

SHORT REPORT

An outbreak of food poisoning due to a genogroup I norovirus

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SUMMARY

Norovirus infection is associated with approximately 90% of epidemic non-bacterial acute gastroenteritis. The objective of this study is to describe an outbreak of norovirus genogroup I gastroenteritis which affected workers in a hospital and was attributed to food prepared by an infected food handler. Forty cases were detected, of whom 80% were interviewed. The index case was the cook employed in the hospital cafeteria. The following symptoms were observed: abdominal pain in 90·6%, vomiting in 71·9%, diarrhoea in 71·9%, general indisposition in 62·5%, headaches in 53·1% and fever in 32·4% of cases. The initial symptoms were abdominal pain in 37% and vomiting in 28%. Of the 14 samples analysed by RT–PCR, 12 (86%) were positive for a genogroup I norovirus. After sequencing the strain was identified as genotype Desert Shield. Many of the foodstuffs consumed were made by hand, favouring transmission from the index case to the cafeteria users.

The Norwalk virus, of the genus *Norovirus* [1] of the Caliciviridae family, was discovered in the 1970s [2] but genetic analysis did not begin until the 1990s with the development of molecular biology techniques for its detection, mostly reverse-transcription polymerase chain reaction (RT–PCR) [3], which permits not only the diagnosis of the disease but also the identification of the genogroup implicated (genogroup I or genogroup II) [4].

The Norwalk virus is the most frequent cause of outbreaks of acute non-bacterial gastroenteritis in some countries [5] and foodborne gastroenteritis due to noroviruses is increasingly recognized as a public health problem. The most common symptoms are

vomiting and diarrhoea, which resolve in 24–72 h, and complications are rare. Transmission is via the faecal–oral route and may be associated with contaminated water or food, person-to-person spread through the aerosol transmission of vomit or through contact with contaminated objects and surfaces [6]. Because the virus is highly infective [7] it may cause widespread epidemic outbreaks in communities, kindergartens, schools, old people's homes, hotels and hospitals [8].

We report an outbreak of norovirus genogroup I gastroenteritis attributed epidemiologically and by sequencing to food prepared by an infected food handler.

On 22 May 2002, an outbreak of acute gastroenteritis affecting at least 18 workers in the Hospital of Mollet was reported to the Epidemiological Surveillance Unit of the Central Region of Catalonia.

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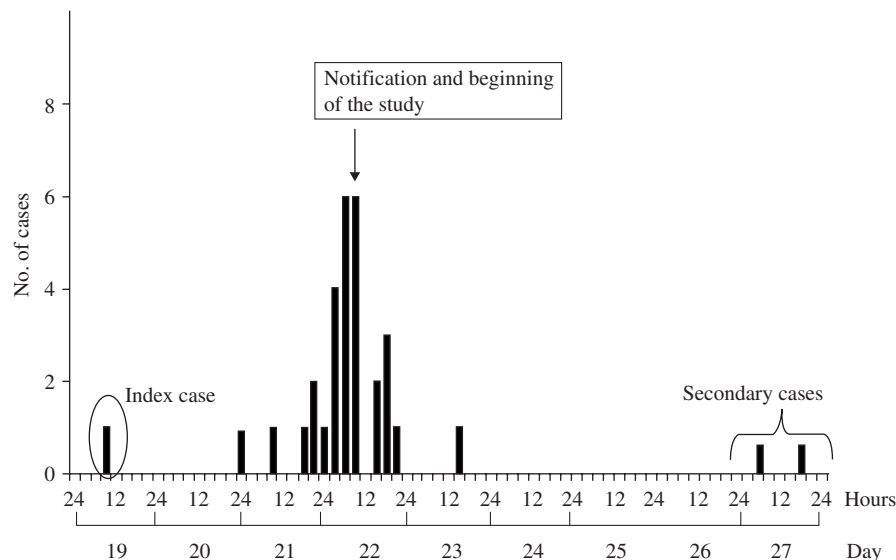


Fig. Epidemic curve of the presentation of cases.

The main symptoms were vomiting and diarrhoea. Most cases became ill during the morning of the day the outbreak was reported or the evening of the previous day. All the subjects affected had consumed some foodstuffs in the hospital cafeteria. The hospital has a canteen restricted to staff and a cafeteria used by both staff and visitors.

Sick subjects were interviewed using a standard questionnaire about symptoms, time of onset, duration of illness, food consumed in the cafeteria and illness among family members. A primary case was defined by the presence of nausea, abdominal pain, vomiting or diarrhoea (>2 episodes in 24 h) beginning during the week 19–26 May and by previous relationship with the hospital (worker, family or in-patient).

A secondary case was defined as a family member or work colleague of a primary case who became ill 24 h after the related probable or confirmed case and had not consumed foodstuffs in the hospital cafeteria.

The cafeteria kitchen facilities were inspected and food handlers were asked about hygienic procedures, the preparation of food items and absenteeism or symptoms of illness. Recommended control measures included exclusion of sick food handlers and hospital employees from work for at least 48 h after the resolution of illness, and an emphasis on hygienic measures, especially hand-washing.

A microbiological study was carried out in the laboratory of the Hospital of Mollet to detect enteropathogenic microorganisms and the Support

Laboratory for Investigation of Foodborne Diseases in Catalonia tested faeces for the presence of virus. Samples were also sent for characterization by RT-PCR for Norwalk-like viruses and for sequencing to the Hospital Clínico Universitario de Valencia.

The epidemic curve was elaborated and the incubation period calculated, taking into account the different times of exposure the day before the onset of symptoms: from 10:00 hours for staff on the morning shift who had breakfast in the cafeteria, from 14:00 hours for staff on the morning–evening shift who had lunch there, and from 19:00 hours for staff on the evening shift who had dinner there. The index case and secondary cases were excluded from the calculation.

Forty cases were detected, of whom 80% were interviewed. Thirty-seven were primary cases and three secondary cases (relatives of primary cases). Of the 40 cases, three were food handlers working in the cafeteria, 34 were hospital staff (of whom 50% were medical staff) and three were relatives. The attack rate among hospital staff was 8% (34/427), although it was not possible to ascertain the number of workers exposed, and, as many staff were not users of the cafeteria, this rate must be assumed to be lower than the real incidence.

Sixty-six per cent of the cases for whom information was available became ill during the 2 days after the appearance of the index case, the cook employed in the cafeteria (Fig.). The median incubation period for primary cases was 27 h (range 15–37 h).

Table 1. *Distribution of cases by symptoms and age groups*

Age group (years)	Abdominal pain	Diarrhoea	Vomiting	General malaise	Headaches	Fever
<10	1 (100%)	—	1 (100%)	—	—	—
20–29	4 (80%)	2 (40%)	5 (100%)	3 (60%)	2 (40%)	3 (60%)
30–39	11 (92%)	9 (75%)	7 (58%)	7 (54%)	7 (54%)	5 (39%)
40–49	7 (100%)	7 (100%)	4 (57%)	5 (71%)	6 (86%)	1 (14%)
50–59	5 (83%)	4 (67%)	5 (83%)	5 (83%)	2 (33%)	3 (50%)
≥60	1 (100%)	1 (100%)	1 (100%)	—	—	—
Total	29 (91%)	23 (72%)	23 (72%)	20 (63%)	17 (53%)	12 (37%)

Table 2. *Distribution of cases according to foods consumed*

Foods consumed	Cases who consumed the food	% Consumption	<i>P</i>
Rissoles	2	6%	—
Squid in batter	5	16%	0.23
Asparagus with eggs	6	19%	0.13
Salads	13	41%	0.001
Sandwiches	22	69%	0.000003

The symptoms reported by the cases were abdominal pain (90.6%), vomiting (71.9%), diarrhoea (71.9%), general malaise (62.5%), headaches (53.1%) and fever (32.4%). The most commonly reported initial symptoms were abdominal pain (37%) and vomiting (28%).

Females accounted for 81% of the cases. The 30–39 years age group was the most affected (Table 1). Seventy-nine per cent of cases worked on the morning, or morning–evening shifts. The foodstuffs consumed by primary cases varied widely, with sandwiches (69%) and salads (41%) being the most frequent (Table 2), with differences being statistically significant.

Faecal samples from 12 cases (three were food handlers) and from two asymptomatic food handlers were analysed. Eleven cases (92%) were positive for norovirus. The three ill food handlers, including the cook, were positive but of the other two asymptomatic handlers, one was negative and the other had a weak positive result. Thus, of the 14 samples analysed by RT-PCR, 12 (86%) were positive for norovirus of genogroup I. All these amplified fragments were sequenced and identified as genotype Desert Shield.

The cook (index case) usually prepared the sandwiches and salads. She became ill on the morning of 19 May with abdominal pain, and suffered diarrhoea in the early morning of 20 May. She continued working in spite of her illness. Her husband was also ill, suffering vomiting on the morning of 19 May while taking his wife to work.

Information from other sources indicated that the cook had dined with the family of her daughter at a popular seaside restaurant in Blanes on 17 May. The cook's son-in-law suffered vomiting and diarrhoea on 18 May.

The other food handlers (both affected and asymptomatic), who were waiters and did not cook, had, however, also eaten sandwiches prepared by the cook.

Inspection of the cafeteria revealed that there were no automatic taps or soap dispensers for sinks and washbasins, and kitchen cloths were used to dry hands. In addition, there was a toilet for the food handlers next to the cafeteria kitchen which did not have a separate washbasin.

The origin of this *Norovirus* outbreak of gastroenteritis among the users of a hospital cafeteria could have been any foodstuff handled by the cook who was the index case in the hospital and in whom the virus was found in faeces. The fact that the cook continued working while ill may have resulted in the contamination of food and the spread of the outbreak. Asymptomatic elimination of the virus can persist for more than a week, meaning that infected food handlers may be a significant source of infection [9].

Transmission of the virus could have occurred through lapses in the personal hygiene of the food handler, increasing the risk of infection, since many of the foodstuffs consumed were made by hand (sandwiches and salads).

Foods are a major cause of outbreaks and any foodstuff can transmit the infection when handled by

any infected or contaminated food handler [10]. Cold foods such as salads and sandwiches are often implicated in this type of transmission and have been reported to be the source of various outbreaks in hospitals [11].

Similarly, the fixtures and fittings of the cafeteria, which were not ideal for complying with suitable hygienic procedures could have contributed to the spread of the outbreak. The food handlers were advised to follow good kitchen hygienic practices, particularly hand washing. On hospital wards, strict control measures aimed at breaking the chain of transmission were implemented as soon as a case was suspected. These included frequent hand washing, adequate disposal of infectious material (vomit and faeces) and cleaning and disinfection of working surfaces, bathrooms and toilets [12].

The Epidemiological Surveillance Unit recommended that food handlers and health workers should not return to work for at least 48 h after recovery. Without control procedures, the outbreak could possibly have continued longer. In fact, 2 weeks after implementation, no further cases in either staff or patients had been detected (the only two cases notified 5 days after were secondary cases, i.e. relatives of primary cases), although epidemiological surveillance was maintained.

The use of RT-PCR techniques to detect *Norovirus* has increased the sensitivity of detection and enabled their molecular characterization. The strain implicated in this outbreak belonged to genogroup I, which is not the most frequent in Catalonia where genogroup II strains predominate [13, 14]. Of the 143 outbreaks of foodborne diseases reported in Catalonia in 2002, 25% were of unknown origin and 11% due to norovirus, of which 92% were of genogroup II. In fact, the outbreak described here is the only one reported in Catalonia to be caused by a genogroup I norovirus, genotype Desert Shield virus. The genogroup I strains do not seem to be as implicated in the appearance of community outbreaks as genogroup II strains [6]. After sequencing, the strain was identified as Desert Shield virus in all the samples, including that from the cook, which implicated the food handler as the source of the outbreak.

Norovirus infection also has a socioeconomic cost, both because of the high number of sporadic cases of gastroenteritis it causes and because of the widespread outbreaks in hospitals, hotels and other institutions. Microbiological confirmation of the aetiology of these outbreaks is important in determining

their real impact and instigating effective control measures.

Education of food handlers should include awareness that they may contaminate food while they have gastrointestinal illness, both before and after the illness, and also following infection or contamination by a sick family member at home. Food hygiene regulations and recommended kitchen practices, if followed, may prevent this type of outbreak.

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REFERENCES

1. van Regenmortel MH, Fauquet CM, Bishop DHL, et al. (eds.) Virus taxonomy, seventh report of the International Committee on Taxonomy of Viruses, 2000. New York: Academic Press.
2. Kapikian AZ, Wyatt RG, Dolin R, Thornhill TS, Kalica AR, Chanock RM. Visualization by immune electron microscopy of a 27-nm particle associated with acute infectious nonbacterial gastroenteritis. *J Virol* 1972; **10**: 1075–1081.
3. Ando T, Jin Q, Gentsch JR, et al. Epidemiologic applications of novel molecular methods to detect and differentiate small round structured viruses (Norwalk-like viruses). *J Med Virol* 1995; **47**: 145–152.
4. Wang J, Jiang X, Madore HP, et al. Sequence diversity of small, round-structured viruses in the Norwalk virus group. *J Virol* 1994; **68**: 5982–5990.
5. Boccia D, Tozzi AE, Cotter B, et al. Waterborne outbreak of Norwalk-like virus gastroenteritis at a tourist resort, Italy. *Emerg Infect Dis* 2002; **8**: 563–568.
6. Glass RI, Noel J, Ando T, et al. The epidemiology of enteric calicivirus from humans: a reassessment using new diagnostics. *J Infect Dis* 2000; **181**: S254–S261.
7. Götz H, Ekdahl K, Lindbäck J, de Jong B, Hedlund KO, Giesecke J. Clinical spectrum and transmission characteristics of infection with Norwalk-like virus: findings from a large community outbreak in Sweden. *Clin Infect Dis* 2001; **33**: 622–628.
8. Chadwick PR, Beards G, Brown D, et al. Management of hospital outbreaks of gastro-enteritis due to small round structured viruses. *J Hosp Infect* 2000; **45**: 1–10.
9. Jiang X, Wang J, Graham DY, Estes MK. Detection of Norwalk virus in stool by polymerase chain reaction. *J Clin Microbiol* 1992; **30**: 2529–2534.

10. Reid JA, Caul EO, White DG, Palmer SR. Role of infected food handler in hotel outbreak of Norwalk-like viral gastroenteritis: implications for control. *Lancet* 1988; **2**: 321–323.
11. Parashar UD, Dow L, Fankhauser RL, et al. An outbreak of viral gastroenteritis associated with consumption of sandwiches: implications for the control of transmission by food handlers. *Epidemiol Infect* 1998; **121**: 615–621.
12. CDC. Norwalk-like viruses: public health consequences and outbreak management. *MMWR* 2001; **50**(no. RR-9).
13. Buesa J, Collado B, Lopez-Andujar P, et al. Molecular epidemiology of caliciviruses causing outbreaks and sporadic cases of acute gastroenteritis in Spain. *J Clin Microbiol* 2002; **40**: 2854–2859.
14. Godoy P, Prat A, Alsedà A, Torres J, Domínguez A, Salleras L. Outbreak of foodborne disease by Norwalk-like genogroup II [in Spanish]. *Med Clin (Barc)* 2002; **119**: 13–15.