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Communicable Diseases Intelligence

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Contents:

VIRUS REPORTING SCHEME - A total of 1368 reports were received this period. The three dengue cases reported by the State Health Laboratory, Brisbane, comprised one imported case in a 29 year old male who had returned recently from Tahiti, and two indigenous cases in patients residing in south Queensland. IgM antibody against dengue was detected in a 73 year old female from Rolleston (about 200 km south east of Rockhampton) and in a 66 year old male with no history of travel outside Kingaroy (about 150 km north east of Brisbane). Extensive cross reactivity prevented accurate serotyping of the infecting viruses. All previous indigenous dengue cases have been reported from northern Queensland.

FLOOD DISASTERS AND IMMUNISATION

(Based on California Morbidity (1983) #9).

The heavy rains and floods in many areas of Queensland and New South Wales this year have disrupted some community water supplies and sewerage systems. Such natural disasters often result in public concern about possible contagion from flood waters due to contamination by sewerage or animal carcasses.

Studies of flood and earthquake disasters have shown that communicable disease outbreaks rarely result from such events. Nevertheless, there is often public demand for emergency mass immunisation, especially against typhoid fever. However, epidemic typhoid has been conspicuously absent following natural disasters. Moreover it takes several weeks for typhoid antibodies to develop and, even then, immunisation provides only moderate protection against the disease. Mass tetanus immunisation programmes are also not indicated as floods, *per se*, pose no additional risk of tetanus. Management of flood-associated wounds should include appropriate evaluation of tetanus immunity (and immunisation if indicated) as at any other time.

Of greatest importance in preventing enteric disease transmission when water and sewerage systems have been compromised is to assure that water and food supplies are safe to consume. Flood victims and relief workers should be careful to wash their hands with soap and water (boiled or disinfected if no regular safe supply is available) before eating or preparing any foods, after toilet use, and after participating in flood cleanup or handling of potentially contaminated articles.

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While communicable disease outbreaks in industrialised countries are rare after flooding, some potential does exist for waterborne disease transmission (e.g. enterotoxigenic E. coli, shigella, salmonella, hepatitis A, Norwalk virus agents, leptospirosis); as flood affected communities should be kept under close surveillance.

Mass immunisation programmes at the time of natural disaster are counterproductive and divert limited manpower and resources from more important relief tasks. Such immunisation programmes may also give the public a false sense of security, leading to neglect of the basic rules of hygiene and sanitation that are far more important than immunisations in preventing the infectious diseases that may be spread by flood waters.

ACQUIRED IMMUNE DEFICIENCY SYNDROME (AIDS) - VICTORIA

(Contributed by C.N. Chesterman, St. Vincent's Hospital, Melbourne).

In August 1982, a 26 year old male presented to St. Vincent's Hospital, Melbourne, with a two week history of painless lumps in the neck, axillae and groin, and vague aches in the back, legs and shoulders. He also complained of a mildly itchy rash around the perineum which had persisted for two years. He gave histories of a pyrexia of unknown origin in May, and a varicella-zoster eruption on the right thoracic wall in June. The patient was an active homosexual with 30-40 partners per year, and who had sometimes inhaled amyl nitrite. He had returned recently from a holiday in Hawaii and California.

Physical examination revealed an unexplained generalised lymphadenopathy (rubbery, non-tender nodes, 1-2 cm diameter). The lymphadenopathy has persisted with little change. Biopsy of an enlarged axillary lymph node revealed non-diagnostic reactive hyperplasia. Rectal examination showed slight excoriation around the anus, perineum and scrotum. Perineum swabs were positive for Trichophyton rubrum, and dermatophytic infection was also evident on the toes and toenails. An examination two weeks later revealed a small (0.5-1.0 mm), raised, yellowish lesion in the upper gingival margin which had persisted but fluctuated in size, and discharged purulent material on pressure. However, swabs of the lesion were negative under dark ground microscopy and in bacterial and virus culture.

Laboratory examination indicated the following; Hb-15.7 g/dl, WBC - 8.1 x 10^9/litre (62% neutrophils, 30% lymphocytes, 5% monocytes, 1% eosinophils), platelets - normal level, ESR - 2 mm/hour. A chest X-ray was normal, as were all tests for liver function, electrolyte balance and urea. Serology performed to date gave a CF titre of 1/80 against cytomegalovirus (CMV), but no reactivity against Toxoplasma or hepatitis B virus antigens. Skin testing to recall antigens Trichophyton/Candida, streptokinase/streptodornase (SKSD), and tuberculosis (PPD) were negative, but positive (8 mm diameter) to mumps. The FTA and TPHA serological tests were positive subsequent to treatment for secondary syphilis in 1977, but the TPI was negative. The basic humoral immune profile was normal, but lymphocyte subpopulation studies revealed normal total T cell numbers but a low T helper/T suppressor cell ratio (OKT4/OKT8); 0.78 in August 1982, and 0.56 in February 1983.

Further microbiological and serological studies have failed to incriminate any specific infectious agent, and although the patient has remained well, he is being followed for periodical review.

Editorial Comment

The Centers for Disease Control (CDC) define a case of AIDS as a disease, at least moderately predictive of a defect in a cell-mediated immunity, occurring in a person with no known cause for diminished resistance to that disease (e.g. Kaposi's sarcoma, Pneumocystis carinii and serious opportunistic infections)[1]. However, the full spectrum of AIDS manifestations may range from absence of symptoms, despite laboratory evidence of immune deficiency, to non-specific symptoms (e.g. fever, weight loss, generalised persistent lymphadenopathy)[2] to specific diseases that are insufficiently predictive of cellular immunodeficiency (e.g. tuberculosis, oral candidiasis, varicella-zoster) to malignant neoplasms that cause, as well as result from, immunodeficiency[3].

Five cases of confirmed (one by CDC) and suspect AIDS have been reported in Australia to date (3 from New South Wales; 2 from Victoria). All cases are in male homosexuals with a history of travel to the USA, presenting with the lymphadenopathy syndrome and a reversed helper/suppressor T-cell ratio.

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ACQUIRED IMMUNE DEFICIENCY SYNDROME (AIDS) - UPDATE

(By J.R. Lake, Commonwealth Department of Health)

From June 1981 to 7 April 1983, 1300 cases of AIDS with an overall case fatality rate of 37.6% have been reported to CDC, Atlanta[1]. Current epidemiological evidence has identified several groups at increased risk of developing AIDS; homosexual or bisexual men with multiple sexual partners (933 cases, 35% fatality), abusers of intravenous (IV) drugs (217 cases, 40%), and Haitians, especially those who entered the USA within the past few years (64 cases, 55%). However, each group contains many persons who probably have little risk of acquiring AIDS. Eleven cases of AIDS (73%) have also been diagnosed in patients with haemophilia. In May 1983, AIDS was also confirmed in a patient with haemophilia in the UK[2]. In addition, 20 cases (50%) of unexplained cellular immunodeficiencies and opportunistic infections in infants born to mothers from groups at high risk for AIDS are under investigation, as well as 75 other cases (43%) comprising patients who have had blood tranfusions, women who were steady sexual partners of men with AIDS, and 36 seemingly risk-free heterosexual men. To date, no person-to-person transmission has been identified other than through intimate contact or blood transfusion[3].

The major feature of the immune deficiency in AIDS is a reversal of the ratio of helper (Leu-3 (OKT4)) to suppressor (Leu-2 (OKT8)) cytotoxic T cells, with a decreased number of Leu-2 cells and, to a greater extent, of Leu-3 cells[4]. Lowered Leu-3/Leu-2 ratios have also been reported recently in homosexual men who have only chronic lymphadenopathy as well as in healthy homosexuals with no symptoms or signs of disease[5,6]. However, these altered ratios in the majority of healthy homosexuals have been shown to be due to an increase, and not a decrease in Leu-2 T cells, so that this acquired immune augmentation may not be a precursor of clinical AIDS. Alternatively, the development of the constellation labelled as AIDS may be a two-stage process, possibly with different risk factors for stage 1 (enlargement of Leu-2 cell population) and stage 2 (reduction in Leu-2 and Leu-3 cell populations with concurrent Kaposi's sarcoma or opportunistic infection). Therefore, care must be taken to distinguish between a changed T-subset ratio due to augmented Leu-2 cells (>1000 per mm^3) and a changed ratio due to deficient Leu-3 cells (<350 per mm^3)[7].

Alterations in lymphocyte populations are also common in haemophilia[8,9,10], and again may not be causally related to AIDS. Thus the recognition of AIDS in a few haemophiliacs may not reflect the tip of an iceberg[11]. In Australia, all blood and blood products are processed from Australian volunteer donors, and no blood products are imported from overseas.

A great deal more needs to be known about the interactions between these various lymphocyte subsets, as well as the cells required for antigen presentation to the T cell classes. Treatment with thymosin, a thymus extract which enhances lymphocytopoiesis and restoring immunological competence in certain situations, has only restored temporarily the helper:supressor T cell ratio[12], suggesting that the defective T cells are under the influence of some persistent agent or combination of chemical and microbial factors that have a persistent and irreversible effect. One study has even indicated consistently elevated serum thymosin α_1 in two male homosexuals with Kaposi's sarcoma and an inverted helper/suppressor T cell ratio[13].

The questions of the aetiology of AIDS, its contemporaneous emergence, and its effect on lymphocyte populations have remain unanswered. If there is a microbial aetiology, and if it is present in patients' body fluids particularly their blood, due care must be exercised in handling material from AIDS patients[14].

Pathological studies of thymus material from patients with a fulminant, lethal evolution of AIDS have shown morphological abnormalities that include a depletion of thymocytes, a lack of a definitive cortex and medulla, scattered plasma cells and mast cells, and an absence of Hassall's corpuscles and clusters of epithelium[15]. The induction of this thymic dysplasia

through injury to thymic epithelium may represent a critical event in relation to the immunological abnormalities. Electron microscopy of lymphoid and other tissues derived from AIDS patients have also shown ultrastructural abnormalities[16,17]. Two types of cytoplasmic inclusions have been detected. Tubuloreticular structure (TRS) consisting of anastomosing branching tubules located within cisternae of endoplasmic reticulum have been detected in several cell types. However, they are also present in lymphocytes and endothelial cells of patients with connective-tissue disease (e.g. systemic lupus erythematosus), in numerous cancers and a large variety of virus infections[18,19]. The second structures are found primarily in lymphocytes, and are termed "test-tube and ring-shaped forms" (TRF) or "vesicular rosettes". These structures have also been described in a Japanese patient with adult T cell leukaemia[20], a cancer that has been linked with human T cell leukaemia virus (HTLV)[21]. Vesicular rosettes occur in B cells, but further cell-surface marker studies are needed to determine whether they occur within T cells. Since no virus particles have been detected in rosette-positive specimens, their relation to possible viral infection is obscure. Although the rosettes may be artifacts of the degenerative process[22], they may also be unique to AIDS and associated conditions.

Epidemiological studies strongly suggest that AIDS is caused by an infectious agent, but efforts to identify it have proved frustrating. Although a single transmissible agent may induce the basic immunological defect, the ultimate expression of AIDS may depend on a cascade of other variables such as nutritional status, other infections or chronic illnesses, environmental agents or genetic predisposition. The diagnosis of the AIDS-like disorder in only one child in a set of identical twins weakens the case for genetic susceptibility, but there is a high incidence of HLA-DR5 antigens in affected individuals[23]. Patients with AIDS have increased titres of antibodies to cytomegalovirus (CMV) and to Epstein-Barr virus (EBV), and a higher prevalence of antibodies to hepatitis B virus and Treponema pallidum. CMV, which was previously considered as a potential cause of the African form of Kaposi's sarcoma[24], has been seriously viewed as a candidate agent because of its immunosuppressive effects[25] and its ubiquity in male homosexuals[26,27]. Contrastingly, one study of seven children with AIDS-like presentations revealed that five of the children and three of their mothers had evidence of a persistent EBV virus infection[28], leading to the speculation that the infectious immunodeficiency was induced by perinatal or in utero transmission of EBV.

The USA Haitian immigrants, which constitute the third largest group of AIDS patients, pose the major puzzle for this syndrome, since most deny both homosexual practices and drug use, and they have not been exposed to clotting factor preparations. Accordingly, research has also been directed to the Caribbean area and equatorial Africa as sources of the

putative AIDS agent. Five cases of AIDS were reported recently in black African immigrants (from Zaire and Chad), two of whom had severe herpes simplex infections[29]. Investigators at the Harvard University School of Public Health, the National Cancer Institute and the Pasteur Institute have now found evidence of HTLV infection in patients with AIDS or at high risk of developing the syndrome[30].

HTLV agents are retroviruses that have recently been associated with certain types of adult T cell lymphoreticular neoplasms of man[31]. There are two types of HTLV; HTLV-1 has been associated with acute T cell leukaemia, and a related, but clearly different, virus HTLV-2 has been isolated from the cells of a patient with hairy cell leukaemia. Retroviruses are RNA viruses containing the enzyme reverse transcriptase, which allows the production of a DNA copy of their RNA genome. This DNA copy can be integrated into the host cell genome. Infections with retroviruses other than HTLV have been associated with a variety of neoplastic diseases in animals including chickens, cats, cattle and gibbons. The feline retrovirus also causes both cell-mediated and humoral immune suppression[32]. HTLV agents are the only presently known retroviruses associated with human diseases. Two regions of the world where both the virus and T cell malignancies occur at increased rates are southern Japan[33] (although AIDS has not been reported in Japan) and the Caribbean islands[34]. The diseases previously associated with HTLV in these endemic areas do not clinically resemble AIDS, and infections are thought rarely to result in malignancies. However, HTLV may spread from some infected persons to their very close contacts[34], and concern has been expressed that it may be transmissible by blood or blood derivatives[35].

HTLV has been isolated from peripheral blood T lymphocytes from several patients with AIDS[30,36]. A retrovirus, related to but clearly distinct from HTLV, has also been isolated in France from cells from a lymph node of a Caucasian homosexual with lymphadenopathy[37]. In addition, antibodies to antigens expressed on the cell surface of HTLV-infected lymphocytes have been detected by an indirect immunofluorescent technique in sera from 19(25%) of 75 AIDS patients, including patients with Kaposi's sarcoma alone (10 of 34), *P. carinii* pneumonia alone (7 of 30) or patients with both diseases (2 of 11)[38]. Similar antibodies were detected in six (26%) of 23 patients with lymphadenopathy. Such antibodies were only found in two of 336 homosexual and blood donor control subjects. Also HTLV nucleic acid sequences have been detected by Southern-blot hydridisation with radiolabelled cloned HTLV DNA probes in lymphocytes of two (6%) of 33 AIDS patients[39].

The relatively low frequency of antibody in AIDS persons might represent a lack of test sensitivity; too stringent criteria for positive tests; infection of AIDS patients with an agent related to, but not identical with, HTLV; non-specific polyclonal B-cell responses; inability of many AIDS patients to

mount antibody responses to these antigens; collection of sera from patients at improper times during disease evolution; or combinations of these and other yet-to-be identified factors. The reported low frequency of detecting HTLV sequences may reflect depletion of infected T-helper lymphocytes, since patients initially positive for such sequences have had negative tests several months later. Alternatively, HTLV or a HTLV-like agent might simply represent yet another opportunistic agent in these multiply infected AIDS patients. Alternatively, HTLV may interact with yet unknown endogenous lymphotropic agents whose expressions depend on co-infection with HTLV (c.f. Delta virus co-infection with hepatitis B virus[40]). However, since HTLV can infect marmoset and other primate T cells, and should this virus be established as the causative agent of AIDS, it may be possible to develop an animal model of the disease. A syndrome closely resembling AIDS has already been identified in a group of rhesus monkeys in California[41].

As long as the cause remains unknown, the ability to understand the natural history of AIDS and to undertake preventive measures is somewhat compromised. The lack of a specific test to detect AIDS at an early stage have made many public health agencies recommend the discouragement of blood donation by all members of groups at increased risk for AIDS, even though it includes many individuals who may be at little risk of transmitting AIDS. The abandonment of promiscuity, homosexuality and drug abuse, could eventually have a profound effect on the elimination of AIDS, but it is unlikely that these solutions would be accepted.

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AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 12/5/83 - 25/5/83 BULLETIN NUMBER . 83/11

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	4				1	5		11
0101 ADENOVIRUS TYPE 1	1								1
0102 ADENOVIRUS TYPE 2				1	5	3			9
0104 ADENOVIRUS TYPE 4						1			1
0105 ADENOVIRUS TYPE 5	2					2			4
0108 ADENOVIRUS TYPE 8				4				1	5
0110 ADENOVIRUS TYPE 10								1	1
0119 ADENOVIRUS TYPE 19				2				17	19
0199 ADENOVIRUS TYPING PENDING			5		6				11
0201 INFLUENZA A VIRUS								2	2
0203 INFLUENZA B VIRUS	1								1
0301 PARAINFLUENZA VIRUS TYPE 1				2	1	7	2		12
0302 PARAINFLUENZA VIRUS TYPE 2							1	1	2
0303 PARAINFLUENZA VIRUS TYPE 3	1	2			1	3		1	8
0399 PARAINFLUENZA VIRUS TYPING PENDING.						7			7
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	1	12	1		7	13	49		83
0500 RHINOVIRUS (ALL TYPES)			1	6	10	17	3		37
0600 MYCOPLASMA PNEUMONIAE	23				2	4	5	4	38
0816 COXSACKIEVIRUS A16							2		2
0899 COXSACKIEVIRUS GROUP A TYPING PENDING							3		3
0903 COXSACKIEVIRUS B3	1		1			2		5	9
0904 COXSACKIEVIRUS B4			1						1
0905 COXSACKIEVIRUS B5			2						2
1006 ECHOVIRUS TYPE 6				1					1
1007 ECHOVIRUS TYPE 7								2	2
1011 ECHOVIRUS TYPE 11	24	1	10	10		1	1		47
1018 ECHOVIRUS TYPE 18				2					2
1022 ECHOVIRUS TYPE 22			1					1	2
1099 ECHOVIRUS TYPING PENDING			1						1
1101 POLIOVIRUS TYPE 1	1								1
1102 POLIOVIRUS TYPE 2			1			2			3
1103 POLIOVIRUS TYPE 3			1						1
1200 MUMPS VIRUS	4			1	1		2		8
1300 HERPES VIRUS GROUP-NOT TYPED	31			4	1	9			45
1301 HERPES SIMPLEX VIRUS NOT-TYPED				1		2	1	8	12
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	5	2						4	11
1303 VARICELLA-ZOSTER VIRUS	2					1			3
1306 HERPES SIMPLEX TYPE 1	9		10	9		14	23	17	82
1307 HERPES SIMPLEX TYPE 2	121		24	57		28	69	37	336
1399 HERPES VIRUS TYPING PENDING			12		2	4			18
1401 COXIELLA BURNETI	2			1			12	1	16
1502 PICORNA VIRUS-NOT TYPED	6							2	8
1521 MEASLES VIRUS	4	1							5
1522 RUBELLA VIRUS				1			1	1	3
1532 HEPATITIS B ANTIGEN	34		7	33		9	5	15	103
1535 HEPATITIS A ANTIBODY	6		3	11		7		23	50
1541 CHLAMYDIA A - C TRACHOMATIS	26					2	29	75	132
1556 CMV - CYTOMEGALOVIRUS	14		2	7	6	2	9	11	51
1563 CORONAVIRUS				1					1
1564 ROTAVIRUS		11	4			20		2	37
1571 ENTEROVIRUS TYPE 71 (BRCR)	1	1		1					3
1599 ENTEROVIRUS TYPING PENDING			5		10	4			19
POXVIRUS GROUP NOT TYPED				1					1
ROSS RIVER VIRUS	1						77	3	81
SMALL VIRUS (LIKE) PARTICLE	6			2					8
DENGUE							3		3
PARAMYXOVIRUS						2			2
KUNJIN VIRUS							1		1
Total	328	34	92	158	52	167	303	234	1,368

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
0101 ADENOVIRUS TYPE 1.....	1										
0102 ADENOVIRUS TYPE 2.....		6		1			2				
0104 ADENOVIRUS TYPE 4.....							1				
0105 ADENOVIRUS TYPE 5.....		2					1				
0201 INFLUENZA A VIRUS.....		1									
0203 INFLUENZA B VIRUS.....	1										
0301 PARAINFLUENZA VIRUS TYPE 1....		13									
0302 PARAINFLUENZA VIRUS TYPE 2....		2									
0303 PARAINFLUENZA VIRUS TYPE 3....		8									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	1	82									
0500 RHINOVIRUS (ALL TYPES).....		34		3							
0600 MYCOPLASMA PNEUMONIAE.....	10	25						1			2
0816 COXSACKIEVIRUS A16.....											2
0903 COXSACKIEVIRUS B3.....		2		1		2	3				
0904 COXSACKIEVIRUS B4.....							1				
0905 COXSACKIEVIRUS B5.....		1					1				
1007 ECHOVIRUS TYPE 7.....							1				
1011 ECHOVIRUS TYPE 11.....		10		18			10				1
1018 ECHOVIRUS TYPE 18.....		1		1							
1022 ECHOVIRUS TYPE 22.....							2				
1101 POLIOVIRUS TYPE 1.....		1									
1102 POLIOVIRUS TYPE 2.....							3				
1103 POLIOVIRUS TYPE 3.....							1				
1200 MUMPS VIRUS.....	4		1	2							
1300 HERPES VIRUS GROUP-NOT TYPED..	1										1
1301 HERPES SIMPLEX VIRUS NOT-TYPED	3	1								1	5
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	2							1			
1303 VARICELLA-ZOSTER VIRUS.....											3
1306 HERPES SIMPLEX TYPE 1.....	3	3					1		1		42
1307 HERPES SIMPLEX TYPE 2.....	9										37
1401 COXIELLA BURNETI.....	2	5									
1502 PICORNA VIRUS-NOT TYPED.....	1		1								
1521 MEASLES VIRUS.....	1	1	1	1							2
1522 RUBELLA VIRUS.....											3
1532 HEPATITIS B ANTIGEN.....	46						1	41		1	
1535 HEPATITIS A ANTIBODY.....	4				1			44			
1541 CHLAMYDIA A - C.TRACHOMATIS...		2									
1556 CMV - CYTOMEGALOVIRUS.....	6	16		1		5		1	1		
1563 CORONAVIRUS.....							1				
1564 ROTAVIRUS.....	2						34				
1571 ENTEROVIRUS TYPE 71 (BRCR)....	2										1
9902 POXVIRUS GROUP NOT TYPED.....											1
9992 ROSS RIVER VIRUS.....	6	3									30
9994 SMALL VIRUS (LIKE) PARTICLE...							8				
9995 DENGUE.....	1		1								
9996 PARAMYXOVIRUS.....		1									
Total.....	106	220	4	28	1	7	71	88	2	2	130

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
0105 ADENOVIRUS TYPE 5.....	1									
0108 ADENOVIRUS TYPE 8.....	4	1								
0110 ADENOVIRUS TYPE 10.....	1									
0119 ADENOVIRUS TYPE 19.....	7	13								
0201 INFLUENZA A VIRUS.....								1		
0301 PARAINFLUENZA VIRUS TYPE 1....								2		
0303 PARAINFLUENZA VIRUS TYPE 3....							1			
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....								1		
0500 RHINOVIRUS (ALL TYPES).....										1
0600 MYCOPLASMA PNEUMONIAE.....				1				2		
0903 COXSACKIEVIRUS B3.....					1					
1006 ECHOVIRUS TYPE 6.....								1		
1007 ECHOVIRUS TYPE 7.....		1								
1011 ECHOVIRUS TYPE 11.....							6	3	1	
1018 ECHOVIRUS TYPE 18.....								1		
1022 ECHOVIRUS TYPE 22.....							1			
1200 MUMPS VIRUS.....			1							
1301 HERPES SIMPLEX VIRUS NOT-TYPED		2								
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			3	2			1		3	
1306 HERPES SIMPLEX TYPE 1.....	3	29						2	2	
1307 HERPES SIMPLEX TYPE 2.....		295								
1401 COXIELLA BURNETI.....					1			13		
1521 MEASLES VIRUS.....							1			
1522 RUBELLA VIRUS.....								1		
1532 HEPATITIS B ANTIGEN.....		1						1	12	
1535 HEPATITIS A ANTIBODY.....				1	1				1	
1541 CHLAMYDIA A - C.TRACHOMATIS...	3	129								
1556 CMV - CYTOMEGALOVIRUS.....	1	10		1			3	3	5	
1564 ROTAVIRUS.....								1		
9992 ROSS RIVER VIRUS.....			1		67		1	15		
9995 DENGUE.....								2		
9997 KUNJIN VIRUS.....					1			1		
Total.....	20	481	5	5	71		14	50	24	1