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## REVIEW ARTICLE

# Congenital varicella syndrome: A systematic review

Ki Hoon Ahn<sup>1\*</sup>, Yun-Jung Park<sup>1\*</sup>, Soon-Cheol Hong<sup>1</sup>, Eun Hee Lee<sup>2</sup>, Ji-Sung Lee<sup>3</sup>, Min-Jeong Oh<sup>1</sup> & Hai-Joong Kim<sup>1</sup>

<sup>1</sup>Department of Obstetrics and Gynaecology, Korea University Anam Hospital, Korea University College of Medicine, Seoul, South Korea,

<sup>2</sup>Department of Paediatrics, Korea University College of Medicine, Seoul, South Korea, and <sup>3</sup>Clinical Research Centre, Asan Medical Centre, Seoul, South Korea

**Varicella-zoster virus (VZV) is a teratogen that can cross the placenta and cause the congenital varicella syndrome (CVS), which is characterised by multi-system anomalies. There have been 130 reported cases of CVS from 1947 to 2013. The estimated incidence of CVS was 0.59% and 0.84% for women infected with VZV during the entire pregnancy and for those infected the first 20 weeks of pregnancy, respectively. Nine cases were reported at 21–27 weeks of gestation and one case was identified at 36 weeks. Herpes zoster caused CVS in two cases. Regarding treatment, varicella zoster immunoglobulin treatment, irrespective of gestational age, should be considered in addition to antiviral drugs for women who have been exposed to or infected with virus.**

**Keywords:** Chickenpox, herpes zoster, pregnancy, congenital varicella syndrome

## Introduction

Varicella-zoster virus (VZV), also known as human herpes virus 3, is a DNA virus that causes chickenpox (varicella). After primary infection, VZV can remain dormant in the sensory nerve ganglia. Herpes zoster (shingles) can result from the reactivation of the virus in the elderly or immunocompromised individuals, and is characterised by painful vesicular skin eruptions in the sensory nerve roots (McCarter-Spaulling 2001).

VZV has teratogenic potential and is associated with low birth weight (Birthistle and Carrington 1998). It can also cause a rare but serious multi-system foetal anomaly called the congenital varicella syndrome (CVS). The purpose of this review was to investigate the vertically transmitted infection rates and management of CVS according to trimester and type of infection (chickenpox or herpes zoster).

## Materials and methods

### Search strategy

A literature search of MEDLINE, PubMed and EMBASE was performed in order to identify the reports that were published from 1947 to 2013. The keywords that were used in the search were 'CVS', 'congenital varicella syndrome', 'VZV infection in pregnancy', 'varicella infection in pregnancy', 'chickenpox in pregnancy' and 'herpes zoster in pregnancy'.

## Inclusion and exclusion criteria

Because CVS is rare, case reports were included in addition to large-scale observational studies. Inclusion criteria were as follows: (1) published in English; (2) provide birth outcomes, vertical transmission and treatment. Studies were excluded if appropriate data cannot be extracted.

## Study selection

Studies were selected in a staged manner. First, the titles and the abstracts of all screened articles were examined by two reviewers (P. Y. J. and H. S. C.) independently. Second, the full text of each article was reviewed for all included articles. Third, if the suitability of the studies was doubtful, two other independent reviewers (A. K. H. and L. E. H.) discussed the study selection. For a reliable review process, we checked the quality of the reviewed papers using the Systematic Review Appraisal Sheet in Critical Appraisal Topic (<http://www.cebm.net/critical-appraisal/>).

## Data extraction and analysis

The following information was extracted from the studies: (1) perinatal outcomes; (2) the presence of CVS; and (3) trimester at the time of diagnosis of CVS. The proportions (%) of confirmed CVS cases were calculated. Data were presented as numbers and percentages. Percentages were calculated using the reported number of a CVS as the numerator, which was divided by all studies that reported live births as the denominator.

## Results

The electronic database searches identified 51 studies that met the inclusion/exclusion criteria, which included 13 cohort studies and 38 case reports (Figure 1). Over 100 cases of CVS have been reported since the first documented case in 1947 (Laforet and Lynch 1947). Since CVS is rare, most reports were from cohort studies. The clinical features and diagnostic criteria used in the studies were described in Tables I and II. The incidence of CVS in the 13 cohort studies was estimated to be 0.59% (16/2705; Table III). The incidence of CVS by infection in the first 20 weeks of pregnancy was estimated to be 0.84% (14/1675;

\*These authors contributed equally to this work and thus are considered co-first author.

Correspondence: Soon-Cheol Hong, Department of Obstetrics and Gynaecology, Korea University Anam Hospital, Korea University College of Medicine, 126-1 Anam-dong 5-ga, Seongbuk-gu, Seoul 136-705, South Korea. Fax: +82-2-921-5357. E-mail: novak082@naver.com

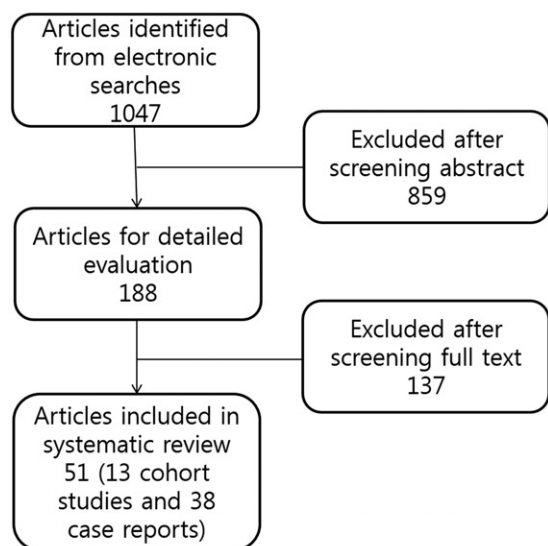


Figure 1. Flow chart of study selection.

Table I. Clinical features of congenital varicella syndrome (Sauerbrei and Wutzler 2000).

Symptoms	Proportion of children (%, <i>n</i> = 96)
Skin lesions (scars, skin loss)	76
Neurologic defects or disease (cortical atrophy, limb hypoplasia, seizures, microcephaly, Horner's syndrome, encephalitis, dysphagia)	60
Eye disease (microphthalmia, chorioretinitis, cataract, nystagmus, anisocoria, optic atrophy)	51
Limb hypoplasia and other skeletal anomalies	49
Intrauterine retardation	22
Muscle hypoplasia	21
Gastrointestinal abnormalities	15
Affections of internal organs	13
Developmental delay	12
Genitourinary abnormalities	12
Defects of the cardiovascular system	8
Defects of other organs	7

CVS, congenital varicella syndrome.

Table II. Diagnostic criteria of congenital varicella syndrome (Alkalay et al. 1987).

(1) Appearance of maternal chickenpox during pregnancy
(2) Presence of congenital skin lesions in dermatomal distribution and/or neurologic defects, eye disease, limb hypoplasia
(3) Proof of intrauterine VZV infection by detection of viral DNA in the infant
(4) Presence of specific IgM
(5) Persistence of IgG beyond 7 months of age
(6) Appearance of zoster during early infancy

CVS, congenital varicella syndrome; VZV, Varicella-zoster virus.

Table IV). Most cases of CVS occurred from infection in the first 20 weeks (Enders et al. 1994), but 10 were reported after 20 weeks. Nine cases (Bai and John 1979; Lambert et al. 1989; Michie et al. 1992; Salzman and Sood 1992; Enders et al. 1994; Ong and Daniel 1998; Deasy et al. 1999; Forrest et al. 2000; Kerkerling 2001; Harger et al. 2002) of CVS that occurred from infection during weeks 21–27 and one case that occurred from infection during week 36 were reported (Tan and Koren 2006).

Although herpes zoster has not been known to be associated with CVS, there have been two CVS cases that resulted from herpes zoster infection. First, a 2.5-year-old female patient presented with chronic progressive small-vessel vasculopathy with radiological features of Moyamoya disease in 2006. She had a localised vesicular rash of the scalp at 2 days of age and was treated with acyclovir IV for 7 days. Her mother had herpes zoster at 36 weeks of gestation. This was a confirmed CVS case (West et al. 2006). The second CVS case presented with a hypoplastic limb covered with a segmental skin scar over the posterior aspect of the left thigh. Her mother had disseminated herpes zoster at 12 weeks of gestation. This was also a confirmed CVS case (Higa et al. 1987).

## Discussion

Chickenpox is common in children under the age of 15 years old. If chickenpox occurs in individuals over the age of 15 years old or in infants under the age of 1 year, there are high rates of complications such as secondary bacterial infection of the skin lesions, pneumonia, dehydration, encephalitis and hepatitis (McCarter-Spaulling 2001). Before the introduction of the chickenpox vaccine, 90% (World Health Organization 1992; Centre for Disease Control and Prevention 1999) and 25–80% (Lee 1998; Lolekha et al. 2001) of the children in countries with temperate and tropical climates, respectively, were seropositive for the virus by the age of 15 years. During the 1990s, the incidence of chickenpox in individuals aged 15–44 years was estimated to be 1.6–4.6/1000 in the United States (Enders and Miller 2000) and 2–3/1000 in the United Kingdom (Fairley and Miller 1996). VZV is highly contagious and is transmitted either by direct contact with vesicular fluid from skin lesions or by secretions from the respiratory tract. The incubation period for VZV ranges from 10 to 21 days. Chickenpox is characterised by fever, malaise, and a generalised vesicular rash that can involve the trunk, face, oropharynx, and/or scalp. The infection period ranges from 1–2 days before to 4–5 days after the onset of the rash (when all the lesions are crusted) (McCarter-Spaulling 2001). Primary infection generally results in lifelong immunity; however, clinical reinfection has been reported (Gershon 1995; Hall et al. 2002). Reinfection may be associated with abnormalities in the memory cells of the immune system or a high inoculum of the virus (Martin et al. 1994). VZV can cross the placenta. Most trans-placental infections are asymptomatic but they can cause CVS or neonatal chickenpox, which have high rates of mortality and morbidity. CVS has multi-system effects characterised by symptoms including skin lesions, neurological defects, eye diseases, limb hypoplasia, and/or skeletal anomalies (Sanchez et al. 2011). These defects may appear as a single symptom or as a spectrum.

One hypothesis is that CVS is caused by reactivation of VZV *in utero* rather than by primary infection of VZV (Higa et al. 1987). Immature foetal cell-mediated immunity would shorten the latent period between primary infection and reactivation (Grose 1989). The dermatomal distribution of the skin lesions (Birthistle and Carrington 1998), segmental maldevelopment and dysfunction of the affected system support this hypothesis (Higa et al. 1987).

The CVS diagnosis can be performed as follows. Both the polymerase chain reaction (PCR) of the amniotic fluid or foetal blood for detecting VZV DNA and the serology tests that detect VZV-specific antibodies are useful in confirming infection, but neither is useful for detecting CVS (Enders et al. 1994; Sauerbrei et al. 1996). Therefore, a detailed ultrasonography, to detect limb deformity, microcephaly, hydrocephalus, polyhydramnios, soft

Table III. Incidence of congenital varicella syndrome in cohort studies.

Study	No. of CVS/no. of live births (%)			
	First trimester	Second trimester	Third trimester	Overall (%)
Hill et al. (1958)				0/30 (0)
Manson et al. (1960)				0/288 (0)
Siegel (1973)	1/27 (3.70)	0/32 (0)	0/76 (0)	1/135 (0.74)
Paryani and Arvin (1986)	1/11 (9.09)	0/11 (0)	0/19 (0)	1/41 (2.43)
Balducci et al. (1992)	0/35 (0)			0/35 (0)
Pastuszak et al. (1994)	1/86 (1.16)		0/14 (0)	1/100 (1)
Enders et al. (1994)	1/469 (0.21)	6/477 (1.26)	0/345 (0)	7/1291 (0.54)
Jones et al. (1994)	1/110 (0.91)	1/46 (2.17)	0/13 (0)	2/169 (1.18)
Dufour et al. (1996) <sup>a</sup>	0/17 (0)		0/3 (0)	0/20 (0)
Figueroa-Damian and Arredondo-Garcia (1997) <sup>a</sup>	0/22 (0)			0/22 (0)
Mouly et al. (1997)	12 weeks	19 weeks		2/94 (2.13)
Harger et al. (2002)		24 weeks		1/231 (0.4)
Sanchez et al. (2011) <sup>a</sup>	1/252 (0.39)			1/252 (0.40)
Mean (%)				16/2708 (0.59)

<sup>a</sup>Focusing on incidence of CVS in first 20 weeks; CVS, congenital varicella syndrome; No., number.

Table IV. Incidence of congenital varicella syndrome in the first 20 weeks.

Study	No. of CVS/no. of live birth (%)
Paryani and Arvin (1986)	1/22 (4.55)
Balducci et al. (1992)	0/35 (0)
Pastuszak et al. (1994)	1/86 (1.16)
Enders et al. (1994)	7/816 (0.86)
Jones et al. (1994)	2/146 (1.37)
Dufour et al. (1996)	0/17 (0)
Figueroa-Damian and Arredondo-Garcia (1997)	0/22 (0)
Mouly et al. (1997)	2/89 (0)
Harger et al. (2002)	0/190 (0)
Sanchez et al. (2011)	1/252 (0.40)
Mean (%)	14/1675 (0.84)

CVS, congenital varicella syndrome; No., number.

tissue calcification and intrauterine growth restriction (Bruder et al. 2000; Enders and Miller 2000; Kerkering 2001; Petignat et al. 2001), is combined with the PCR and serology tests. Because CVS may be caused by the reactivation of VZV (Higa et al. 1987), an interval of at least 5 weeks is needed between the ultrasonography and maternal onset of the rash (Pretorius et al. 1992). Herpes simplex virus (HSV) and VZV have cross-reactivity (Gershon 1995) and both coxsackie B virus and HSV type 2 can cause clinical symptoms that are similar to CVS (Koskimies et al. 1978; Sauerbrei et al. 2000; Johansson et al. 2004). Thus, these factors should be considered during diagnosis.

Treatment and prevention of CVS are comprised of vaccination, varicella zoster immunoglobulin, and antiviral drugs. The VZV vaccine is a live attenuated virus that is not secreted in breast milk. Postpartum vaccination should not be delayed (Bohlke et al. 2003). Immunity to chickenpox usually results from natural infection. Therefore, vaccination is needed in only 3.9% of adults (Hanaoka et al. 2013). However, VZV vaccination can prevent infection of the mother and foetus and reduce the incidence of both CVS and neonatal chickenpox (Hanaoka et al. 2013). Although post-exposure vaccination during pregnancy or vaccination 3 months before pregnancy is contraindicated (Royal College of Obstetricians and Gynaecologists 2015), a 10-year study involving vaccination during pregnancy or within the 3 months before pregnancy demonstrated that none of the 944 infants that were studied showed any clinical features of chickenpox (Wilson et al. 2008).

If a pregnant woman has contact with a person infected with chickenpox within the infection period, her sero-status must be

evaluated. If she has a history of chickenpox vaccination, reassurance is needed (Watson et al. 2007). If she was not vaccinated, her sero-status must be tested. If the woman is sero-negative, she should receive post-exposure prophylaxis immediately. Varicella zoster immunoglobulin (VZIG) is recommended both for women who are exposed and for women who are susceptible to infection with the virus, because it can prevent maternal disease and complications (Marin et al. 2007). Additionally, VZIG can reduce the risk of foetal infection (Cohen et al. 2011). It should be administered within 96 h of chickenpox exposure (Marin et al. 2007).

Antiviral drugs such as acyclovir and valacyclovir, may be used as post-exposure prophylaxis. Antiviral treatment is indicated for the mother (Heuchan and Isaacs 2001; Tan and Koren 2006) in late pregnancy and if there are respiratory symptoms (Lamont et al. 2011). However, in early pregnancy, the foetal benefit is controversial (Mandelbrot 2012).

## Conclusions

It appears that the incidence of CVS is slightly increasing compared with prior studies. Despite its rarity, CVS can occur in the second half of pregnancy. Since our analyses revealed the interesting information that herpes zoster can cause CVS, we presented an updated review of CVS for the readers' benefit.

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