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Contents:

Editor
Dr Robert Hall

VIRUSES, CHLAMYDIAS, COXIELLAS, RICKETTSIAS AND MYCOPLASMAS REPORTING SCHEME: A total of 1232 reports were processed during this period. Reports from Westmesd Hospital which missed the processing deadline for the last issue of CDI have been included in this period.

Two cases of Q fever (one male aged 44 years and one female aged 29 years) were reported. No occupational exposure details have been provided.

Two Hundred and thirty-five reports of Ross River virus infection were received during this period bring the years total to 1041 so far. A breakdown by state is shown below. The level of reporting for the first three months of the year is approaching that of the early part of 1984 (Jan, 395; Feb, 568; Mar, 306) - the highest level of reporting of RRV since the inception of the CDI.

- The Bulletin is compiled and distributed by the Communicable Diseases Section, Communicable Diseases and Social Health Branch, Telephone: (062) 89 1555, Department of Community Services and Health.
- Contributions are solicited, and do not preclude later publication elsewhere.

- Figures given may be subject to revision.

State	Date of sample collection						
	1988				1989		
	Sep	Oct	Nov	Dec	Jan	Feb*	Mar*
Western Australia	0	4	19	114	209	186	88
Victoria	0	2	25	104	146	192	50
South Australia	0	0	2	2	3	22	16
New South Wales	3	0	0	1	67	55	1
Queensland	6	1	0	0	0	4	2
Total	9	7	46	221	425	459	157

* Data are incomplete and may be amended at a later date.

OVERSEAS BRIEFS:

1. JAPANESE ENCEPHALITIS IN INDIA

Increased Japanese encephalitis activity has been reported in Bihar and Uttar Pradesh in northern India and in southern Tamil Nadu and Kerala in southern India.

2. DENGUE FEVER IN NEW CALEDONIA AND FRENCH POLYNESIA

In New Caledonia, an estimated 18,000 cases of dengue fever have been reported between 16 February and 5 April 1989. The epidemic (previously reported in CDI 89/6) appears to be on the wane with urban cases decreasing since the end of February and rural cases decreasing since the end of March. Although dengue type 3 predominates, dengue types 4 and 1 have also been identified.

The dengue type 1 epidemic in French Polynesia peaked in early February and the numbers of reported cases are now decreasing regularly. Over 20,000 clinical cases were reported between 22 December 1988 and 5 April 1989. No deaths and no haemorrhagic symptoms have been reported.

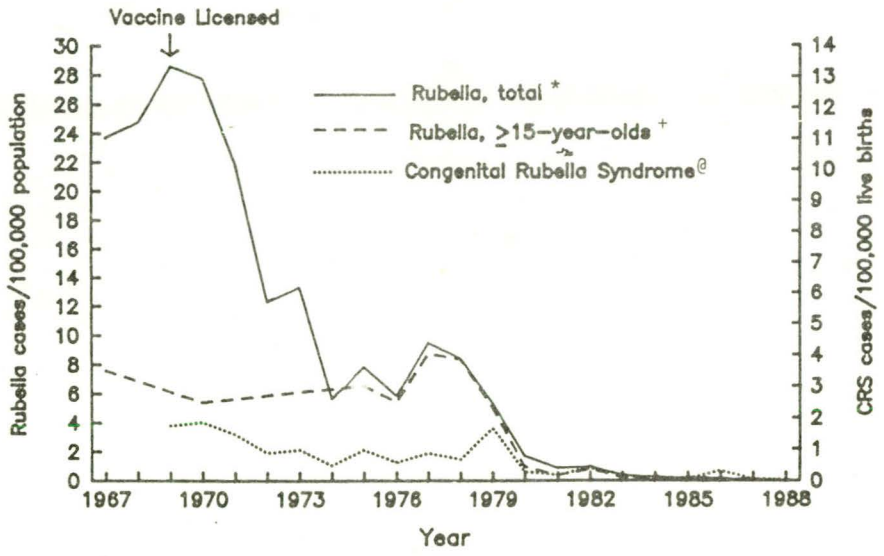
RUBELLA AND CONGENITAL RUBELLA SYNDROME - UNITED STATES, 1985 - 1988

(Based on MMWR 1989;38:173-178)

Rubella

A provisional total of 221 cases of rubella was reported in the United States in 1988 (0.1 cases per 100,000 population), the lowest since rubella became a nationally notifiable disease in 1966. In 1987, 306 cases of rubella (0.1/100,000) were reported. The incidence of rubella has declined by more than 99% since 1969, the year rubella vaccine was licensed in the U.S. (Figure 1).

Figure 1: Incidence rates of reported rubella and congenital rubella syndrome (CRS) cases - United States, 1967-1988



* 1988 provisional data.

\+ Includes patients 15 years of age or older for whom age was not reported. Average annual U.S. estimate based on data from Illinois, Massachusetts, and New York City for the 3-year periods 1966-1968, 1969-1971, and 1972-1974.

@ Confirmed and compatible cases, by year of birth. Provisional data due to delayed diagnosis and reporting.

In 1987, the last year for which complete data are available, 20 of 52 reporting areas (which comprise the 50 states, District of Columbia, and New York City [NYC]) reported no rubella cases, compared with 18 reporting areas in 1986 and 14 in 1985. One hundred and five (3.3%) counties reported rubella cases in 1987, compared with 152 (4.8%) in 1985. The reported age-specific incidence rates of rubella declined for all age groups during these 3 years (Table 1). In 1987, children under 5 years of age continued to have the highest incidence rate (0.5 cases/100,000 population) and accounted for 28% of the total number of patients with known ages. The rate for persons 15 years of age or older, who accounted for 49% of the patients with known ages in 1987, declined most dramatically - by 59% (0.19/100,000 in 1985 to 0.08/100,000 in 1987).

Long-term trends of rubella incidence among specific age groups can be assessed by comparing recent data from the total United States with those from three areas for which age-specific data were available before 1975 - Illinois, Massachusetts, and NYC (Table 2). In the 3-year period before vaccine licensure (1966-1968), the estimated risk of acquiring rubella was highest in children 5-9 years of age. Of the patients with known ages, children under 10 years of age accounted for 60%, while only 23% of the total was reported among those 15 years of age or older. By comparison, the reported incidence rates for 1985-1987 have declined by 95% or more for all age groups, with the greatest decreases occurring among persons under 20 years of age. Persons 20 years of age or older accounted for just over half of all patients with known ages. Although the decrease in incidence rates was smallest for this age group, their risk of acquiring rubella still declined more than 95%, relative to prevaccine licensure years.

Table 1: Age distribution of reported rubella cases and estimated incidence rates* - United States, 1985-1987

Age group (yrs)	1985			1986			1987			Rate change+ (%) 1985-1987
	No.	(%)	Rate*	No.	(%)	Rate*	No.	(%)	Rate*	
<1	47	(8.6)	1.5	50	(10.5)	1.6	33	(11.0)	0.9	-37.9
1-4	69	(12.6)	0.6	79	(16.7)	0.6	50	(16.7)	0.3	-41.8
5-9	60	(11.0)	0.4	48	(10.1)	0.3	47	(15.7)	0.3	-32.1
10-14	23	(4.2)	0.2	21	(4.4)	0.1	24	(8.0)	0.1	-27.2
15-19	34	(6.2)	0.2	44	(9.3)	0.3	27	(9.0)	0.2	-24.2
20-24	69	(12.6)	0.4	80	(16.9)	0.5	24	(8.0)	0.1	-69.7
25-29	96	(17.6)	0.5	72	(15.2)	0.4	48	(16.0)	0.2	-55.4
>30	148	(27.1)	0.1	80	(16.9)	0.1	47	(15.7)	0.0	-63.3
Total, known age	546	(100.0)	-	474	(100.0)	-	300	(100.0)	-	-
Total, unknown age	84	-	-	77	-	-	6	-	-	-
Total cases reported	630	-	0.3	551	-	0.2	306	-	0.1	-58.1

* Cases/100,000 population (projected census data) derived from extrapolating the age distribution of patients with known age to total cases.

\+ Based on actual rates.

Congenital rubella syndrome

Data on congenital rubella syndrome (CRS) are available from reports submitted weekly to the MMWR and from the National Congenital Rubella Syndrome Registry (NCRSR) maintained at the Division of Immunization, Center for Prevention Services, Centers for Disease Control (CDC). The MMWR CRS reports are case counts with no accompanying data and are tabulated by year of report. The NCRSR contains clinical and laboratory information on cases of CRS that are reported by state and local health departments. The NCRSR cases are monitored by year of patient's birth and are classified into six clinical categories [1], the most specific of which are 'CRS-confirmed' (i.e. cases with both congenital anomalies and laboratory evidence of rubella infection) and 'CRS-compatible' (i.e. cases that satisfy selected clinical criteria without laboratory confirmation). Beginning in 1984, information was routinely collected to evaluate whether a CRS case was 'indigenous' or 'imported'**. Since the NCRSR cases are classified by year of patient's birth, data are considered provisional for any given year; delays in diagnosis and/or reporting may result in the updating of figures. This summary updates previous reports on surveillance of CRS in the United States [1].

** Based on definitions approved by the Council of State and Territorial Epidemiologists, an imported case of CRS is defined as CRS in a U.S. or non-U.S. citizen whose mother was outside the United States during her presumed exposure to rubella. If the timing of exposure to rubella cannot be determined, the mother must have been outside the United States throughout the 21 days before conception and the first 20 weeks of her pregnancy.

For infants born in 1987, six CRS cases were reported to the NCRSR, of which three were considered indigenous. All three were confirmed CRS cases, and one of them occurred in a mother who had had at least one previous pregnancy. Only one CRS case has been reported thus far for 1988. Recent declines in rates of CRS recorded by NCRSR have paralleled the decline in overall rubella incidence and, more specifically, in the incidence for persons 15 years of age or older (Figure 1). During 1970-1987, the reported rate of rubella among persons in this age group declined 97%, from 2.3 to 0.1 cases/100,000 population. In 1970, 67 CRS cases occurred (1.80/100,000 live births), and three have been reported as of 22 March 1989, for 1987 (0.08/100,000 live births), representing a 96% decline (Table 3). This downward trend was interrupted in 1986, when 12 CRS cases were reported [2]. In that year, eight of these cases were reported to the NYC Department of Health 8-10 months after the peak of a rubella outbreak in NYC [3].

Table 2: Age distribution of reported rubella cases and estimated incidence rates* - Illinois, Massachusetts, and New York City, 1966-1968+, and total United States, 1985-1987+

Age group (yrs)	1966-1968 average@		1985-1987 average#		Rate change** (%) 1966-1987
	%	Rate	%	Rate	
<5	21.6	63.3	24.8	0.6	-99.1
5-9	38.5	101.3	11.8	0.3	-99.7
10-14	17.0	44.0	5.2	0.1	-99.7
15-19	12.7	35.7	8.0	0.2	-99.5
>20	10.2	3.7	50.2	0.1	-96.5
Total	100.0	24.3	100.0	0.2	-99.2

* Reported cases/100,000 population. Patients with unknown age excluded.
\+ Average annual figures over 3-year period.
@ Represents prevaccine years. National age data were not available before 1975 and were not consistently reported (i.e. over 75% of cases) until 1980.
\# Total U.S. data (1986 population projections) are used for 1985-1987; because the overall number of reported rubella cases is currently small, fluctuations (such as the epidemic in NYC in 1985) in only these three reporting areas skewed the data for this period.
** Based on actual rates.

MMWR Editorial Note

As part of the 1990 health objectives for the nation, the Public Health Service set a goal to reduce the number of rubella cases to less than 1000 and to reduce CRS to less than 10 cases annually [4]. The former goal was achieved for the first time in 1983, when 970 rubella cases were reported [5]. Although the goal for CRS has also been reached, unacceptable morbidity is still occuring. The primary aim of rubella vaccination programs is to prevent congenital rubella infection, which can result in miscarriages, abortions, stillbirths, and CRS in infants. When rubella vaccine was licensed in 1969, the United States adopted a policy of

universal immunisation of children of both sexes. The focus of this rubella vaccination strategy was to control rubella in preschool-aged and young school-aged children, the primary source of rubella transmission. This strategy was designed primarily to reduce and interrupt circulation of the virus, thereby reducing the risk of exposure to susceptible pregnant women. Also, vaccinated children would be protected immediately, and their immunity was expected to persist at least through their childbearing years [6]. Secondary emphasis was placed on vaccinating susceptible adolescents and adults, especially women.

The success of the rubella control program is apparent. In 1966-1987, the reported incidence rates of CRS and of rubella among persons 15 years of age or older declined in parallel by 95%-96% to all-time low levels. Meanwhile, incidence rates of rubella in children under 15 years of age have continued their downward trend. As the highly immune cohorts of young children enter the childbearing years, CRS should disappear from the United States.

However, concern continues despite the dramatic success of the U.S. rubella immunisation program. In 1987, 48% of reported rubella cases were in persons 15 years of age of older (32% of all cases were in persons 15-29 years of age). Most serological surveys of various postpubertal populations carried out during the 1970s and early 1980s found rates of rubella susceptibility comparable to the prevaccine years: 10%-20% of persons still lacked serological evidence of immunity to rubella [7-9]. Updated population-based serological surveys are needed to fully characterise the magnitude and extent of risk for this adolescent and young adult population. The NYC experience during 1985-1986 [2,3] and several recent college outbreaks [10] highlight the possibile risk of disease in postpubertal women. The continued occurrence of rubella in childbearing-aged populations suggests that potentially preventable cases of CRS may continue to occur during the next 10-30 years. Such concerns led CDC to announce an initiative in February 1985 to hasten elimination of rubella and CRS by targeting susceptible childbearing-aged populations for vaccination [11].

In addition, the reported figure for CRS cases is believed to underestimate the actual total, perhaps capturing only 10% of the actual total [12]. The NCRSR is a passive reporting system that, by its nature, results in underreporting of actual disease incidence and selective reporting of infants with severe and obvious CRS recognised and reported early in life. The limitations of current CRS surveillance underscore the need for all specialists who treat children with congenital anomalies compatible with CRS to continue to consider it in the differential diagnosis and to report all suspected cases to their state health departments.

As with other adult immunisations, creative approaches are necessary to enhance rubella immunisation levels in the childbearing-aged population. Adopting and enforcing comprehensive kindergarten through 12th grade school immunisation laws (especially for postpubertal elementary and secondary school students) and requiring proof of immunity to

rubella as a condition for college entry can minimise the risk of rubella outbreaks in the populations [13]. Another way to reach susceptible postpubertal women is to offer rubella vaccine at any encounter with the health-care system. After excluding patients who say they may be pregnant and counselling about the advisability to avoid conception for 3 months after vaccination, practitioners should not hesitate to vaccinate childbearing-aged women against rubella. No CRS-like defects have been detected in 212 infants born to susceptible mothers inadvertently vaccinated with RA27/3 live rubella virus vaccine during pregnancy [14; CDC, unpublished data]. NCRSR surveillance data indicate that one third to one half of mothers delivering CRS infants had had a previous live birth, suggesting that both postpartum vaccination and use of rubella vaccine in family-planning clinics could have an important impact on the overall occurrence of reported CRS. Physicians and other health-care personnel should offer rubella vaccine whenever they encounter a potentially susceptible woman lacking contraindications for vaccination. Susceptible persons identified through preemployment, premarital, or prenatal screening should be offered vaccine at follow-up visits.

Table 3: Incidence rate of congenital rubella syndrome* reported to the National Congenital Rubella Syndrome Registry (NCRSR) - United States, 1969-1988

Year	NCRSR cases+	Incidence rate#	Year	NCRSR cases+	Incidence rate#
1969	62	1.72	1979	57	1.63
1970	67	1.80	1980	14	0.39
1971	44	1.24	1981	10	0.28
1972	32	0.98	1982	13	0.36
1973	30	0.96	1983	7	0.19
1974	22	0.70	1984	2	0.05
1975	32	1.02	1985	2	0.05
1976	22	0.69	1986	13	0.35
1977	29	0.87	1987	3	0.08
1978	30	0.90	1988	1	0.03

* Confirmed and compatible cases only, reported by year of birth. Data are provisional because of delay reporting.
\+ Excluded are the following imported cases: 1984 (1 case), 1985 (1), 1986 (2), and 1987 (3). No imported cases have been reported for 1988.
\# Cases/100,000 live births/year.

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RISKS ASSOCIATED WITH HUMAN PARVOVIRUS B19 INFECTION

(Based on MMWR 1989;38:81-8,93-7)

General information

B19 was discovered in England in 1975 in serum specimens from healthy blood donors [1]. Since its discovery, B19 has been shown to be the causative agent of erythema infectiosum (EI)(also known as fifth disease) and is the primary etiological agent of transient aplastic crisis (TAC) in patients with chronic haemolytic anaemias [2-4]. B19 has also been associated with fetal death (both spontaneous abortions and stillbirths), acute arthralgias and arthritis, and chronic anaemia in immunodeficient patients [5-14].

The virus belongs to the family Paroviridae. It is heat-stable and can survive at 60°C (140°F).

Clinical features of B19 infection

Erythema infectiosum (fifth disease)

The most commonly recognised illness associated with B19 infection is EI. EI is a mild childhood illness characterised by a facial rash ('slapped cheek' appearance), and a reticulated or lacelike rash on the truck and extremities [15]. Reappearance of the rash may occur for several weeks following nonspecific stimuli such as change in temperature, sunlight, and emotional stress. Typically, the patient is otherwise well at rash onset but often gives a history of mild systemic symptoms 1-4 days before rash onset. In some EI outbreaks, pruritis has been a common clinical feature. In addition to typical EI, B19 infection has been associated with a variety of other exanthems, including those that are rubella-like, vesicular, and purpuric [15].

Asymptomatic infection

In outbreak investigations, asymptomatic infection has been reported in approximately 20% of children and adults [16-17].

Arthropathy

In some outbreaks of EI, arthralgias and arthritis have been commonly reported [7,8,18]. Infection may produce a symmetrical peripheral polyarthropathy. Joints in the hands

are most frequently affected, followed by the knees and wrists. Symptoms are usually self-limited but may persist for several months. Joint symptoms, more common in adults, may occur as the sole manifestation of infection.

Transient aplastic crisis (TAC) and severe anaemia

B19 is the primary aetiological agent causing TAC in patients with chronic haemolytic anaemias (eg, sickle cell disease, haemoglobin SC disease, hereditary spherocytosis, B-thalassemia, and autoimmune heamolytic anaemia)[19,20]. It can also cause TAC in other conditions in which increased red cell production is necessary to maintain stable red cell indices, as may occur in anaemia due to blood loss. Patients with TAC typically present with pallor, weakness, and lethargy and may report a nonspecific prodromal illness in the preceding 1-7 days. Few patients with TAC report a rash. In the acute phase of the illness, patients usually have a moderate to severe anaemia with absence of reticulocytes, and bone marrow examination shows a hypoplastic or an aplastic erythroid series with a normal myeloid series. Recovery is indicated by a return of reticulocytes in the peripheral smear approximately 7-10 days after their disappearance. TAC may require transfusion and hospitalisation and can be fatal if not treated promptly.

B19 infection in immunodeficient patients

A B19-related severe chronic anaemia associated with red cell aplasia has been described in patients on maintenance chemotherapy for acute lymphocytic leukaemia, patients with congenital immunodeficiencies, and patients with human immunodeficiency virus (HIV)-related immunodeficiency [9-14]. It is not yet known how often B19 causes chronic anaemia in immunodeficient patients or which patients are most susceptible to this complication of infection. Chronic B19 infection should, however, be included in the differential diagnosis of chronic anaemia in the immunodeficient patient.

Infection in the pregnant woman

Intrauterine infection and fetal death

In most of the reported B19 infections occurring during pregnancy, the fetus has not been adversely affected [5,6,21-27]. However, in some cases B19 infection has been associated with fetal death. The risk of fetal death attributable to parvovirus infection following documented maternal infection (B19 IgM-antibody-positive) is not known, but preliminary results of one study from the United Kingdom suggest that it is less than 10% [27; SM Hall, unpublished data].

Results from an ongoing study in the United States also suggest that B19-attributable fetal deaths are infrequent (CDC, unpublished data). In this study, 95 pregnant women with IgM antibody to B19 are being followed prospectively. Fetal loss has so far occurred in two (4.1%) of 49 women followed to term. It is not known whether the two fetal deaths were caused by B19 infection. One fetus was hydropic; the other was not described. No tissues for either fetus were available for B19 hybridisation studies.

A study of 96 women who had stillbirths, 96 women who had spontaneous abortions, and controls matched by age, duration of pregnancy, and location suggests that B19 is not responsible for a substantial proportion of fetal deaths in the general population [28]. In this study, the rate of serologically confirmed B19 infection was the same (1%) in cases and controls. In a survey of 50 fetuses with nonimmunological hydrops fetalis, an uncommonly diagnosed cause of fetal death, four (8%) were positive for B19 DNA [22].

Congenital abnormalities

Since some of the animal parvoviruses are teratogens [29], the possibility that infection may also be associated with congenital abnormalities in humans is a concern. However, there is no evidence that the rate of congenital anomalies following B19 infection exceeds background rates. B19-associated congenital anomalies have not been reported among several hundred liveborn infants of B19-infected mothers. One aborted fetus with eye anomalies and histological evidence of damage to multiple tissues born to an B19-infected woman has been reported [30]. An anencephalic fetus was reported in a B19 infected woman, but the timing of infection made it unlikely that B19 contributed to the defect [31].

Pathogenesis

The pathogenesis of the rash in EI unknown, but the rash may be immunecomplex-mediated. The other, more serious, manifestations of B19 infection are related to the propensity of the virus to infect and lyse erythroid precursor cells and interrupt normal red cell production [32]. In a person with normal haematopoiesis, B19 infection produces a self-limited red cell aplasia that is clinically inapparent. Transient leukopenia, lymphocytopenia, and thrombocytopenia have also been reported with B19 infection in the normal host [33,34].

In patients who have increased rates of red cell destruction or loss and who depend on compensatory increases in red cell production to maintain stable red cell indices, B19 infection may lead to TAC. Patients at risk for TAC include those with chronic haemolytic anaemias and those with anaemias associated with acute or chronic blood loss. In immunodeficient persons, B19 infection may persist, causing chronic red cell aplasia, which results in chronic anaemia; chronic neutropenia has also been described [10].

B19 DNA-positive tissues have been reported in 20 fetal deaths; in all 17 cases in which pathological findings were described, the fetuses had nonimmunological hydrops fetalis [6,22-24,27,31,35-40]. The precise pathogenesis of fetal death remains unclear. Severe anaemia may precipitate congestive heart failure, generalised oedema, and ultimately fetal death. The fetus may be particularly vulnerable to B19 infection because red cell survival is short, and the red cell volume is rapidly expanding. Severe anaemia, B19 viraemia, and cytological changes in erythroid precursor cells have been described in fetuses just before death [23,24,35]. Chronic infection may occur in the fetus (one fetus was viraemic for at least 4 weeks)[23]. In one case report, infection of myocardial cells was noted, suggesting that direct damage to myocardial tissue may also contribute to the disease process in the fetus [26].

Epidemiological features of B19 infection

Prevalence

B19 infection occurs worldwide [41,42]. Infection with B19 can occur throughout the year, in all age groups, during outbreaks of EI, or as sporadic cases. B19 infection is most frequently recognised during outbreaks of EI in schools. The level of EI activity in a community varies from year to year; periods of increased activity lasting several years are generally followed by several years of decreased activity [43-46]. The reported seroprevalence ranges from 2% to 15% in children 1-5 years old, 15% to 60% in children 5-19 years old, and 30% to 60% in adults [15,36,47,48].

Incubation period

Studies of secondary illness in households suggest that the incubation period for clinical EI and TAC is usually 4-14 days but can be as long as 20 days [15]. In volunteer studies, rash illness occurred 17-18 days after inoculation [33,34].

Transmission

B19 DNA has been found in respiratory secretions in viraemic patients, which suggest that these secretions are involved in transmission [16,17,33].

The virus is transmitted effectively after close contact exposures. The secondary attack rate for infection among susceptible household contacts of patients with TAC or EI is about 50% [16,17]. In school outbreaks, 10%-60% of students may develop EI. In outbreaks in which student involvement is widespread, preliminary data suggest 20%-30% of susceptible (IgG-antibody-negative) staff may develop serological evidence of B19 infection during the course of the outbreak (CDC, unpublished data).

In outbreak settings, it is not known whether the primary mode of transmission involves direct person-to-person contact, fomites, large-particle droplets, or small-particle droplets. The virus can also be transmitted parenterally by transfusion of blood or blood products and vertically from mother to fetus [1,49,50]. Transmission rarely occurs during transfusion with single-donor blood products but is common during treatment with clotting-factor concentrates, even after steam- or dry-heat treatment of the clotting factor concentrate [1,49,50]. Tattooing was suspected as the source of B19 transmission in two instances [51].

Diagnosis

Diagnostic tests include:

- detection of B19 IgM antibody using capture-antibody radioimmunoassay or enzyme immunoassay [52,53];
- detection of B19 DNA using nucleic acid hybridisation [54-56];
- detection of eosinophilic nuclear inclusions with peripheral condensation of Chromatin in erythroid pre cursor cells of infected patients using light microscopy [25,37,57];

- electron microscopic detection of parvovirus-like particles in serum or eosinophilic nuclear inclusions;
- demonstration in tissue of replicative forms of B19 DNA and non-structural proteins by Southern and Western blot analysis [58-59].

Diagnostic testing is available at only a few sites in the United States (primarily research laboratories and the Division of Viral Diseases, Center for Infectious Diseases, CDC).

Prevention of infection

Risk groups

Although B19 infection usually produces a mild, self-limited illness, three groups of persons are at risk for serious complications of infection:

1. persons with chronic haemolytic anaemias;
2. persons with congenital or acquired immunodeficiencies; and
3. pregnant women.

Since infection in these persons can lead to substantial morbidity and some mortality, consideration should be given to preventing or ameliorating disease.

Health-care settings

Guidelines for isolation precautions in hospitals have been published for EI [60], but recent information suggests that these guidelines should be modified. Most patients with EI are past their period of infectiousness and do not present a risk for further transmission; thus isolation precautions are not indicated. However, there is risk for nosocomial transmission of B19 from patients with TAC and from immunodeficient patients with chronic B19 infection. These patients should be considered infectious and placed on isolation precautions for the duration of their illness or until the infection has been cleared. Nosocomial transmission of B19 has been associated with one case of TAC [61]. Transmission of B19 infection has also occurred in medical research laboratories [4,62].

Patients with TAC or chronic B19 infection should be admitted to private rooms. Persons in close contact with the patients should wear masks. Gloves should be worn by persons likely to touch infective material such as respiratory secretions, and gowns should be worn when soiling is anticipated (contact isolation) [60]. Hands should be washed after the patient or potentially contaminated articles are touched and before care is provided to another patient. B19-infected patients may share a room with another B19-infected patient unless sharing is contraindicated by another infection or condition.

Health-care workers should be advised that they are at risk of B19 infection after exposure in the hospital or in the community and that there may be a risk for further transmission to patients. Routine infection-control practices should minimise the risk of transmission.

Personnel who may be pregnant or who might become pregnant should know about potential risks to the fetus from B19 infection and about preventive measures that may reduce those risks.

Homes, schools, and workplaces

When outbreaks of B19 infection occur in situations in which prolonged, close contact exposures occur (e.g., at home, in schools, or in day-care centres), options for preventing transmission are limited. The greatest risk of transmitting the virus occurs before symptoms of EI develop; therefore, transmission cannot be prevented by identifying and excluding persons with EI. The efficacy of decontaminating toys and environmental surfaces to decrease B19 transmission has not been studied. The efficacy of handwashing to decrease B19 transmission has not been studied either, but handwashing is recommended as a practical and probably effective measure.

When outbreaks occur, parents of school-aged children and employees should be advised about the risk of transmitting and acquiring infection and about who is at risk for serious complications.

The decision to try to decrease any person's risk of infection by avoiding a workplace or school environment in which an EI outbreak is occurring should be made by the person after discussions with family members, health-care providers, public health officials, and employers or school officials. A policy to routinely exclude members of high-risk groups is not recommended.

Patient management

Patients with chronic haemolytic anaemia

The exposed patient with chronic haemolytic anaemia should be managed by alerting the patient or his/her parents or guardians about the exposure, the symptoms and signs associated with TAC (pallor, weakness, and lethargy), and the need to consult a physician immediately if symptoms or signs of TAC develop. Management of the patient with TAC is based on treating symptoms of the associated anaemia and may require blood transfusion.

Patients with congenital and acquired immunodeficiencies

The exposed patient with a congenital or acquired immunodeficiency should be managed by advising the patient or his/her parents or guardians about the exposure and the possibility that B19 infection may lead to chronic anaemia. The physician should consider B19 infection in the differential diagnosis of chronic anaemia in this group of patients, especially if there is an outbreak of EI in the community.

In several patients with acute lymphocytic leukemia, the administration of immunoglobulin resulted in disappearance of viraemia and improvement in red cell indices [10]. In other patients, the infection and associated anaemia resolved when immune function returned [12,14]. The role of immunoglobulin in the treatment of these patients needs further study.

Pregnant women

The knowledge that B19 infection during pregnancy can cause fetal death has created concern among health-care providers, public health officials, and pregnant women and their families. In managing exposed pregnant women, risks should be considered in the context of other risks to the pregnancy and the risks associated with intervention.

For women with a documented infection, maternal serum alpha-fetoprotein levels and diagnostic ultrasound examinations have been used to identify adversely affected fetuses, but the sensitivity and specificity of these tests, their appropriate timing, and the risks and benefits of their use in managing infected pregnant women have not yet been determined [35,37]. Interpretation of the ultrasound is difficult early in pregnancy and should be supervised by a physician experienced in diagnosing fetal abnormalities. Intrauterine blood transfusion (IBT) has been proposed as treatment for the fetus with B19-induced severe anaemia. However, IBT is a high-risk, specialised procedure of unproven benefit in this situation and cannot be recommended for routine treatment of B19-related hydrops fetalis [63].

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AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES
BASED ON DATE OF REPORTING

PERIOD 30/3/89 TO 12/4/89

1. CODE 019 - FAIRFIELD(VIC)
2. CODE 065 - STATE LAB(WA) PMH(WA)
3. CODE 110 - IMVS(SA)
4. CODE 111 - RCH(VIC)
5. CODE 112 - ICPMR(NSW) WVH(ACT)
6. CODE 113 - PHH POW(NSW)
7. CODE 114 - RAHC(NSW)
8. CODE 115 - STATE LAB(QLD)

		019	065	110	111	112	114	115	TOTAL
0100	ADENOVIRUS NOT TYPED	1	1	2	0	4	0	7	15
0101	ADENOVIRUS TYPE 1	1	0	1	0	0	0	0	2
0102	ADENOVIRUS TYPE 2	1	1	2	0	1	0	0	5
0103	ADENOVIRUS TYPE 3	1	0	2	0	8	0	0	11
0104	ADENOVIRUS TYPE 4	3	1	0	0	0	0	0	4
0105	ADENOVIRUS TYPE 5	1	0	0	0	0	0	0	1
0106	ADENOVIRUS TYPE 6	0	0	2	0	0	0	0	2
0107	ADENOVIRUS TYPE 7	0	0	1	0	0	0	0	1
0108	ADENOVIRUS TYPE 8	1	0	0	0	4	0	0	5
0111	ADENOVIRUS TYPE 11	0	0	0	0	1	0	0	1
0119	ADENOVIRUS TYPE 19	0	0	0	0	1	0	0	1
0122	ADENOVIRUS TYPE 22	0	0	0	0	1	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	0	0	13	0	0	0	13
0201	INFLUENZA A VIRUS	0	0	0	0	1	0	0	1
0203	INFLUENZA B VIRUS	1	1	0	0	0	0	0	2
0302	PARAINFLUENZA VIRUS TYPE 2	0	0	2	6	1	0	4	13
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	0	10	0	0	0	10
0400	RESPIRATORY SYNCYTIAL VIRUS (R	2	4	2	1	0	2	5	16
0500	RHINOVIRUS (ALL TYPES)	2	1	14	25	1	0	1	44
0600	MYCOPLASMA PNEUMONIAE	2	2	4	2	1	0	0	11
0700	ORNITHOSIS-PSITTACOSIS	1	0	1	0	0	0	0	2
0901	COXSACKIEVIRUS B1	0	0	1	0	0	0	0	1
0904	COXSACKIEVIRUS B4	1	3	0	0	0	0	0	4
1005	ECHOVIRUS TYPE 5	0	1	0	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	0	2	1	0	0	0	0	3
1007	ECHOVIRUS TYPE 7	0	0	1	0	0	0	0	1
1009	ECHOVIRUS TYPE 9	3	0	0	0	0	0	0	3
1018	ECHOVIRUS TYPE 18	0	0	0	0	1	0	0	1
1022	ECHOVIRUS TYPE 22	0	0	1	0	0	0	0	1
1030	ECHOVIRUS TYPE 30	5	3	1	0	1	0	0	10
1100	POLIOVIRUS NOT TYPED	0	1	0	0	0	0	0	1
1101	POLIOVIRUS TYPE 1	1	0	1	0	0	0	0	2
1102	POLIOVIRUS TYPE 2	0	0	1	0	0	0	0	1
1200	MUMPS VIRUS	0	0	0	0	1	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	2	1	0	2	0	1	6
1301	HERPES SIMPLEX VIRUS - NOT TYP	3	4	0	0	84	0	0	91
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	2	12	12	4	38	4	0	72
1303	VARICELLA-ZOSTER VIRUS	4	1	1	2	7	0	0	15
1306	HERPES SIMPLEX TYPE 1	35	23	15	0	0	0	22	95
1307	HERPES SIMPLEX TYPE 2	56	47	16	0	0	0	21	140
1399	HERPES VIRUS TYPING PENDING	7	0	0	6	0	0	0	13
1401	COXIELLA BURNETI	0	0	0	0	2	0	0	2
1502	PICORNIA VIRUS - NOT TYPED = E	0	3	0	0	3	0	11	17
1514	MOLLUSCUM CONTAGIOSUM	0	1	0	0	0	0	0	1
1521	MEASLES VIRUS	0	0	0	1	0	0	0	1
1522	RUBELLA VIRUS	4	1	1	0	2	0	0	8
1532	HEPATITIS B ANTIGEN	10	13	18	1	45	0	15	102
1535	HEPATITIS A ANTIBODY	3	3	3	0	2	0	0	11
1541	CHLAMYDIA A - C. TRACHOMATIS	8	35	9	2	28	0	44	126
1556	CMV - CYTOMEGALOVIRUS	27	5	3	7	20	1	10	73
1564	ROTAVIRUS	9	3	0	0	2	0	1	15
1599	ENTEROVIRUS TYPING PENDING	0	0	0	15	0	3	0	18
9992	ROSS RIVER VIRUS	40	65	8	0	120	0	2	235
TOTAL		235	239	127	95	382	10	144	1232

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1.

PERIOD 30/3/89 TO 12/4/89

1. CODE 00, 99 ..... - NO ILL OR DATA
2. CODE 01, 02, 11, 12 - RESPIRATORY
3. CODE E3 ..... - ENCEPHALITIS
4. CODE M3 ..... - MENINGITIS
5. CODE 04 ..... - PARALYSIS
6. CODE 05, 13 ..... - CNS OTHER UNSPEC
7. CODE 07, 49 - GASTRO INTESTINAL
8. CODE 17, 47 - HEPATIC
9. CODE 19 ... - CVS
10. CODE 89 ... - URINARY TRACCT
11. CODE 06 ... - SKIN MUCOUS

		1	2	3	4	6	7	8	9	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	2	3	0	0	1	5	0	0	0	0	11
0101	ADENOVIRUS TYPE 1	0	1	0	0	0	0	0	0	0	0	1
0102	ADENOVIRUS TYPE 2	1	2	0	1	0	1	0	0	0	0	5
0103	ADENOVIRUS TYPE 3	1	2	0	0	0	2	0	0	0	0	5
0105	ADENOVIRUS TYPE 5	0	1	0	0	0	0	0	0	0	0	1
0106	ADENOVIRUS TYPE 6	0	0	0	0	0	2	0	0	0	0	2
0107	ADENOVIRUS TYPE 7	1	0	0	0	0	0	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	0	0	0	0	0	1	0	0	0	0	1
0119	ADENOVIRUS TYPE 19	1	0	0	0	0	0	0	0	0	0	1
0122	ADENOVIRUS TYPE 22	0	0	0	0	0	1	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	1	9	0	0	0	0	0	0	0	0	10
0201	INFLUENZA A VIRUS	0	1	0	0	0	0	0	0	0	0	1
0203	INFLUENZA B VIRUS	1	0	0	0	0	0	0	0	0	0	1
0302	PARAINFLUENZA VIRUS TYPE 2	0	13	0	0	0	0	0	0	0	0	13
0303	PARAINFLUENZA VIRUS TYPE 3	0	8	0	0	0	0	0	0	0	0	8
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	16	0	0	0	0	0	0	0	0	16
0500	RHINOVIRUS (ALL TYPES)	1	36	0	2	0	0	0	0	0	0	39
0600	MYCOPLASMA PNEUMONIAE	0	9	0	0	0	0	0	0	0	0	9
0700	ORNITHOSIS-PSITTACOSIS	0	2	0	0	0	0	0	0	0	0	2
0901	COXSACKIEVIRUS B1	0	0	0	0	0	1	0	0	0	0	1
0904	COXSACKIEVIRUS B4	1	1	0	0	1	0	0	0	0	0	3
1005	ECHOVIRUS TYPE 5	0	0	0	1	0	0	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	0	0	0	1	1	0	0	0	0	0	2
1007	ECHOVIRUS TYPE 7	0	1	0	0	0	0	0	0	0	0	1
1009	ECHOVIRUS TYPE 9	0	0	0	3	0	0	0	0	0	0	3
1018	ECHOVIRUS TYPE 18	1	0	0	0	0	0	0	0	0	0	1
1022	ECHOVIRUS TYPE 22	1	0	0	0	0	0	0	0	0	0	1
1030	ECHOVIRUS TYPE 30	0	2	0	7	0	1	0	0	0	0	10
1100	POLIOVIRUS NOT TYPED	0	1	0	0	0	0	0	0	0	0	1
1101	POLIOVIRUS TYPE 1	0	2	0	0	0	0	0	0	0	0	2
1300	HERPES VIRUS GROUP - NOT TYPED	1	0	0	0	0	0	1	0	0	4	6
1301	HERPES SIMPLEX VIRUS - NOT TYP	20	0	0	0	0	0	0	0	1	20	41
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	12	5	0	0	2	1	8	0	0	1	29
1303	VARICELLA-ZOSTER VIRUS	1	1	0	0	1	0	0	0	0	11	14
1306	HERPES SIMPLEX TYPE 1	2	5	0	1	0	0	0	0	0	57	65
1307	HERPES SIMPLEX TYPE 2	2	0	0	0	0	0	0	0	0	54	56
1399	HERPES VIRUS TYPING PENDING	0	2	0	0	0	0	0	0	1	5	8
1502	PICORNIA VIRUS - NOT TYPED = E	0	7	1	0	4	3	0	1	0	0	16
1514	MOLLUSCUM CONTAGIOSUM	0	0	0	0	0	0	0	0	0	1	1
1521	MEASLES VIRUS	0	0	0	0	0	0	0	0	0	1	1
1522	RUBELLA VIRUS	1	0	0	0	0	0	0	0	0	7	8
1532	HEPATITIS B ANTIGEN	52	0	0	0	0	1	40	0	0	0	93
1535	HEPATITIS A ANTIBODY	3	0	0	0	0	0	8	0	0	0	11
1541	CHLAMYDIA A - C. TRACHOMATIS	19	1	0	0	0	2	0	0	0	0	22
1556	CMV - CYTOMEGALOVIRUS	7	17	0	0	2	1	0	0	4	2	33
1564	ROTAVIRUS	0	0	0	0	0	14	0	0	0	0	14
1599	ENTEROVIRUS TYPING PENDING	0	8	0	5	0	1	0	1	0	0	15
9992	ROSS RIVER VIRUS	25	1	0	0	0	0	0	0	0	54	80
TOTAL		157	157	1	21	12	37	57	2	6	217	667

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2.

PERIOD 30/3/89 TO 12/4/89

12. CODE 10 - EYE
13. CODE 59 - GENITAL
14. CODE 39 - ENDOCRINE/SALIVARY GL.
15. CODE 38 - RETICULO-ENDOTHELIAL
16. CODE 29 - MUSCLE/JOINT
17. CODE 69 - CONGENTIAL
18. CODE P8 - PUO
19. CODE G8 - FEVER/MALAISE
20. CODE 09 - OTHER
21. CODE A1 - SIDS

		12	13	14	15	16	17	18	19	20	21	TOTAL
0100	ADENOVIRUS NOT TYPED	2	0	0	0	0	0	1	0	1	0	4
0101	ADENOVIRUS TYPE 1	1	0	0	0	0	0	0	0	0	0	1
0103	ADENOVIRUS TYPE 3	4	0	0	0	0	0	0	1	0	1	6
0104	ADENOVIRUS TYPE 4	4	0	0	0	0	0	0	0	0	0	4
0108	ADENOVIRUS TYPE 8	5	0	0	0	0	0	0	0	0	0	5
0199	ADENOVIRUS TYPING PENDING	2	0	0	0	0	0	0	1	0	0	3
0203	INFLUENZA B VIRUS	0	0	0	0	0	0	0	1	0	0	1
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	0	1	0	0	0	1	0	0	2
0500	RHINOVIRUS (ALL TYPES)	0	0	0	2	0	0	0	0	1	2	5
0600	MYCOPLASMA PNEUMONIAE	0	0	0	0	0	0	0	1	1	0	2
0904	COXSACKIEVIRUS B4	0	0	1	0	0	0	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	0	0	0	0	0	0	0	0	0	1	1
1102	POLIOVIRUS TYPE 2	0	0	0	0	0	0	0	1	0	0	1
1200	MUMPS VIRUS	0	0	1	0	0	0	0	0	0	0	1
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	50	0	0	0	0	0	0	0	0	50
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	23	3	2	0	2	5	8	0	43
1303	VARICELLA-ZOSTER VIRUS	1	0	0	0	0	0	0	0	0	0	1
1306	HERPES SIMPLEX TYPE 1	2	21	0	0	0	0	2	0	5	0	30
1307	HERPES SIMPLEX TYPE 2	1	79	1	0	0	0	0	0	3	0	84
1399	HERPES VIRUS TYPING PENDING	0	1	0	0	0	0	0	1	3	0	5
1401	COXIELLA BURNETI	0	0	0	0	1	0	0	1	0	0	2
1502	PICORNIA VIRUS - NOT TYPED = E	0	0	0	0	0	0	0	0	1	0	1
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	0	0	9	0	9
1541	CHLAMYDIA A - C. TRACHOMATIS	1	103	0	0	0	0	0	0	0	0	104
1556	CMV - CYTOMEGALOVIRUS	0	6	0	1	0	1	4	8	20	0	40
1564	ROTAVIRUS	0	0	0	0	0	1	0	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	0	1	0	0	2	3
9992	ROSS RIVER VIRUS	0	0	0	0	127	0	2	24	2	0	155
TOTAL		23	260	26	7	130	2	12	45	54	6	565