

Bullous neonatal varicella, acquired postpartal from a household member, treated successfully with i.v acyclovir: a case report

Abstract

Neonatal varicella (NV) is extremely rare, with an incidence of 2–6 per 100,000 live births per year, which is often severe and fatal. Bullous NV (BNV) is a rarer variant of varicella. Only 61 cases of NV and 16 cases of BV have been reported; most of them were due to streptococcal infections, also not true bullous varicella; we were not able to find one case of BNV. Herein, we report a case of a 13-day-old male patient presented with severe extensive bullous varicella. On the 9th day postpartum, the mother developed symptoms of a primary varicella zoster virus (VZV) infection while the infant was breastfeeding. Two days later, the infant developed a fever, followed by extensive bullous exanthema involving the entire skin and mucous membranes. He was admitted to the intensive care unit and administered intravenous acyclovir 10 mg/kg/8 h for 7 days and topical fusidic acid/betamethasone cream three times daily. The patient showed prompt improvement and was discharged after 1 week without any sequelae. This is the first case of BNV, treated successfully with only i.v. acyclovir, without VZV IgG or IVIG, which highlights the significance of early intervention with antiviral therapy in neonatal VZV infections.

Keywords: varicella-zoster virus, newborn, neonatal varicella, acyclovir, VZV IgG

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Introduction

Neonatal varicella (NV) is extremely rare, with an incidence of 2–6 per 100,000 live births per year, which is often severe and fatal.¹ It is caused by the varicella zoster virus (VZV), also known as human herpesvirus 3, an exclusively human neurotropic virus belonging to the family Alpha-herpesvirinae.¹ VZV does not have an animal reservoir and primarily infects T lymphocytes, epithelial cells, and ganglia. Neonates get infected either transplacental from the infected mother or postpartum via the respiratory system, which can be from the infected mother or other infected households.² Symptoms appear 10–21 days after exposure and typically last 5–7 days. Two factors determine the course and severity of NV, namely the viral load and the maturity of the neonate immune system. If the neonate gets infected transplacentally, the high virus load from the infected mother and the absence of protective maternal antibodies will lead to a severe and eventually fatal course.² In contrast, if the neonate got infected postnatally through the respiratory system, then the virus load is low, and the relatively immature immune system of the neonate can master the infection, leading to a much milder course. Primary infections usually confer lifelong immunity against the disease. The initial infection, mainly acquired via inhalation or less commonly by direct contact with vesicles, results in varicella (chickenpox), most commonly affecting children. In contrast, herpes zoster results from the reactivation of the latent VZV inside the sensory ganglion cells. Varicella is a childhood disease that occurs primarily during the first decade of life. Varicella infection in the mother during the last week of pregnancy increases the possibility of transplacental infection. To the best of our knowledge, this is the first case of bullous NV (BNV), acquired postnatally, simultaneously with his mother, from a household member.

Case report

A 13-day-old infant, weighing 2800 g and delivered vaginally, presented to our clinic with a disseminated confluent vesiculobullous rash covering most of the skin surface, including the scalp, palms, and soles (Figures 1a-1f & 2a-2d). His mother developed varicella on the 9th day postpartum, which suggested that she acquired varicella during the last five days of pregnancy, considering the typical incubation period of 10–28 days. The infant started to develop typical skin lesions 2 days after his mother, which ruled out the possibility of postnatal infection from his mother. Upon further questioning, the mother confirmed postnatal contact with an infected household member. She explained that it is a tradition for women to congratulate the new mother, and she distinctly remember a woman with VZV infection among the visitors, one day postpartum. Based on the typical history and clinical presentation, a diagnosis of BNV was made, and the baby was administered intravenous acyclovir 10 mg/kg/8 h for 7 days, followed by supportive therapy, to which he responded well and recovered within 1 week. He was followed up at 3, 12, 24, and 36 months after discharge; no complications were found, and the shallow scars disappeared completely by 12 months. The infection was severe in this infant, as he was born to a seronegative mother, who presented with a rash only nine days after delivery, thus having no possibility for the production and transfer of protective IgM and IgG antibodies to the baby. He was treated successfully with 10 mg/kg intravenous acyclovir for 7 days, without VZV-specific hyperimmunoglobulin (VZIG) or IVIG, due to their unavailability. The present case highlights the importance of rapid diagnosis and intravenous acyclovir administration, even in the absence of varicella-zoster immune globulin, to protect the neonate, from severe and eventually fatal complications like, encephalitis, pneumonia, hepatitis, and secondary bacterial infections.



Figure 1a Back of the neonate at presentation, almost monomorphic vesiculobullous exanthem, covering most of the back. Note that some of the bullae are flat due to pressure from laying on the back.



Figure 1e Chest and abdomen at presentation, almost monomorphic vesiculobullous eruption. Note large bullae over upper chest, presternal, and paraumbilical.



Figure 1b Back of the child 9 days after treatment. Most of the lesions have healed, with only a few hemorrhagic crusts visible.



Figure 1f Chest and abdomen 9 days after treatment. Most of the lesions have healed, with only a few hemorrhagic crusts visible.



Figure 1c Face, scalp, and chest of the patient before treatment, covered with yellow crusts, vesicles, and tense bullae mainly on the scalp and upper chest.



Figure 2a Right palm at presentation, tense vesicles and bullae, filled with clear fluid.



Figure 1d Face of the patient 9 days after treatment. Most of the lesions have healed, with only a few hemorrhagic crusts visible.



Figure 2b Right palm 9 days after treatment. Most of the lesions have healed, with only a few hemorrhagic crusts visible.



Figure 2c Right sole at presentation, almost monomorphic vesicles, and bullae, covering most of the sole.

Figure 2d Right sole 9 days after treatment. Most of the lesions have healed, with only a few hemorrhagic crusts visible.

Discussion

VZV infection during pregnancy is associated with an increased risk for both the mother and fetus. The outcome of such an infection in infants depends on the timing of the maternal infection.³ Near-term maternal infections are associated with a high risk of neonatal varicella. Serious disseminated infections with visceral involvement may occur in the affected infants.⁴ NV can be expected if a mother contracts chickenpox during the final 3 weeks of pregnancy. Maternal chickenpox near-term or soon after delivery can cause severe or fatal illness in newborns. Neonatal chickenpox within the first 10–12 days of life could be attributed to the intrauterine transmission of VZV

because of the incubation period of varicella. Chickenpox occurring 10–12 days after birth can be attributed to postnatal VZV infection and has a low morbidity rate because maternally derived antibodies protect most neonates.¹ The severity of intrauterine-acquired neonatal chickenpox is closely associated with the time of onset of maternal infection because transplacentally transmitted antibodies can reduce the severity of symptoms in newborns. Generalized NV, which can be fatal, is highly likely if mothers develop a varicella rash 4 days before to 2 days after delivery. Some studies have reported an extended risk period for maternal rash onset from 5 days antepartum to 2 days postpartum. Only 61 cases of NV and 16 cases of BV have been reported; we were not able to find one case of BNV (Table 1 & 2).

Table 1 Reported cases of bullous varicella. Out of 16 reported cases of bullous varicella, none involved bullous neonatal varicella

Pub. year	Ist Author	Title	Journal	Country	Age 8Y 11Y ?	Sex M F ?	Number I I I
1963	Canby JP	Bullous Chickenpox (Varicella Bullosa)	Clin Pediatr (Phila)	USA	? 11M 12M	? M M	I I I
1960	Saslaw S	Varicella bullosa	JAMA	USA	17M	F	I
1963	Seigerman H	Varicella bullosa, a case report	J Pediatr	USA	9M	M	I
1970	Glenn MP	Varicella bullosa associated with measles vaccine	Br J Dermatol		3Y	F	I
1973	Melish ME	Bullous varicella. its association with the staphylococcal scalded skin syndrome	J Pediatr	USA	2YM	M	I
2008	Sulik A	Bullous varicella in a 5-month-old infant	Clin Exp Dermatol	Poland	5MF	F	I
2018	Suvirya S	Monomorphic Bullous Hemorrhagic Varicella in a Patient on Methotrexate	Indian J Paediatr Dermatol	India	14YF	F	I
2019	Mansouri S	Bullous varicella in an immunocompetent infant	BMJ Case Rep	Morocco	6M	M	I
1994	White GM	Vesicles and bulla in an infant. Bullous varicella (chicken pox complicated by bullous impetigo)	Arch Dermatol	USA	9M	F	I
2003	Sathynarayana BD	Varicella bullosa	Indian J Dermatol Venerol Leprol	India	3Y	M	I
2013	Sharma CM	A Classical Case of Neonatal Varicella	J Clin Neonatol	India	5D	M	I
Total							16

Table 2 Reported cases of neonatal varicella. Out of 61 reported cases of neonatal varicella, none involved bullous varicella

Pub. year	Ist Author	Title	Journal	Country	Age	Sex	Nr.
1878	Hubbard TW	Varicella occurring in an infant twenty-four hours after birth	Br Med J	UK	1D	M	1
1947	Lucchesi PF	Varicella neonatorum	Am J Dis Child	USA	10D	F	1
1958	Ehrlich RM	Neonatal varicella. A case report with isolation of the virus	J Pediatr	Canada	12D	F	1
1967	Hyatt HW	Neonatal varicella. Report of a case in a five-day-old infant and review of the literature	J Natl Med Assoc	USA	5D	M	1
1968	Cutter BG	Neonatal varicella	Med J Aust	Australia	3D	M	1
1986	Carter PE	Neonatal varicella infection	Lancet	UK			10
1986	Holland P	Fatal neonatal varicella infection	Lancet	UK	7D	F	1
1999	Singalavanija S	Neonatal varicella. a report of 26 cases	J Med Assoc Thai	Thailand		F M	13 13
2009	Click JW	Picture of the month. neonatal varicella infection	Arch Pediatr Adolesc Med	USA	12D	F	1
2010	Thakur AK	Neonatal varicella	Indian Pediatr	India	6D	M	1
2011	Bhardwaj AK	Neonatal varicella. A case report	Australas Med J	India	5D	M	1
2011	Jackson C	Rash in a neonate	J Clin Virol	UK	17D	F	1
2012	Kluthe M	Neonatal vaccine-strain varicella-zoster virus infection 22 days after maternal postpartum vaccination	Pediatr Infect Dis J	USA	25D	F	1
2012	Singh SN	Varicella infection in a neonate with subsequent staphylococcal scalded skin syndrome and fatal shock	BMJ Case Rep	India	23D	M	1
2012	Hon KL	Neonatal herpes. what lessons to learn	Hong Kong Med J	12DM			1
2013	Sharma CM	A Classical Case of Neonatal Varicella	J Clin Neonatol	India	5D	M	1
2014	Marwah P	Fatal newborn varicella despite varicella zoster immunoglobulin prophylaxis	Indian J Dermatol Venereol Leprol	India	9D	M	1
2017	Choudhary P	Late neonatal varicella	Indian J Paediatr Dermatol	India	14D	F	1
2018	Machi H	Neonatal varicella. Probable transmission from a vaccinated mother	Pediatr Int	Japan	10D	M	1
2020	Piyanonpong W	Effects of Intravenous Immunoglobulin and Acyclovir in Preventing Neonatal Varicella	Case Rep Inf Dis	Thailand	3D	M	1
2020	Reddy R	Neonatal Varicella	J Clin Diag Res	India	15D	F	1
2021	Lai JW	Case report. neonatal varicella acquired from maternal zoster	Front Pediatr	Australia	21D	M	1
2022	Earlia N	Neonatal varicella. a rare case	Bali MedJ	Indonesia	14D	F	1
2023	Frantzis I	Varicella in the neonatal ICU due to the Varicella vaccine Oka strain	J Neonatal Perinatal Med	USA	7D	M	1
2024	Chap C	Neonatal varicella, treated with oral acyclovir. A Rare and Challenging Case Report in a Limited-Resource Country like Cambodia	Clin Case Rep	Cambodia	15D	M	1
2024	Li H	Case Report. Taking action or standing by. managing a preterm neonate at the risk of neonatal varicella by metagenomic next-generation sequencing	Front Pediatr	China	1D	M	1
2024	Alhwayan AA	Intravenous Immunoglobulin and Intravenous Acyclovir as an Alternative Therapy to Varicella Zoster Immunoglobulin in the Prevention of Serious Complications of Neonatal Varicella	Cureus	Jordan	2D	M	1
						Total	61

After maternal varicella infection during this period, the risk of severe neonatal chickenpox is generally estimated at 20–50%, with a fatal outcome reported in approximately 20% of cases.⁵ Unfortunately, these infants are exposed to maternal viremia without acquiring protective antibodies. Additionally, the cell-mediated immune response of the neonate is likely insufficient to prevent hematogenous dissemination of VZV after transplacental spread.² As a result, a fatal outcome is more likely if neonatal disease occurs 5–10 days after delivery. According to available literature, 23% of infants with disseminated and fulminant infections have died.⁶ NV within the first 4 days after birth is typically milder. Fetuses exposed to VZV between 20 and 6 days before delivery may develop non-fatal neonatal chickenpox due to the presence of maternal antibodies, which reduce the risk of complications. Because of neonatal chickenpox, zoster may occur during infancy, usually with an uncomplicated course. The relatively short viral latency period may be explained by the immature cell-mediated immune response in young children. Maternal zoster during the perinatal period does not generally affect newborn infants because the infants possess specific maternal IgG class antibodies and do not experience the viremic spread of VZV. Treatment options include: (1) IVZIG, (2) IVIG, (3) oral, and (4) intravenous acyclovir, where the best outcomes are achieved by combination of option 1, and 4, followed by option 2, and 4, while the less favorable outcomes are achieved by option 3 alone followed by option 4 alone, which depends on the availability of the drugs.

To conclude, the presented case is the first BNV that is treated successfully with only i.v. acyclovir for 7 days, without VZIG or IVIG.

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Conflicts of interest

The authors declare that there are no conflicts of interest.

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