Epidemiol. Infect. (2002), **129**, 355–360. © 2002 Cambridge University Press
DOI: 10.1017/S0950268802007446 Printed in the United Kingdom

An outbreak of viral gastroenteritis following environmental contamination at a concert hall

M. R. EVANS[1]*, R. MELDRUM[2], W. LANE[3], D. GARDNER[3], C. D. RIBEIRO[2], C. I. GALLIMORE[4] AND D. WESTMORELAND[2]

[1] *Public Health Directorate, Bro Taf Health Authority, Cardiff CF10 3NW, UK*
[2] *Department of Medical Microbiology & Public Health Laboratory, University Hospital of Wales, Heath Park, Cardiff CF14 4XW, UK*
[3] *Environment and Public Protection Division, Cardiff County Council, Cardiff CF10 1NQ, UK*
[4] *Enteric Virus Unit, Enteric Respiratory and Neurological Virus Laboratory, CPHL, London, NW9 5HT, UK*

(*Accepted 17 May 2002*)

SUMMARY

In January 1999, an outbreak of viral gastroenteritis affected more than 300 people who attended a metropolitan concert hall over a 5-day period. Norwalk-like virus (NLV) was confirmed in faecal samples by reverse transcription polymerase chain reaction assay. The index case was a concert attendee who vomited in the auditorium and adjacent male toilet. Gastrointestinal illness occurred among members of 8/15 school parties who attended the following day. Children who sat on the same level of the auditorium as the index case were much more likely to be ill than those seated elsewhere (relative risk 7·1, 95% confidence interval 5·4–9·2, $P < 0{\cdot}001$). The majority of other reported cases had not been present on the evening of the vomiting incident. Disinfection procedure was poor and the disinfectant used contained no sodium hypochlorite. Transmission most likely occurred through direct contact with contaminated fomites. The outbreak has implications for disinfection procedures following vomiting incidents at public venues.

INTRODUCTION

Norwalk-like viruses (NLV) are a leading cause of outbreaks of acute gastroenteritis in a variety of settings [1]. The virus is usually transmitted directly from person-to-person by faecal-oral spread, or through contaminated food and water [2]. In a closed environment it may be spread by airborne droplets produced during vomiting [3–5]. Gastroenteritis probably occurs at a low background level in the community until an infected individual contaminates a common source, and an explosive outbreak occurs. Outbreaks are generally limited in extent unless transmission is facilitated by a closed environment (e.g. a nursing home) or prolonged by renewal of the susceptible population (e.g. a new set of passengers on a cruise ship) [6]. Environmental contamination has long been suspected to play a role in hospital outbreaks and the development of reverse–transcriptase polymerase chain reaction assay (RT–PCR) has confirmed that such contamination does occur, at least in the immediate environment of symptomatic patients [7]. The role of environmental contamination in causing NLV outbreaks, however, remains unclear.

On 29 January 1999 the Environment and Public Protection Division, Cardiff County Council was notified of outbreaks of gastroenteritis, characterized by vomiting, at two primary schools affecting 90 of 120 (75%) children and 73 of 195 (37%) children

* Author for correspondence: PHLS Communicable Disease Surveillance Centre (Wales), Abton House, Wedal Road, Cardiff CF14 3QX, UK.

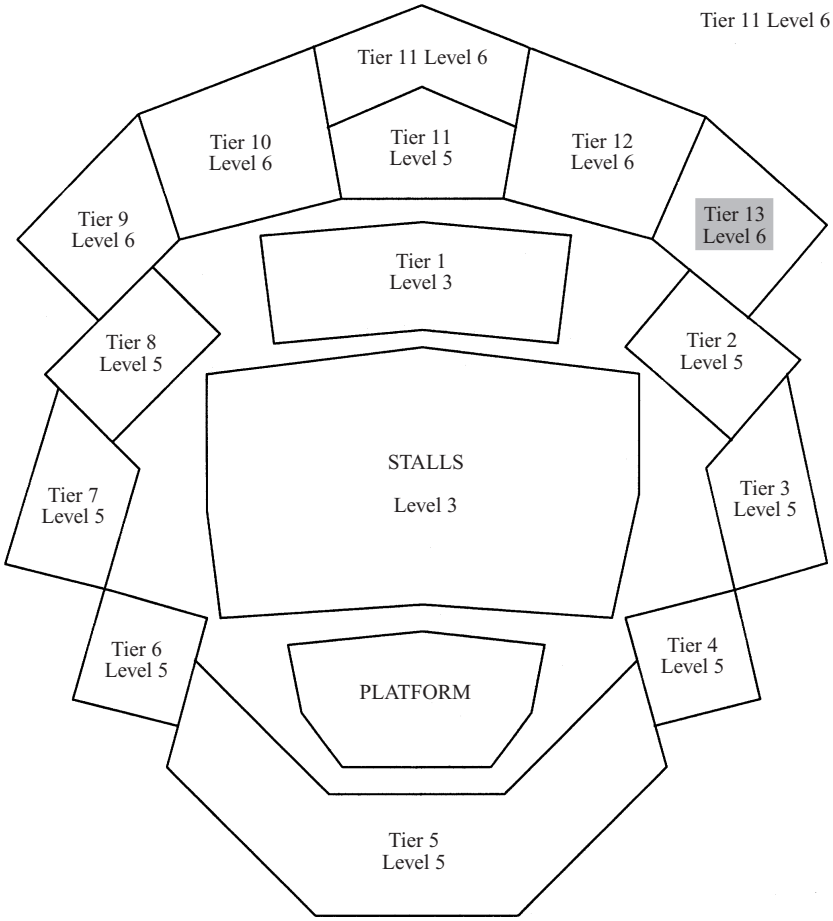

Fig. 1. Plan of the concert hall involved in an outbreak of viral gastroenteritis, Cardiff, UK, January 1999, showing location of index case ▩.

respectively. Both groups of children had attended the same lunchtime concert in Cardiff on 27 January. The concert had taken place at a large metropolitan concert hall with seating capacity for 2000 persons, and had been attended by children from a large number of primary schools in the area. An investigation was started to identify the causal agent, the source of infection and mode of spread; to assess the possibility of ongoing transmission; and to recommend control measures.

METHODS

Epidemiological investigation

The initial investigation centred upon the lunchtime concert on 27 January. A plan of the concert hall auditorium was obtained (Fig. 1), and a list of all schools that had attended the concert was sought from the concert organisers. School headteachers were interviewed by telephone and asked about where the school party had been seated in the auditorium, the number of children who had attended the concert and the number who had subsequently been ill. Details of any food or drink consumed at the concert hall were also obtained. A case was defined as any person who had attended the concert hall from 26 January onwards and who had developed vomiting and/or diarrhoea within 24–72 h of the visit.

The outbreak was publicised in the local press and several cases among other concert attendees were identified either after people contacted the concert hall or reported symptoms to the Council. Details of illness and location of seating within the auditorium were recorded. No active case searching through box office records was carried out.

Environmental investigation

An environmental health officer inspected the concert hall and reviewed food hygiene standards, food preparation practices and disinfection procedures. All auditorium and catering staff were interviewed in detail about their job, where they worked within the

https://doi.org/10.1017/S0950268802007446 Published online by Cambridge University Press

concert hall, whether they were aware of any incidents of vomiting in concert hall attendees during or before the concert on 27 January, and what disinfection procedures were followed for dealing with such incidents. They were also asked if they had been ill themselves.

Microbiological investigation

Faecal specimens were submitted to the Public Health Laboratory Service (PHLS) for bacterial and viral examination. Samples were cultured for Salmonellae, Shigellae, *Campylobacter* species, *E. coli* O157:H7, *Staphylococcus aureus*, *Clostridium perfringens*, and *Bacillus cereus*. Fixed stained films were examined for the presence of *Cryptosporidium* species and wet preparations were examined for other ova, cysts and parasites. Samples were examined for the presence of NLV RNA using reverse transcriptase polymerase chain reaction (RT–PCR). Briefly, nucleic acid was extracted using guanidium thiocyanate and silica and the RNA was converted to complementary DNA using random primers and reverse transcriptase. PCR was performed using a broadly reactive primer pair Ni/E3 [8]. PCR amplicons were cloned using a TOPO cloning vector (Invitrogen, UK) and sequenced using a Beckman capillary sequencer (Beckman Coulter, UK), according to the manufacturer's instructions. Contiguous sequences were compared to known NLV sequences on the Enteric Virus Unit NLV sequence databank and GenBank by pairwise alignment.

RESULTS

Interviews of the concert hall staff identified a vomiting incident during the concert on the evening of 26 January, but none on 27 January. The index case was a male concert attendee seated in tier 13, level 6 (Fig. 1) who vomited four times in the concert hall. Further details about the case were ascertained after a family member reported the incident in response to press publicity about the outbreak. The case had been ill before attending the concert with his family. At 8.30 p.m., during the concert, he left his seat because he felt nauseous. He reached the corridor outside the main arena and vomited into a waste bin, and then went to the toilets on level 5 where he vomited into the toilet and used the wash hand basin. At around 9.30 p.m. he left his seat to vomit again. On this occasion he reached the emergency fire escape and vomited on the floor. He proceeded to the same toilet as before and washed himself. At the end of the performance, as he was leaving, he suffered a bout of violent vomiting on a carpeted area on the top tier walkway on level 6. All three members of his family who accompanied him reported illness within 48 h of the vomiting incident. Concert hall staff cleaned up the vomit using an emergency spillage compound after the auditorium had been cleared. The carpeted areas were also cleaned with an ordinary vacuum cleaner the next day, but not until after the lunchtime school concert. No hypochlorite-based product was used.

The lunchtime concert on 27 January was attended by 1229 children from 15 primary schools (Table 1). Symptoms of illness meeting the case definition were reported in 257 (20·9%) children, but attack rate by school was 0·6–75%. Five schools reported no cases of illness. Illness was characterized mainly by vomiting and in most children duration of illness was 12–24 h. Most children had become ill on 29 January. Children had not been served with any food or drink during the concert. The single highest attack rate occurred in school A who were seated in tier 13, the same tier as the index case (Fig. 1). High attack rates also occurred in schools B, C and E who were seated in the other tiers (tiers 9–12) on the same level as the index case (level 6), particularly those on the same side of the auditorium (tiers 11–12). Individuals seated in these tiers would have used the level 6 walkway where the index case vomited the previous evening. There were also cases in pupils of school F who sat in tier 2, situated immediately below (and overhung by) tier 13. There were virtually no cases from schools that sat distant to tier 13, particularly those that sat in the stalls, three levels (storeys) below. In all, 199/387 (51·4%) children who sat in tiers 9–13 were ill compared to a total of 58/797 (7·3%) of children who sat elsewhere (relative risk 7·1, 95% CI 5·4–9·2, $P < 0{\cdot}001$).

Illness meeting the case definition was also reported by 37 other concert attendees including 10 who attended an event on 26 January, 9 on 29 January, 12 on 30 January and 6 on 31 January (there was no event on 28 January). All the cases except one had sat in the upper tiers (tiers 11–13) of the auditorium (Fig. 1). Sixteen concert hall staff met the case definition: 9 became ill on 28 January, 4 on 29 January and 3 on 30 January. All staff with a date of onset on 28 January had either helped to clear up after the vomiting incident on the evening of 26 January or had worked on the upper tiers on the same night.

https://doi.org/10.1017/S0950268802007446 Published online by Cambridge University Press

Table 1. *Illness attack rates in primary school pupils who attended the lunchtime concert on 27 January 1999 at a concert hall in Cardiff, UK*

School	No. of pupils attending	No. (%) of pupils ill	Median date of onset of symptoms	Seating location in concert hall
A	120	90 (75)	29 Jan	Tiers 12 and 13
B	195	73 (37)	29 Jan	Tiers 9–12
C	72	36 (50)	29 Jan	Tier 11
D	45	22 (49)	29 Jan	Tiers 3, 7 and 8
E	66	20 (30)	29 Jan	Tiers 8 and 11
F	110	10 (9)	29 Jan	Tiers 2 and 8
G	70	2 (3)	29 Jan	Stalls
H	20	2 (10)	29 Jan	Stalls
I	172	1 (0·6)	29 Jan	Stalls
J	43	1 (2)	29 Jan	Tier 3
K	90	0 (0)	—	Tiers 1 and 7
L	88	0 (0)	—	Tier 1
M	52	0 (0)	—	Stalls
N	32	0 (0)	—	Stalls
O	54	0 (0)	—	Stalls

No faecal samples were obtained from the index case as the time elapsed between the incident and the identification of the individual was considered to be too long. Faecal samples from two ill school children were negative for all bacterial and parasitic pathogens, but both specimens were positive for NLV RNA by RT–PCR. The NLV strain responsible for this outbreak is most similar to a German strain Hu/NLV/Pfaffenhofen028/2000 (GenBank AF3125198) and a UK strain Hu/NLV/Girlington/1993/UK [9], with 98% nucleotide identity over this region of the RNA polymerase gene.

DISCUSSION

This was a large point source outbreak of NLV affecting over 300 people in which transmission occurred over several days, almost certainly as a consequence of environmental contamination. Though NLV was identified from only two faecal samples, the clinical features of the illness were typical of a viral gastroenteritis. There were several reasons for the paucity of faecal specimens including the delay in obtaining information from schools (over a weekend), the predominance of vomiting as the main symptom, and the rapid resolution of illness. Since no active case searching based on box office records was conducted, there may have been many more cases among members of the general public who attended concerts between 26 and 31 January than were reported to the investigating team. Under-reporting of viral gastroenteritis is common because it is usually a mild illness. Although no specimen was obtained from the index case, it is clear from the time-scale of the vomiting incident and the spatial distribution of cases that he was the most likely cause of the outbreak. Most of the cases were children, staff and other concert attendees seated or working in the top tiers on the same level as the index case. Highest attack rates were in the school parties that sat closest to the area where the index case sat, particularly those that used the walkway where one of the vomiting episodes occurred.

Concert attendees and staff seated or working in the upper tiers on the evening of the vomiting incident were probably infected by exposure to aerosol. However, aerosol transmission does not adequately explain bridging from the vomiting incident on the 26 January to cases exposed only between 27 and 31 January. If aerosol had persisted, it would have been distributed throughout the auditorium by the overhead vents of the air-conditioning system, giving rise to widespread distribution of cases, rather than cases localised to people seated in tiers 9–13. The common link between these cases is use of the same exit on level 6 via the top tier walkway, the exact spot where the index case vomited. Schoolchildren, concert attendees and staff that were exposed after 26 January are therefore most likely to have been infected through persisting environmental contamination. Males may have been infected when using the contaminated male

toilet on level 6, but female cases could only have been exposed when using the contaminated upper tier walkway. Viral ingestion then occurred either from direct contact with contaminated hands, clothing or footwear, or possibly as a result of the virus becoming airborne again as people walked across contaminated floor areas. The most likely cause of the outbreak was inadequate cleaning and disinfection of hard surfaces, carpets and soft furnishings contaminated by vomit.

The NLV strain detected in this outbreak (Hu/Pfaffenhofen028/2000/DE) was more common than the Hu/NLV/Grimsby/1995/UK in 1998/9 [9]. Hu/NLV/Grimsby/1995/UK is the predominant NLV strain currently circulating and has been the predominant strain since 1992 [10], except for 1993/4 where Hu/NLV/Mexico/1989/MX (GenBank U22498) was an epidemic strain. Both Grimsby and Pfaffenhofen are genogroup II Norwalk-like viruses.

NLV was first found in vomit in studies on volunteers deliberately inoculated with the virus under controlled circumstances [11]. More recently, NLV from vomit specimens has been further characterized by RT–PCR during investigation of hospital outbreaks of gastroenteritis [7, 12]. Infected vomit may contain up to 10^6 infectious particles/ml [13], and incidents of projectile vomiting can give rise to infectious aerosols that may remain suspended for significant periods of time [3]. The infective dose of NLV may be as low as 10–100 particles [13], and in confined spaces ingestion of aerosol is likely. It has been proposed that aerosols formed during vomiting are, in fact, one of the main routes of transmission in outbreaks not related to food [13, 14].

Infection by both person-to-person contact and airborne droplets has been postulated to explain recurrent outbreaks on a cruise ship [15], and secondary spread within the home [16]. Several NLV outbreaks associated with airborne transmission after vomiting incidents have been described including in hospital [3, 12, 17] on an air-conditioned coach [4], and at a restaurant [5]. Transmission has also been documented from food prepared in a sink contaminated with vomit by a foodhandler [18].

The role of environmental contamination in NLV outbreaks is less clear. Prolonged outbreaks on ships [6, 15, 19] suggest that NLV survives well in the environment. Investigation of recurrent outbreaks on a cruise ship found that illness was associated with sharing bathrooms and further outbreaks were only prevented after repeated and thorough bathroom cleaning [15]. Extensive environmental contamination of a hospital ward during an NLV outbreak has been demonstrated by RT–PCR testing of swabs from commodes, lockers and curtains [7]. One report in which two carpet-fitters developed gastroenteritis after removing a contaminated carpet suggests that the virus is not eliminated by repeated vacuum cleaning and can remain viable for at least 12 days [14]. It has been suggested that even light contamination of surfaces is enough to allow transmission to occur, and that environmental contamination may be the cause of bridging between cases [20].

In response to this outbreak, it was recommended that all hard surfaces, particularly the toilets, be cleaned with a hypochlorite-based solution and that all carpets, seats and soft furnishings between level 4 and 6 in the auditorium, stairwells and communal areas be steam cleaned. Detailed advice was also given to the management staff on procedures and cleaning regimens for dealing with any future vomiting incidents in public areas.

This outbreak demonstrates the highly infectious nature of NLV and the potential for viral particles to survive in the environment for up to 5 days after an incident as a consequence of inadequate environmental cleaning. It also illustrates that outbreaks associated with vomiting are not just confined to closed communities such as hospitals, or to confined spaces such as a ship's cabin. Rigorous cleaning procedures have been developed based on experience in managing hospital outbreaks of gastroenteritis [21]. To ensure effective cleaning after a vomiting incident in a public place we recommend that staff undertaking cleaning should wear a mask, that all hard surfaces be disinfected with freshly prepared 0·1% (1000 ppm) hypochlorite and that all carpets and soft furnishings be cleaned with hot water and detergent using a disposable cloth, followed by steam cleaning.

ACKNOWLEDGEMENTS

The authors gratefully acknowledge the assistance of the Enteric Virus Unit, ERNVL at the PHLS Central Public Health Laboratory, Colindale, London for their help and assistance in analysing the samples.

REFERENCES

1. Kaplan JE, Gary GW, Baron RC, et al. Epidemiology of Norwalk gastroenteritis and the role of Norwalk virus in outbreaks of acute nonbacterial gastroenteritis. Ann Intern Med 1982; **96**: 756–61.

2. Centers for Disease Control. Viral agents of gastroenteritis. Public health importance and outbreak management. MMWR 1990; 39 (No. RR-5): 1–23.
3. Sawyer LA, Murphy JJ, Kaplan JE, et al. 25 to 30 nm virus particles associated with a hospital outbreak of acute gastroenteritis with evidence of airborne transmission. Am J Epidemiol 1988; **127**: 1261–71.
4. Chadwick PR, Walker M, Rees AE. Airborne transmission of a small round structured virus. Lancet 1994; **343**: 171.
5. 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.
6. Gunn RA, Terranova WA, Greenberg HL, et al. Norwalk virus gastroenteritis aboard a cruise ship: an outbreak on five consecutive cruises. Am J Epidemiol 1980; **112**: 820–7.
7. Green J, Wright PA, Gallimore CI, et al. The role of environmental contamination with small round structured viruses in a hospital outbreak investigated by reverse-transcriptase polymerase chain reaction assay. J Hosp Infect 1998; **39**: 39–45.
8. Green J, Gallimore CI, Norcott JP, Lewis D, Brown DWG. Broadly reactive reverse transcriptase polymerase chain reaction (RT–PCR) for the diagnosis of SRSV-associated gastroenteritis. J Med Virol 1995; **47**: 392–8.
9. Vinje J, Green J, Lewis DC, Gallimore CI, Brown DW, Koopmans MP. Genetic polymorphism across regions of the three open reading frames of Norwalk-like viruses. Arch Virol 2000; **145**: 223–41.
10. Hale A, Mattick K, Lewis D, et al. Distinct epidemiological patterns of Norwalk-like virus infection. J Med Virol 2000; **62**: 99–103.
11. Greenberg HB, Wyatt RG, Kapikian AZ. Norwalk virus in vomitus. Lancet 1979; **i**: 55.
12. Marx A, Shay DK, Noel JS, et al. An outbreak of acute gastroenteritis in a geriatric long-term-care facility: combined application of epidemiological and molecular diagnostic methods. Infect Control Hosp Epidemiol 1999; **20**: 306–11.
13. Caul EO. Small round structured viruses: airborne transmission and hospital control. Lancet 1994; **343**: 1240–2.
14. Cheesborough JS, Barkess-Jones L, Brown DW. Possible prolonged environmental survival of small round structured viruses. J Hosp Infect 1997; **35**: 325–6.
15. Ho M-S, Monroe SS, Stine S, et al. Viral gastroenteritis aboard a cruise ship. Lancet 1989; **ii**: 961–5.
16. Heun EM, Vogt RL, Hudson PJ, Parren S, Gary GW. Risk factors for secondary transmission in households after a common-source outbreak of Norwalk gastroenteritis. Am J Epidemiol 1987; **126**: 1181–6.
17. Chadwick PR, McCann R. Transmission of small round structured virus by vomiting during a hospital outbreak of gastroenteritis. J Hosp Infect 1994; **26**: 251–9.
18. Patterson W, Haswell P, Fryers PT, Green J. Outbreak of small round structured virus gastroenteritis arose after kitchen assistant vomited. CDR Rev 1997; **7**: 101–3.
19. Corwin AL, Soderquist R, Edwards M, et al. Shipboard impact of a probable Norwalk virus outbreak from coastal Japan. Am J Trop Med Hyg 1999; **61**: 898–903.
20. Caul EO, Sellwood NJ, Brown DW, et al. Outbreaks of gastroenteritis associated with SRSVs. PHLS Microbiology Dig 1993; **10**: 2–8.
21. Chadwick PR, Beards G, Brown D, et al. Management of hospital outbreaks of gastroenteritis due to small round structured viruses. J Hosp Infect 2000; **45**: 1–10.