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Contents:

- Paediatric classification system for HIV infection.
- Whooping cough.
- Non-group A rotaviruses (NGAR) novel agents of adult gastroenteritis.
- Infant botulism - 3 cases in a small town (USA).
- Infant botulism - California's experience '85 and '86.

VIRUS REPORTING SCHEME: A total of 1 375 reports were processed for this period.

19 cases of Q fever were reported, 1 from South Australia, 9 from Queensland and 9 from New South Wales. Occupational exposure data were only available for the South Australian case, a 29 year old male feral goat handler. None of these 19 patients was involved in the Q fever vaccine field trial conducted in South Australia.

Adenovirus, untyped, was isolated from the nasal aspirate of a 4 year old female with acute lymphoblastic leukaemia (ALL), who had atypical pneumonia.

Cytomegalovirus was isolated from the post mortem lung tissue of a 9 year old female who died from a severe 'viral illness' following bone marrow transplantation.

Herpes simplex virus type 1 was isolated from the saliva of a 25 year old, human immunodeficiency virus (HIV) antibody positive male, who presented with cryptococcal meningitis and a concurrent pneumonia.

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CLASSIFICATION SYSTEM FOR HUMAN IMMUNODEFICIENCY VIRUS (HIV) INFECTION IN CHILDREN UNDER 13 YEARS OF AGE
(Based on MMWR Vol. 36, No. 15, 24 April 1987)

INTRODUCTION

With the identification of the causative agent of the acquired immunodeficiency syndrome (AIDS), a broad spectrum of clinical manifestations has been attributed to infection with the human immunodeficiency virus (HIV). With the exception of the Centers for Disease Control (CDC) surveillance definition for AIDS, no standard definitions for other manifestations of HIV infection have been developed for children. Classification systems published to date have been developed primarily to categorise clinical presentations in adult patients and may not be entirely applicable to infants and children.

Physicians from institutions caring for relatively large numbers of HIV-infected children report that only about half of their patients with symptomatic illness related to the infection fulfil the criteria of the CDC surveillance definition for AIDS.

To develop a classification system for HIV infection in children, CDC convened a panel of consultants consisting of clinicians experienced in the diagnosis and management of children with HIV infection; public health physicians, representatives from the American Academy of Paediatrics, the Council of State and Territorial Epidemiologists, the Association for Maternal Child Health and Crippled Children's Programs, the National Institute on Drug Abuse/Alcohol, Drug Abuse and Mental Health Administration, the National Institute of Allergy and Infectious Diseases/National Institutes of Health, and the Division of Maternal and Child Health/Health Resources and Services Administration, and CDC.

GOALS AND OBJECTIVES OF THE CLASSIFICATION SYSTEM

The system was designed primarily for public health purposes, including epidemiologic studies, disease surveillance, prevention programs, and health-care planning and policy. The panel attempted to devise a simple scheme that could be subdivided as needed for different purposes.

DEFINITION OF HIV INFECTION IN CHILDREN (Table 1)

Ideally, HIV infection in children is identified by the presence of the virus in blood or tissues, confirmed by culture or other laboratory detection methods. However, current tests - including culture - for detecting the virus or its antigens are not standardised and are not readily available. Detection of specific antibody to the virus is a sensitive and specific

indicator of HIV infection in adults, since the majority of adults with antibody have had culture evidence of infection. Similar studies involving children have not been reported. Also, the presence of passively transferred maternal antibody in infants limits the interpretation of a positive antibody test result in this age group. Most of the consultants believed that passively transferred maternal HIV antibody could sometimes persist for up to 15 months. For this reason, two definitions for infection in children are needed: one for infants and children up to 15 months of age who have been exposed to their infected mothers perinatally, and another for older children with perinatal infection and for infants and children of all ages acquiring the virus through other means.

Infants and children under 15 months of age with perinatal infection -
Infection in infants and children up to 15 months of age who were exposed to infected mothers in the perinatal period may be defined by one or more of the following:

1) the identification of the virus in blood or tissues,
2) the presence of HIV antibody as indicated by a repeatedly reactive screening test (eg enzyme immunoassay) plus a positive confirmatory test (eg Western blot, immunofluorescence assay) in an infant or child who has abnormal immunologic test results indicating both humoral and cellular immunodeficiency (increased immunoglobulin levels, depressed T4 [T-helper] absolute cell count, absolute lymphopenia, decreased T4/T8 ratio) and who meets the requirements of one or more of the subclasses listed under class P-2 (described below), or
3) the confirmation that a child's symptoms meet the previously published CDC case definition for paediatric AIDS.

The infection status of other perinatally exposed seropositive infants and children up to 15 months of age who lack one of the above immunologic or clinical criteria is indeterminate. These infants should be followed up for HIV-related illness, and they should be tested at regular intervals for persistence of antibody to HIV. Infants and children who become seronegative are virus-culture negative (if blood or tissue samples are cultured), and continue to have no clinical or laboratory-confirmed abnormalities associated with HIV infection are unlikely to be infected.

Older children with perinatal infection and children with HIV infection acquired through other modes of transmission
- HIV infection in these children is defined by one or more of the following:

1) the identification of virus in blood or tissues,
2) the presence of HIV antibody (positive screening test plus confirmatory test) regardless of whether immunologic abnormalities or signs or symptoms are present, or
3) the confirmation that the child's symptoms meet the previously published CDC case definition for paediatric AIDS.

These definitions apply to children under 13 years of age. Persons 13 years of age and older should be classified according to the adult classification system.

TABLE 1. **Summary of the definition of HIV infection in children**

Infants and children under 15 months of age with perinatal infection

1) Virus in blood or tissues
or

2) HIV antibody and evidence of both cellular and humoral immune deficiency and one or more categories in Class P-2
or

3) Symptoms meeting CDC case definition for AIDS

Older children with perinatal infection and children with HIV infection acquired through other modes of transmission

1) Virus in blood or tissues
or

2) HIV antibody
or

3) Symptoms meeting CDC case definition for AIDS

CLASSIFICATION SYSTEM (Table 2)

Children fulfilling the definition of HIV infection discussed above may be classified into one of two mutually exclusive classes based on the presence or absence of clinical signs and symptoms (Table 2). Class Paediatric-1 (P-1) is further subcategorised on the basis of the presence or absence of immunologic abnormalities, whereas Class P-2 is subdivided by specific disease patterns. Once a child has signs and symptoms and is therefore classified in P-2, he or she should not be reassigned to class P-1 if signs and symptoms resolve.

Perinatally exposed infants and children whose infection status is indeterminate are classified into class P-0.

Class P-0. Indeterminate infection.

Includes perinatally exposed infants and children up to 15 months of age who cannot be classified as definitely infected according to the above definition but who have antibody to HIV, indicating exposure to a mother who is infected.

Class P-1. Asymptomatic infection.

Includes patients who meet one of the above definitions for HIV infection but who have had no previous signs or symptoms that would have led to classification in Class P-2.

These children may be subclassified on the basis of immunologic testing. This testing should include quantitative immunoglobulins, complete blood count with differential, and T-lymphocyte subset quantitation. Results of functional testing of lymphocytes (mitogens, such as pokeweed) may also be abnormal in HIV-infected children, but it is less specific in comparison with immunoglobulin levels and lymphocyte subset analysis, and it may be impractical.

Subclass A - Normal immune function.
Includes children with no immune abnormalities associated with HIV infection.

Subclass B - Abnormal immune function.
Includes children with one or more of the commonly observed immune abnormalities associated with HIV infection, such as hypergammaglobulinemia, T-helper (T4) lymphopenia, decreased T-helper/T-suppressor (T4/T8) ratio, and absolute lymphopenia. Other causes of these abnormalities must be excluded.

Subclass C - Not tested.
Includes children for whom no or incomplete (see above) immunologic testing has been done.

Class P-2. Symptomatic infection. Includes patients meeting the above definitions for HIV infection and having signs and symptoms of infection. Other causes of these signs and symptoms should be excluded. Subclasses are defined based on the type of signs and symptoms that are present. Patients may be classified in more than one subclass.

Subclass A - Nonspecific findings.
Includes children with two or more unexplained non-specific findings persisting for more than 2 months, including fever, failure-to-thrive or weight loss of more than 10% of baseline, hepatomegaly, splenomegaly, generalised lymphadenopathy (lymph nodes measuring at least 0.5 cm present in two or more sites, with bilateral lymph nodes counting as one site), parotitis, and diarrhoea (three or more loose stools per day) that is either persistent or recurrent (defined as two or more episodes of diarrhoea accompanied by dehydration within a 2-month period).

Subclass B - Progressive neurologic disease.
Includes children with one or more of the following progressive findings:

1) loss of developmental milestones or intellectual ability,
2) impaired brain growth (acquired microcephaly and/or brain atrophy demonstrated on computerized tomographic scan or magnetic resonance imaging

scan), or
3) progressive symmetrical motor deficits manifested by two or more of these findings: paresis, abnormal tone, pathologic reflexes, ataxia, or gait disturbance.

Subclass C - Lymphoid interstitial pneumonitis.
Includes children with a histologically confirmed pneumonitis characterised by diffuse interstitial and peribronchiolar infiltration of lymphocytes and plasma cells and without identifiable pathogens, or, in the absence of a histologic diagnosis, a chronic pneumonitis - characterized by bilateral reticulonodular interstitial infiltrates with or without hilar lymphadenopathy - present on chest X-ray for a period of at least 2 months and unresponsive to appropriate antimicrobial therapy. Other causes of interstitial infiltrates should be excluded, such as tuberculosis, Pneumocystis carinii pneumonia, cytomegalovirus infection, or other viral or parasitic infections.

Subclass D - Secondary infectious diseases.
Includes children with a diagnosis of an infectious disease that occurs as a result of immune deficiency caused by infection with HIV.

Category D-1. Includes patients with secondary infectious disease due to one of the specified infectious diseases listed in the CDC surveillance definition for AIDS: Pneumocystis carinii pneumonia; chronic cryptosporidiosis; disseminated toxoplasmosis with onset after 1 month of age; extra-intestinal strongyloidiasis; chronic isosporiasis; candidiasis (oesophageal, bronchial, or pulmonary); extrapulmonary cryptococcosis; disseminated histoplasmosis; noncutaneous, extrapulmonary, or disseminated mycobacterial infection (any species other than leprae); cytomegalovirus infection with onset after 1 month of age; chronic mucocutaneous or disseminated herpes simplex virus infection with onset after 1 month of age; extrapulmonary or disseminated coccidioidomycosis; nocardiosis; and progressive multifocal leucoencephalopathy.

Category D-2. Includes patients with unexplained, recurrent, serious bacterial infections (two or more within a 2-year period) including sepsis, meningitis, pneumonia, abscess of an internal organ, and bone/joint infections.

Category D-3. Includes patients with other infectious diseases, including oral candidiasis persisting for 2 months or more, two or more episodes of herpes stomatitis within a year, or multidermatomal or disseminated herpes zoster infection.

Subclass E - Secondary cancers.
Includes children with any cancer described below in categories E-1 and E-2.

Category E-1. Includes patients with the diagnosis of one or more kinds of cancer known to be associated with HIV infection as listed in the surveillance definition of AIDS and indicative of a defect in cell-mediated immunity: Kaposi's sarcoma, B-cell non-Hodgkin's lymphoma, or primary lymphoma of the brain.

Category E-2. Includes patients with the diagnosis of other malignancies possibly associated with HIV infection.

Subclass F - Other diseases. Includes children with other conditions possibly due to HIV infection not listed in the above subclasses, such as hepatitis, cardiopathy, nephropathy, haematologic disorders (anaemia, thrombocytopenia), and dermatologic diseases.

TABLE 2. Summary of the classification of HIV infection in children under 13 years of age

Class P-0. Indeterminate infection

Class P-1. Asymptomatic infection

- Subclass A. Normal immune function
- Subclass B. Abnormal immune function
- Subclass C. Immune function not tested

Class P-2. Symptomatic infection

- Subclass A. Nonspecific findings
- Subclass B. Progressive neurologic disease
- Subclass C. Lymphoid interstitial pneumonitis
- Subclass D. Secondary infectious diseases
  - Category D-1. Specified secondary infectious diseases listed in the CDC surveillance definition for AIDS
  - Category D-2. Recurrent serious bacterial infections
  - Category D-3. Other specified secondary infectious diseases
- Subclass E. Secondary cancers
  - Category E-1. Specified secondary cancers listed in the CDC surveillance definition for AIDS
  - Category E-2. Other cancers possibly secondary to HIV infection
- Subclass F. Other diseases possibly due to HIV infection

Editorial Note:

This classification system is based on present knowledge and understanding of paediatric HIV infection and may need to be revised as new information becomes available. New diagnostic tests, particularly antigen detection tests and HIV-specific IgM tests, may lead to a better definition of HIV infection in infants and children. Information from several natural history studies currently under way may necessitate changes in the subclasses based on clinical signs and symptoms.

A definitive diagnosis of HIV infection in perinatally exposed infants and children under 15 months of age can be difficult. The infection status of these HIV-seropositive infants and children who are asymptomatic without immune abnormalities cannot be determined unless virus culture or other antigen-detection tests are positive. Negative virus cultures do not necessarily mean the child is not infected, since the sensitivity of the culture may be low. Decreasing antibody titres have been helpful in diagnosing other perinatal infections, such as toxoplasmosis and cytomegalovirus. However, the pattern of HIV-antibody production in infants is not well defined. At present, close follow-up of these children (Class P-0) for signs and symptoms indicative of HIV infection and/or persistence of HIV antibody is recommended.

The parents of children with HIV infection should be evaluated for HIV infection, particularly the mother. The child is often the first person in such families to become symptomatic. When HIV infection in a child is suspected, a careful history should be taken to elicit possible risk factors for the parents and the child. Appropriate laboratory tests, including HIV serology should be offered. If the mother is seropositive, other children should be evaluated regarding their risk of perinatally acquired infection. Intrafamilial transmission, other than perinatal or sexual, is extremely unlikely. Identification of other infected family members allows for appropriate medical care and prevention of transmission to sexual partners and future children.

The nonspecific term AIDS-related complex has been widely used to describe symptomatic HIV-infected children who do not meet the CDC case definition for AIDS. This classification system categorises these children more specifically under Class P-2.

The development and publication of this classification system does not imply any immediate change in the definition of paediatric AIDS used by CDC for reporting purposes. Changes in this definition require approval by State and local health departments. However, changes in the definition for reporting cases have been proposed by CDC and are awaiting State and local approval.

Written comments are encouraged. They should be mailed to the AIDS Program, Center for Infectious Diseases, Centers for Disease Control, Atlanta, GA 30333.

WHOOPING COUGH

Whooping cough is a protracted and distressing disease with particular risks to infants aged less than 6 months.

The prevalence and severity of the disease have declined this century due to the availability of effective whooping cough vaccines. However, publicity concerning the adverse effects of whooping cough vaccination has greatly reduced the uptake of immunisation in many countries.

In England in the 1982/83 epidemic there were over 65 000 natural pertussis cases with 14 deaths [1]. Vaccination against whooping cough has ranged from 78% in 1971 to 37% in 1974 to 65% in 1986 [2].

Whooping cough affected as many as 265 000 persons and killed 9 000 to 12 000 at its peak in the United States in the 1930s. By the mid 1980s there are some 1 000 to 2 000 cases with 5 to 20 deaths each year[3]. There is about a 90% vaccination rate among school children[4].

In Japan, following the deaths of 2 infants in 1974/75, pertussis vaccination was discontinued then reintroduced for older children. The rate of vaccination decreased from 60% to 10% while the number of cases increased with 13 105 cases and 41 deaths recorded in 1979 [5].

The Disease

Clinical illness begins 7 to 10 days after infection of the respiratory tract with Bordetella pertussis. Three stages can usually be recognised: 1) the highly infectious catarrhal stage resembling a minor respiratory illness. The cough, initially dry, increases in severity over time with greater frequency at night.

In the second or paroxysmal stage, of 1 to 2 months duration, the characteristic whoop is heard at the end of a series of coughs. In adults and very young infants the whoop may be absent. Cyanosis and apnoea may accompany bouts of coughing and vomiting after paroxysms of coughing appears to assist in dislodging tenacious mucous from the respiratory tract. A marked leukocytosis with an increase in lymphocytes characterises this stage.

The third or convalescent stage dates from the reduction in frequency and severity of coughing bouts and may last from 1 to several months.

Antibiotic Use

No effective treatment is available for whooping cough and young adults with a mild whooping cough infection can constitute a major reservoir of infection.

Antibiotic use has been recommended for children with whooping cough to reduce infectivity[6, 7]. This treatment does not necessarily prevent the spread of infection.

Seven days of erythromycin therapy failed to eradicate B pertussis from the pharynx of an infant and a second infant exposed to this child also developed the disease despite prophylactic use of erythromycin[8].

In a study [9] of 22 children at Royal Alexandra Hospital for Children in Sydney both erythromycin and co-trimoxazole appeared to be effective inhibiting agents against B pertussis. Children with culture positive B pertussis swabs were considered contagious even with antimicrobial treatment. However, the inhibition of growth without eradication of the organism suggests that a longer course of erythromycin is desirable.

There is no general agreement about chemotherapy for pertussis infection of index cases and exposed children under 1 year of age. However, a 14 day course of erythromycin therapy should be considered in such cases as it may suppress the organism until specific secretory IgA develops.

Vaccination

Vaccination is the most effective prophylaxis. Genuine contraindications are few and children up to the age of 6 years can be vaccinated if they have missed earlier immunisation[10].

There is no transfer of passive immunity to pertussis from the mother to the new born infant, and the pertussis vaccination is therefore ideally carried out in early infancy to give protection when most needed. Pertussis itself is not followed by long-standing immunity and pertussis vaccine is less effective than other vaccines used in childhood immunisation, providing about 80-90 per cent protection which wanes over several years. Pertussis vaccine is also responsible for most of the side effects which occur with diphtheria-tetanus-pertussis vaccine, including, very rarely, severe neurological damage. However the likelihood of severe neurological damage or death in unimmunised individuals is much greater than that from the vaccine[11].

Pertussis vaccine used in Australia is a suspension of killed Bordetella pertussis organisms in a mixed vaccine also containing diphtheria and tetanus vaccines. Acellular pertussis vaccines are being developed, with the possibility of fewer side-effects and greater effectiveness.

The National Health and Medical Research Council recommends immunisation at 2, 4, 6 and 18 months with Triple Antigen which protects against diphtheria and tetanus as well as pertussis.

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NON-GROUP A ROTAVIRUSES (NGAR), NOVEL AGENTS OF ADULT GASTROENTERITIS

(Based on California Morbidity #1, 16 January 1987)

Rotaviruses are enteric viruses known to cause the majority of nonbacterial gastroenteritis in infants and children worldwide. In temperate climates the virus displays a striking predilection, causing illness almost exclusively during the winter months. Transmission is most likely by the faecal-oral route and the incubation period ranges from 1 to 3 days. Symptoms generally subside within one week, and complications are extremely rare. Peak incidence occurs in infants and children 6 months to 2 years of age. At least 80% of children will demonstrate serologic evidence of past infection and immunity by 3 years of age, but sporadic adult reinfections have been reported.

In 1984, two unusual outbreaks of gastrointestinal illness among 12 000 adults living in two coal mining districts in Mainland China were reported[1,2]. The illness was characterised by severe cholera-like watery diarrhoea. Epidemiologic studies inplicated faecal contamination of water supplies as the source of infection. Bacteriologic and parasitic examinations of stools were negative but immunoelectron microscopy consistently revealed rotavirus-like particles in the stool samples. However, testing for rotavirus by other conventional means such as ELISA and complement-fixation serology or tissue culture yielded negative results. In addition, RNA genome segments from these new agents were found to be distinct from RNA of typical rotaviruses.

These novel agents are now thought to represent variant rotaviruses which are morphologically identical to typical rotaviruses but do not share any antigenic determinants. They have been termed non-group A rotaviruses (NGAR) to distinguish them from typical group A rotaviruses which are the agents of infantile gastroenteritis. NGAR have also been isolated from rats, pigs, chickens, lambs, cows and birds.

Clinical features of infection with NGAR are similar to those of typical rotavirus infection, except for increased severity of diarrhoeal symptoms. However, the full clinical spectrum of this new agent may not be completely apparent until further outbreaks are identified. Diagnosis of infection with NGAR can be considered after exclusion of bacterial, parasitic or other viral agents as causes of illness. NGAR particles can be visualised by electron microscopy if acute and convalescent sera are also available. Experimental ELISA kits for detection and antigenic characterisation of NGAR have been developed for research purposes.[3]

Preliminary seroepidemiologic surveys indicate that exposure to typical rotaviruses does not confer immunity to the newly discovered NGAR. Thus, adults and children alike are potentially susceptible to infection with this new agent. To date, however, the only large outbreaks of human illness with NGAR have been reported in China. Further epidemiologic studies of outbreaks of nonbacterial gastroenteritis will be necessary in order to determine the importance of this agent as a cause of gastroenteritis worldwide.

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INFANT BOTULISM (USA) - THREE CASES IN A SMALL TOWN

A cluster of three cases of infant botulism has been reported in a small Colorado town (population 800, 20-30 live births per year) over a six month period in 1981[1]. Clusters of infant botulism cases are rarely reported.

Infant botulism has been linked to ingestion of honey, and is commonly thought to be acquired from soil or house dust from which the aetiological agent, Clostridium botulinum, is often recovered. However, the source of most infections is unknown.

CASE HISTORIES

Patient 1: A male infant (aged 4.5 months) became irritable and began to refuse food about 8 June 1981. Oral amoxicillin therapy was commenced on 11 June following examination by a paediatrician who diagnosed only bilateral otitis media. On 12 June the child was admitted to hospital as he was lethargic, had a weak cry and a poor sucking response. On admission it was noted that the child had not had a bowel movement for 8 days, was breast fed and had not been given honey or corn syrup.

Neurological examination revealed the child had a generalised hypotonia, bilateral ptosis, loss of head control and extraocular muscle palsy. Deep tendon reflexes were symmetrical but diminished. Bilateral otitis media was noted. Results of a complete blood cell count (CBC), urinalysis and cerebrospinal fluid (CSF) analysis were normal. Normal values were obtained for serum urea nitrogen, electrolytes, creatine kinase and aspartate aminotransferase.

Blood, CSF and urine cultures yielded no bacteria. However, a stool specimen was positive for type A toxin of C botulinum (determined by Centers for Disease Control (CDC), Atlanta) and culture from the same specimen yielded the organism.

Supportive therapy, including intravenous and orogastive tube feeding was given and there was a gradual improvement in muscular weakness such that the child was able to be discharged on 10 July 1981. The parents continued orogastric feeding for one month at home and the child had a normal neurological examination four months after discharge.

Patient 2: A female infant (aged 8 months) was seen on 26 December 1981 after the parents noted that the child was lethargic and had a mild fever. Bilateral otitis media and generalised weakness was noted and oral amoxicillin therapy was commenced. CSF values, CBC, electrolyte and blood glucose values were all normal.

On 28 December 1981, the child had increasing weakness, difficulty in feeding, and was admitted to hospital. The infant was breast fed, supplemented with cow's milk-based formula and commercial baby foods. She had not received honey or corn syrup.

Physical examination on admission revealed generalized muscular weakness with bilateral ptosis, a weak cry, poor sucking response, poor head control and a disconjugate gaze. Deep tendon reflexes were normal and symmetrical. Bilateral otitis media was noted. CBC values were normal and results of triiodothyronine, thyroxine, and creatine kinase levels were also normal. Urine and CSF cultures were negative. Stool cultures were negative for enteric pathogens. A stool specimen was positive for the type A toxin of C botulinum (as determined by CDC, Atlanta) and the organism was cultured from the same sample.

The infant received supportive therapy including orogastric tube feeding. She was discharged after eight days with the parents being instructed to maintain orogastric tube feeding. Four days after discharge, the child apparently aspirated vomitus following a feed and had cardiopulmonary arrest. Resuscitation at home and in hospital were unsuccessful.

Patient 3: The patient, a five month old female infant, had difficulty with feeding two days prior to admission. The child was exclusively breast fed except for a few unsuccessful attempts at feeding with cereals. She had not been given honey or corn syrup. The child had had no bowel movements for three days prior to admission.

Physical examination on admission revealed the infant had generalised hypotonia with loss of head control. Deep tendon reflexes were symmetrical and normal. A stool specimen contained type A toxin of C botulinum and the organism was cultured from the specimen.

The infant was intubated shortly after admission and required mechanical ventilatory support for seven weeks. At discharge (eight weeks after admission) the child had residual generalised weakness, and required occasional nasogastric tube feedings. The child had a five month delay in motor development, but has since shown progressive improvement.

All three patients lived within 800m of each other, in trailer homes. All infants were breast fed and none had been given honey or corn syrup. There was no social contact between any of the three families. A common link between cases 1 and 2 was established in that they had used the same crib: case 1 used the crib until approximately one month after his illness. The crib was passed to another family who, in turn, passed it to the family of case 2 about two months later. Case 3 had no contact with the crib.

C botulinum producing type A toxin was cultured from the base of the crib. A culture of house dust from the home of patient 2 was negative, but a similar sample from the home of her grandmother proved positive. House dust from the home of patient 3 also yielded the organism. Four soil samples from 1982 and seven from 1985 all yielded C botulinum producing type A toxin.

The source of C botulinum in the three cases reported is not clear. Each infant may have aquired the infection from separate sources or from the environment.

Type A is the predominent type of C botulinum in the western United States and is frequently found in soil. The cluster of cases was not correlated with unusual climatic conditions, or with any excavation work in or near the town where the cases were exposed. Clusters such as the one reported suggest that as yet undefined environmental factors may play an important role in the occurrence of infant botulism.

The aetiology of most cases of sudden infant death syndrome (SIDS) is never defined. However, botulinum toxin was found in 9 of 70 children considered to have died with SIDS[2].

C botulinum may cause a SIDS-like presentation as well as the more slowly evolving neurological disorder seen in the children reported in this USA study.

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INFANT BOTULISM - CALIFORNIA'S EXPERIENCE - 1985/86
(Based on California Morbidity #5, 13 February 1987)

California continues to report approximately half the cases of infant botulism recognised in the United States. In the past two years, a total of 83 cases were identified in California, 43 cases in 1985 and 40 in 1986. Infants of all races were affected:-

- 47% are white;
- 42% are hispanic
- 8% are asian
- 1% are black, and
- 1% belong to other racial groups

The geographic distribution of the 83 cases was as follows:-

- 41 cases (49%) lived in the greater Los Angeles basin (Los Angeles, Orange, Riverside and Ventura Counties);
- 15 cases (18%) lived in the San Francisco Bay Area (Alameda, Contra Costa, San Francisco, Santa Clara, Sonoma);
- 10 cases (12%) lived in the Central Valley (Butte, Fresno, Kern, San Joaquin, Sacramento, Tulare, Yolo); and
- The remaining 17 cases (21%) lived in the Sierra foothills (Placer, Nevada) or in coastal countries (Monterey, San Diego, Santa Barbara, San Luis Obispo)

Type A cases (59%) outnumbered type B cases (41%). Median age at onset was 11.7 weeks. (range 2.0 - 30.5 weeks). Although cases occurred in all calendar months, two thirds occurred in the summer and fall.

Eighty-one of the 83 patients were hospitalised; no patients died. Hospital stay averaged six weeks (mean) and ranged from three days to over three months. Aggregate hospital costs for the 81 in-patients exceeded A $2.8m.

Thirteen patients (16%), or approximately one in six infants had been fed honey before onset of illness, and Clostridium botulinum type B was identified in two of the 19 tested samples of honey eaten by two patients, each of whom had type B illness, but no C botulinum was identified in the 26 corn syrups from patients' homes that were available for testing. Approximately three quarters of the hospitalised patients had been breast-fed at birth, and approximately two-thirds of them were still being nursed at onset.

Editorial Comment:-

As in past years, California continues to report as many cases of infant botulism as the rest of the United States combined. This fact may effect the following circumstances:-

1. a more general awareness of the disease among the States' medical care providers;

2. convenient availabilty of diagnostic laboratory services;

3. the widespread distribution of C botulinum spores in the California environment; and

4. the continued feeding of honey to babies.

Despite the relatively low numbers of recorded cases, the economic impact of infant botulism remains substantial.

In 1986, two mildly-affected infants, both breast-fed, were managed as outpatients. These cases, the first out-patients with infant botulism to be recognised in California in five years suggest that the milder end of the clinical spectrum may still be overlooked in non specific categories as "failure to thrive" and "viral syndrome". Although California has the most cases of infant botulism, its incidence (10/100 000 births) is not the highest, a distinction that belongs to Hawaii (15-20/100 000 births).

The disparity in reported incidence between California, Hawaii and other States suggests that regional factors are important epidemiologically and underscores the need for continuing surveillance in a variety of geographical settings. In California the continuing absence of reported cases from Marin and Imperial Counties remains puzzling.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 4-5-87 TO 17-5-87 BULLETIN NUMBER 87/10
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	4	1	3	1	3	3	9		24
0101 ADENOVIRUS TYPE 1						3			3
0102 ADENOVIRUS TYPE 2						2			2
0103 ADENOVIRUS TYPE 3				1					1
0104 ADENOVIRUS TYPE 4				1					1
0106 ADENOVIRUS TYPE 6						1			1
0108 ADENOVIRUS TYPE 8						1			1
0111 ADENOVIRUS TYPE 11					1				1
0123 ADENOVIRUS TYPE 23				1					1
0137 ADENOVIRUS TYPE 37				1					1
0201 INFLUENZA A VIRUS	1								1
0203 INFLUENZA B VIRUS	1		1				1		3
0301 PARAINFLUENZA VIRUS TYPE 1				1	1	1			3
0302 PARAINFLUENZA VIRUS TYPE 2				2			13	2	17
0303 PARAINFLUENZA VIRUS TYPE 3	1	2		2	9				14
0399 PARAINFLUENZA VIRUS TYPING PENDING					1				1
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	9	5	3	3	5	11	8	2	46
0500 RHINOVIRUS (ALL TYPES)			1	3	12	4		2	22
0600 MYCOPLASMA PNEUMONIAE	22		9		2	2	6	7	48
0700 ORNITHOSIS-PSITTACOSIS	1								1
0824 COXSACKIEVIRUS A24				1					1
0903 COXSACKIEVIRUS B3			1			4			5
1004 ECHOVIRUS TYPE 4	1								1
1005 ECHOVIRUS TYPE 5		1						1	2
1009 ECHOVIRUS TYPE 9	1	1							2
1011 ECHOVIRUS TYPE 11			1	5					6
1018 ECHOVIRUS TYPE 18								1	1
1100 POLIOVIRUS NOT TYPED			4						4
1101 POLIOVIRUS TYPE 1				3					3
1102 POLIOVIRUS TYPE 2	1								1
1103 POLIOVIRUS TYPE 3						1			1
1300 HERPES VIRUS GROUP-NOT TYPED	58			1	1		2	1	63
1301 HERPES SIMPLEX VIRUS NOT-TYPED				1				1	2
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	13	1				1	18	15	48
1303 VARICELLA-ZOSTER VIRUS	4		1	1			3	2	11
1306 HERPES SIMPLEX TYPE 1	18			39		32	43	22	154
1307 HERPES SIMPLEX TYPE 2	88			48		19	52	83	290
1399 HERPES VIRUS TYPING PENDING				3	5				8
1401 COXIELLA BURNETI	9					1	9		19
1502 PICORNA VIRUS-NOT TYPED	12		11				18	2	43
1521 MEASLES VIRUS				1	2		1		4
1522 RUBELLA VIRUS			1			2	9	1	13
1532 HEPATITIS B ANTIGEN	78		15	1	1	11	15	30	151
1535 HEPATITIS A ANTIBODY	17		1			5	1	10	34
1541 CHLAMYDIA A - C TRACHOMATIS	45		1	22		28	20	53	169
1543 CHLAMYDIA A - LGV TYPE							13		13
1556 CMV - CYTOMEGALOVIRUS	3	1	8	17	2	5	2	19	57
1564 ROTAVIRUS	9	2	1	5	4	16	7		44
1599 ENTEROVIRUS TYPING PENDING			3		9				12
9992 ROSS RIVER VIRUS			9					10	19
9997 KUNJIN VIRUS							1		1
9998 ARBO. GROUP B. ...							1		1
Total	396	14	74	164	58	153	252	264	1,375

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 4-5-87 TO 17-5-87 BULLETIN NO 87/10

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.....						1					
0102 ADENOVIRUS TYPE 2.....		2									
0106 ADENOVIRUS TYPE 6.....		1									
0111 ADENOVIRUS TYPE 11.....				1							
0123 ADENOVIRUS TYPE 23.....								1			
0201 INFLUENZA A VIRUS.....		1									
0203 INFLUENZA B VIRUS.....		2									
0301 PARAINFLUENZA VIRUS TYPE 1....		3									
0302 PARAINFLUENZA VIRUS TYPE 2....	1	15									
0303 PARAINFLUENZA VIRUS TYPE 3....		14									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	4	43									
0500 RHINOVIRUS (ALL TYPES).....	1	2									
0600 MYCOPLASMA PNEUMONIAE.....	7	35									
0700 ORNITHOSIS-PSITTACOSIS.....		1									
0824 COXSACKIEVIRUS A24.....							1				
0903 COXSACKIEVIRUS B3.....	1	4									
1004 ECHOVIRUS TYPE 4.....	1										
1005 ECHOVIRUS TYPE 5.....			1	2							
1009 ECHOVIRUS TYPE 9.....	1			1							
1011 ECHOVIRUS TYPE 11.....	2	1		2		1					
1018 ECHOVIRUS TYPE 18.....	1										
1101 POLIOVIRUS TYPE 1.....		1					1				1
1102 POLIOVIRUS TYPE 2.....							1				
1103 POLIOVIRUS TYPE 3.....							1				
1300 HERPES VIRUS GROUP-NOT TYPED..	1										
1301 HERPES SIMPLEX VIRUS NOT-TYPED	1										1
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	10	3	1					3			4
1303 VARICELLA-ZOSTER VIRUS.....	4					1					5
1306 HERPES SIMPLEX TYPE 1.....	9	8									7
1307 HERPES SIMPLEX TYPE 2.....	11										10
1401 COXIELLA BURNETI.....	3	1									
1521 MEASLES VIRUS.....											4
1522 RUBELLA VIRUS.....	1	1									10
1532 HEPATITIS B ANTIGEN.....	62							79			
1535 HEPATITIS A ANTIBODY.....	19							11			
1541 CHLAMYDIA A - C.TRACHOMATIS...	22									1	2
1543 CHLAMYDIA A - LGV TYPE.....	13										
1556 CMV - CYTOMEGALOVIRUS.....	8	11					1			4	1
1564 ROTAVIRUS.....	7						37				
9992 ROSS RIVER VIRUS.....	6										
Total.....	196	149	2	6		3	43	94		5	210

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 4-5-87 TO 17-5-87 BULLETIN NO 87/10

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			
0103 ADENOVIRUS TYPE 3.....								1		
0104 ADENOVIRUS TYPE 4.....	1							1		
0108 ADENOVIRUS TYPE 8.....	1									
0137 ADENOVIRUS TYPE 37.....	1									
0203 INFLUENZA B VIRUS.....					1			1		
0302 PARAINFLUENZA VIRUS TYPE 2....					1			2		
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....								1		
0600 MYCOPLASMA PNEUMONIAE.....					1			6	4	
1011 ECHOVIRUS TYPE 11.....								1		
1101 POLIOVIRUS TYPE 1.....								1		
1300 HERPES VIRUS GROUP-NOT TYPED..	1									
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			7	8	4		1	9	5	
1303 VARICELLA-ZOSTER VIRUS.....								1	1	
1306 HERPES SIMPLEX TYPE 1.....	4	50						2	4	
1307 HERPES SIMPLEX TYPE 2.....		176								
1401 COXIELLA BURNETI.....			1		4		3	10	2	
1502 PICORNA VIRUS-NOT TYPED.....								1		
1521 MEASLES VIRUS.....					1					
1522 RUBELLA VIRUS.....					5			3		
1532 HEPATITIS B ANTIGEN.....		1		1				1	8	
1535 HEPATITIS A ANTIBODY.....								1	4	
1541 CHLAMYDIA A - C.TRACHOMATIS...	2	142							1	
1556 CMV - CYTOMEGALOVIRUS.....				1	1	8	1	9	17	
1564 ROTAVIRUS.....								1		
9992 ROSS RIVER VIRUS.....					13					
9997 KUNJIN VIRUS.....								1		
9998 ARBO. GROUP B.								1		
Total.....	10	369	8	10	31	8	8	54	46	