Fetal and neonatal infections

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Abstract

Several organisms cross the placenta, causing infections in the fetus that manifest differently depending on the organism and the time of acquisition during pregnancy. Neonates are relatively immunocompromised, and prematurity increases the risk of infection. Newborns acquire infections during delivery and breastfeeding (vertical infections) or in the neonatal period from the environment (horizontal acquisition). Hospital-acquired infections are common in neonatal intensive care units and can pose serious infection control issues. This chapter addresses the most common agents causing congenital and neonatal infections, their clinical manifestations, management and prophylaxis.

Keywords CMV; cross-infection; fetus; GBS; HSV; MRCP; newborn; sepsis; syphilis; *Toxoplasma*; VZV; Zika

Key points

- Oral valganciclovir is now recommended for 6 months to treat congenital CMV infections when diagnosed in the newborn period
- A new congenital syndrome likely to be caused by Zika virus has been described and is characterized by microcephaly and CNS abnormalities, travellers to the affected countries needs to be counselled about the need for barrier contraception methods
- The incidence of herpes simplex virus is increasing. New prophylaxis guidelines have been recently published
- In low- and middle-income countries, neonatal infections are responsible for just under 1 million deaths in the under-5s. In high-income countries, hospital-acquired infections are more common in neonates than any other age group. Emergence of antimicrobial resistance needs to be considered when empirical antibiotic choices are made

Infections in the fetus – intrauterine infections

Several pathogens affect pregnant women and can cause disease in the fetus. These range from early intrauterine death resulting in miscarriage and stillbirth to intrauterine growth restriction (IUGR), congenital malformations and congenital infections. Infections in the first trimester are generally more likely to cause severe defects and can result in stillbirth. Infants can be symptomatic in early life, or symptoms and signs can manifest later in life (Table 1). These protean manifestations depend on the organism, the time of infection in relation to the pregnancy and maternal immune status.

Pathogenesis

Infectious agents can affect the developing fetus in different ways (Figure 1):

- by infecting the placenta and interfering with fetal nutrition and gas exchange, causing intrauterine death and compromising fetal growth (IUGR) (e.g. placental malaria)
- by compromising the development of specific organs for which the organism has a specific tropism (e.g. rubella)
- by infecting the fetus (e.g. hepatitis B or HIV).

The likelihood of an organism affecting the fetus depends on the time of acquisition during pregnancy and varies with different pathogens: congenital toxoplasmosis is more likely if infection is later in pregnancy (30–75%), while rubella transmission occurs early.

The mother's immune status is important, and primary infection during pregnancy is associated with a high likelihood of fetal involvement. In the case of rubella, where the risk of transmission to the fetus is limited to the first infection and an effective vaccine is available, congenital infections have almost disappeared in high-income countries.

The most common congenital infection in high-income countries is currently cytomegalovirus (CMV; the incidence of congenital CMV in the UK is three per 1000). Worldwide, HIV and syphilis are the most common congenital infections, with about 330,000 new HIV infections per year and about 500,000 adverse pregnancy outcomes resulting from syphilis each year.[1]

In 2015, Zika virus infection was linked to a newly described congenital syndrome characterized by microcephaly, although causality has yet to be conclusively proven. Zika cases in adults have currently been reported in more than 80 countries.

Clinical manifestations, investigations and management

The clinical manifestations of congenital infections are variable. Infection during the early stages of pregnancy severely compromises embryogenesis and can result in fetal demise, congenital malformations and fetal growth restriction, resulting in IUGR. Infections later in pregnancy can result in asymptomatic babies at birth who might remain asymptomatic or progress to develop signs of infection at a later stage. Pathogens can cause a constellation of signs such as rashes, lymphadenopathy, hepatosplenomegaly, jaundice and intracranial calcifications. Characteristic congenital malformations are described for some infections (Table 1).

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Congenital infections

Viruses	Time of acquisition	Maternal immunity	Manifestation at birth	Treatment and prevention
Cytomegalovirus (CMV)	Mostly during third trimester	Primary infection > reinfections	Asymptomatic (90%) Blueberry muffin, petechial rash, hepatosplenomegaly, microcephaly, ocular involvement, intracranial calcifications, jaundice, thrombocytopenia, abnormal liver function tests Later development of hearing loss	Oral valganciclovir for 6 months if congenital CMV is diagnosed in the neonatal period and the infant is symptomatic
HIV	Mostly during delivery and breastfeeding	Risk increases with high viral load and low CD4 cell count	Asymptomatic or lymphadenopathy, hepatosplenomegaly	Highly active antiretroviral combination therapy in mothers and avoidance of breastfeeding where safe, affordable and sustainable, exclusive breastfeeding if not possible
Hepatitis B	Typically chronic carriage in mother	Risk of transmission: HbSAg+ HbeAg−: 5−20% HbSAg+ HbeAg+: 70−90%	Asymptomatic	Vaccination at birth if mother HbSAg+ HbeAg− Passive and active vaccination at birth if HbSAg+ HbeAg+ *Prevention:* vaccination
Zika virus	Unknown	Likely primary infection	Microcephaly, cortical atrophy, brainstem abnormality, craniofacial disproportion, seizures, spasticity, cardiac malformations, digestive system malformations	Prevention of infection in pregnancy through avoidance of travel to high-risk areas, protection against mosquito bites, avoidance of pregnancy No treatment is available
Parvovirus B19	Mostly during the first 20 weeks	Primary infection	Hydrops fetalis Anaemia	There are case reports of successful intravenous Ig use for treatment
Varicella-zoster	Mostly during the first 20 weeks	Primary infection	Asymptomatic, congenital scarring of the skin, cutaneous defects, bullous lesions, asymmetrical limb hypoplasia and autonomic dysfunction, eye defects, seizures and mental retardation	*Prevention*: • Vaccination before pregnancy • Varicella-zoster Ig after exposure
Bacteria				
Rubella	Mostly in the first trimester (80%)	Primary infection	Asymptomatic, IUGR, thrombocytopenic purpura, hepatosplenomegaly, lymphadenopathy, jaundice, eye involvement, cardiac abnormalities, pneumonitis, meningo-encephalitis, bone lesions, cryptorchidism, haemolytic anaemia Progressive disease in some cases	*Prevention:* vaccination before pregnancy
Treponema pallidum	From 14 weeks' gestation, risk increases progressively	Primary infection 70−100% transmission Secondary infection 40% transmission	Asymptomatic Petechial, vesciculo-bullous or macular rash affecting also palms and soles, hepatosplenomegaly, lymphadenopathy, rhinorrhoea, jaundice, anaemia, CSF abnormalities, bone abnormalities on X-ray	Parenteral benzylpenicillin for 10 days
Mycobacterium tuberculosis	Rare, transmission can be *in utero* and at delivery		Asymptomatic Miliary tuberculosis	Quadruple antimycobacterial therapy

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