Microbiology

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Parvoviruses

- **Structure**
  - Parvoviruses are *very small* (18 to 26 nm), *naked virions* that contain a *linear single-stranded DNA* (the smallest DNA animal viruses)
  - *Icosahedral* with 32 capsomers and 2 protein coats (VP2 and VP1)
  - Parvovirus *B19* is pathogenic for humans

- **Replication and control**
  - Because of the limited coding capacity of their genome, viral replication is dependent on functions supplied by replicating host cells or by co-infecting helper viruses
  - Autonomously *replicating* and defective parvoviruses that require a *helper virus* for replication.
  - It is difficult to culture human B19 parvovirus
  - Viral *DNA* replication occurs in the *nucleus*
  - Viral *replication* results in *cell death*
  - Virions are extremely *resistant* to inactivation.
    - ✓ They are stable between a *pH* of 3 and 9
    - ✓ Withstand *heating* at 56°C for 60 minutes
    - ✓ Can be *inactivated* by formalin, and oxidizing agents

- **Parvovirus B19 Infections**
  - ***Viral target***
    - ✓ The cellular receptor for B19 is blood group P antigen (globoside).
    - ✓ P antigen is expressed on mature erythrocytes, erythroid progenitors, megakaryocytes, endothelial cells, placenta, and fetal liver and heart, which helps explain the narrow tissue tropism of B19 virus
    - ✓ The major sites of virus replication in patients are assumed to be the adult marrow, some blood cells, and the fetal liver
    - ✓ A primary site of replication appears to be the nucleus of an immature cell in the erythrocyte lineage.

- **Epidemiology**
  - The viral infection is common among *children* 5-15 years old
  - Epidemiologic evidence suggests that spread of the virus is primarily by the respiratory route, and high transmission rates occur in *households*
  - Once *skin rash* appear the virus is *no* more *contagious*

- **Pathogenesis**
  - Viral replication causes cell death interrupting red blood cell production (anemia)
  - Bone marrow biopsies from infected patients show erythrocyte maturation arrest, with erythroblast intranuclear inclusions
  - Both virus-specific IgM and IgG antibodies are made after B19 infections which form immune complex
  - The clinical consequences of the viral effect on erythrocytes are generally trivial, unless patients are already compromised by a chronic hemolytic process, such as sickle cell disease or thalassemia or in immunocompromised patients

1. *Erythema infectiosum*

- ✓ Erythema infectiosum (also referred to as fifth disease, slapped check, apple face, or academy rash)
- ✓ After an incubation period of 4 to 12 days, a mild illness appears, characterized by fever, malaise, headache, myalgia, and itching in varying degrees
- ✓ Viremia occurs 1 week after infection and persists for about 5 days
- ✓ A confluent, indurated rash appears on the face, giving a "slapped-cheek" appearance. The rash spreads in a day or two to other areas, particularly exposed surfaces such as the arms and legs, where it is usually macular and reticular
- ✓ During the acute phase, generalized lymphadenopathy or splenomegaly may be seen, along with a mild leukopenia and anemia
- ✓ The illness lasts 1 to 2 weeks, but rash may recur for periods of 2 to 4 weeks thereafter, exacerbated by heat, sunlight, exercise, or emotional stress
- ✓ Arthralgia sometimes persists or recurs for weeks to months, particularly in adolescent or adult females
- ✓ Serious complications, such as hepatitis, thrombocytopenia, nephritis or encephalitis are rare

2. *Transient aplastic crisis*

- ✓ Transient aplastic crisis may complicate chronic hemolytic anemia (sickle cell disease, thalassemias), acquired hemolytic anemias in adults, and after bone marrow transplantation.
- ✓ Abrupt cessation of RBCs synthesis in the bone marrow (reduction of erythroid precursors), accompanied by a rapid worsening of anemia.
- ✓ The infection lowers production of erythrocytes, causing a reduction in the hemoglobin level.
- ✓ The temporary arrest of production of RBCs becomes apparent only in patients with chronic hemolytic anemia because of the shortened life span of their erythrocytes

3. *Pure red cell aplasia*

- ✓ B19 may establish persistent infections and cause chronic suppression of bone marrow and chronic anemia in immunocompromised patients.
- ✓ The anemia is severe, and patients are dependent on blood transfusions.

4. *Hydrops fetalis*

- ✓ Maternal infection with B19 virus may pose a serious risk to the fetus, resulting in hydrops fetalis and fetal death due to severe anemia.
- ✓ The overall risk of human parvovirus infection during pregnancy is low; fetal loss occurs in fewer than 10% of primary maternal infections.
- ✓ Fetal death occurs most commonly before the 20th week of pregnancy.

- **Diagnosis**
  - ➢ Viremia usually lasts 7 to 12 days but can persist for months in some individuals
  - ➢ CBC (low Hb)
  - ➢ Polymerase chain reaction (PCR)
  - ➢ IgM-specific antibody late in the acute phase or during convalescence strongly supports the diagnosis
  - ➢ Antigen detection assays
  - ➢ Bone marrow biopsy

- **Management**
  - Fifth disease and transient aplastic crisis are treated symptomatically
  - Severe anemia due to the latter may require transfusion therapy
  - Commercial immunoglobulin preparations contain neutralizing antibodies to human parvovirus. These can sometimes ameliorate persistent B19 infections in immunocompromised patients and in those with anemia
  - There is *no vaccine* against human parvovirus
  - There is *no antiviral* drug therapy

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