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V. Background

Guideline for the Prevention and Control of Norovirus Gastroenteritis Outbreaks in Healthcare Settings (2011)

AT A GLANCE

Background from the Guideline for the Prevention and Control of Norovirus Gastroenteritis Outbreaks in Healthcare Settings (2011).

ON THIS PAGE

Background

[The Pathogenesis of Human Norovirus Infection](#)

Background

Norovirus is the most common etiological agent of acute gastroenteritis and is often responsible for outbreaks in a wide spectrum of community and healthcare settings. These single-stranded RNA viruses belong to the family *Caliciviridae*, which also includes the genera Sapovirus, Lagovirus, and Vesivirus.¹ Illness is typically self-limiting, with acute symptoms of fever, nausea, vomiting, cramping, malaise, and diarrhea persisting for 2 to 5 days.^{2,3} Noteworthy sequelae of norovirus infection include hypovolemia and electrolyte imbalance, as well as more severe medical presentations such as hypokalemia and renal insufficiency. As most healthy children and adults experience relatively mild symptoms, sporadic cases and outbreaks may be undetected or underreported. However, it is estimated that norovirus may be the causative agent in over 23 million gastroenteritis cases every year in the United States, representing approximately 60% of all acute gastroenteritis cases.⁴ Based on pooled analysis, it is estimated that norovirus may lead to over 91,000 emergency room visits and 23,000 hospitalizations for severe diarrhea among children under the age of five each year in the United States.^{5,6}

Noroviruses are classified into five genogroups, with most human infections resulting from genogroups GI and GII.⁶ Over 80% of confirmed human norovirus infections are associated with genotype GII.4.^{7,8} Since 2002, multiple new variants of the GII.4 genotype have emerged and quickly become the predominant cause of human norovirus disease.⁹ As recently as late 2006, two new GII.4 variants were detected across the United States and resulted in a 254% increase in acute gastroenteritis outbreaks in 2006 compared to 2005.¹⁰ The increase in incidence was likely associated with potential increases in pathogenicity and transmissibility of, and depressed population immunity to these new strains.¹⁰ CDC conducts surveillance for foodborne outbreaks, including norovirus or norovirus-like outbreaks, through voluntary state and local health reports using the Foodborne Disease Outbreak Surveillance System (FBDSS). CDC summary data for 2001-2005 indicate that caliciviruses (CaCV), primarily norovirus, were responsible for 29% of all reported foodborne outbreaks, while in 2006, 40% of foodborne outbreaks were attributed to norovirus.¹¹ In 2009, the National Outbreak Reporting System (NORS) was launched by the CDC after the Council of State and Territorial Epidemiologists (CSTE) passed a resolution to commit states to reporting all acute gastroenteritis outbreaks, including those that involve person-to-person or waterborne transmission.

Norovirus infections are seen in all age groups, although severe outcomes and longer durations of illness are most likely to be reported among the elderly.² Among hospitalized persons who may be immunocompromised or have significant medical comorbidities, norovirus infection can directly result in a prolonged hospital stay, additional medical complications, and, rarely, death.¹⁰ Immunity after infection is strain-specific and appears to be limited in duration to a period of several weeks, despite the fact that seroprevalence of antibody to this virus reaches 80-90% as populations transition from childhood to adulthood.² There is currently no vaccine available for norovirus and, generally, no medical treatment is offered for norovirus infection apart from oral or intravenous repletion of volume.²

Food or water can be easily contaminated by norovirus, and numerous point-source outbreaks are attributed to improper handling of food by infected food-handlers, or through contaminated water sources where food is grown or cultivated (e.g., shellfish and produce) ([Updated Norovirus Outbreak Management and Disease Prevention Guidelines](#) [PDF](#)) The ease of its transmission, with a very low infectious dose of 10 -100 virions, primarily by the fecal-oral route, along with a short incubation period (24-48 hours) ^{12,13}, environmental persistence, and lack of durable immunity following infection, enables norovirus to spread rapidly through confined populations.⁶

Institutional settings such as hospitals and long-term care facilities commonly report outbreaks of norovirus gastroenteritis, which may make up over 50% of reported outbreaks.¹¹ However, cases and outbreaks are also reported in a wide breadth of community settings such as cruise ships, schools, day-care centers, and food services, such as hotels and restaurants. In healthcare settings, norovirus may be introduced into a facility through ill patients, visitors, or staff. Typically, transmission occurs through exposure to direct or indirect fecal contamination found on fomites, by ingestion of fecally-contaminated food or water, or by exposure to aerosols of norovirus from vomiting persons.^{2,6} Healthcare facilities managing outbreaks of norovirus gastroenteritis may experience significant costs relating to isolation precautions and PPE, ward closures, supplemental environmental cleaning, staff cohorting or replacement, and sick time.

The Pathogenesis of Human Norovirus Infection

The P2 subdomain of the viral capsid is the likely binding site of norovirus, and is the most variable region on the norovirus genome.¹⁴ The P2 ligand is the natural binding site with human HBGA, which may be the point of initial viral attachment.¹⁴ HBGA is found on the surfaces of red blood cells and is also expressed in saliva, in the gut, and in respiratory epithelia. The strength of the virus binding may be dependent on the human host HBGA receptor sites, as well as on the infecting strain of norovirus. Infection appears to involve the lamina propria of the proximal portion of the small intestine,¹⁵ yet the cascade of changes to the local environment is unknown.

Clinical diagnosis of norovirus gastroenteritis is common, and, under outbreak conditions, the Kaplan Criteria are often used to determine whether gastroenteritis clusters or outbreaks of unknown etiology are likely to be attributable to norovirus.¹⁶ These criteria are:

1. Submitted fecal specimens negative for bacterial and if tested, parasitic pathogens,
2. Greater than 50% of cases reporting vomiting as a symptom of illness,
3. Mean or median duration of illness ranging between 12 and 60 hours, and
4. Mean or median incubation period ranging between 24 and 48 hours.

The current standard for norovirus diagnostics is reverse transcriptase polymerase chain reaction (RT-PCR), but clinical laboratories may use commercial enzyme immunoassays (EIA), or electron microscopy (EM).⁶ ELISA and transmission electron microscopy (TEM) demonstrate high sensitivity but lower specificities against the RT-PCR gold standard. The use of enzyme-linked immunosorbent assays (ELISA) and EM together can improve the overall test characteristics—particularly test specificity.¹⁷ Improvements in PCR have included the development of multiple nucleotide probes to detect a spectrum of genotypes as well as methods to improve detection of norovirus from dilute samples or low viral loads and those containing PCR-inhibitors.¹⁸ While the currently available diagnostic methods are capable, with differing degrees of sensitivity and specificity, of detecting the physical presence of human norovirus from a sample, its detection does not directly translate into information about residual infectivity.

A significant challenge to controlling the environmental spread of norovirus in healthcare and other settings is the paucity of data available on the ability of human strains of norovirus to persist and remain infective in environments after cleaning and disinfection.¹⁹ Identifying the physical and chemical properties of norovirus is limited by the fact that human strains are presently uncultivable *in vitro*. The majority of research evaluating the efficacy of both environmental and hand disinfectants against human norovirus over the past two decades has primarily utilized feline calicivirus (FCV) as a surrogate. It is still unclear whether FCV is an appropriate surrogate for human norovirus, with some research suggesting that human norovirus may exhibit more resistance to disinfectants than does FCV.²⁰ Newer research has identified and utilized a murine norovirus (MNV) surrogate, which exhibits physical properties and pathophysiology more similar to those of human norovirus.²⁰ Currently, the Environmental Protection Agency (EPA) offers a list of approved disinfectants demonstrating efficacy against FCV, and the Federal Drug Administration (FDA) is responsible for evaluating hand disinfectants with label-claims against FCV as a surrogate for human norovirus (among other epidemiologically significant pathogens). It is unknown whether there are variations of physical and chemical tolerances to disinfectants and other virucidal agents among the various human norovirus genotypes. Other research pathways are evaluating the efficacy of fumigants, such as vapor phase hydrogen peroxides, as well as fogging methods as virucidal mechanisms to eliminate norovirus from environmental surfaces.

[READ NEXT](#)
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TABLE OF CONTENTS

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