

Communicable Diseases Intelligence

Bulletin number 84/6
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VIRUS REPORTING SCHEME - A total of 1057 reports were received this period. Unfortunately, a delay in the mail prevented the processing of data from the State Health Laboratory, Brisbane, although reference to them are made in the following text.

- . Arbovirus infections - The State distribution of the 281 clinical and serologically confirmed cases of Ross River virus infection reported this period is New South Wales (89), Queensland (74), Victoria (34), Western Australia (17), South Australia (12) and Northern Territory (10). In addition to the 43 cases in which the location was unspecified, two cases were diagnosed in residents of the Australian Capital Territory, although details of their recent itineraries were not provided. The Brisbane laboratory also diagnosed dengue in a 24 year old female who had returned recently from Bali, and a Kunjin virus infection in a 56 year old male.
- . Influenza - Unseasonal influenza activity was demonstrated by the four reports of influenza A and two reports of influenza B virus infections, all diagnosed by complement-fixation. The two influenza B infections reported by the Institute of Clinical Pathology and Medical Research, Sydney, were in a 43 year old male and a baby girl. The laboratory also diagnosed influenza A in a 28 year old female and a 56 year old male with myalgia; and the State Health Laboratory Services, Perth, reported influenza A infection in a 16 year old asthmatic, febrile male who had returned from Nigeria, and in a 43 year old female with hypertension and rash. Influenza B virus resembling B/Singapore/222/79 was also isolated by the OIC WHO Influenza Reference Centre from a 22 year old male student from Melbourne University with fever and myalgia.

During the 1983-84 season in the northern hemisphere, influenza activity has been very low except in Japan, USA and USSR where it has been widespread (WER (1984) 59 : 53). Antigenic analysis of H₁N₁ viruses have indicated that several distinct, but closely related, variants are in circulation (A/Brazil/11/78; A/England/333/80; A/Chile/1/83; A/Dunedin/27/83 and A/Victoria/7/83), and are frequently detected from the same outbreak. Approximately 70% of the H₃N₂ viruses have been characterised as A/Belgium/2/81 in that they reacted equally well with antisera against A/Bangkok/1/79-like and A/Texas/1/77-like strains, although as the season progressed, strains showing a slightly different reaction pattern, typified by A/Philippines/2/82, became more common. Some of the influenza B viruses isolated recently from USA and USSR have reacted poorly with antisera to B/Singapore/222/79.

MELIOIDOSIS - SYDNEY

(Contributed by R. Munro, Institute of Clinical Pathology and Medical Research, Sydney.)

In January 1984, a 61 year old Indian Hindu from Fiji was admitted to the Ryde Hospital, Sydney, with a four day history of fever, sore throat, polydipsia and polyuria with mild dysuria. The patient was diabetic managed with oral hypoglycaemic agents, and had a recent history of intermittent medication. He had been in Australia on holiday for the past two months. He was commenced on intravenous ampicillin, but over the next three days became progressively worse with the development of a non-productive cough and evidence of bilateral bronchopneumonia on chest X-ray. Treatment with erythromycin, followed by cephalothin, gentamicin and flucloxacillin had no affect on his rapid deterioration. Nine days after admission he developed arthritis and effusion in one knee. The patient died on day 12, clinically septicaemic with overwhelming bronchopneumonia.

A Gram-negative rod was isolated from blood cultures, knee aspirate and sputum, which was identified as Pseudomonas pseudomallei using the API identification system (Carter Wallace) and by oxidation-fermentation media reactions. Colonies had the characteristic wrinkled appearance and pungent odour after a few days incubation. However, on primary isolation the colonies were mucoid in appearance after 24 hours.

Melioidosis is endemic in Northern Queensland and parts of the Northern Territory (1,2). Otherwise, cases occurring in Australia are likely to have been imported from South East Asia, Asia Minor, Papua New Guinea or other areas where melioidosis is endemic. The disease may present clinically as an acute septicaemia with pneumonia and multiple abscesses; a more subacute process evolving over weeks or months with multiple abscesses and pneumonia; or a chronic suppurative condition similar to tuberculosis. Latent infection can occur from months to many years after exposure, and in endemic areas serological testing has revealed frequent subclinical infection.

The patient probably represented a latent infection which reactivated in relation to poor control of diabetes. The organism is usually resistant to penicillins, cephalosporins and aminoglycosides. Recommended treatments include tetracycline, co-trimoxazole or chloramphenicol. Combinations of antibiotics are usually given for periods of weeks to months.

References

1. MJA (1980) 1 : 314
2. Trans. Roy. Soc. Trop. Med. (1980) 74 : 138

COCCIDIOIDOMYCOSIS

(Based on CDR (1984) 84/05 : 4)

A fatal case of coccidioidomycosis was diagnosed recently at the Royal United Hospital, Bath, UK. On 29 June 1983, a 58 year old retired chauffeur was referred to an out-patient clinic with a seven week history of dyspnoea, dry cough and weight loss. He had experienced a flu-like illness while travelling in the USA in April 1983, where he visited Salt Lake City, Utah, and Los Angeles, Las Vegas, San Francisco and the San Joaquin Valley in California. His symptoms began to improve following treatment with oral prednisolone and several courses of antibiotics prescribed by his general practitioner. He had

been using salbutamol and beclomethasone inhalers since 1981 when emphysema and asthma were diagnosed, the latter apparently dating from cessation of smoking the previous year.

He was admitted for further investigations and was found to have a total white cell count of $10.9 \times 10^9/l$ with an eosinophilia of $1.2 \times 10^9/l$ (11%) and raised plasma viscosity of 1.96 (normal range 1.50-1.72). When chest X-ray examinations showed wide-spread nodular shadows and a transbronchial lung biopsy revealed interstitial fibrosis, a diagnosis of fibrosing alveolitis was made. He was discharged home on 21 July taking oral prednisolone 30 mg once daily. Subsequent attempts to reduce the steroid dosage resulted in exacerbation of cough.

He was re-admitted on 9 October complaining of a worsening dry cough and feverish shivering episodes. He failed to respond to steroids and antibiotics, developed a high swinging pyrexia and deteriorated rapidly, dying from respiratory failure on 17 October. A post mortem diagnosis of disseminated coccidioidomycosis was made when spherules of Coccidioides immitis were found in lung, spleen, kidney and thyroid tissue. Serum samples taken on 29 June, 15 July and 14 October were examined by the PHLS Mycological Reference Laboratory and all were coccidioides precipitin test positive but complement fixation (CF) test negative. C. immitis was subsequently grown by the Reference Laboratory from post mortem specimens of lung and spleen.

Comment

Coccidioidomycosis is endemic in parts of Utah and California, particularly in the San Joaquin Valley. Approximately four cases a week are notified to the Department of Health Services, California, with annual totals of 305, 389 and 462 for the years 1983, 1982 and 1981 respectively (1). C. immitis is a dimorphic fungus that propagates in soil, especially in and around rodent burrows, and can infect man, cattle, cats, dogs, horses and other animals (2). Transmission is by the inhalation of spores from soil; there is no direct transmission from man, or animal to man. Laboratory cultures of C. immitis represent a severe biohazard, and suspect isolates should only be handled by experienced laboratories prepared to deal with them using appropriate safety equipment and procedures (3). Dusty fomites from endemic areas can transmit infections elsewhere, and infections occur in persons who have merely travelled through endemic areas. The internal mycosis begins as a respiratory infection, although the primary infection may be entirely asymptomatic or resemble an acute influenzal illness with fever, chills, cough and pleural pain. Primary infection may heal completely without detectable residuals, or may leave fibrosis, calcification of pulmonary lesions, a persistent thin-walled cavity, or, most rarely, progress to the granulomatous disseminated form characterised by lung lesions and abscesses throughout the body.

Coccidioidomycosis is rarely diagnosed in the UK, with only four cases confirmed during the past four years. Serological tests are often, but not invariably helpful. A positive precipitin test is usually indicative of a recent primary infection. Precipitating antibodies appear earlier than CF antibodies. Although they generally do not persist, they may do so in some patients with disseminated infection. CF antibodies usually correlate with severity of infection. High and rising CF titres are usually associated with dissemination, but death caused by fulminating infection may occur without a

rise in CF titre. Persistence of precipitins and absence of CF antibodies is unusual, but the diagnosis of coccidioidomycosis in the above case was supported by the serological findings and the isolation of C. immitis from post mortem tissue.

Coccidioidomycosis endemic areas coincide generally with the Lower Sonoran Life Zone which is characterised by arid to semi-arid climates, hot summers, few winter freezes, low altitude, alkaline soil and sparse flora, so that the fungus could become established in Australia. To the Editor's knowledge there have been no published Australian cases of C. immitis infection in persons returning from the endemic south western states of the USA. Coccidioidomycosis was suspected in an American tourist presenting with pneumonia at the Royal Melbourne Hospital in 1981, but despite exhaustive laboratory tests, the aetiological agent was not determined. The woman made an uneventful recovery and returned to the USA.

References

1. California Morbidity (1983) No. 51
2. Coccidioidomycosis. In: "Control of Communicable Diseases in Man". Ed. A.S. Benenson (1980). pp 86-88. The American Public Health Association, Washington D.C.
3. Ann. Intern. Med. (1964) 60 : 941
4. Ariz. Med. (1965) 22 : 261

FOOD VIROLOGY

(Based on WER (1984) 59 : 58)

Viruses may occur in foods and sometimes cause illness among consumers. The record of these diseases is limited because many cases are not reported and because the diagnosis of viral diseases is even more complicated than that of many other foodborne infectious illnesses. The majority of viruses causing foodborne disease emanate from the human intestines and are introduced into food either through mishandling by an infected individual or by way of water or sewage contaminated with human faeces. However, a few viral zoonoses are known to be transmitted through foods on occasion, and several viral agents of animal disease may be disseminated by way of the human food supply.

For many years the Veterinary Public Health Unit (WHO Headquarters) has coordinated activities on food virology which are based at the WHO Collaborating Centres in Brno, Czechoslovakia, and Madison, USA.

The "Manual on food virology" (VPH/83/46) is written by Dr. D. Cliver, Head of the WHO Collaborating Centre on Food Virology, University of Wisconsin, Madison, and attempts to summarise the successful activity of the Food Virology Program and gives information in a form coherent enough to make a significant contribution to the protection of human health. All food vehicles, including drinking water, and all viruses known to be transmitted through foods are considered, and other viruses which appear to show a significant potential for contaminating food are also discussed. There is no doubt that clarification of the role of food as a source or vehicle of viral diseases and understanding of the ecology of viruses in food, as well as the selection of appropriate laboratory methods and decontamination measures, can contribute to the elaboration and implementation of programs on the prevention and control of foodborne diseases caused by viruses, which should be considered an important part of primary health care.

This document, which will be valuable for clinicians, veterinarians, laboratory virologists, epidemiologists, students and many others, can be obtained free of charge from the Chief, Veterinary Public Health, Division of Communicable Diseases, World Health Organisation, 1211 Geneva, 27, Switzerland.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE
 REPORTING PERIOD - 1/3/84 - 14/3/84 BULLETIN NUMBER . 84/6
 VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPMR		PHH/	FAIR-			STATE	STATE	Total
	(NSW)/ WVH (ACT)	RAHC (NSW)	POW (NSW)	FIELD (VIC)	RCH (VIC)	IMVS (SA)	LAB (QLD)	LAB (WA)	
0100 ADENOVIRUS NOT TYPED.....	3	4	2			3	1	2	15
0101 ADENOVIRUS TYPE 1.....	1		1				1		3
0102 ADENOVIRUS TYPE 2.....	2			1			2		5
0103 ADENOVIRUS TYPE 3.....				2					2
0105 ADENOVIRUS TYPE 5.....		1					1		2
0107 ADENOVIRUS TYPE 7.....							1		1
0108 ADENOVIRUS TYPE 8.....			1						1
0110 ADENOVIRUS TYPE 10.....								2	2
0137 ADENOVIRUS TYPE 37.....								1	1
0199 ADENOVIRUS TYPING PENDING.....		1	4			5	2		12
0201 INFLUENZA A VIRUS.....	2							2	4
0203 INFLUENZA B VIRUS.....	2								2
0301 PARAINFLUENZA VIRUS TYPE 1.....						3			3
0302 PARAINFLUENZA VIRUS TYPE 2.....								1	1
0303 PARAINFLUENZA VIRUS TYPE 3.....	2			1	3	1		3	10
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)...	1		2						3
0500 RHINOVIRUS (ALL TYPES).....		1		4	5	3		6	19
0600 MYCOPLASMA PNEUMONIAE.....	87	3	6	9	1	14		1	121
0700 ORNITHOSIS-PSITTACOSIS.....			1	1					2
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED.....							1		1
0902 COXSACKIEVIRUS B2.....						1		2	3
0905 COXSACKIEVIRUS B5.....		1							1
1003 ECHOVIRUS TYPE 3.....								1	1
1009 ECHOVIRUS TYPE 9.....				2					2
1011 ECHOVIRUS TYPE 11.....			1						1
1014 ECHOVIRUS TYPE 14.....	1								1
1017 ECHOVIRUS TYPE 17.....	1								1
1025 ECHOVIRUS TYPE 25.....				1					1
1030 ECHOVIRUS TYPE 30.....			1						1
1100 POLIOVIRUS NOT TYPED.....			5						5
1101 POLIOVIRUS TYPE 1.....							3		3
1102 POLIOVIRUS TYPE 2.....	4								4
1103 POLIOVIRUS TYPE 3.....	2	1					1		4
1104 POLIOVIRUS-VACCINAL STRAIN.....				1	3				4
1200 MUMPS VIRUS.....	4	1		1					6
1300 HERPES VIRUS GROUP-NOT TYPED.....	11			2			6	3	22
1301 HERPES SIMPLEX VIRUS NOT-TYPED.....								2	2
1302 EPSTEIN-BARR VIRUS (EB VIRUS).....	6	1						12	19
1303 VARICELLA-ZOSTER VIRUS.....	4							4	8
1306 HERPES SIMPLEX TYPE 1.....	12			27			20	15	74
1307 HERPES SIMPLEX TYPE 2.....	76		1	73			18	35	203
1399 HERPES VIRUS TYPING PENDING.....					3		1		4
1401 COXIELLA BURNETI.....	3								3
1502 PICORNA VIRUS-NOT TYPED.....	15		4						19
1514 MOLLUSCUM CONTAGIOSUM.....							1		1
1521 MEASLES VIRUS.....							3		3
1522 RUBELLA VIRUS.....	8		2	3	1			2	16
1532 HEPATITIS B ANTIGEN.....	51		1	14			11	9	86
1535 HEPATITIS A ANTIBODY.....	1		1	2			1	5	10
1541 CHLAMYDIA A - C TRACHOMATIS.....	10							81	91
1556 CMV - CYTOMEGALOVIRUS.....	17	3	2	29	8	2		10	71
1564 ROTAVIRUS.....	7	1	9		11	7			35
1599 ENTEROVIRUS TYPING PENDING.....		1	12		4	1			18
9901 ARBO. GROUP A.(UNSPECIFIED).....				44		17			61
9902 POXVIRUS GROUP NOT TYPED.....				1					1
9992 ROSS RIVER VIRUS.....			9	27				22	58
9994 SMALL VIRUS (LIKE) PARTICLE.....	3								3
Total.....	336	19	65	246	51	119		221	1,057

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 1/3/84 to 14/3/84

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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/ muc memb
0100 ADENOVIRUS NOT TYPED.....		3					7				1
0101 ADENOVIRUS TYPE 1.....	1						1				
0102 ADENOVIRUS TYPE 2.....	3	2									
0103 ADENOVIRUS TYPE 3.....		1		1							
0105 ADENOVIRUS TYPE 5.....		1					1				
0107 ADENOVIRUS TYPE 7.....		1									
0201 INFLUENZA A VIRUS.....	1								1		
0203 INFLUENZA B VIRUS.....	1	1									
0301 PARAINFLUENZA VIRUS TYPE 1....		3									
0302 PARAINFLUENZA VIRUS TYPE 2....		1									
0303 PARAINFLUENZA VIRUS TYPE 3....	2	9									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	2	1									
0500 RHINOVIRUS (ALL TYPES).....		17					2				
0600 MYCOPLASMA PNEUMONIAE.....	26	78					1		1		4
0700 ORNITHOSIS-PSITTACOSIS.....		2									
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED.....											1
0902 COXSACKIEVIRUS B2.....		2									
0905 COXSACKIEVIRUS B5.....							1				
1009 ECHOVIRUS TYPE 9.....				2							
1011 ECHOVIRUS TYPE 11.....				1							
1017 ECHOVIRUS TYPE 17.....							1				
1025 ECHOVIRUS TYPE 25.....		1		1							
1030 ECHOVIRUS TYPE 30.....				1							
1101 POLIOVIRUS TYPE 1.....		1					2				
1102 POLIOVIRUS TYPE 2.....	4										
1103 POLIOVIRUS TYPE 3.....	2						2				
1104 POLIOVIRUS-VACCINAL STRAIN....		1					2				1
1200 MUMPS VIRUS.....				2							
1300 HERPES VIRUS GROUP-NOT TYPED..	4							1			13
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1									
1302 EPSTEIN-BARR VIRUS (EB VIRUS)..	5	4						1			
1303 VARICELLA-ZOSTER VIRUS.....			1	1							
1306 HERPES SIMPLEX TYPE 1.....	4	4	1					1			
1307 HERPES SIMPLEX TYPE 2.....	1		1	1						1	27
1502 PICORNA VIRUS-NOT TYPED.....	1		1	1			5		2		1
1514 MOLLUSCUM CONTAGIOSUM.....											1
1521 MEASLES VIRUS.....											3
1522 RUBELLA VIRUS.....	2										11
1532 HEPATITIS B ANTIGEN.....	48			1				22			1
1535 HEPATITIS A ANTIBODY.....	3							7			
1541 CHLAMYDIA A - C.TRACHOMATIS...		1									
1556 CMV - CYTOMEGALOVIRUS.....	15	11			5		2	2		7	1
1564 ROTAVIRUS.....		1					35				
9901 ARBO. GROUP A.(UNSPECIFIED)...	16										38
9902 POXVIRUS GROUP NOT TYPED.....											1
9992 ROSS RIVER VIRUS.....	6			1							16
9994 SMALL VIRUS (LIKE) PARTICLE...							3				
Total.....	147	147	4	13	6		65	34	4	8	156

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 1, 3, 84 to 14, 3, 84 ...

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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	Gen-ital	Endo/sal gland	RES	Muscle/joint	Con-genital	PUO	Fever/mal-aise	Other	SIDS
0101 ADENOVIRUS TYPE 1.....	1									
0103 ADENOVIRUS TYPE 3.....			1					1		
0108 ADENOVIRUS TYPE 8.....	1									
0110 ADENOVIRUS TYPE 10.....	1	1								
0137 ADENOVIRUS TYPE 37.....	1									
0201 INFLUENZA A VIRUS.....					1			1		
0303 PARAINFLUENZA VIRUS TYPE 3....								1		
0600 MYCOPLASMA PNEUMONIAE.....					1		7	4	1	
0902 COXSACKIEVIRUS B2.....								1		
1003 ECHOVIRUS TYPE 3.....								1		
1014 ECHOVIRUS TYPE 14.....									1	
1200 MUMPS VIRUS.....			4				1		1	
1300 HERPES VIRUS GROUP-NOT TYPED..		2								
1302 EPSTEIN-BARR VIRUS (EB VIRUS)..			8					1	1	
1303 VARICELLA-ZOSTER VIRUS.....				1						
1306 HERPES SIMPLEX TYPE 1.....		32		1				3	4	
1307 HERPES SIMPLEX TYPE 2.....		176							2	
1401 COXIELLA BURNETI.....							3			
1502 PICORNA VIRUS-NOT TYPED.....			1		2		1	3		
1513 COWPOX VIRUS.....		1								
1522 RUBELLA VIRUS.....								1	2	
1532 HEPATITIS B ANTIGEN.....									14	
1541 CHLAMYDIA A - C.TRACHOMATIS...		87						2		
1556 CMV - CYTOMEGALOVIRUS.....	1	2	2	2		3	4	10	9	
9901 ARBO. GROUP A.(UNSPECIFIED)...					30			4		
9992 ROSS RIVER VIRUS.....					50			3		
Total.....	5	301	16	4	84	3	16	36	35	