Parvovirus B19 infection and its significance in pregnancy

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Abstract

Parvovirus B19 causes prolonged epidemics of erythema infectiosum, particularly in primary school-aged children. Infection causes clinically significant anaemia in individuals with high red cell turnover, including the fetus. Approximately 40% of women of childbearing age are susceptible, and annual seroconversion rates vary from 1.5% during endemic periods to 10-15% during epidemics. Infection occurs in around 50% of susceptible women exposed at home and 20-30% following occupational exposure (for example, at a primary school). Maternal infection in the first half of pregnancy is associated with 10% excess fetal loss and hydrops fetalis in 3% of cases (of which up to 60% resolve spontaneously or with appropriate management). No congenital abnormalities or long-term sequelae have been attributed to parvovirus B19 infection. The overall risk of serious adverse outcome from occupational exposure to parvovirus B19 infection during pregnancy is low (excess early fetal loss in 2-6/1,000 pregnancies and fetal death from hydrops in 2-5/10,000 pregnancies). It is not recommended that susceptible pregnant women be excluded routinely from working with children during epidemics. ***Commun Dis Intell*** **2000;24:69-71.**

Keywords: parvovirus B19, pregnancy, erythema infectiosum, hydrops fetalis

Clinical features and pathogenesis

The clinical features and pathogenesis of parvovirus B19 have been described in two reports.[1,2] Human parvovirus B19 causes an acute, usually self-limiting, infection which is often asymptomatic. The usual clinical manifestation is erythema infectiosum (fifth disease), characterised by a mild prodrome – mild fever, malaise, myalgia – followed by a biphasic rash. A bright red malar eruption, 'slapped cheek syndrome', with circumoral pallor is followed by a maculopapular rash on the extremities and trunk, which fades to a reticular appearance and often recurs, transiently, for weeks. In adult women particularly, parvovirus infection can cause symmetrical polyarthralgia or arthritis, predominantly affecting peripheral joints,

ISSN 0725-3141
Volume 24
Supplementary
March 2000

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which usually lasts 1-3 weeks or occasionally longer.

The virus primarily infects erythroid precursors and causes haemolytic anaemia, which is subclinical and spontaneously reversible in otherwise normal people. In individuals with high red cell turnover, as in sickle cell anaemia, parvovirus infection can cause acute aplastic crisis, which can be life threatening but is ultimately self-limiting. In people who are immunocompromised, parvovirus infection can cause chronic infection and red cell aplasia or pancytopenia. Fetal infection is usually benign and self-limiting but, in a small proportion, causes severe anaemia and hydrops fetalis, usually in the second trimester.

Epidemiology

The spread of parvovirus B19 is by the respiratory route and usually occurs immediately before the onset of rash. The incubation period is from one to three weeks. Epidemics of erythema infectiosum occur over extended periods. Limited evidence suggests that approximately two-yearly epidemic periods alternate with endemic periods of similar length.[3] Young children are most commonly involved, but 30-50% of adults are susceptible. The only local data are from a recent study in Victoria, which demonstrated a two-yearly endemic/epidemic periodicity. The highest rates of infection were in children aged 5-9 years and 60% of women of child-bearing age (20-39 years) were immune (Heath Kelly, personal communication). Annual seroconversion rates among women of childbearing age vary from about 1.5% during endemic periods, to about 10 times higher during epidemics.[4]

The risk of infection generally depends on the degree of exposure to children. It is highest (at least 50%) in susceptible women with an infected child at home. Generally, primary school teachers and child-care workers are at somewhat greater risk of infection (10-30%) than the general population (10-15%) during epidemics, depending on the ages, number of children and degree of contact.[4-7] Nosocomial transmission of parvovirus from chronically infected, immunocompromised patients or those with acute aplastic crisis to health care workers can occur, but the risk is apparently low and difficult to distinguish from community spread.[8,9]

Potential consequences of parvovirus B19 infection during pregnancy

Asymptomatic fetal infection occurs in up to 50% of cases following proven maternal infection in pregnancy.[10] The small risk of fetal damage is virtually confined to the first half of pregnancy. There is an excess early fetal loss, following maternal infection in the first 20 weeks, of about 15% compared with a background rate of 5%; that is, the excess is about 10%.[11] Approximately 3% of maternal infections between 9 and 20 weeks are complicated by hydrops fetalis, due to severe anaemia and cardiac failure. When it occurs, hydrops presents, on average, around 5 weeks (range 2-17) after maternal infection.[11,12] Chronic congenital anaemia, following intrauterine transfusion for hydrops fetalis, has been reported.[1] No specific developmental abnormalities or increase in their incidence, and no long-term sequelae in otherwise normal infants, have been attributable to maternal parvovirus infection.[11,13] The risks of occupational exposure of a pregnant woman, to parvovirus B19 infection during an epidemic, are summarised in Figure 1.

Diagnosis of parvovirus infection

Pregnant women who have been exposed to parvovirus infection (erythema infectiosum/fifth disease) should be offered serological testing for parvovirus-specific IgG to determine their susceptibility.

The diagnosis of parvovirus infection is usually made, serologically, by demonstration of IgG seroconversion and/or the presence of parvovirus IgM. IgM is usually detectable within 1-3 weeks of exposure and lasts for 2-3 months. Various serological methods are available, of which enzyme immunoassay and immunofluorescence are most commonly used. The sensitivities of IgM assays vary; they are highest in adults with typical acute parvovirus infection and arthropathy but lower in children. They are generally highly specific (specificity at least 95%) in asymptomatic controls but false positive results can occur (specificity 70-85%) in patients with other acute infections, including rubella.[14]

Management of proven maternal parvovirus infection in pregnant women

No intervention is available to prevent fetal infection or damage. Because of the low risk of fetal damage neither termination of pregnancy nor amniocentesis for diagnosis of asymptomatic intrauterine fetal infection is recommended. Repeated ultrasound examination by an experienced specialist to detect hydrops is recommended.[12,15] If hydrops is detected, further assessment is indicated to determine the need for treatment (intrauterine transfusion).

The role of parvovirus infection in hydrops fetalis

Parvovirus is implicated in 5-15% of cases of non-immune hydrops fetalis. Investigations for possible previous maternal parvovirus infection include maternal history of viral illness or contact, serological testing for maternal parvovirus IgG seroconversion* and IgM (the latter is often negative by the time hydrops develops). The diagnosis of intrauterine parvovirus infection can be confirmed by amniocentesis and polymerase chain reaction for parvovirus DNA, if indicated.

* Serum collected at the time of presentation should be tested for IgG in parallel with stored serum collected for antenatal screening (if available; laboratories generally store serum from pregnant women for at least 12 months).

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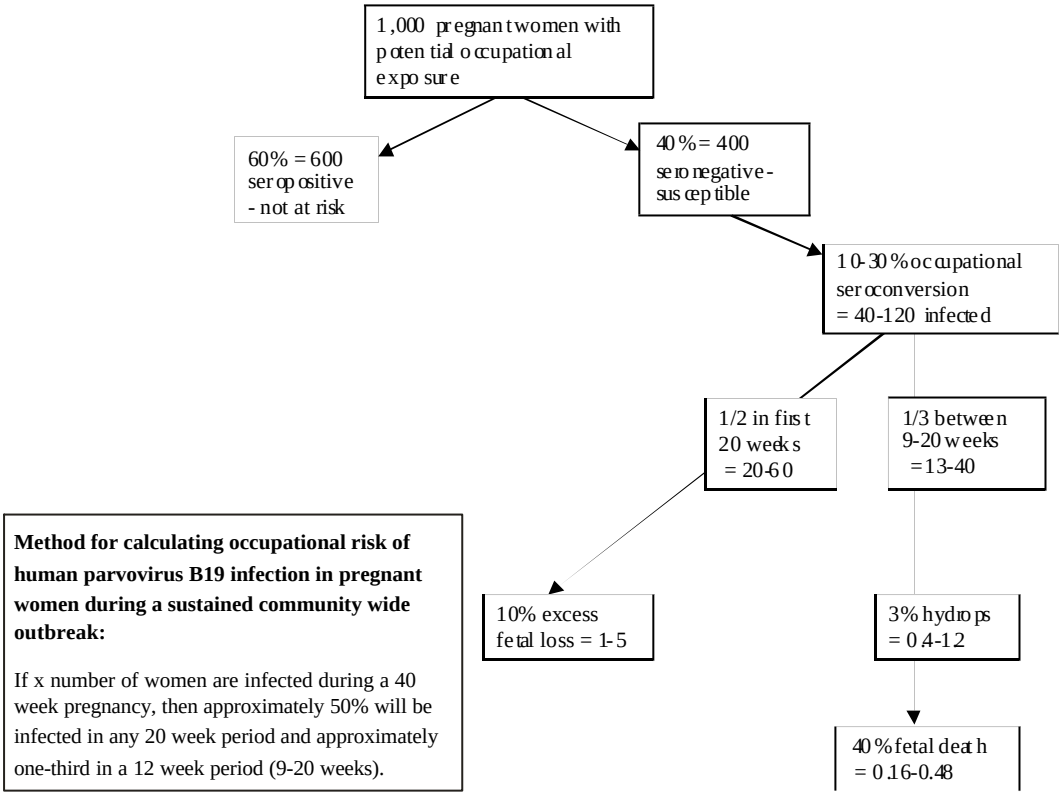
Figure 1. Risks of occupational exposure of 1,000 pregnant women to parvovirus B19 infection during an epidemic (all data are approximate).



Outcome from hydrops fetalis

Hydrops should be managed by a specialist with experience in intrauterine transfusion. Limited data from several reviews indicate the following outcomes, depending on the severity of fetal anaemia and hydrops:[12,15,16]

- spontaneous resolution in about one-third of cases;
- fetal death occurs within a few days of diagnosis in about one-third of cases;
- intrauterine transfusion in about one-third of cases, with a success rate of about 80% and fetal death in a small minority; and
- there is circumstantial evidence that the prognosis can be significantly improved by intrauterine transfusion in cases with severe anaemia and hydrops.

Conclusion

Parvovirus B19 infection is generally benign. There is a small risk of serious adverse sequelae in some high risk individuals, including the fetus. However, the low risk following infection in pregnancy does not justify routine exclusion of susceptible pregnant women from working with children during epidemics.

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