- During normal development, the fetus is in a protected intrauterine environment, with fetal membranes serving as a physical barrier to external infection and the placenta contributing, with maternal immunity, to protection against many blood-borne infections. Transplacental transmission of specific immunoglobulins, particularly of the IgG class, continues to provide some immunologic protection to the infant for weeks to months after birth, while cytokines from the mother can provide transient cell-mediated immune support. If the infant is breast-fed, specific immunoglobulins (predominantly of the IgA class) in maternal colostrum afford some protection against pathogens that involve or invade through the infant's gastrointestinal tract.
- On the other hand, the fetal immune system is immature, and there is relative suppression of maternal cell-mediated immunity as pregnancy progresses. These immune deficiencies serve an important biological purpose; they protect fetus and mother from activation of specific immunologic recognition and response mechanisms to differences in their histocompatibility locus antigens. If these processes did not occur normally, the fetus could be immunologically rejected by the mother or the fetal immune mechanisms activated to respond against maternal antigens in a form of "graft versus host" disease.
- Specific and nonspecific immune responses begin to develop in early fetal life, perhaps as early as 8 weeks' gestation; however, a nearly normal immunocompetent state is usually not achieved until the infant is more than 2 years of age. Deficiencies commonly seen in the early period include poor antibody response to polysaccharide antigens, decreased phagocytic capability and variability in intracellular killing of certain infectious agents, lower levels of complement components, and decreased opsonic capacity.

Definitions:
Prenatal infections include those acquired by the mother and/or fetus at any time before birth.
Natal infections are those acquired during delivery.
Postnatal infections: acquired after delivery throughout the newborn (or neonatal) period, defined as the first 4 weeks of life.
C**ongenital** infection, which describes infection occurring at any time before or at birth (prenatal or natal).

Modes of Infection and Major Agents

Mode	Bacteria
Prenatal transplacental	*Listeria monocytogenes*, *Mycobacterium tuberculosis* (rare), *Treponema pallidum*
Ascending	Group B streptococci, *Escherichia coli*, *L. monocytogenes*
Natal	Group B streptococci; *E. coli*, *L. monocytogenes*, *Neisseria gonorrhoeae*
Postnatal	E. coli, group B streptococci, *L. monocytogenes*, miscellaneous Gram-negative bacteria, *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Clostridium tetani*

C. trachomatis and gonococci produce severe conjunctivitis

Chlamydia infant pneumonia syndrome occurs in infants up to 6 months of age

Postnatal infections by *S. aureus* may cause scalded skin syndrome

Infection of Immunocompromised patients

Immunocompromised patients are those whose host defense mechanisms are impaired by an inherited deficit, disease, or treatment. The immunocompromised state increases the risk of infection with many of the common pathogens as well as with low-virulence organisms present in the normal flora or environment.

The organisms involved are those mostly able to take advantage of situations such as disruption of the skin or mucosal barriers and the more specific immune defects, including (1) defects in the phagocytic response, (2) defects in the complement system, (3) defects in antibody-mediated immunity, (4) defects in cell-mediated immunity, and (5) loss of reticuloendothelial function.

Each of these defects tends to be associated with infections caused by specific groups of organisms. For example, neutropenia and disorders of phagocytosis are associated with infections by Gram-positive cocci, Enterobacteriaceae, *Pseudomonas,* and fungi. In contrast, patients with defects in cell-mediated immunity tend to have severe viral, parasitic, and fungal infections or disease caused by bacteria that can multiply intracellularly (eg, mycobacteria). Those with defects in antibody production, such as agammaglobulinemia, are prone to infection with encapsulated organisms such as *Streptococcus pneumoniae* and *Haemophilus influenzae* type b.

DIAGNOSIS

Clinical recognition and treatment of infections in the immunocompromised patient are often difficult, because the infection may be relatively silent due to impairment of the immune response. Laboratory diagnosis can also be difficult, because many of the organisms involved require special culture media and grow slowly; others cannot be grown at all. The increased involvement of low-virulence organisms commonly found in the normal flora may make it difficult to distinguish colonization from infection.