A large, prolonged outbreak of human calicivirus infection linked to an aged–care facility

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Abstract

This report investigates an outbreak of acute gastrointestinal illness, microbiologically and epidemiologically linked to an aged–care facility and seeks to determine if there was a point source of infection. A register of cases that included onset date and time of illness and symptoms was maintained by nursing staff. Faecal specimens were tested for conventional gastrointestinal pathogens and for human calicivirus (HuCV). There were 81 cases reported. Specimens were received for testing from 25 cases. Twenty–three of the 25 (92%) specimens were positive for HuCV RNA by reverse transcriptase polymerase chain reaction (RT–PCR). The 2 negative samples contained RT–PCR inhibitors. Descriptive epidemiology suggested that staffing practices were important in prolonging the outbreak. No point source of infection was identified. Instead environmental contamination, aerosol transmission and work practices that fail to take account of the natural history of HuCV infection probably contributed to the size (81 cases) and duration (3 weeks) of this outbreak among the residents, staff and visitors of an aged–care facility and their contacts. Institutional outbreaks caused by HuCV, formerly called Norwalk–like or small round structured viruses, are extremely difficult to control. Infected staff may contribute significantly to the amplification of outbreaks. Rapid confirmation of HuCV infection is now routinely possible using polymerase chain reaction diagnostics but progress in laboratory technology has not yet translated into faster or more effective interventions. Commun Dis Intell 2002;26:261–264.

Keywords: outbreak, Norwalk-like virus; calicivirus, small round structured virus

Introduction

Human caliciviruses (HuCVs), formerly called Norwalk–like viruses (NLVs) or small round structured viruses (SRSVs), have long been suspected to cause outbreaks of acute gastroenteritis.1 Until recently determining the specific aetiology of these outbreaks has been hampered by the insensitivity of microbiological diagnostics. In deciding whether cases of acute gastroenteritis were caused by HuCVs, epidemiologists have had to depend on the combination of laboratory tests being negative for all other pathogens and the occurrence of characteristic symptoms of HuCV infection: an illness duration of 12 to 60 hours, an incubation period 15 to 48 hours and vomiting being more prominent than diarrhoea.2 The development of antigen detection methods and, more significantly, polymerase chain reaction (PCR) diagnostics now make it possible to definitively determine the cause of many outbreaks previously only suspected as being the result of HuCV infection. We present the investigation of a 3 week long outbreak of acute HuCV gastroenteritis which affected 81 people associated with an aged–care facility in Adelaide.

The outbreak

On 24 August 2000, the Communicable Disease Control Branch (CDCB) was notified of an acute outbreak of gastrointestinal illness in an aged–care facility in metropolitan Adelaide. The initial report was of gastrointestinal illness among several hostel residents and staff but not among residents of the associated nursing home. The symptoms were consistent with the classic presentation of HuCV.2
Methods

Case definition
A case was defined as a person living, working, visiting or epidemiologically linked to the aged-care facility with acute onset of diarrhoea or vomiting between 14 August and 3 September 2000.

Epidemiological investigation
Staff of the aged-care facility maintained an illness register. Staff from CDCB visited the aged-care facility and gathered data on resident seating arrangements at meal-time and residents’ room numbers. Residents were not interviewed because of cognitive impairment.

Environmental investigation
A local environmental health officer reviewed food preparation, food storage and hygiene practices among food handlers at the facility and arranged the collection of faecal specimens.

Microbiological investigation
Faeces from 25 symptomatic residents, staff and their contacts were tested for conventional parasitic, bacterial and viral (rotavirus and adenovirus) pathogens by the Institute of Medical and Veterinary Science (IMVS), using routine methods. In addition, tests were conducted for HuCVs by RT–PCR assays using primers specific for each of the three main groups of HuCV: Norwalk–like virus group 1 (NLV–1), NLV–2 and Sapporo–like viruses (SLV) and for astroviruses.3

Infection control measures
Standard infection control practices were routine at this institution. Additional infection control measures were initiated on 24 August 2000 when the outbreak was first reported to the CDCB in accordance with published recommendations.2 As well, ill hostel residents were isolated and signs were erected informing visitors about the outbreak. Staff were advised not to return to work for 48 hours after symptoms resolved.

Results

Epidemiological investigation
Setting
The aged-care facility had 107 (17 males, 90 females) residents, of whom 64 (60%) were resident in the hostel. The other residents lived in the nursing home section. Hostel residents had their own rooms, however, meals were eaten in a common dining room and residents had specific seating arrangements. Nursing home residents slept in single rooms with the exception of two, who shared a room. Only 16 (37%) of the 43 nursing home residents ate meals in a dining room (separate to the dining room used by hostel residents). Food was prepared in a central kitchen on site for both the nursing home and hostel residents. Seventy–five staff members were employed at the aged-care facility. Nursing staff worked between the 2 areas, particularly during periods of staff shortages.

Outbreak description
Of the 107 residents, 65 (61%) reported gastrointestinal illness. The epidemic curve (Figure) shows a slow start and protracted course (over a 3 week period). The first person to become ill was a hostel resident who had not left the facility prior to illness. The first wave, observed from 14 to 23 August 2000, occurred mainly among hostel residents and included 2 nursing staff members. The second wave from 24 August to 3 September 2000 affected mainly nursing home residents and staff. One visitor and an indirect contact (daughter of a case from the hostel and the daughter’s grandson who did not visit the hostel) reported illness at the beginning of the second wave.

Figure. Cases of human calicivirus in an aged-care facility, 14 August to 3 September 2000, by date of onset.
Hostel residents
The attack rate among hostel residents was 73 per cent (47/64). Of these, 31 (66%) experienced diarrhoea and vomiting, 8 (17%) vomiting and 8 (17%) had symptoms of diarrhoea. The mean age was 85 years.

Nursing home residents
Of the 43 nursing home residents, 18 (42%) were ill. Of these, 8 (44%) experienced vomiting, 8 (44%) diarrhoea and 2 (11%) had diarrhoea and vomiting. The mean age was 87 years.

Staff
Of the 75 staff, 14 (19%) were ill. Of these, 7 (50%) experienced diarrhoea, 5 (36%) vomiting and diarrhoea and 2 (14%) had vomiting. The first 2 staff members to become ill were a nurse who would visit all areas of the facility and a nurse who worked in the hostel. The onset of illness in kitchen staff did not occur until the beginning of the second wave. Staff who worked in the nursing home were the last to become ill. The only staff member with NLV–2 detected in faeces was a nurse who worked in the hostel section prior to onset of illness.

Visitor
A visitor to the facility reported contact with vomit on her mother’s nightdress and on the bedroom floor. The soiled nightdress was removed from the facility and washed at home in the presence of a grandson. The grandson had not accompanied his grandmother to the complex earlier in the day. Within 18 hours, the daughter and grandson experienced vomiting and diarrhoea. In both, the duration of illness was 24 hours.

Environmental investigation
The environmental health inspection revealed problems with food quality and food handling practices. Serving utensils had been left in foods on the stove, in the cool-room and on the preparation bench. Cracked wooden spoons were in use accumulating food particles and potentially harbouring microbes. Inadequate cleaning was observed in the kitchen, for example, the accumulation of grime and dirt on surfaces and equipment. A further follow-up inspection was conducted by the environmental health officer.

Microbiological investigation
No parasites or bacterial pathogens were detected in any of the 25 individual case specimens. All adenovirus antigen assays were negative as were RT–PCR assays for NLV–1, SLV and astroviruses. The RT–PCR assay for NLV–2 RNA was positive in 23 of 25 (92%) specimens, with specimens from the remaining 2 cases (1 from a hostel resident and 1 from a nurse) containing RT–PCR inhibitors which could not be removed. Two hostel residents who were positive for NLV–2 RNA were also positive for rotavirus antigen.

Discussion
We can expect in Australia that institutional outbreaks of acute gastroenteritis proven definitively to be caused by HuCVs will now be reported regularly.\textsuperscript{4,5} The chief aim of investigating such outbreaks is to determine if transmission has occurred person to person or through contaminated food or drink. HuCVs in oysters and orange juice have caused large outbreaks in Australia.\textsuperscript{6} Kitchen workers infected with HuCV may cause more circumscribed outbreaks through food contamination.\textsuperscript{7} Although the investigation reported here revealed problems with food quality and handling, the descriptive epidemiology of this outbreak strongly suggests that transmission occurred by person-to-person contact. It was particularly significant that, although the hostel and the nursing home residents were fed the same food from the same kitchen, the outbreak followed a biphasic pattern: only 4 new cases occurred in hostel residents after the first case in a nursing home resident. Most of the affected staff only became ill after the outbreak spread to the nursing home, perhaps because of the necessarily closer contact between staff and these dependent, ill residents. The sudden increase of cases among residents of the hostel only after the first staff member became ill suggests that staff had a crucial role in amplifying the outbreak. By contrast, contact between residents at meal times was unrelated to the time of onset of disease. Single room accommodation, which would reduce resident-to-resident contact, did not prevent spread of the infection suggesting the importance of ill staff in prolonging the outbreak.

Controlling person-to-person transmission of HuCV gastroenteritis in an institution is very difficult. It is almost certain that these viruses can be spread by airborne transmission, so in contrast to other
gastrointestinal pathogens which spread strictly by
the faecal–oral route, handwashing alone is not
efficacious. Also because these viruses are
relatively hardy, they can survive for some time on
contaminated bedding and clothes and presumably could be re–aerosolised. The
grandson of the visitor who became ill after contact
with the nightdress of one of the residents
illustrates the extreme infectivity of the agent.

Because virus excretion commences some hours
prior to the onset of symptoms it is recommended
that staff from a ward affected by an HuCV
outbreak not be transferred to new work areas for
48 hours after their last shift in the affected ward.2
Also HuCVs continue to be excreted some days
after symptoms have ceased and ill health care
workers and food–handlers should not
recommence duty until at least 48 hours after their
last symptoms.10 The public health message is that
conditions of employment that encourage early
return to work after HuCV illness may contribute to
prolonging outbreaks. In practice, especially when
symptomatic illness reduces staff numbers, it is
probably difficult for administrators to adopt these
restrictions with the result, as in the situation
reported here, that the outbreak may spread
further and be prolonged.

**References**

1. Glass RI, Noel J, Ando T, et al. The epidemiology of
enteric caliciviruses from humans: a reassessment

of hospital outbreaks of gastroenteritis due to small

3. Ratcliff RM, Doherty C, Higgins GD. Detection of
human calicivirus and astrovirus in human faeces

4. Penson MJ, Ressler KA, McIver CJ, Issacs M,
Rawlinson WD. Norwalk–like virus as a cause of a
gastroenteritis outbreak in a childcare centre. *Aust N

R. Three nursing home outbreaks of Norwalk–like
virus in Brisbane in 1999. *Comm Dis Intell*

6. Fleet GH, Heiskanen P, Reid I, Buckle KA. Foodborne
viral illness in Australia. *Int J Food Microbiol*

7. Patterson W, Haswell P, Fryers PT, Green J. Outbreak
of small round structured virus gastroenteritis arose
after kitchen assistant vomited. *Commun Dis Rep CDR*

viruses: public health significance. *IMVS Newsletter*

9. 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.

10. White KE, Osterholm MT, Mariotti JA, Korlath JA. A
foodborne outbreak of Norwalk virus gastroenteritis.