Myths and Misconceptions: Varicella-Zoster Virus Exposure, Infection Risks, Complications, and Treatments

Alexander M. Newman, MD; and Ravi Jhaveri, MD, FAAP

Northwestern University Feinberg School of Medicine, Chicago, IL, USA

ABSTRACT

Varicella zoster and herpes zoster are infections caused by the highly contagious varicella-zoster virus (VZV). Despite widespread availability of vaccines against VZV, as well as varicella vaccination rates >95%, VZV remains a public health concern because of several common myths and misconceptions. Because of the success of routine varicella vaccination programs, some people mistakenly believe that varicella and herpes zoster are now no longer a threat to public health. Another common misconception is that shingles is less infectious than varicella; however, clinical evidence indicates otherwise. Several knowledge gaps exist around VZV transmission and the availability and use of varicella zoster immune globulin (human) for postexposure prophylaxis against VZV. To help reduce the incidence of severe disease in high-risk individuals (eg, elderly people, pregnant women, unvaccinated persons, infants, and immunocompromised children and adults), this article addresses misbeliefs and broadens awareness of VZV exposure, infection risks, complications, and treatments. (*Clin Ther*. 2019;41:1816–1822) © 2019 Published by Elsevier Inc.

Keywords: herpes zoster, public health, post-exposure prophylaxis, varicella.

INTRODUCTION

Varicella zoster (ie, chickenpox) is an acute infection caused by the varicella-zoster virus (VZV). VZV is a member of the alphaherpesviruses, a group of double-stranded DNA viruses that lead to human infection and latency in dorsal root ganglia.[1] Primary varicella (PV) infection is thought to occur through contact with respiratory secretions from a person with active varicella or from direct contact with varicella vesicular lesions.[2] Despite an observed 10- to 21-day incubation period from infection to clinical symptoms, the host is contagious and can spread VZV through the respiratory route 24 to 48 hours before initial skin eruption.[3]

Those infected with PV may experience prodromal symptoms before the classic varicella vesicular rash.[4] Although the primary eruption is uncomfortable, PV can lead to major complications that cause morbidity and mortality, including bacterial superinfections that progress to necrotizing fasciitis, central nervous system diseases (eg, cerebellar ataxia, meningoencephalitis, and Guillain-Barre syndrome), pneumonitis, and death.[2] PV is more severe among high-risk populations, such as older adolescents and adults, pregnant women, neonates, unvaccinated and previously unexposed persons, and individuals with impaired cellular immunity (eg, those with leukemia). Before the implementation of VZV vaccination programs, 100 VZV-related deaths were reported annually in the United States, mostly in previously healthy children.[5]

As the host ages and innate immunity wanes, VZV can reactivate from latency, causing herpes zoster (HZ; ie, shingles). HZ differs from PV in that it is often a localized infection and preceded by pain, hyperesthesia, or pruritus in the location where vesicles will soon erupt.[5] New lesions occur a mean of up to 7 days, and the infection can be spread through multiple routes (detailed below).[5] The risk of developing HZ after PV infection increases after 50 years of age.[3] More than 95% of immunocompetent individuals 50 years and older are seropositive for VZV and, therefore, are at risk for developing HZ (if

Accepted for publication June 18, 2019
https://doi.org/10.1016/j.clinthera.2019.06.009
0149-2918/$ - see front matter

not vaccinated). Reactivation can also occur in those with impaired cellular immunity, including those with leukemia, and those who have undergone solid organ transplant and stem cell transplant.[1] These populations are at increased risk for disseminated HZ and further morbidity and mortality from the disease.[5]

There are tolerable and effective vaccines to prevent varicella and HZ. The United States first licensed the varicella vaccine (Varivax®, Merck & Co, Inc, North Wales, Pennsylvania) for all healthy children 1 year and older in 1995, and initial studies found that 1 dose provided children with 85% protection from contracting primary disease.[6] In 2006, the Centers for Disease Control and Prevention (CDC) recommended a second dose to children older than 4 years, which increased protection up to 95% in postvaccine studies.[5] Currently, the CDC's Advisory Committee on Immunization Practices recommends a 2-dose vaccination series to all immunocompetent children, starting at 1 year of age.[7] Unfortunately, immunization does not prevent disease in individuals already exposed to VZV. There are also 2 vaccines approved for adults 50 years and older to reduce the risk of HZ (Zostavax®, Merck & Co, Inc, and Shingrix®, GlaxoSmithKline Biologicals, Rockville, Maryland). Fortunately, there are viable treatment options for those that develop PV and HZ, those ineligible to receive vaccines, and individuals at high risk for complications after exposure to VZV. Passive immunization, with anti-VZV immunoglobulin medications, such as varicella zoster immune globulin (VZIG) (human) (VARIZIG®, Saol Therapeutics, Roswell, Georgia), can prevent PV or reduce the severity of disease if given in a timely manner.[8] The decision to use medications such as VARIZIG depends on multiple factors, including the type of exposure, the risk of severe disease, and the exposed person's own immunity to VZV.[3] In individuals with decreased T-cell immunity, passive immunization is recommended to prevent primary disease or to decrease severity of disease.[3] VARIZIG is most efficacious if given within 96 hours after exposure, but it can be administered up to 10 days after exposure.[9] Acyclovir therapy has become a mainstay in high-risk populations to prevent disease progression and more severe complications after disease onset.[5] Although acyclovir is not recommended for postexposure prophylaxis in otherwise healthy children, acyclovir reduces the risk of reactivation in previously exposed people with depressed T-cell function.[3] Prevention of PV can also be achieved through isolation of infected individuals.[7]

Despite the availability of varicella vaccines and the fact that varicella vaccination rates are >95%, infection with VZV varicella and HZ remains a public health concern. There are several common myths and misconceptions that serve to undermine the most effective VZV prevention measures from being used. This article addresses those misbeliefs and broadens awareness of VZV exposure, infection risks, complications, and treatments.

MYTH 1: THE RISK OF EXPOSURE TO VARICELLA OR HZ IS LOW BECAUSE OF VACCINATION

Myth

Because of the success of widespread routine VZV vaccination programs, some people believe that varicella and HZ are now no longer public health issues.

Facts

Since the implementation of the 2-dose varicella vaccination program, the mean annual incidence of varicella has decreased by 85%, from 25.4 per 100,000 during 2005 to 2006 to 3.9 per 100,000 during 2013 to 2014 ($P < 0.001$).[6] However, in the United States alone, approximately 100,000 cases of varicella and nearly 1 million new cases of HZ still occur each year.[6–10] Furthermore, the incidence of HZ increased by 98% during 13 years, from 1.7 per 1000 individuals in 1993 to 4.4 per 1000 in 2006 ($P < 0.001$).[11] Nearly 1 in 3 people in the United States will develop HZ in their lifetime,[10] and the risk of getting HZ increases with age.[12] Unfortunately, 2017 data from the CDC indicate that only 40% of adults 60 years and older have received 1 of the HZ vaccines.[13]

Despite a high rate of >95% in 2-dose varicella vaccination uptake, breakthrough varicella disease, defined as VZV infection occurring >42 days after varicella vaccination,[14] still occurs.[15–18] Among children who receive 1- or 2-dose vaccine regimens, breakthrough varicella rates range from 2.2% to 7.3%.[7] Rates of breakthrough varicella are significantly higher in North America compared with Asia and Europe ($P = 0.005$).[16] Typically, breakthrough varicella disease is relatively mild (ie,

shorter duration and fewer lesions), although still contagious to susceptible individuals.[14] However, 25% to 30% of breakthrough varicella has resulted in severe disease, with complications similar to those in unvaccinated individuals.[7,19]

Medical and nonmedical vaccination exemption rates are increasing nationwide. According to the CDC, the number of kindergartners with medical and nonmedical exemptions from ≥1 required vaccine (including varicella vaccines) increased from 2.0% during the 2016 to 2017 school year to 2.2% in 2017 to 2018.[20] An overall increase in exemptions has been observed for the last 3 consecutive years.[20,21] In a birth cohort of approximately 3 million, this increase translates to 6000 children per year, or an increase of almost 20,000 exemptions during the 3 years. From 2009 to 2017, the number of nonmedical vaccination exemptions from vaccination significantly increased in 12 of the 18 states that grant nonmedical vaccination exemption ($P < 0.05$). Nonmedical vaccination exemptions were also heterogeneously distributed within individual states, leading to concentrated areas of high nonmedical vaccination exemption rates in select counties and metropolitan areas across the country, resulting in hotspots of unvaccinated individuals susceptible to preventable infection.[22] Similarly, results from a retrospective cohort analysis of children born between 2000 and 2009 within Kaiser Permanente of Northern California primary care indicated that underimmunization with varicella vaccines and vaccine refusal clustered in geographically similar areas.[23] Data from a case–control study of children aged 12 months to 8 years found that varicella vaccine refusal significantly increased the risk of varicella infection that required medical care ($P = 0.004$).[24]

Using data from the 2013 National Health Interview Survey, researchers estimated that in the United States 2.7% of the population was immunosuppressed, and the prevalence of immunosuppression may be increasing because of new immunosuppressive treatment indications and increased life expectancy.[25] Old age and immunosuppression are risk factors for complications attributable to varicella; therefore, as more immunosuppressive medications become available, the number of susceptible individuals at risk for severe complications from infection with VZV will likely increase.[26]

MYTH 2: TRANSMISSION OF VZV VIA HZ ONLY OCCURS THROUGH DIRECT CONTACT AND TRANSMISSION VIA HZ EXPOSURE IS LESS LIKELY THAN THROUGH VARICELLA EXPOSURE

Myth

People commonly misbelieve one cannot contract VZV from a person with active HZ if his/her rash is covered. However, VZV is transmitted from person to person via multiple routes.

Facts

VZV is highly infectious. According to the CDC, a person infected with PV is contagious beginning 1 to 2 days before rash onset (typically 14 days after VZV exposure) and continuing until all lesions are scabbed (typically 5 days after rash onset). In 2012, compelling epidemiologic data indicated that breakthrough varicella in vaccinated children is just as likely to occur after exposure to those with HZ as in those with PV: nearly 10% of HZ cases (n = 27 of 290 cases) versus 15% of varicella cases (n = 205 of 1358) caused exposed individuals to develop breakthrough varicella infection.[27,28] Additional long-term studies are needed to further analyze VZV transmission rates between individuals with varicella and individuals with zoster.

VZV is primarily transmitted via direct contact with varicella or HZ lesions but may also be transmitted via respiratory exposure of aerosolized virus. There are limited data suggesting that VZV aerosolization from HZ occurs, as indicated by studies using air samples from rooms of patients with active varicella and patients with active HZ infection.[29,30] Multiple case reports from acute and long-term health care facilities have found VZV transmission from isolated patients with HZ to health care workers or other patients or residents without direct contact, suggesting the possibility of aerosolized viral transmission.[14,31,32] Scratching the lesions may aerosolize the VZV virus and consequently facilitate airborne transmission.[14] This finding suggests that VZV can be transmitted from a person with HZ, even if his/her rash is covered. Furthermore, it has been reported that individuals with covered HZ rashes (eg, on the trunk) have the same probability of transmission as those with uncovered rashes (eg, on the arms or legs): the relative risk of transmission was 1.0 (95% CI,

0.8−1.3) and 1.1 (95% CI, 0.8−1.3) when comparing trunk rash with rash on the arms and legs.[27]

Nosocomial transmission of VZV among patients, health care professionals, and visitors can be life threatening, necessitating that isolation protocols exist for prevention.[33,34] Since the widespread implementation of the 2-dose varicella vaccination program, reports of nosocomial VZV transmission have become less common in the United States.[2] In addition to transmission originating in a hospital, long-term care settings, or other health care facilities, household transmission of VZV is also a problem.[35] Close indoor contact (ie, in the same room or sharing airspace) by one person with an individual with varicella or HZ increases the risk of the other person contracting VZV, especially in high-risk populations (ie, pregnant women, infants, and immunocompromised people). Typically, contagiousness of breakthrough varicella correlates with lesion number. Vaccinated individuals with <50 lesions were found to be one-third as contagious as unvaccinated individuals, and vaccinated individuals with ≥50 lesions were found to be just as contagious as unvaccinated individuals.[35]

MYTH 3: VARIZIG IS DIFFICULT TO OBTAIN SO ANTIVIRALS AND INTRAVENOUS IMMUNOGLOBULIN SHOULD BE USED FOR POSTEXPOSURE PROPHYLAXIS

Myth

According to recent survey data, several knowledge gaps exist among physicians about the availability and use of VARIZIG for postexposure prophylaxis against VZV.

Facts

For >50 years, a previous formulation of VZIG was used to prevent or attenuate clinical varicella and its complications. However, in 2006, VZIG was discontinued and replaced by a new VZIG (human) formulation, VARIZIG. In 2012, the US Food and Drug Administration approved VARIZIG for postexposure prophylaxis of VZV in high-risk populations, including immunocompromised persons, newborns of mothers with perinatal and postnatal varicella, premature infants, infants 1 year and older, adults without proven seroprotection, and pregnant women.[8] Specifically, VARIZIG is for individuals who are exposed to PV or HZ and are unable to be vaccinated against varicella, who lack evidence of seroprotection against VZV, who have a high likelihood of VZV exposure leading to infection, and who are at high risk for severe disease.[2,9] The CDC and American Academy of Pediatrics guidelines recommend the use of VARIZIG ideally within 96 hours, but VARIZIG can be administered up to 10 days after VZV exposure.[9,36]

According to a recent survey, most patients are prophylactically treated after VZV (varicella or HZ) exposure with antivirals (eg, acyclovir) (Figure). However, there is limited clinical evidence to support the use of antivirals for varicella prophylaxis.[37] In some patients, intravenous immunoglobulin was reportedly used as postexposure prophylaxis to VZV; however, intravenous immunoglobulin titer levels are variable and unreliable for prophylaxis.[38] There was confusion regarding VZIG, the first commercial VZIG preparation available in the United States, and VARIZIG. Most physicians surveyed were not aware that VZIG was discontinued and replaced by VARIZIG, and many did not know that VARIZIG was in fact widely distributed and available or stocked in their practice setting (Figure). The CDC website now indicates VARIZIG is widely available from a broad network of specialty distributors in the United States and can be easily obtained within 24 hours for high-risk patients after VZV exposure.[2]

DISCUSSION

Varicella and HZ remain global public health issues in large part because of several common myths and misconceptions about VZV exposure risk, transmission, and current treatment options. Addressing these myths and misconceptions is challenging but important, especially for high-risk populations.

Although national varicella vaccinations rates are high, exposure to varicella or HZ is more common than most people realize. Increased incidence of HZ heightens the risk of exposure for susceptible individuals. VZV transmission occurs through direct contact with varicella or HZ lesions and respiratory exposure to aerosolized virus. Survey data from the CDC indicate that medical and nonmedical vaccination exemption rates are increasing nationwide. Overall, nonmedical exemptions weaken herd or community immunity, placing those who are unable to get vaccinated for medical reasons (eg, persons immunocompromised because of disease or

A

Physician survey respondents		
Patient population	Specialty	No.of surveys (N = 215)
Adult treaters (57%)	Hematology-oncology, non-SCT	30
	Hematology-oncology, SCT	31
	Infectious disease specialist	31
	OB/GYN	32
Pediatric treaters (43%)	Hematology-oncology	30
	Infectious disease specialist	30
	Neonatologist	31

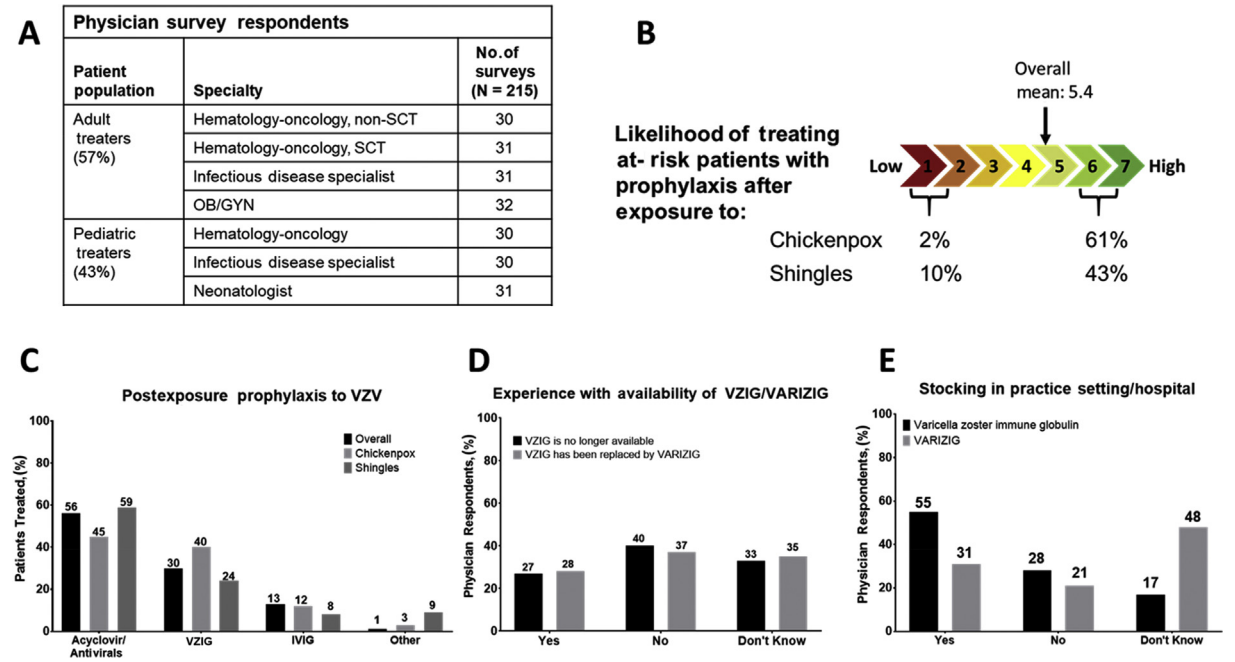

Figure. Self-administered internet survey of physicians in full-time clinical practice who see or manage patients at high risk for complications after exposure to varicella-zoster virus (VZV). (A) Physician survey respondent stratification. (B) Likelihood of treating at-risk patients with prophylaxis after exposure to chickenpox (primary varicella) or shingles (herpes zoster [HZ]) on a scale of 1 (low likelihood) to 7 (high likelihood). (C) Prophylaxis after VZV (varicella/chickenpox or HZ/shingles) exposure. (D) Physician experience with varicella zoster immune globulin (VZIG) or varicella zoster immune globulin (human) (VARIZIG) availability. (E) Physician understanding of VZIG or VARIZIG stocking in their practice setting or hospital. SCT = stem cell transplant; OB/GYN = obstetrician/gynecologist.

immunosuppressive therapy) at increased risk for exposure and infection. In addition, vaccinated individuals still face breakthrough disease because of waning immunity. HZ exposure represents a significant risk, especially in high-risk individuals, and HZ vaccine uptake is low. With increased prevalence of immune-related disorders and new immunosuppressive treatment indications, the incidence of PV after VZV exposure may increase concomitantly.

VARIZIG is the only passive immunization product approved for postexposure prophylaxis of VZV in high-risk individuals without immunity. Administration of VARIZIG is intended to reduce varicella severity. VARIZIG is widely available and easily obtained for high-risk patients after VZV exposure.[2] Increased awareness of the common misconceptions and facts of VZV transmission and the appropriate form of postexposure prophylaxis may help reduce the incidence of severe disease in susceptible individuals.

FUNDING SOURCES

Funding was provided by Saol Therapeutics.

CONFLICTS OF INTEREST

Dr Newman has received funding/grant support from Merck. Dr Jhaveri has received funding/grant support from AbbVie, Gilead, and Merck and honorarium for consultancy from MedImmune/Astra Zeneca. He is also an unpaid consultant for Saol. The authors have indicated that they have no other conflicts of interest regarding the content of this article.

ACKNOWLEDGMENTS

We thank Saol Therapeutics for support of this article. Medical writing support, funded by Saol Therapeutics, was provided by Caroline Walsh Cazares, PhD, of JB Ashtin, who developed the first draft based on an author-approved outline and assisted in implementing author revisions throughout the editorial process. All authors contributed equally to the research concept and study design; data acquisition and interpretation; and wrote, edited, reviewed, and approved the manuscript for publication.

REFERENCES

1. Gershon AA, Breuer J, Cohen JI, et al. Varicella zoster virus infection. *Nat Rev Dis Primers*. 2015;1:15016.
2. Centers for Disease Control and Prevention. *Chickenpox (Varicella) for Healthcare Professionals*. Atlanta, GA: Centers for Disease Control and Prevention; 2018.
3. Long SS, Prober CG, Fischer M. *Principles and Practice of Pediatric Infectious Diseases*. 5th ed. Philadelphia, PA: Elsevier; 2018.
4. Mueller NH, Gilden DH, Cohrs RJ, Mahalingam R, Nagel MA. Varicella zoster virus infection: clinical features, molecular pathogenesis of disease, and latency. *Neurol Clin*. 2008;26:675–697. viii.
5. Cherry JD, Harrison GJ, Kaplan SL, Hotez PJ, Steinbach WJ. *Feigin and Cherry's Textbook of Pediatric Infectious Diseases*. 8th ed. Philadelphia, PA: Elsevier; 2019.
6. Lopez AS, Zhang J, Marin M. Epidemiology of varicella during the 2-dose varicella vaccination program - United States, 2005-2014. *MMWR Morb Mortal Wkly Rep*. 2016;65:902–905.
7. Marin M, Guris D, Chaves SS, et al. Prevention of varicella: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Recommen Rep Morb Mortal Wkly Rep*. 2007;56:1–40.
8. *VARIZIG Prescribing Information*. Roswell, GA: Saol Therapeutics; 2018.
9. Centers for Disease Control and Prevention. Updated recommendations for use of VariZIG–United States, 2013. *MMWR Morb Mortal Wkly Rep*. 2013;62:574–576.
10. Yawn BP, Saddier P, Wollan PC, St Sauver JL, Kurland MJ, Sy LS. A population-based study of the incidence and complication rates of herpes zoster before zoster vaccine introduction. *Mayo Clin Proc*. 2007;82:1341–1349.
11. Leung J, Harpaz R, Molinari NA, Jumaan A, Zhou F. Herpes zoster incidence among insured persons in the United States, 1993-2006: evaluation of impact of varicella vaccination. *Clin Infect Dis*. 2011;52:332–340.
12. Centers for Disease Control and Prevention. Shingles Surveillance. https://www.cdc.gov/shingles/surveillance.html. Accessed 28 January 2019.
13. Centers for Disease Control and Prevention. *Adult Vaccination Coverage General Population Dashboard*; 2017. https://www.cdc.gov/vaccines/imz-managers/coverage/adultvaxview/data-reports/general-population/dashboard/2017.html. Accessed 28 January 2019.
14. Lopez AS, Marin M. *Strategies for the Control and Investigation of Varicella Outbreaks Manual*; 2008. https://www.cdc.gov/chickenpox/outbreaks/manual.html. Accessed 28 January 2019.
15. Papaloukas O, Giannouli G, Papaevangelou V. Successes and challenges in varicella vaccine. *Ther Adv Vaccin*. 2014;2:39–55.
16. Zhu S, Zeng F, Xia L, He H, Zhang J. Incidence rate of breakthrough varicella observed in healthy children after 1 or 2 doses of varicella vaccine: results from a meta-analysis. *Am J Infec Contr*. 2018;46:e1–e7.
17. Centers for Disease Control and Prevention. Outbreak of varicella among vaccinated children–Michigan, 2003. *MMWR Morb Mortal Wkly Rep*. 2004;53:389–392.
18. Tugwell BD, Lee LE, Gillette H, Lorber EM, Hedberg K, Cieslak PR. Chickenpox outbreak in a highly vaccinated school population. *Pediatrics*. 2004;113:455–459.
19. Leung J, Broder KR, Marin M. Severe varicella in persons vaccinated with varicella vaccine (breakthrough varicella): a systematic literature review. *Expert Rev Vaccin*. 2017;16:391–400.
20. Mellerson JL, Maxwell CB, Knighton CL, Kriss JL, Seither R, Black CL. Vaccination coverage for selected vaccines and exemption rates among children in kindergarten - United States, 2017-18 school year. *MMWR Morb Mortal Wkly Rep*. 2018;67:1115–1122.
21. US Department of Health and Human Services, National Center for Immunization and Respiratory Disease. *SchoolVaxView Interactive!*. Atlanta, GA: US Department of Health and Human Services, National Center for Immunization and Respiratory Disease; 2017.
22. Olive JK, Hotez PJ, Damania A, Nolan MS. The state of the antivaccine movement in the United States: a focused examination of nonmedical exemptions in states and counties. *PLoS Med*. 2018;15, e1002578.
23. Lieu TA, Ray GT, Klein NP, Chung C, Kulldorff M. Geographic clusters in underimmunization and vaccine refusal. *Pediatrics*. 2015;135:280–289.
24. Glanz JM, McClure DL, Magid DJ, Daley MF, France EK, Hambidge SJ. Parental refusal of varicella vaccination and the associated risk of varicella infection in children. *Arch Pediatr Adolesc Med*. 2010;164:66–70.

25. Harpaz R, Dahl RM, Dooling KL. Prevalence of immunosuppression among US adults, 2013. *JAMA*. 2016;316:2547–2548.
26. Malaiya R, Patel S, Snowden N, Leventis P. Varicella vaccination in the immunocompromised. *Rheumatology*. 2015;54:567–569.
27. Viner K, Perella D, Lopez A, et al. Transmission of varicella zoster virus from individuals with herpes zoster or varicella in school and day care settings. *J Infect Dis*. 2012;205:1336–1341.
28. Bloch KC, Johnson JG. Varicella zoster virus transmission in the vaccine era: unmasking the role of herpes zoster. *J Infect Dis*. 2012;205:1331–1333.
29. Sawyer MH, Chamberlin CJ, Wu YN, Aintablian N, Wallace MR. Detection of varicella-zoster virus DNA in air samples from hospital rooms. *J Infect Dis*. 1994;169:91–94.
30. Suzuki K, Yoshikawa T, Tomitaka A, Matsunaga K, Asano Y. Detection of aerosolized varicella-zoster virus DNA in patients with localized herpes zoster. *J Infect Dis*. 2004;189:1009–1012.
31. Josephson A, Gombert ME. Airborne transmission of nosocomial varicella from localized zoster. *J Infect Dis*. 1988;158:238–241.
32. Depledge DP, Brown J, Macanovic J, Underhill G, Breuer J. Viral genome sequencing proves nosocomial transmission of fatal varicella. *J Infect Dis*. 2016;214:1399–1402.
33. Weber DJ, Rutala WA, Hamilton H. Prevention and control of varicella-zoster infections in healthcare facilities. *Infect Contr Hosp Epidemiol*. 1996;17:694–705.
34. Advisory Committee on Immunization P, Centers for Disease Control and Prevention. Immunization of health-care personnel: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Recomm Rep*. 2011;60:1–45.
35. Seward JF, Zhang JX, Maupin TJ, Mascola L, Jumaan AO. Contagiousness of varicella in vaccinated cases: a household contact study. *JAMA*. 2004;292:704–708.
36. Pediatrics AAO. Varicella-zoster infections. In: Pickering LKBC, Kimberlin DW, Long SS, eds. *Red Book: 2012 Report of the Committee on Infectious Diseases*. 29th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2012:774–789.
37. Johansen JS, Westergren T, Lingaas E. Post-exposure varicella prophylaxis [in Norwegian]. *Tidsskr Nor Laegeforen*. 2011;131:1645–1648.
38. Paryani SG, Arvin AM, Koropchak CM, et al. Comparison of varicella zoster antibody titers in patients given intravenous immune serum globulin or varicella zoster immune globulin. *J Pediatr*. 1984;105:200–205.

Address correspondence to: Ravi Jhaveri, MD, FAAP, Northwestern University Feinberg School of Medicine, Ann & Robert H Lurie Children's Hospital of Chicago, 225 E Chicago Ave, Box 20, Chicago, IL 60611, USA. E-mail: ravi.jhaveri@northwestern.edu