13				
1571 ENTEROVIRUS TYPE 71 (BRCR)....				1							
9901 ARBO. GROUP A.(UNSPECIFIED)...											1
9992 ROSS RIVER VIRUS.....											1
9994 SMALL VIRUS (LIKE) PARTICLE...	1						3				
Total.....	120	128	7	50	3	5	29	82	1	11	120

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

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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	1	1
0102 ADENOVIRUS TYPE 2.....								1	1	
0105 ADENOVIRUS TYPE 5.....							2			
0107 ADENOVIRUS TYPE 7.....	2									
0108 ADENOVIRUS TYPE 8.....	1									
0119 ADENOVIRUS TYPE 19.....	2	3								
0201 INFLUENZA A VIRUS.....			1		2		1	10		
0203 INFLUENZA B VIRUS.....								1	1	
0302 PARAINFLUENZA VIRUS TYPE 2....							1			
0303 PARAINFLUENZA VIRUS TYPE 3....								1		
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....								3		
0500 RHINOVIRUS (ALL TYPES).....										2
0600 MYCOPLASMA PNEUMONIAE.....			1		1			19		
0905 COXSACKIEVIRUS B5.....		1								
1011 ECHOVIRUS TYPE 11.....							2	4	2	
1022 ECHOVIRUS TYPE 22.....						1	1			
1200 MUMPS VIRUS.....			3	1	1			1		
1301 HERPES SIMPLEX VIRUS NOT-TYPED		19						1	2	
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			4					1		
1303 VARICELLA-ZOSTER VIRUS.....		1						2		
1306 HERPES SIMPLEX TYPE 1.....	3	27						1		
1307 HERPES SIMPLEX TYPE 2.....		172								
1401 COXIELLA BURNETI.....								9		
1521 MEASLES VIRUS.....								1		
1522 RUBELLA VIRUS.....					1			7	1	
1532 HEPATITIS B ANTIGEN.....					1			1	19	
1541 CHLAMYDIA A - C.TRACHOMATIS...		111								
1556 CMV - CYTOMEGALOVIRUS.....		1		1		3		3	1	
1564 ROTAVIRUS.....								8		
9992 ROSS RIVER VIRUS.....								1		
Total.....	8	335	9	2	6	4	10	76	28	3