Parvovirus B 19 constitutes a non-enveloped human DNA virus of world-wide distribution whose single-stranded genome comprises 5.6 kb (Shade et al, 1986). It was first discovered in the sera of healthy blood donors and patients, one of whom had acute hepatitis (Cossart et al, 1975). Human parvovirus B 19 infection is rather common, with seroprevalence rates in adults amounting to approximately 50%. At the age of 70, seroprevalence reaches to more than 80% (van Elsacker-Niele and Kroes, 1999). It displays a broad spectrum of clinical manifestations such as erythema infectiosum in children, aplastic crisis in patients with hemolytic anemia, chronic bone marrow failure in immunocompromised hosts, and hydrops fetalis after intrauterine infection (Yoto et al, 1996). Infants surviving parvovirus B 19 induced hydrops fetalis can have congenital hepatic dysfunction (Metzman et al, 1989) and those who die have hepatitis (Naides, 1993).

Altogether, parvovirus B 19 is a ubiquitous virus so that by 15 years of age, approximately 50% of individuals display serologic evidence of past infection (van Elsacker-Niele and Kroes, 1999) which may have presented as the common childhood disease erythema infectiosum. Infection with this agent can induce several clinical manifestations of varying severity, depending on the respective patient’s immune status. Along those lines, aplastic crisis in chronic hemolytic anemia, exanthematous disease and arthropathy, mainly in women, and chronic anemia in the immunocompromised host have been observed. After initial replication, probably in the respiratory tract, the virus enters its target cells in the bone marrow, erythroid precursor cells, through its receptor, the blood group P antigen (van Elsacker Niele and Kroes, 1999). Ensuing viral replication leads to an arrest in erythropoiesis which usually lasts approximately 1 week. At this stage, patients under ‘erythropoietic stress’ can experience an aplastic crisis. Viremia ceases as virus-specific antibodies appear in the patient’s serum, which may trigger potentially immune-mediated symptoms such as a rash or arthralgia. Thereupon, at least in immunologically normal individuals, the infection is cleared within several weeks by humoral immune response, with detectable specific IgG conferring lifelong immunity to reinfection. In patients with dysfunctional or altogether absent humoral immunity, on the other hand, persistent infection resulting in chronic suppression of erythropoiesis with chronic anemia can occur (van Elsacker-Niele and Kroes, 1999).
In addition, in a pediatric patient group surprising similarities of presentation between human parvovirus B 19 infection and systemic lupus erythematosus (SLE) have been observed in that the patients not only displayed SLE-like symptomatology but also positive serology suggestive of SLE (Moore et al., 1999).

Since its discovery in 1975 (Cossart et al., 1975), the prevalence of parvovirus B 19 infection has been investigated in various geographic locations, such as the United Kingdom (Cohen and Buckley, 1989), Hong Kong (Lim et al., 1997), Taiwan (Lin et al., 1999) and Brazil (Nascimento et al., 1990), among patients positive for the human immunodeficiency virus in Spain (Negredo et al., 1998) and Sweden (Gyllensten et al., 1994), as well as among patient groups with defined hematologic diseases in the United States (Pardi et al., 1998; Ragni et al., 1996). In contrast, data for Thailand are altogether non-existent. Hence, particularly in the context of transfusion- and transplant-related viral hepatitis which might affect pediatric patients receiving immunosuppressive therapy, our group initiated an investigation into the prevalence of parvovirus B 19 for epidemiological evaluation in Thailand.

The population investigated comprised three groups consisting of (a) 30 healthy children who attended the well-baby clinic, Chulalongkorn Hospital, for the purpose of hepatitis B immunization and/or follow-up, (b) 11 children with acute illness or elective surgery admitted to the Department of Pediatrics, Chulalongkorn Hospital, as well as 53 admitted to Hat Yai Hospital, Songkhla Province, and (c) 35 voluntary blood donors between 16 and 51 years at the National Blood Center, Thai Red Cross.

Blood samples were obtained in the course of two years, between 1998 and 1999, sera were separated by centrifugation and stored at -20°C until further analysis. The sera were subjected to enzyme linked immunosorbent assay (ELISA) using the commercially available Human Parvovirus B19 (recombinant) ELISA kit (Genzyme Virotech GmbH, Germany) according to the manufacturer’s specifications.

As shown in Fig 1, immunity to parvovirus B 19 increases with age among healthy individuals in Thailand. Consequently, the percentage of anti-parvovirus B 19 IgG rose steadily from 11.9% (5/42) within the lowest age group (0-6 years) over 19.05% (8/42) and 25% (3/12) within the intermediate age groups (7-12 and 13-19 years, respectively) to 30.3% (10/33) within the highest age group tested (20-51 years). The overall prevalence amounted to 20.16% (26/129).

From the age-specific prevalence of anti-parvovirus B 19 IgG established for Thailand, we can conclude that the local population encounters this agent at frequencies comparable with those determined for some other countries in Asia. Along those lines, the overall prevalence of anti-parvovirus B 19 IgG and IgM was found to amount to 32.8% and 0.35%, respectively, in Taiwan (Lin et al., 1999). In Hong Kong, between 1983 and 1993, a low incidence of parvovirus infection leading to a shift in the prevalence rate among the general population was observed in that from 1991 to 1996, only 2.5% of patients presenting with illness potentially caused by parvovirus B 19 were positive for IgM and 19.6% for IgG (Lim et al., 1997). A seroepidemiological survey conducted in Singapore showed the prevalence of anti-parvovirus B 19 IgG at 0% among children below the age of 5 years, 3.5% among the 5-to-14-year olds, 7.7% among teenagers between 15 and 19 years, 10.3% among those between 20 and 24, 28% among those between 25 and 34, and 65% among those above the age of 35 years (Matsunaga et al., 1994). This study also very clearly mirrors the age dependent frequency of anti-parvovirus B 19 which we have observed in our study. Age-dependent antibody prevalence has also been demonstrated, along with a drastically increased frequency, among the population of Rio de Janeiro (Nascimento et al., 1990) where anti-parvovirus B 19 IgG was demonstrated at 35% among children below the age of 5 years, at almost 80% in children between 11 and 15 years old, and above 90% among those older than 50 years.

Particularly with reference to the Brazilian study, it appears as though parvovirus B 19 infection were generally less prevalent in Southeast Asia than else-
where. Elucidation of the reasons underlying this difference, as well as the clinical significance attributable to the virus infecting immunocompromised hosts in the same environment will require further investigations.

ACKNOWLEDGEMENTS

We would like to thank Dr Chupuppakran, Hat Yai Hospital, National Blood Center, Thai Red Cross, for providing the blood specimens, and the Thailand Research Fund, Senior Research Scholar for supporting the study presented.

REFERENCE


