

Air Sickness: Vomiting and Environmental Transmission of Norovirus on Aircraft

Ben Lopman

Division of Viral Diseases, National Center for Immunization and Respiratory Diseases, Atlanta, Georgia

(See the Article by Thornley et al, on pages 515–20.)

Vomiting is a common—perhaps the defining—feature of norovirus gastroenteritis. Indeed, the syndrome was once known as “winter vomiting disease.” Vomiting often occurs with no prodromic forewarning; thus, public vomiting appears to be the spark that starts the rapid spread of many outbreaks. Both classic outbreak investigations [1] and sophisticated modeling studies [2] have demonstrated the role of vomiting in direct transmission. This suggests a role for vomitus-oral transmission through droplets, more frequently associated with transmission of respiratory pathogens. Cases occurring through this mode of transmission occur within 1 incubation period (<48 hours for norovirus) from the time of the vomiting incident. In addition, after being aerosolized by vomiting, norovirus settles on surfaces and may persist and remain infectious, from which cases may occur days or even weeks later.

After a public vomiting incident aboard a long haul flight on a Boeing 777-200, 27

flight attendants who worked on the aircraft on 8 flight sectors over 6 days suffered symptoms of gastroenteritis consistent with norovirus infection [3]. The evidence that these illnesses were a result of a common exposure to norovirus is circumstantial, but compelling nonetheless. Aside from the crew on the first flight sector on which the vomiting incident occurred, there was no apparent opportunity for direct transmission from symptomatic crew to each other. No flight attendants worked on the aircraft while ill or within 48 hours after they recovered. Although both pre- and postsymptomatic transmission of norovirus have been documented in other studies [4], this is not likely to have played a major role in this situation. Two flight attendants from different sectors had laboratory-confirmed infections with the rare genogroup 1 genotype 6 (G1.6) norovirus. The chances that both individuals were infected with this particular norovirus strain and were not part of the same chain of transmission are improbably small. The long-standing norovirus sequence database of the viruses causing outbreaks in New Zealand and worldwide [5] was crucial to making this molecular epidemiological inference.

One particularly revealing observation was the dose-response relationship between norovirus exposure and the risk of

developing disease. Attack rates among flight attendants decreased as exposure became more distant in time from the vomiting incident. This indeed may be a good proxy for the exposure dose. At the time of shedding, through vomitus in this case, most particles are likely to be infectious. In circumstances in which infectious particles still suspended in air can be inhaled and ingested, norovirus spreads very rapidly [6], which probably happened on the first flight sector, from which the attack rate among flight attendants was phenomenally high (90%). However, when the virus has to pass through the environment, transmission may not be as efficient. After landing on a surface, some of the infectious virus particles are lost for transmission, because they are on locations that cannot be touched or wiped off or because they are irreversibly attached. Surface-attached virus dies at a certain rate because of drying, exposure to sunlight, or other conditions adverse for the virus. Any virus particle that is picked up by humans touching contaminated surfaces can only cause infection when it is ingested [7]. This process likely explains why attack rates in the second 2-flight sector remained high (78%; 7 cases) and subsequently decreased over the next 6 sectors (≤ 3 cases per sector). This complex process is what underlies Thornley et al's

Received 6 June 2011; accepted 10 June 2011.

Correspondence: Ben Lopman, MSc, PhD, Centers for Disease Control and Prevention, 1600 Clifton Rd MS-G04, Atlanta, GA 30333 (blopman@cdc.gov).

Clinical Infectious Diseases 2011;53(6):521–522

© The Author 2011. Published by Oxford University Press on behalf of the Infectious Diseases Society of America. All rights reserved. For Permissions, please e-mail: journals.permissions@oup.com.

1058-4838/2011/536-0004\$14.00

DOI: 10.1093/cid/cir486

dose-response: with each subsequent flight sector, the amount of infectious virus that a flight attendant was exposed to decreased, as did their risk of infection [3].

Direct transmission from public vomiting, such as the event that ostensibly sparked this outbreak, is very difficult to interrupt in such confined settings. In this outbreak, the cabin was cleaned using a range of approaches, including surface disinfection by parachlorometaxenol. This agent is not registered by the US Environmental Protection Agency as effective against norovirus. Regardless, this effort was seemingly undertaken after the outbreak was well underway. Even effective disinfection would not stop the initial cases that were a result of direct transmission. However, in this outbreak, the majority of cases in flight attendants did not occur on the first flight sector and were likely a result of environmentally mediated spread. Among the many challenges posed by our inability to culture norovirus in the laboratory is that it has not been possible to develop a direct evidence base for the efficacy of hand sanitizers and surface disinfectants. Practical guidelines have been developed largely on the basis of experiments with surrogate viruses (feline calicivirus and murine norovirus) [8] that focus on the use of sodium hypochlorite, at least for surfaces where

its use is acceptable. Additional studies are needed to find disinfectants that are both effective and safe for use in the range of settings where norovirus outbreaks occur.

Copious shedding in feces (10^6 – 10^9 viruses/gram of stool), potential for widespread and rapid dissemination by vomit, a short incubation period, environmental stability, resistance to chemical disinfection, broad genetic diversity, and limited cross-protective immunity of short duration make norovirus a pernicious pathogen. Swift effective action is required for its control. Unfortunately, we are still some way off from quantifying the full burden of norovirus gastroenteritis, much less have an evidence base for its control. As we move toward that goal, rigorous outbreak investigations, such as the one reported by Thornley et al [3], can be illuminating and teach us something new about this important pathogen.

Acknowledgments

I thank Dr. Peter Teunis, Dr. Aron Hall, Dr. Jan Vinjé, and Dr. Umesh Parashar for their constructive comments on this commentary.

The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the Centers for Disease Control and Prevention.

Potential conflicts of interest. Author certifies no potential conflicts of interest.

The author has submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed in the Acknowledgments section.

References

1. Marks PJ, Vipond IB, Carlisle D, Deakin D, Fey RE, Caul EO. Evidence for airborne transmission of Norwalk-like virus (NLV) in a hotel restaurant. *Epidemiol Infect* **2000**; 124:481–7.
2. O'Neill PD, Marks PJ. Bayesian model choice and infection route modelling in an outbreak of Norovirus. *Stat Med* **2005**; 24: 2011–24.
3. Thornley CN, Emslie NA, Sprott TW, Greening GE, Rapana JP. Recurring norovirus transmission on an airplane. *Clin Infect Dis* **2011**; 53:521–26.
4. Lo SV, Connolly AM, Palmer SR, Wright D, Thomas PD, Joynson D. The role of the pre-symptomatic food handler in a common source outbreak of food-borne SRSV gastroenteritis in a group of hospitals. *Epidemiol Infect* **1994**; 113:513–21.
5. Siebenga JJ, Vennema H, Zheng DP, et al. Norovirus illness is a global problem: emergence and spread of norovirus GII.4 variants, 2001–2007. *J Infect Dis* **2009**; 200:802–12.
6. Glass RI, Parashar UD, Estes MK. Norovirus gastroenteritis. *N Engl J Med* **2009**; 361: 1776–85.
7. Teunis PF, Brienens N, Kretzschmar ME. High infectivity and pathogenicity of influenza A virus via aerosol and droplet transmission. *Epidemics* **2010**; 2:215–22.
8. Updated norovirus outbreak management and disease prevention guidelines. Division of Viral Diseases, National Center for Immunization and Respiratory Diseases, Centers for Disease Control and Prevention. **2011**. MMWR Recomm Rep 60:1–18.