ABSTRACT

Congenital infections refer to a group of perinatal infections that are caused by pathogens transmitted from mother to child during pregnancy (transplacentally) or delivery (peripartum) which may have similar clinical presentations, including rash and ocular findings. TORCH is the acronym that covers these infections (toxoplasmosis, other [syphilis], rubella, cytomegalovirus, herpes simplex virus). Other important causes of intrauterine/perinatal infection include human immunodeficiency virus, varicella-zoster virus, Treponema pallidum, Zika virus, and parvovirus B19. This overview aims to describe various congenital infections beyond TORCH with a Hong Kong perspective. Intrauterine and perinatal infections are a major cause of in utero death and neonatal mortality, and an important contributor to childhood morbidity. A high index of suspicion for congenital infections and awareness of the prominent features of the most common congenital infections can help to facilitate early diagnosis, tailor appropriate diagnostic evaluation, and initiate appropriate early treatment. Intrauterine infections should be suspected in newborns with clinical features including microcephaly, seizures, cataract, hearing loss, congenital heart disease, hepatosplenomegaly, small for gestational age, and/or rash. Primary prevention of maternal infections during pregnancy is key to the prevention of congenital infection, and resources (if available) should focus on public health promotion and pre-marital counselling.

Introduction

Congenital infections are those that can cross the placenta and damage the fetus in utero or transmit to the infant during delivery in the peripartum period, resulting in neonatal infection.1 Congenital infections also account for 2% to 3% of all congenital anomalies apart from miscarriage, stillbirth, and neonatal deaths and are a significant cause of childhood morbidity.2-4 Immunologist Andres Nahmias first used the acronym ToRCH in 1971 to describe perinatal infections associated with toxoplasma (To), rubella (R), cytomegalovirus (C), and herpes simplex virus (H).5 Subsequently, the ‘O’ in TORCH has been broadened and now stands for ‘Others’, including the following pathogens: syphilis, parvovirus B19, coxsackievirus, listeriosis, hepatitis virus, varicella-zoster virus (VZV), Trypansomoma cruzi, enterovirus, human immunodeficiency virus (HIV), and the latest addition, Zika virus (ZIKV).1,4

Congenital infections have remained a major global health issue, those in developing countries are particularly vulnerable. Congenital infections can lead to significant consequences, such as severe disabilities or even the death of the fetus. We recently overviewed “TORCH” in detail,6 and this overview focuses on the following congenital infections beyond TORCH, namely, HIV, VZV, ZIKV, and parvovirus B19. References were searched using key terms (“congenital infection”) and (“Hong Kong”) or (“Zika”) and (“Hong Kong”) in PubMed, limited to ‘human’, with no filters on article type or publication time. Discussion is based on but not limited to the search results.

Congenital human immunodeficiency virus infection

The HIV strains HIV-1 and HIV-2 are cytopathic lentiviruses belonging to the family Retroviridae.7 Paediatric HIV infection remains a significant global health issue: the Joint United Nations Programme on HIV/AIDS reported that an estimated 3.1 million children were living with HIV globally.8 Congenital infection with HIV can occur via the transplacental route to the developing fetus. Over 90% of HIV infections in children worldwide are caused by MTCT, which is one of the three general modes of HIV transmission.9 The first case of HIV infection in Hong Kong was reported in 1984. The Department of Health has had a voluntary and anonymous HIV/AIDS
前諮詢。先天性感染的關鍵，並在資源許可的情況下應集中推廣公共衛生和婚病、肝脾腫大、胎齡細小及皮疹。妊娠期母親感染的一級預防是預防兒宮內感染，包括小頭畸形、癲癇、白內障、聽力下降、先天性心臟制定合適的診斷評估和及早治療。當出現以下臨床特徵時應懷疑新生高警覺以及加強對最常見先天性感染顯著特徵的認識有助早期診斷、以外的各種先天性感染。宮內和圍產期感染是子宮內死亡和TORCH水痘帶狀皰疹病毒、梅毒、寨卡病毒和細小病毒。本文概述香港TORCH以外的先天性感染概述。宮內和圍產期感染的其他重要因素包括HIV、巨細胞病毒（C）、其他包括梅毒（O）、德國麻疹（B）、巨細胞病毒（C）和單純皰疹病毒（H）感染的首字母縮寫。宮內和圍產期感染的其他重要因素包括HIV、水痘帶狀皰疹病毒、梅毒、寨卡病毒和細小病毒。本文概述香港TORCH以外的各種先天性感染。宮內和圍產期感染是子宮內死亡和新生兒死亡的主因，也是導致兒童發病的重要因素。對先天性感染提高警覺以及加強對最常見先天性感染顯著特徵的認識有助早期診斷、制定合適的診斷評估和及早治療。當出現以下臨床特徵時應懷疑新生兒宮內感染，包括小頭畸形、癲癇、白內障、聽力下降、先天性心臟病、肝脾腫大、胎齡細小及皮疹。妊娠期母親感染的一級預防是預防先天性感染的關鍵，並在資源許可的情況下應集中推廣公共衛生和婚前諮詢。

報名香港以外的先天性感染概述

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Congenital infections

of the upper respiratory tract. Varicella is highly contagious through the conjunctiva or the mucosal surface, occasionally through airborne spread, entering the infected respiratory tract secretions, direct contact of VZV can occur via multiple routes, including transmission of VZV from maternal infection can result in a range of adverse sequelae. Low birth weight is universal, and intrauterine growth restriction is observed in around 23% of cases. Further, approximately 20% of infants with in utero VZV infection develop neonatal or infantile herpes zoster. Congenital anomalies resulting from congenital VZV infection are known collectively as CVS, which is expected in approximately 12% of infected fetuses. Maternal infection within the first 20 weeks of gestation is responsible for most cases of CVS, with newborns of mothers infected between the 7th and 20th weeks having the highest risk. No cases of CVS have been reported when maternal varicella infection occurs after the 28th week of gestation. The clinical manifestations of CVS include cutaneous scars in a dermatomal distribution, limb hypoplasia, microcephaly, cortical atrophy, hydrocephaly, mental retardation, microphthalmia, chorioretinitis, cataracts, muscle hypoplasia, developmental delay, and anomalies of the cardiovascular system, gastrointestinal tract, and genitourinary tract. During the first few months of life, CVS has a 30% mortality rate. Neonatal VZV infection can result from transplacental passage of VZV and ascending or postnatal infections. If VZV infection occurs during the first 10 to 12 days of life, it is often the result of intrauterine transmission, whereas VZV with later onset is usually acquired postnatally. Appropriate treatment with varicella-zoster immunoglobulin (VZIG) and antivirals has reduced the mortality rate of neonatal VZV to 7%. Prenatal diagnosis of CVS is often based on detection of VZV DNA in the amniotic fluid or fetal blood by PCR. In addition, ultrasonography findings consistent with CVS, such as microcephaly, limb deformities, polyhydramnios, soft tissue calcification, and intrauterine growth restriction can be diagnostic when there is a history of maternal VZV infection. An ultrasound examination should be performed at least 5 weeks after the onset of rash in the mother.

Congenital varicella syndrome

Varicella-zoster virus is a member of the herpesvirus family. Pregnant women are several times more likely to develop fatal VZV than non-pregnant ones. Although rare, the fetus is at high risk of congenital varicella syndrome (CVS) if the mother is infected, and the neonate is at high risk of a severe or fatal form of VZV. Varicella is the most frequently reported notifiable disease in Hong Kong. More than 95% of the population not vaccinated against VZV has been infected, mostly before 20 years of age. Approximately 95% of pregnant women were found to be seropositive for VZV in a 2009 study. With the vast majority of the population immune to VZV at childbearing age, the risk of primary infection during pregnancy (and thus the risk of congenital VZV infection) is relatively low. A worldwide systemic review of the literature from 1947 to 2013 indicated that there were only 130 reported cases of congenital VZV infection in that period. Only two cases of neonatal VZV infection were reported in Hong Kong from 2008 to 2010. Transmission of VZV can occur via multiple routes, including infected respiratory tract secretions, direct contact with infectious vesicular fluid from moist lesions, and occasionally through airborne spread, entering the host through the conjunctiva or the mucosal surface of the upper respiratory tract. Varicella is highly contagious from 1 to 2 days before the onset of the rash, until all lesions are crusted. Transplacental passage of VZV from maternal infection can result in congenital or neonatal VZV infection. The risk of vertical transmission reaches 25% if maternal primary infection occurs during the first or second trimesters, with around 12% risk of congenital anomalies among newborns of mothers who were infected during that period. The rate of vertical transmission reaches up to 50% when maternal VZV infection occurs within 1 to 4 weeks before delivery, with 23% developing neonatal VZV infection. Fetal VZV infection in utero can result in a range of adverse sequelae. Low birth weight is universal, and intrauterine growth restriction is observed in around 23% of cases. Further, approximately 20% of infants with in utero VZV infection develop neonatal or infantile herpes zoster. Congenital anomalies resulting from congenital VZV infection are known collectively as CVS, which is expected in approximately 12% of infected fetuses. Maternal infection within the first 20 weeks of gestation is responsible for most cases of CVS, with newborns of mothers infected between the 7th and 20th weeks having the highest risk. No cases of CVS have been reported when maternal varicella infection occurs after the 28th week of gestation. The clinical manifestations of CVS include cutaneous scars in a dermatomal distribution, limb hypoplasia, microcephaly, cortical atrophy, hydrocephaly, mental retardation, microphthalmia, chorioretinitis, cataracts, muscle hypoplasia, developmental delay, and anomalies of the cardiovascular system, gastrointestinal tract, and genitourinary tract. During the first few months of life, CVS has a 30% mortality rate. Neonatal VZV infection can result from transplacental passage of VZV and ascending or postnatal infections. If VZV infection occurs during the first 10 to 12 days of life, it is often the result of intrauterine transmission, whereas VZV with later onset is usually acquired postnatally. Appropriate treatment with varicella-zoster immunoglobulin (VZIG) and antivirals has reduced the mortality rate of neonatal VZV to 7%. Prenatal diagnosis of CVS is often based on detection of VZV DNA in the amniotic fluid or fetal blood by PCR. In addition, ultrasonography findings consistent with CVS, such as microcephaly, limb deformities, polyhydramnios, soft tissue calcification, and intrauterine growth restriction can be diagnostic when there is a history of maternal VZV infection. An ultrasound examination should be performed at least 5 weeks after the onset of rash in the mother.

Congenital varicella syndrome is diagnosed postnatally when there is history of maternal VZV during pregnancy, in the presence of skin lesions distributed along dermatome(s), and in the presence of other clinical manifestations consistent with CVS or neonatal seizures. The presence of VZV DNA in the newborn, detection of immunoglobulin M antibodies against VZV in the cord blood or fetal blood, immunoglobulin G antibodies against VZV persisting longer than the first 7 months of life, or development of herpes zoster during early infancy
incidence during pregnancy remain to be elucidated.

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95%. Because the incidence of varicella in younger
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vaccine into the HKCIP, as coverage of all other
exposure can effectively prevent infection, especially
age who are not immune to VZV, vaccination after
exposed to VZV within 72 and up to 96 hours of
prophylaxis if the mother has perinatal varicella
infection regardless of CVS development.30
Oral acyclovir with or without VZIG is
recommended for pregnant women infected with
varicella, as it can shorten the duration of fever and
reduce the symptoms of varicella when antiviral
therapy is initiated within 24 hours after the onset
of rash.38,39 Acyclovir is effective at lowering the
morbidity and mortality rates of both the fetus and
the mother when administered within 24 and up to
72 hours of the onset of rash, and VZIG has been
reported to lower the incidence and severity of
varicella.30 A 10-day course of intravenous acyclovir
is effective in neonates affected by varicella and
complications of CVS.7 Prophylactic use of VZIG
is also possible in susceptible pregnant mothers
exposed to VZV within 72 and up to 96 hours of
exposure but is not effective after clinical signs have
appeared.37 Administration of VZIG in neonates
should occur within 10 days of initial exposure for
prophylaxis if the mother has perinatal varicella
rash and absence of antibodies against VZV in the
mother or the neonate.7 In women of childbearing
age who are not immune to VZV, vaccination after
exposure can effectively prevent infection, especially
when administered within 3 days of exposure.38

Vaccination against VZV was incorporated
into the Hong Kong Childhood Immunisation
Programme (HKCIP) in 2014.39 For eligible children
born on or after 1 January 2013, the first vaccination
is given at age 12 months, and the second dose
(at around age 6–7 years) in primary one, given as
part of the measles mumps, rubella, and varicella
vaccine. Before being incorporated into the HKCIP,
a varicella vaccine was available on the private
market. A local study conducted in 2012 found
that the parent-reported VZV vaccination uptake
was 65% in kindergarten students, and the rate was
69% of preschool and schoolchildren in another
study conducted in 2013.40,41 The vaccination rate is
expected to rise after incorporation of the varicella
vaccine into the HKCIP, as coverage of all other
vaccines in the HKCIP has been maintained at over
95%. Because the incidence of varicella in younger
children is likely to drop because of increased
immunisation, the effects of the vaccination on
incidence during pregnancy remain to be elucidated.

Congenital Zika virus infection
Zika virus is a neurotropic flavivirus that particularly
infests neural progenitor cells. Approximately 80%
of ZIKV infection is asymptomatic.42 Symptomatic
infections can be categorised as either Zika fever or
congenital Zika syndrome. Zika fever is a relatively
mild disease that presents with low-grade fever,
generalised maculopapular rash, non-purulent
conjunctivitis, myalgia, and arthralgia of the small
joints of the hands and feet, although more severe
complications, including Guillain-Barre syndrome,
seem to be associated as well.42 The disease is usually
self-limiting and resolves within 3 to 7 days.42 Zika
virus infection during pregnancy is associated
with a higher rate of fetal loss, including stillbirths.
In addition, serious sequelae can be observed in
infants with congenital Zika syndrome, making it an
important public health concern in some areas.

Congenital Zika syndrome raised international
public health concern after the increase in cases of
congenital microcephaly in neonates in October 2015
following an outbreak of ZIKV infection in Brazil.
Fortunately, since ZIKV infection became a
notifiable disease in February 2016, the number of
cases in Hong Kong has remained low, with only two
cases in 2016 and one case in 2017.43 Therefore, the
risk of congenital Zika syndrome is extremely low in
Hong Kong.

Transmission of ZIKV primarily occurs via infected Aedes mosquitoes. It may also be transmitted through sexual contact, blood
transfusion, and organ transplantation.44 Vertical
transmission through the placenta is a major concern
because of the resulting congenital Zika syndrome.
A report by the United States Zika Pregnancy
Registry found congenital defects in 10% of 250 cases
with laboratory-confirmed ZIKV infection during
pregnancy.45,46

Neurological findings seen in congenital
Zika syndrome include microcephaly, cutis gyrata,
ventriculomegaly, subcortical calcifications,
polyvalformative syndrome (including craniofacial
disproportion and craniostenosis), brainstem
dysfunction, developmental delay, sensorineural
hearing loss, seizures, marked hypertonia,
hyperreflexia, and dysphagia.45,46 Ophthalmological
findings include cataracts, asymmetrical ocular size,
intracocular calcifications, macular abnormalities
(pigmentary retinal mottling, chorioretinal
atrophy/scarring), optic nerve abnormalities (optic
nerve hypoplasia, increased cup-to-disk ratio),
subretinal haemorrhage, coloboma, microcornea,
microphthalmia, cataracts, glaucoma, and lens
subluxation.45,46 Congenital heart disease occurs in
10% to 15% of cases.45 Although the full spectrum of
the syndrome has not been completely delineated,
a review study identified some characteristic features
of congenital Zika syndrome, including severe
microcephaly with partially collapsed skull, thin
cerebral cortices with subcortical calcifications,
macular scanning, focal pigmentary retinal
mottling, congenital limb contractures, and marked
early hypertonia with signs of extrapyramidal
involvement.51 In one study of 117 liveborn infants
born to pregnant women with confirmed ZIKV
infection, the overall rate of functional and structural
abnormalities was 42%.51
Definitive diagnosis of congenital ZIKV infection can only be established within the first few days of life by detecting ZIKV RNA in the serum, urine, or cerebrospinal fluid via a real-time reverse transcription PCR nucleic acid test.\textsuperscript{54} Detection of immunoglobulin M antibodies against ZIKV in the serum or CSF by enzyme-linked immunosorbent assay can support the diagnosis.\textsuperscript{54} Because there is currently no effective antiviral treatment for ZIKV infection, supportive treatment is mainly offered to manage the complications of congenital Zika syndrome. Continuous healthcare support and referral to developmental specialists to monitor the condition are recommended.\textsuperscript{54}

As no vaccine against ZIKV is currently available, avoidance of ZIKV infection by personal protection against mosquito bites and vector control remains the most effective means to prevent congenital Zika syndrome. Pregnant mothers should avoid travelling to places with known ZIKV transmission by mosquitoes and maintain mosquito prevention practices, such as removing mosquitoes' breeding grounds, wearing long-sleeved clothing, and using mosquito repellents. Protective measures against possible sexual transmission should also be adopted to minimise the risk of ZIKV infection during pregnancy.

**Parvovirus B19 infection**

Parvovirus B19 is the only pathogenic human parvovirus. In Hong Kong, the prevalence of antibodies to the B19 virus has been reported as 20%, which is much lower than the corresponding value for the Western population, where >60% of adults have been reported to be seropositive.\textsuperscript{55,56} The epidemiology of parvovirus B19 infection during pregnancy in Hong Kong is unknown, but it is likely to have low prevalence in light of the low population prevalence rate.\textsuperscript{57} Only three local case reports of hydrops fetalis due to parvovirus B19 were found in the literature.\textsuperscript{57-59}

Parvovirus B19 infection in adults is usually asymptomatic.\textsuperscript{60} Parvovirus infection in pregnant women is associated with hydrops fetalis because it causes severe fetal anaemia, sometimes leading to miscarriage or stillbirth.\textsuperscript{60-62} This is caused by a combination of haemolysis of red blood cells and the virus directly negatively affecting red blood cell precursors in the bone marrow. The risk of fetal loss is about 10% if infection occurs before week 20 of pregnancy (especially between weeks 14 and 20) but minimal after then. In contrast, parvovirus B19 infection during the second trimester is more commonly associated with hydrops fetalis or fetal loss. If the fetus does not develop any of these acute complications, or if intrauterine blood transfusion is successful in saving the fetus, the risk to the fetus presented by chronic parvovirus B19 infection is negligible.

Knowledge of the mother’s parvovirus B19 immune status could allow her to avoid contact with individuals suspected or known to have ongoing parvovirus B19 infection. However, antenatal immunity testing is not currently recommended, as there is no effective means to prevent infection, no specific therapy, and no vaccines available. Thus, testing may increase maternal anxiety and fear without proven benefit.

The best approach would be to recommend that all pregnant women avoid contact with children who currently have symptoms of parvovirus B19 infection. However, if a pregnant woman is exposed to parvovirus B19, serological testing should be performed as soon as possible to determine whether she should be monitored for seroconversion.\textsuperscript{63} If the results are suggestive of an acute parvovirus B19 infection, the fetus should be monitored by regular ultrasound assessment for signs of fetal hydrops and fetal anaemia. The peak systolic velocity of the fetal middle cerebral artery is an accurate predictor of fetal anaemia,\textsuperscript{63} and intrauterine blood transfusions can be considered in severe cases. Although some case reports have suggested that intrauterine parvovirus B19 infection caused developmental abnormalities in childhood, epidemiologic studies have not supported this association.\textsuperscript{64,65} The bulk of the available data suggest that parvovirus B19 is not teratogenic.\textsuperscript{19,61} No human vaccine against parvovirus B19 has been approved to date.\textsuperscript{66,67}

**Conclusion**

Although Hong Kong still has relatively low rates of congenital infections among countries in more developed regions despite being densely populated, it is important to remain vigilant against any possible infections during pregnancy, which may lead to severe morbidities and mortality of the fetus or infant.

The antenatal screening programme in Hong Kong is tailored to the local epidemiology of infectious and hereditary diseases, aiming to detect significant diseases that are potentially damaging to the fetus as early as possible. At present, the congenital infections for which the programme screens include rubella, hepatitis B, syphilis, and HIV, and the hereditary diseases screened for are thalassemia and Down syndrome.

Health authorities (eg, the Centre for Health Protection), obstetricians, and paediatricians should collaborate to establish a central congenital infection disease registry. This registry could be used for disease surveillance and monitoring of the outcomes of congenital infections. These results would be useful for detection of disease clusters and determination of their prevalence, morbidity, and mortality. Such a registry would help to estimate
the burden of these congenital infections on the healthcare system and guide resource allocation.

The data for such a registry could be captured from the notifiable disease database and laboratory surveillance of antenatal blood samples. Congenital rubella syndrome and Zika virus are notifiable diseases in Hong Kong, as their risk of transmission is high, outbreaks of these diseases can impose a significant risk to the community, and public health measures can be implemented if an outbreak is detected early. In terms of other congenital infections, the universal antenatal screening programme in Hong Kong should be able to identify most cases of congenital syphilis, rubella, and HIV. If recourses are available, health authorities can consider including congenital HIV and congenital syphilis as notifiable diseases. At present, the Hong Kong Department of Health has a surveillance programme intended to detect most congenital HIV and syphilis infections.44 Mothers with herpes simplex virus who are symptomatic should present during an antenatal visit. Toxoplasmosis and cytomegalovirus are not part of the antenatal screening programme, but serological tests can be performed if there is suspicion of congenital infection.

A congenital infection registry could act as a platform to provide information for disease surveillance. Public health promotion for primary prevention of maternal infections during pregnancy is the key to avoiding congenital infections.

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All authors had full access to the data, contributed to the study, approved the final version for publication, and take responsibility for its accuracy and integrity.

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Congenital infections


