In 1972, a virus was discovered in fecal specimens collected during the investigation of a community outbreak of gastroenteritis in Norwalk, Ohio. Although it became known as the Norwalk virus, as more viral gastroenteritis outbreaks were investigated it became clear that Norwalk virus was only one of a large family of viruses. Hence, subsequent terminology for these viruses included Norwalk-like viruses and small, round-structured viruses (SRSVs), describing their electron microscopical appearance. SRSVs are now recognized as the most common cause of outbreaks of acute nonbacterial gastroenteritis in the community.

Outbreaks typically occur in institutional settings, including personal-care homes, nursing homes, day-care centres, schools, dormitories, campgrounds and cruise ships. Although transmission occurs year-round, there is a winter peak, historically described as “winter vomiting disease.”

More recently, SRSVs have been renamed noroviruses, belonging to the norovirus genus of the Caliciviridae family of viruses. There are five genogroups of noroviruses (G-I to G-V), three of which (G-I, G-II, G-IV) are known to cause human disease. The original Norwalk virus belongs to genogroup G-I. Each genogroup can be further divided into several genetic clusters. Noroviruses are very stable and can withstand freezing, heating to 60°C and disinfection with chlorine at concentrations below 10 parts per million. A person can become infected with as few as 100 viral particles or less.

What are the clinical manifestations?
Viral gastroenteritis caused by norovirus is so typical that a definitive diagnosis can often be made based on clinical presentation alone. Typically the illness is of sudden onset with marked nausea and projectile vomiting, as well as non-bloody, watery diarrhea with the absence of fever in most instances. Symptom onset usually occurs after a short incubation and the illness is usually of short duration.

A Gut Feeling: Viral GI Infections

A Family Affair

• At 3:30 p.m., Elizabeth, 30, phones her husband, Shawn, at work to come home urgently because she “passed out” on the bathroom floor after experiencing protracted vomiting.

• She was too weak to attend to their two children (aged 2 and 8).

• Her husband arrived home at 4:00 p.m. to find his wife in stable condition, but exhausted in bed.

• At 4:30 p.m., Shawn experienced abrupt onset of projectile vomiting and voluminous watery diarrhea. He managed to crawl into bed and, too exhausted to move, made a phone call to a neighbour to come and attend to the children. The neighbour removed the children from the household for the night, noticing that the parents were practically incapacitated by around 5:00 p.m.

What happens to Shawn and Elizabeth? Go to page 96 for the answer.
Although clinical resolution is usually rapid, symptoms can last for 10 days. The illness is believed to be most communicable from symptom onset to 48 hours after symptom resolution. Duration of symptoms decrease with age and vomiting is a more prominent clinical manifestation in adults. Mortality is uncommon, but may occur in the very young and the very old if dehydration is not managed aggressively.

Antibodies against noroviruses are obtained early in life, often before the age of two in 70% to 80% of the infant population. This suggests a very high population prevalence of the disease. Unfortunately, immunity conferred by antibodies is short-lived (up to 14 weeks) and is likely genogroup-specific. It is, therefore, unlikely that an effective vaccine can be developed in the near future.

What are the modes of transmission?

There are multiple modes of acquisition of noroviruses, however, it is primarily a microorganism with fecal-oral transmission. Direct person-to-person spread is the most commonly recognized mode of transmission in norovirus outbreaks, accounting for 50% to 75% of cases. There is evidence that projectile vomiting may also result in airborne transmission, with aerosolized virus being swallowed after inhalation, leading to intestinal infection.

Foodborne transmission is well-recognized with contaminated bivalve shellfish (especially oysters), foods contaminated by infected foodhandlers and fruits/vegetables contaminated through irrigation, being the more common vehicles of transmission. Outbreaks have also occurred as a result of contaminated drinking water, including contaminated private and public wells and large community water systems. As noroviruses are resistant to chlorine inactivation, it is not surprising that municipal water supplies can become contaminated.

Transmission within hospitals, personal care homes and nursing homes is particularly efficient. Attack rates of 30% to 50% in both staff and patients/residents are common. Modes of transmission in these settings are likely a combination of person-to-person, airborne and heavily contaminated environments.

What is the molecular epidemiology?

Noroviruses are so diverse that, until 1995, no two strains from different outbreaks were identical. However, in early 1995, a shift was noted in the US with more than 50% of norovirus outbreak strains isolated from geographically separated unrelated outbreaks indistinguishable by RNA polymerase chain reaction, belonging to the genogroup G-II family of noroviruses. Also

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noted during the winter of 1995-96 was a significant increase in the number of genogroup G-II norovirus outbreaks reported in the US, Europe, South America, China, Australia and Canada. No explanation was put forward to explain this global phenomenon, as subsequent years revealed a great diversity of norovirus outbreak strains, with no single genogroup cluster predominating.

In 2002, the US Center for Disease Control and Prevention noticed an increase in reports of norovirus outbreaks in long-term care facilities, hospitals, restaurants, schools, residential summer camps and cruise ships. These outbreaks were otherwise typical, except for the observation that 41% of them were associated with a genogroup G-II, cluster 4 (G-II/4) norovirus that was named the Farmington Hills strain, after the location where the first gastroenteritis outbreaks had been reported.

Also in 2002, a large number of cruise ship outbreaks were reported to the CDC and were found to be attributed to this same G-II/4 norovirus, although no consistent epidemiologic links were identified. This G-II/4 norovirus was practically indistinguishable from the 1995 to 1996 genogroup G-II strain that caused global outbreaks. Furthermore, throughout 2002, several European countries observed atypical summer peaks of norovirus outbreaks also caused by a G-II/4 norovirus indistinguishable from the Farmington Hills strain.

In retrospect, it is evident that a global pandemic of norovirus outbreaks occurred in the years 1995-96 and 2002, caused by a unique norovirus clone of the G-II/4 cluster. The explanation for these pandemics is speculative. It is possible that this new norovirus variant might be more virulent or environmentally stable than previous strains. It may also be possible that this virus is more transmissible, as it has been described as having a higher prevalence of projectile vomiting, which could lead to more efficient transmission (i.e., by the airborne route). Similar to what has been seen with other micro-organisms, such as methicillin-resistant Staphylococcus aureus and Clostridium difficile in recent years, might this be an epidemic strain of norovirus?

What can we do about prevention and control?

Since little is understood about norovirus epidemiology, including why the G-II/4 cluster strain has not been prevalent since 2002, basic infection control precautions remain the mainstay of prevention and control. Routine practices, include:

- meticulous hand hygiene,
- appropriate use of personal protective

What’s wrong with Shawn and Elizabeth?

The following morning, Shawn and Elizabeth had near complete resolution of symptoms, managing to maintain oral rehydration throughout the night. The children were returned to their household and remained asymptomatic.

Although no clinical specimens were obtained to confirm the etiology, there was a known concurrent community outbreak of viral gastroenteritis. Neither Elizabeth nor Shawn were able to determine the source of their enteric illness as there had been no recent contact with restaurants, symptomatic children or suspect water sources and neither had consumed shellfish or travelled on a cruise ship.

The sudden violent nature of the illness was as impressive as its rapid resolution.
equipment (gloves, masks, gowns) as necessary and
• environmental hygiene used for all direct patient/resident care in all health-care settings to prevent the transmission of all micro-organisms.

Routine practices, therefore, remain the best standard of care. Whether enhanced environmental cleaning beyond routine practices is necessary during a norovirus outbreak scenario is unknown. Staff exclusion policies have been attempted in long-term care facilities and hospitals, to keep symptomatic staff away from the workplace until 48 hours after symptom resolution to reduce norovirus transmission during outbreaks.

References

What control measures are effective in preventing the spread of norovirus?

Routine practices, including meticulous hand hygiene, appropriate use of personal protective equipment (gloves, masks, gowns) as necessary and environmental hygiene for all direct patient/resident care in all health-care settings to prevent the transmission of microorganisms remains the best standard of care to manage institutional norovirus outbreaks.

Whether enhanced environmental cleaning beyond routine practices or staff exclusion policies are necessary during a norovirus outbreak scenario is unknown. Cancellation of group events and limitation of admissions to affected wards/institutions might be another measure to attempt to reduce transmission during outbreaks. Ultimately, the most cost-effective prevention and control measure for norovirus infection is likely good hand-washing practice.