Eurosurveillance
Europe's journal on infectious disease epidemiology, prevention and control

Vol. 16 | Weekly issue 30 | 28 July 2011

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ecdc EUROPEAN CENTRE FOR DISEASE PREVENTION AND CONTROL

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Food-borne outbreak of norovirus infection in a French military parachuting unit, April 2011

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Citation style for this article:
Mayet A, Andréo V, Bédubourg G, Victorion S, Plantec JY, Soullié B, Meynard JB, Dedieu JJ, Polvèche PY, Migliani R. Food-borne outbreak of norovirus infection in a French military parachuting unit, April 2011.
Euro Surveill. 2011;16(30):pii=19930. Available online: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=19930

Article published on 28 July 2011

On 13 April 2011 the medical service of a French military parachuting unit reported an outbreak of acute gastroenteritis involving 147 persons among the military personnel. Meals suspected to have caused the outbreak (pasta and some raw vegetables) were tested for norovirus by PCR. The same norovirus (genogroup I) was found in some of the food items consumed by the cases and in a cook who prepared the meals.

On 13 April 2011, the medical service of a French military parachuting unit reported to the French military epidemiological centre an outbreak of acute gastroenteritis involving more than 100 persons who had fallen ill on 12 and 13 April 2011. The clinical symptoms reported by the patients were fever, vomiting and diarrhoea. Food from the military base canteen was suspected to be a potential vehicle of infection. An investigation was initiated on 14 April to describe the extent of the outbreak, to identify the causative pathogen, the mode of transmission and the source of infection, and to implement infection control measures. We report the results of this investigation as a reminder that food-borne disease outbreaks can present an important problem, particularly in settings such as military establishments where people live in close proximity and share the same facilities [1,2].

Methods

We collected information on demographics, clinical symptoms and disease onset from personnel with acute gastroenteritis among the staff present on site at the time of the investigation (n=295). A case was defined as a member of the military unit staff who presented at least one measurable symptom of the following: diarrhoea defined by three or more liquid stools in 24 hours, vomiting, and oral temperature of ≥38°C between 11 and 15 April 2011. In order to test the hypothesis that food items served in the canteen were the vehicles of infection, we conducted a case–control study which only targeted the meals served between 11 and 12 April because the unit had been closed on 9 and 10 April. Because part of the staff in the unit was not present at the time of the investigation, a retrospective cohort study was not possible. On 14 April, we distributed a standardised questionnaire to all members of the staff present in the unit. These included cases but also other staff members who did not experience any symptoms and who were therefore considered as controls. This questionnaire collected information on dates of onset of illness, symptoms and types of food consumed. Participation was voluntary and anonymous. In parallel, another investigation was initiated on 13 April by the veterinary department of the Military Health Service. This investigation consisted in the inspection of the catering process and the verification of hygiene procedures in place. Data were analysed using Stata (Stata Statistical Software: Release 9, Texas). The statistical strength of the associations was estimated by odds ratios (OR) with 95% confidence intervals (CI). In addition to the bivariate analysis, a multivariate analysis was performed using logistic regression.

At French military base canteens, meal items are routinely sampled and samples are kept for five days. We tested for norovirus the water of the drinking fountains and the food items served and sampled in the canteen on 11 and 12 April, which were suspected to be associated with the outbreak following the analytical study. The extracted RNA was tested for norovirus by real-time RT-PCR [3]. Pasta was tested by culture for *Bacillus cereus* which was initially suspected to have caused the outbreak by the physicians who treated the cases. In addition, water from the drinking fountain was tested by culture for coliform germs. For logistical reasons, no samples were requested from the cases, apart from a cook who had prepared the meals and who had fallen ill before the outbreak. The stool sample from the cook was tested for norovirus by PCR as described [3].

Results

During the investigation, 295 individuals (all staff present in the unit at the time of the investigation) were interrogated (58% of 466 persons who had eaten in the canteen on 11 and 12 April). The information regarding the total number of people who had eaten in the canteen on 11 and 12 April was obtained from the sales register of the canteen. Among 169 individuals who reported symptoms, 147 met the case definition. This corresponds to 147 cases of 295 persons who were interrogated. The remaining persons were used as controls in our study and this results in approximately one control per case. Symptoms reported by the patients included vomiting (85%), diarrhoea (79%) with a mean of four liquid stools a day, abdominal pain (91%), nausea (87%) and fever (73%). An oral temperature of ≥38°C was objectively measured in three patients. Date of symptom onset was known only for 138 cases of the 147 persons who met the case definition. The first cases occurred on 11 April in the evening and the last in the morning of 14 April, with an epidemic peak during the night of 12 April (Figure). The first cases included the cook who prepared cold dishes in the canteen on 11 and 12 April and who experienced abdominal pain and nausea starting with 11 April.

The analytical study performed on 69 food items used in four meals, showed a significant association between the occurrence of illness and the consumption of salad (OR: 2.1; 95% CI: 1.0–4.4; p=0.03) and raw vegetables (OR: 2.1; 95% CI: 1.1–3.8; p=0.01) which were prepared by the ill cook and served on 11 April at lunch and dinner. Other associations were found with water taken from a drinking fountain for the dinner of 11 April (OR: 2.6; 95% CI: 1.0–7.0; p=0.03) and pasta served on 12 April dinner (OR: 2.7; 95% CI: 1.1–7.1; p=0.01). All these statistical links disappeared in the multivariate analysis, which was not surprising due to the high number of food items tested.

As this outbreak involved a large number of cases (including a majority who occurred during a parachuting training on 12 April at night and some cases among physicians of the medical service), no samples were taken from any of the cases. However, a stool sample was tested from the ill cook and was found positive for norovirus genogroup I by PCR. Following the results of the analytical study, uncooked vegetables served on 11 April were tested for norovirus. The same genogroup I norovirus was found in carrots and salad served at lunch, and in the tomatoes served at dinner. No further genotyping was performed. Among the cases, 72% had eaten at least one of these food items. The culture of *Bacillus cereus* from pasta served on 12 April was negative. The pasta was also negative for norovirus. Analyses performed on the water from the drinking fountains did not find noroviruses or any other pathogen but retrieved coliform germs. The investigation performed by the veterinary services also revealed some dysfunction in the cold chain concerning the preparation of the cold starters and in the maintenance of the drinking fountains. Water fountains were closed until disinfection and recommendations for hygiene were given to the company responsible for catering.

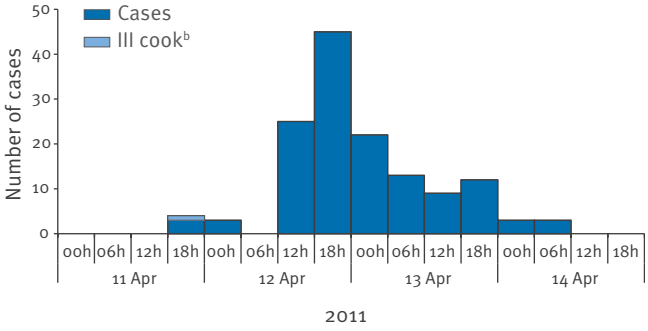
Discussion and conclusion

This norovirus-related food-borne disease outbreak involving 147 cases occurred during a parachuting exercise on the night of 12 April and affected significantly the activities of the military unit. It is interesting to note that another outbreak of acute gastroenteritis occurred between 10 and 12 April among residents of a retirement home in the same geographical area, in which the same cook involved in the outbreak in the military unit prepared food on 9 and 10 April. However, the outbreak in the nursing home was only suspected after interrogation of the ill cook; it had not been reported to the health authorities and consequently, it had not been investigated, but it is likely that it was also caused by norovirus considering that around 50% of acute gastroenteritis outbreaks in industrialised countries are related to this agent [4]. Other norovirus outbreaks related to raw vegetables have been described in the past in other military units [5,6]. The episode described here illustrates once more that food-borne disease outbreaks can easily occur in such settings and stricter hygiene measures may need to be considered.

Despite the fact that no samples were taken from cases, the presence of genogroup I norovirus in the cook who had fallen ill 24 hours before the outbreak and in some food items which he prepared, implicate him as the source of the outbreak. As the norovirus incubation period ranges from 6 to 48 hours [7] we may assume the following hypothetical sequence of events: likely contamination of the cook at the retirement home, contamination of the food items prepared by the cook for lunch and dinner on 11 April in the military unit, the occurrence of the first cases among military staff on 11 April in the evening (six hours after the first assumed

Figure

Acute gastroenteritis outbreak due to norovirus infection in a French military parachuting unit, April 2011 (n=138 cases[a])



[a] With known date of symptom onset

[b] The cook presented only subjective symptoms and did not meet the case definition, but was added to the curve for a better understanding of the outbreak.

food contamination), and the epidemic peak during the night of 12 April (24-36 hours after supposed food contamination). However, considering the date of symptom onset of the cook, it may also be possible that he contaminated both places.

The symptoms reported by the patients, particularly the fever, are compatible with a norovirus infection [7]. The infectious dose of norovirus is known to be low; therefore many food items could have been contaminated by one person only [4]. Due to the fact that in military settings people live in close proximity and share the same facilities, secondary human-to-human transmission of norovirus appears possible, which may account for the large number of cases involved in this outbreak [8]. Statistical associations observed between consumption of water and symptoms could account for a human-to-human contamination via the drinking fountains shared by the cases.

The fact that the investigation occurred 48 hours after the outbreak and that only 58% of the staff present during the episode could be interrogated, may have resulted in a lack of power of the analytical study and may also be a source of potential bias. This could explain the fact that not all dishes prepared by the ill cook were significantly associated with illness.

The fact that norovirus was detected in a non-diarrhoeic stool sample from the cook who presented only few symptoms, underlines the importance of testing samples from cases even when they are non symptomatic. Recent laboratory techniques permit the detection of norovirus from faeces up to approximately seven days following the infection [9,10]. Therefore physicians should be encouraged to collect samples from patients even if the outbreak is over.

In conclusion, this investigation demonstrates that food-borne disease outbreaks may have certain impact on the operational activities of settings such as military units but, on the other hand, the operational context may perturb the investigation and create difficulties in the identification of the vehicle of infection.

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Outbreak of norovirus infection in a hotel in Oslo, Norway, January 2011

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Citation style for this article:
Guzman-Herrador B, Heier BT, Osborg EJ, Nguyen VH, Vold L. Outbreak of norovirus infection in a hotel in Oslo, Norway, January 2011. Euro Surveill. 2011;16(30):pii=19928. Available online: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=19928

Article published on 28 July 2011

A total of 56 people were affected with gastroenteritis after attending a one-day meeting in a high-quality hotel in the centre of Oslo, Norway, at the end of January 2011. A complete outbreak investigation was carried out. The microbiological investigation confirmed that the outbreak was caused by norovirus. All participants at the meeting were invited by email to complete an online questionnaire asking for information on demographic data, symptoms and food consumption. The results of the epidemiological investigation of the food items served were inconclusive and the source and transmission route of this outbreak remains unclear. However, the environmental investigation highlighted several irregularities in the kitchen that may have enabled the spread of the virus. Specific cleaning procedures and rules were set up for the kitchen staff. As a consequence of this outbreak investigation, the hotel is planning to change its internal routine protocols, for example, samples of food items served at every meal during an event will be stored.

Introduction

Noroviruses are a group of RNA viruses belonging to the Caliciviridae family that cause gastroenteritis in humans. They are highly contagious and as few as 10 viral particles may be sufficient to infect an individual [1]. During outbreaks of gastroenteritis due to norovirus infection, several modes of transmission have been documented, the most frequent being food-borne, followed by subsequent secondary person-to-person transmission [1]. Norovirus is known to be responsible for most gastroenteritis outbreaks in winter in industrialised countries [2], such as in the European Union. In Norway, it is the most frequently reported suspected cause of food-borne outbreaks [3].

On 31 January 2011, the Norwegian Institute of Public Health (NIPH) was informed about a possible outbreak of gastroenteritis among people attending a one-day meeting organised by an international company. The meeting was held on 28 January, in a hotel in the centre of Oslo: it included around 900 participants from all over Norway. According to the initial information received, at least 30 participants had fallen ill with vomiting and diarrhoea after attending the meeting, but none required hospitalisation. The Department of Infectious Disease Epidemiology of the NIPH, in collaboration with the Food Safety Authority and the Municipal medical officer of Oslo, decided to carry out an outbreak investigation in order to measure the extent of the outbreak, identify the source, pathogen and the vehicle of transmission, and implement control measures to prevent further outbreaks. The investigation was started on 31 January.

Methods

A retrospective cohort study was conducted among all the persons who attended the meeting.

We defined an outbreak case as a person who attended the one-day meeting at the hotel in Oslo on 28 January 2011 and developed diarrhoea and/or vomiting within the following three days. The Food Safety Authority gathered information on food and drink served during the meeting. There were four servings (breakfast, lunch, snack and dinner); some of the dishes were prepared in the kitchen of the hotel, while others were cooked in other places and delivered to the hotel, ready to be served.

The NIPH Outbreak Team adapted a standard food-borne disease Internet-based questionnaire for the current outbreak, to be completed by the attendees. The questionnaire was partly based on the information from the Food Safety Authority on what was served, and contained questions on demographic data in addition to symptoms and food consumption during the meeting. On 4 February, a link to the questionnaire was sent to all the attendants of the meeting via email by the human resources department of the company who organised the meeting. One week later, on 10 February, a reminder was sent to those who had not answered yet, to try to increase the response rate. On 21 February, the online questionnaire was closed. Once the data from the participants were collected, we carried out a descriptive and univariate analysis.

On 31 January, the NIPH contacted the human resources department of the company and asked them to encourage all people who reported being sick after attending the meeting to go to a medical facility to submit a stool sample.

The Food Safety Authority went to the hotel on 2 February to carry out a routine environmental inspection of the kitchen.

Results

Epidemiological investigation

A total of 880 people from all over Norway attended the one-day meeting on 28 January in the hotel. The questionnaire was sent to all of them and 391 replied (response rate: 44%): 358 answered the questionnaire within the first week, while 33 replied after the reminder. Of the respondents, 206 (53%) were female and 64% (n=250) were between 40 and 59 years old (range: 20–74 years). They included people working in various offices around the country. Regarding symptoms, 90 respondents (23%) reported to have had at least one of the symptoms listed in the questionnaire (vomiting, diarrhoea, nausea, abdominal pain and fever), but only 56 matched the case definition (attack rate: 14%). One person reported to have had diarrhoea during the night before the meeting and thus did not meet the case definition. Of the 56 cases, 30 reported having had only diarrhoea, seven only vomiting and 19 both symptoms. As seen in Table 1, several cases reported having had more symptoms than those included in the case definition.

Table 1

Clinical features of outbreak cases due to norovirus infection, one-day meeting, Oslo, Norway, 28 January 2011 (n=56)

Clinical feature	Number
Type of symptom[a]	
Only diarrhoea	30
Only vomiting	7
Both diarrhoea and vomiting	19
Nausea	30
Abdominal pain	26
Fever	21
Duration of symptoms	
Less than one day	6
1–2 days	31
More than 2 days	19
Deaths	**0**
Hospitalisation	**0**
Went to the doctor	**4**

[a] Symptoms were not further defined in the questionnaire.

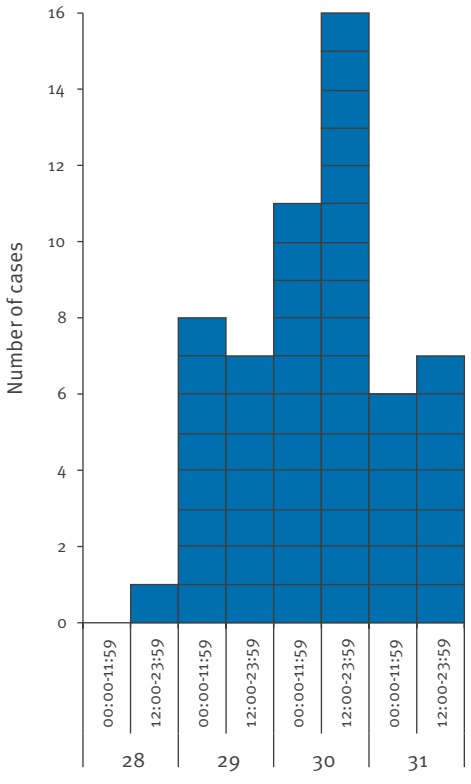
The date and time of onset of symptoms for the 56 cases are shown in the Figure. The first case became ill the same evening as the meeting. Most cases became ill 48 hours after the meeting, on 30 January, with most falling ill between noon and midnight. The last cases reported symptom onset 72 hours after the meeting.

There was no difference in the risk of infection between female and male cases. The cases worked in several different offices around Norway and there was no cluster of cases from any particular office or city. Those aged 60 years and older (n=11) had a higher attack rate (34%) and were almost three times more likely to have been sick than younger people. Very few cases reported having had contact with one or more persons who were sick during the meeting or in the four days before the meeting (Table 2). Only six cases reported having eaten something outside of the hotel during the meeting. We did not gather information on whether participants had stayed in the hotel the night before or the night after the meeting.

People exposed to seven food items served during the meeting had a higher risk of developing symptoms (Table 3). Items eaten by most of the cases were those eaten during the dinner. However, the results were not statistically significant since most of the attendants were exposed to the same foods. The two food items leading to the highest attack rate among those

Figure

Outbreak cases due to norovirus infection, one-day meeting, Oslo, Norway, 28 January 2011 (n=56)



exposed and the lowest p value were wraps and sandwiches. However, they only accounted for 52% and 70% of the cases, respectively (Table 3).

In a second univariate analysis we excluded cases who reported having had contact with people who were sick before or during the meeting. This was done in order to separate cases who were symptomatic or incubating the disease before the meeting from those who became ill as a result of the meeting. The results of this second analysis were similar to those previously calculated in the first analysis.

We also considered whether attending only one specific serving represented a higher risk of becoming a case. Results of a stratified analysis by meal were inconclusive since almost all the cases had more than one meal.

Microbiological investigation

Stool samples were taken from three of the four cases who visited a doctor: all were positive for norovirus. No further genotyping was carried out. The samples were also analysed for *Campylobacter* spp. *Salmonella* spp., *Yersinia* spp. and *Shigella* spp. – such tests are routinely performed on faecal samples in Norway. No further tests were carried out. No samples were taken from the food handlers or other kitchen employees.

It was not feasible to perform a microbiological analysis of the food items served during the meeting as there were no leftovers available when food items at the time the outbreak investigation was initiated.

Environmental investigation

The Food Safety Authority found several irregularities during their inspection of the hotel's kitchen. In particular, they observed incorrect washing routines and storage of dishes, there was inadequate management and control of the cooling of heat-treated foods, only one operative hand-washing point in the whole kitchen, and insufficient cleaning or disinfection of work surfaces, crockery and cutlery. Following the outbreak, the Food Safety Authority gave specific orders and rules to the hotel regarding correct methods of cleaning to be carried out by the kitchen staff. None of the food handlers or kitchen staff reported having been sick in the days or weeks before to the outbreak.

Discussion and conclusion

This outbreak did not have serious public health consequences: the number of people affected was low (attack rate: 14%), very few people consulted their doctor, nobody was hospitalised and there was no media attention. We carried out a complete outbreak investigation in order to prevent possible future outbreaks, since the hotel where the outbreak happened is a very popular location for national and international meetings and events. As this hotel also provides accommodation for tourists from other parts of Norway and other countries, any outbreak occurring in the hotel could potentially be of international concern. Although these types of outbreaks are preventable, they still happen in places where they would not be expected, due to supposedly high quality of service, such as in this hotel.

The results of the microbiological analysis confirmed that the outbreak was caused by norovirus. Although we only had three positive stool samples, there were no indications that other pathogens were involved. Furthermore, most of the cases reported becoming ill between 24 and 72 hours after the meeting, which is in accordance with the incubation period for norovirus [2,4]. The clinical presentation of the disease also matched symptoms previously described for norovirus

Table 2

Characteristics of outbreak cases due to norovirus infection, one-day meeting, Oslo, Norway, 28 January 2011 (n=56)

Characteristic	Number of cases	Denominator	Attack rate, as percentage	Relative risk (95%CI)
Sex				
Male	25	182	13.7	–
Female	31	206	15.0	1.09 (0.67–1.78)
Age, in years				
20–39	16	107	15.0	–
40–59	29	250	11.6	0.75 (0.38–1.44)
60–74	11	32	34.4	2.98 (1.18–7.51)
Contact with sick people[a]				
During the four days before the meeting	6	37	16.2	0.98 (0.89–1.08)
During the meeting	4	34	11.8	1.02 (0.94–1.10)
All cases	**56**	**391**	**14.3**	–

[a] Persons with diarrhoea, vomiting, fever and/or abdominal pain.

infections [5-7]. Outbreaks of norovirus infection in hotels have been reported elsewhere, such as in [8,9].

The specific source of this outbreak remains unclear. We know that a common source of infection was present during the meeting since the epicurve suggests a point-source transmission pattern, with a sudden increase of cases occurring just several hours after the meeting. It is not very likely that food served outside the hotel played a role in the outbreak since only six cases reported having eaten something not served during the meeting. Other people could have been infected more than 72 hours after the meeting, due to person–to-person transmission, which is very common in outbreaks due to norovirus [9,10]. However, in the questionnaire, we only asked about symptom onset during the 72-hour period following the meeting, as our main goal was to look for a possible common exposure during the meeting.

One of the main challenges we faced in this investigation related to the type of menu that was served. Since it was a set menu, almost everybody ate the same items, so for some of the food items very few people were unexposed. Therefore no further stratification and multivariable analysis was feasible. The results of the univariate analysis did not lead to strong conclusions. Those items that were closer to statistical significance were the sandwiches served for lunch and the wraps served during breakfast. The specific attack rates of the different food items were not very high, which suggests that there was not massive contamination of one specific food item, but that potentially several different items were contaminated. People who developed symptoms might have been sitting next to each other in the same area where contaminated food was served by the same waiter. However, we were not able to check this hypothesis.

We did not find any explanation as to why people aged 60 years and older were more likely to become ill. We found no specific food item that was more frequently eaten by meeting participants from this age group. It is possible that these participants had more underlying conditions, making them more prone to infection, as has been described previously in norovirus outbreaks [11,12], but we did not collect information on this.

We consider that the outbreak was probably caused by contaminated food either from food handlers, kitchen staff, waiters or meeting participants who were shedding the virus. One person who attended the meeting reported having had diarrhoea when they arrived at the hotel and might have contributed to the spread of the virus. We also have to take into account that some of the food items, such as the wraps, were produced

Table 3

Exposure to foods[a], outbreak cases due to norovirus infection, one-day meeting, Oslo, Norway, 28 January 2011 (n=56)

Food items[a] at each meal	Food eaten			Food not eaten			Relative risk (95% CI)	P value	Percentage of cases exposed[b]
	Number of cases	Total number of participants	Attack rate, as percentage	Number of cases	Total number of participants	Attack rate, as percentage			
Breakfast									
Wraps with cheese, ham and salad	29	159	18.2	27	229	11.8	1.5 (0.9–2.5)	0.075	51.8
Lunch									
Sandwiches with cheese and salad	39	204	19.1	4	47	8.5	2.3 (0.8–6.0)	0.082	69.6
Borek with spinach and cheese	24	144	16.7	6	59	10.2	1.6 (0.7–3.8)	0.236	42.9
Pastries	11	79	13.9	9	84	10.7	1.3 (0.6–3.0)	0.532	19.6
Dinner									
Starter: pickled cod	54	349	15.5	1	10	10.0	1.5 (0.2–10.1)	0.636	96.4
Main dish: reindeer médaillons	54	348	15.5	0	10	0.0	–	–	96.4
Dessert: chocolate-flavoured liquorice	53	339	15.6	1	18	5.6	2.8 (0.4–19.2)	0.245	94.6

[a] Only food items with a relative risk greater than one are shown.

[b] Calculations were carried out using as the numerator the number of cases who answered that they were sure that they had eaten a specific food item and the total number of cases (n=56) as the denominator.

outside of the hotel, so the contamination could have happened before or during delivery to the hotel.

The irregularities that the Food Safety Authority's inspection found in the kitchen may have enabled the spread of the virus. Handling of ready-to-eat foods by infected food handlers is commonly identified as a contributing factor in outbreaks caused by norovirus [13-15]. However, the role of kitchen employees or food handlers in the outbreak reported here remains unclear since none of those in the hotel reported any symptoms to the Food Safety Authority and no information was available regarding the health status of the food handlers who produced some of the food items outside the hotel. The importance of identifying asymptomatic food handlers shedding the virus is also well described in the literature: such people can also be a contributing factor in norovirus outbreaks [16,17]. We do not know if asymptomatic food handlers were involved in the spread of the virus in this outbreak as the employees were not asked to provide stool samples.

We would like to emphasise the importance of performing a complete outbreak investigation, looking at all epidemiological, environmental and microbiological components, when norovirus outbreaks occur. All three are equally important and complementary. In this outbreak, the epidemiological investigation of the food items served was inconclusive, but the microbiological analysis revealed the identity of the pathogen and the environmental investigation revealed several irregularities in the kitchen.

Specific recommendations, orders and rules were given by the Food Safety Authority for the correct cleaning and management of the kitchen. The Food Safety Authority followed up with the hotel to ensure implementation of the recommendations and to verify that all the irregularities had been addressed within the deadline proposed. As a consequence of this outbreak investigation, the hotel is planning to change their internal, routine protocols, for example, samples of food items served at every meal in an event will be stored, in case a similar situation happens again and analysis of the food is needed.

Food handlers and other personnel who present with gastrointestinal symptoms should avoid involvement with the preparation of food while they are symptomatic in order to prevent spread of the pathogen, and they should also adhere to appropiate hygiene and hand-washing routines.

Acknowledgements

We would like to acknowledge the following colleagues for their contribution to this outbreak investigation: Katrine Borgen (Norwegian Institute of Public Health and EPIET supervisor, Oslo, Norway), Emily MacDonald (Norwegian Institute of Public Health, Oslo, Norway) and Alicia Barrasa (EPIET coordinator, Instituto Carlos III Madrid, Spain).

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Potential role of asymptomatic kitchen food handlers during a food-borne outbreak of norovirus infection, Dublin, Ireland, March 2009

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Citation style for this article:
Nicolay N, McDermott R, Kelly M, Gorby M, Prendergast T, Tuite G, Coughlan S, McKeown P, Sayers G. Potential role of asymptomatic kitchen food handlers during a food-borne outbreak of norovirus infection, Dublin, Ireland, March 2009.
Euro Surveill. 2011;16(30):pii=19931. Available online: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=19931

Article published on 28 July 2011

In March 2009, the Department of Public Health in Dublin, Ireland, was notified of a cluster of four gastroenteritis cases among people who attended a family lunch in a Dublin hotel. A retrospective cohort study was carried out. An outbreak case was defined as an attendee who developed diarrhoea and/or vomiting in the 60 hours following the lunch. Of 57 respondents, 27 met the case definition. Consumption of egg mayonnaise, turkey with stuffing or chicken sandwiches were each associated with increased risk of gastroenteritis: (risk ratio (RR): 2.3; 95% CI: 1.4–3.9), (RR: 1.9; 95% CI: 1.2–3.2), (RR: 1.9; 95% CI: 1.1–3.1), respectively. An environmental investigation established that before notification of the cluster, there had been unreported gastroenteritis among staff at the hotel. The earliest symptomatic person identified was a staff member who had vomited in the staff toilets but had not reported it. The sandwiches had most likely been contaminated by three asymptomatic kitchen food handlers who had used the same toilets. Stool samples were submitted by eight cases and 10 staff members. All eight cases and three asymptomatic food handlers on duty at the lunch tested positive for norovirus genogroup II.4 2006. Our analysis suggests that asymptomatic food-handlers can be responsible for norovirus transmission.

Introduction

Norovirus causes self-limiting gastroenteritis that is usually characterised by sudden and abrupt vomiting followed by more prolonged diarrhoea [1]. A low infectious dose, a capacity to survive long periods in the environment, thermal stability and resistance to many common disinfectants contribute to the considerable outbreak potential of this virus [2]. Transmission primarily occurs through environmental contamination following direct soiling and indirect aerosolisation resulting from projectile vomiting. It can be introduced into a particular setting by contaminated drinking water or food [3]. Subsequent person-to-person transmission will lead to onward propagation in the original setting or in other linked settings, often making the original contamination event difficult to identify [4]. It is important to investigate outbreaks due to norovirus in order to ascertain the source of the infection and mode of transmission. However, finding the initial event that allows the linkage of cases is often problematic, making the epidemiological investigation challenging [5]. Immunity against norovirus occurs post infection but may be short lived. This, plus the existence of several viral antigenic types, means later re-infection is possible [2].

In Ireland, individual cases as well as outbreaks of norovirus infection have been legally notifiable since 2004 (Infectious Diseases (Amendment) (No. 3) Regulations 2003). Norovirus caused 115 outbreaks – 48% of outbreaks of infectious intestinal disease in Ireland in 2009 [6]. In March 2009, the Department of Public Health in Dublin, was informed by local environmental health officers of four gastroenteritis cases. Environmental health officers initially interviewed the informant, a family member who had organised the event. The person indicated that all cases had attended an extended-family lunch three days earlier: 100 family and friends attended the lunch in a hotel in Dublin. The menu consisted of soup, a variety of handmade sandwiches (egg mayonnaise, tuna mayonnaise, ham, salad, turkey with stuffing, chicken and cheese), followed by tea or coffee. Guests were free to help themselves from communal platters. The food was prepared by five kitchen food handlers. There was no history of illness among the guests prior to the family function. The guests attended a church service before the lunch. Apart from family groupings, there had been no gathering of guests before that day. Given the clustering of people with symptoms, the hotel was suspected as the location of exposure and food served at the hotel the

vehicle of contamination. A multidisciplinary outbreak control team was established: this report describes the epidemiological investigation that was carried out to determine the extent of the outbreak, to identify the aetiological agent and mode of transmission and to take appropriate control measures.

Methods

Study design

A retrospective cohort study was carried out among attendees at the lunch. As no list of the guests was available, active participant finding was facilitated by the organiser of the lunch. Each respondent was asked to provide contact details for all other attendees known to them. Every person identified was contacted and interviewed.

Using a standardised structured questionnaire, food and beverage consumption was assessed, as well as exposure to any person with diarrhoea (defined as three or more loose stools per day) or vomiting in the week before the lunch. Environmental health officers administered the questionnaire by telephone, from day 3 to day 6 after the lunch. Information on socio-demographic characteristics of the respondents and the spectrum of symptoms was also recorded.

Case definition

An outbreak case was defined as a lunch attendee who developed diarrhoea (defined as three or more loose stools per day) or vomiting or both during the 60 hours following the lunch.

Environmental investigation

Local environmental health officers inspected the hotel on the day they were notified of the cluster of illness (three days after the lunch). Staff on duty for the lunch and any staff who reported any gastrointestinal symptoms one week before and/or after the lunch were interviewed.

An environmental assessment was undertaken at the hotel, as well as a review of work practices including the Hazard Analysis and Critical Control Points (HACCP) system [7]. The inspection included sampling of food items (e.g. sliced turkey and ham used for sandwiches on the day of the event), mains water and ice. The food items were tested for indicator bacteria (aerobic colony count), *Salmonella* spp. and *Escherichia coli* and toxins of *Staphylococcus aureus*, *Bacillus cereus* and *Clostridium perfringens*. Water samples were taken from the bar and the kitchen at the hotel and tested for indicator bacteria (coliforms, *E. coli* and *Enterococcus* spp.). Ice samples were also tested for coliforms, *E. coli* and *Enterococcus* spp.

Clinical microbiological investigation

Following notification of the outbreak, stool samples were collected from eight cases, from the five food handlers responsible for the sandwich preparation, and any staff members who reported having had gastrointestinal illness one week before and up to one week after the lunch. Faecal specimens were tested for *Salmonella* spp., *Shigella*, *Campylobacter* and enterohaemorrhagic *E. coli*. and for the toxins of *C. perfringens*, *S. aureus* and *B. cereus*. They were also examined for norovirus by reverse transcription-polymerase chain reaction (RT-PCR) [8,9]. The DNA sequences were analysed using SeqMan and CLUSTAL W. Genotype information was obtained by comparing the sequences against those available in GenBank using BLAST. Rotavirus was not tested for as this outbreak mainly involved adults.

Statistical analysis

A data matrix was constructed in EpiData Entry version 2.0 software (EpiData Association, Denmark) and analysis undertaken using Stata version 9.0 (StataCorp LP, United States). Age according to illness status was compared using Student's t-test Specific attack rates (ARs) and crude risk ratios (RRs) with 95% confidence intervals (CIs) were calculated according to the sex of those affected and for each specific food and beverage exposure. Research of effect modifier was performed using stratified analysis by variable of exposure.

Results

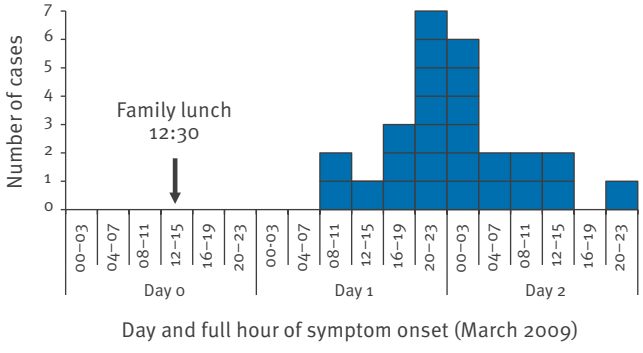
Epidemiological investigation

The cohort study recruited 57 attendees who completed the questionnaire, out of a total of 100 attendees (response rate: 57%). Of these, 27 met the case definition (AR: 47%). One attendee presented with symptoms more than 72 hours after the lunch and was considered as a secondary case and was excluded from the cohort study.

The median age of cases was 51 years (range: 13–87) and the median age of attendees who were not cases (n=30) was 47 years (range: 11–78). Among the cases, 11 were male and among those who were not cases, 14

Figure

Outbreak cases due to norovirus infection, family lunch, Dublin, Ireland, March 2009 (n=26)[a]



[a] One case was unable to recall the exact time of symptom onset.

were male. The differences in median age and proportion of cases who were male in the two groups were not statistically significant.

The time of symptom onset ranged from less than 24 hours to 60 hours after the lunch. The epidemic curve showed a peak in the number of cases between 28 hours and 44 hours after the lunch (Figure).

All cases reported diarrhoea; 16 cases reported vomiting. Additionally, 18 cases reported nausea, 15 abdominal pain, 11 chills, 10 headache, nine reported fever and seven reported muscle pain. The median duration of acute symptoms was 44 hours (range: 3–72). Two cases consulted their general practitioner following the occurrence of symptoms; none were hospitalised.

In a univariate analysis (Table), cases who reported eating egg mayonnaise sandwiches (AR: 78% exposed vs 33% not exposed) were associated with the highest risk of gastroenteritis (RR: 2.3, 95% CI: 1.4–3.9). Eating turkey stuffing sandwiches (73% vs 38%) or chicken sandwiches (67% vs 36%) was associated with an increased risk of gastroenteritis (RR: 1.9; 95% CI: 1.2–3.2), (RR: 1.9; 95% CI: 1.1–3.1), respectively. Eating cheese sandwiches (65% vs 35%) was marginally associated with an increased risk of gastroenteritis (RR: 1.8; 95% CI: 1.0–3.2). Consumption of salad, tuna mayonnaise or ham sandwiches were not associated with illness. No significant association was found between the consumption of beverages and gastroenteritis.

We then carried out an analysis stratified by consumption of each sandwich type. Eating of chicken was significantly associated with an increased risk of gastroenteritis in attendees who had not eaten turkey with stuffing (RR: 3.3; 95% CI: 1.5–7.3; p=0.002), but was not in attendees who had eaten turkey with stuffing (RR: 0.6; 95% CI: 0.3–1.3; p=0.18). No other association was found.

Environmental investigation

A HACCP Food Safety Management System was in place in the hotel. Food appeared to be prepared in a safe and hygienic manner and the staff members were highly trained. No skin lesions were noted on food handlers during the inspection. Overall, the environmental health officers were of the opinion that the food premises were well managed.

All the food used in the sandwiches was freshly prepared at the hotel. The meats were cooked and cooled on the day before the lunch and were sliced on the function day. The preparation and cutting of the sandwiches took place just before and during the function. A variety of sandwiches were made on an ongoing basis, rather than in batches of one type and then another.

Five food handlers (Food handlers 1 to 5) had been allocated to prepare the sandwiches: all the food handlers prepared various types of sandwiches. Hotel staff mentioned that the attendees arrived earlier and in greater numbers than expected and that the five food handlers were under time pressure when preparing the

Table

Univariate analysis of risks associated with food and beverage consumption, family lunch, Dublin, Ireland, March 2009 (n=57)

Item tested	Exposed			Not exposed			Risk ratio	95% CI
	Total number of attendees exposed	Number of cases	Attack rate (%)	Total number of attendees not exposed	Number of cases	Attack rate (%)		
Food								
Sandwiches								
Egg mayonnaise	18	14	78	39	13	33	2.3	1.4–3.9
Turkey with stuffing	15	11	73	42	16	38	1.9	1.2–3.2
Chicken	21	14	67	36	13	36	1.9	1.1–3.1
Cheese	23	15	65	34	12	35	1.8	1.0–3.2
Salad	14	5	36	43	22	51	0.7	0.3–1.7
Tuna mayonnaise	18	10	56	39	17	44	1.3	0.7–2.2
Ham	8	4	50	49	23	47	1.1	0.5–2.3
Soup	50	25	50	7	2	29	1.8	0.5–5.8
Beverage								
Mains water	4	3	75	53	24	45	1.7	0.9–3.1
Ice	9	5	56	48	22	46	1.2	0.6–2.3
Coffee	15	6	40	42	21	50	0.8	0.4–1.6
Tea	30	15	50	27	12	44	1.1	0.7–2.0

sandwiches. The family lunch was the only function held in the hotel at lunchtime that day; another function was held that evening. Hotel staff contacted representatives of this party and no illness was reported in association with this event. No further cases of norovirus infection associated with the hotel were reported to the public health authorities around that time.

In-depth face-to-face interviews were conducted with the five food handlers during the inspection of the premises on day 3 after the lunch. They reported no gastrointestinal symptoms and had no known contact with a symptomatic person. They were employed full time on the premises and had worked full time the week before the lunch.

The on-site investigation by environmental health officers including face-to-face interviews with hotel management and staff (on day 3 after the lunch) indicated that there had been some symptoms of gastroenteritis among other staff who were off duty the day of the lunch.

One food handler (Food handler 6) had vomited in the staff toilets seven days before the lunch but had not reported this to the management at that time. This person was then off duty for 48 hours and had no involvement in the lunch. Another food handler (Food handler 7) developed gastrointestinal symptoms at home while off duty three days before the lunch and did not return to work before the lunch. Investigations by environmental health officers revealed that gastrointestinal symptoms had also occurred in some hotel staff not involved in food preparation: two bar workers had been symptomatic two days before the lunch while off duty and neither worked on the day of the lunch. A third bar worker became symptomatic one week after the lunch. This worker had not been present at the lunch and reported contact with a person with gastroenteritis in their own household before becoming symptomatic.

Laboratory investigation

Bacteriological analysis of all food, water and ice samples and of all clinical samples was negative.

Eight lunch attendees and the 10 relevant staff (Food handlers 1 to 7 and Bar workers 1 to 3) submitted stool samples (five to six days after the lunch). The samples from all eight attendees tested and five of the 10 staff were positive for norovirus RNA genogroup II.4 2006. The staff members who tested positive were three of the five asymptomatic food handlers (Food handlers 1 to 3) who were on duty at the lunch and two of the staff who had been symptomatic at home: a bar worker (Bar worker 2) and a food handler (Food handler 7). The staff member who had vomited on the premises seven days before the lunch tested negative (Food handler 6).

Control measures

Food-safety advice on preparation techniques, temperature control, risk analysis based on the HACCP system, and cleaning and personal hygiene, including hand washing, was given to all available staff members during the initial inspection on day 3 following the lunch. Staff and management were also given written advice on enteric precautions. The management were specifically advised to only use bought-in pre-prepared turkey, ham, chicken and egg mayonnaise, to minimise handling and shorten the preparation chain while the investigation was ongoing. Further advice on cleaning and decontamination of the hotel was also given.

As the hotel had good hygiene and cleaning management systems already in place, the environmental health officers initially considered that intensive decontamination of the premises by a specialist contractor was unnecessary. However, when the diagnosis of norovirus infection was confirmed, the hotel, including the gym and all bedrooms, was disinfected by a specialist contractor in accordance with national guidance [10].

Discussion

The epidemic curve of this outbreak suggested a single, common, point source and the cohort study identified several types of sandwiches served during the family lunch as possible vehicles of contamination, with egg mayonnaise, turkey with stuffing and chicken sandwiches being the most likely vehicles of the outbreak. Sandwiches (as a ready-to-eat food) are recognised to be potential vehicles for norovirus outbreaks [3,11,12]. Since a tiny inoculum (as few as 10 virus particles) is sufficient to cause infection [13], norovirus outbreaks can easily occur. The results of our study are consistent with previous reports where multi-ingredient foods were implicated in norovirus outbreaks [14,15]. The viral strain isolated in attendees and staff members was the commonly circulating strain in Ireland [16].

In the outbreak investigated here, the food handler who vomited in the staff toilets one week before the lunch was the earliest symptomatic person identified. The vomiting episode was not reported at the time and appropriate cleaning did not take place. Other staff members could have been infected via direct contact with fomites, such as the contaminated surfaces in the toilets, as has occurred in other outbreaks [17-20] Therefore, it is extremely important that staff report any vomiting episode and that it be managed as infectious [10]. A response team should immediately decontaminate and clean the area after the vomiting episode has occurred. Members of this team must not be food handlers. Hot water (≥60 °C) plus disinfectant such as 0.1% bleach solution should be used for the cleaning. Rapid implementation of such enhanced hygiene measures is the only way to prevent transmission via the environment [21].

In this outbreak, direct person-to-person transmission by the initial symptomatic person was possible but was probably limited, as this person left work soon after the vomiting episode. Transmission of the virus from

contaminated kitchen surfaces was unlikely, given the cleaning schedule in place at the hotel (daily cleaning of all areas of the hotel, particularly the kitchen), which reduced the likelihood of the kitchen being a continuing source of norovirus contamination. Widespread contamination in the kitchen would have led to more cases among people who had eaten at the hotel. It is worth noting that no further cases of norovirus infection associated with the hotel were reported to the public health authorities around that time. The same facilities were used the same evening for another family party and there were no reports of gastrointestinal disease. It has been established that swabbing environmental surfaces is helpful in formally excluding their role in norovirus transmission [22]. However, the resources are not currently available in Ireland to carry out such investigations.

From our investigation, it would appear that asymptomatic food handler(s) contaminated sandwiches during their preparation. A Japanese study demonstrated that food handlers could be infected with norovirus without displaying any symptoms and could shed a similar number of virus particles as those who were symptomatic, which could potentially lead to widespread dissemination of the virus [23]. An outbreak investigation detected the presence of norovirus RNA on the hands of food handlers, which demonstrated the feasibility of norovirus transmission by virus-shedding food handlers [22]. Our investigation found three asymptomatic food handlers who tested positive for norovirus and who probably were the source of this outbreak at the lunch. The workers appeared to be highly trained, but the time pressure resulting from the early arrival and increased number of attendees is likely to have contributed to a lapse in personal hygiene before and during the ongoing preparation of sandwiches during the lunch, resulting in the contamination of the food. The handling of multiple foods by three asymptomatic carriers is reflected in the findings that several sandwich types were significantly associated with illness.

The response rate of 57% in our cohort study was less than optimal: there was no guest list for this function and, despite exhaustive efforts, it was not possible to interview all attendees. While we cannot rule out the introduction of bias due to this, we consider that a larger sample size would not alter the overall findings of this investigation that asymptomatic food handlers can cause a substantial norovirus outbreak.

It is impossible to obtain retrospectively objective confirmation of the absence of symptoms; however, the three asymptomatic food handlers always maintained that they had had no symptoms. They were interviewed by experienced professionals and appeared to be unembarrassed, very straightforward and truthful during their interview and provided stool samples without reservation. They were well informed about the risk of infection associated with gastroenteritis. The hotel was well run and the management and the staff were very compliant and cooperative throughout the course of investigation.

It is common practice in the leisure industry that staff members are not paid during periods of sick leave, which could act as a barrier to reporting; however, in Ireland, sick staff can claim a state allowance that covers their unpaid days.

In conclusion, our study indicates that asymptomatic food handlers who shed norovirus can be responsible for food-borne outbreaks particularly when preparing ready-to-eat foods during busy work periods in circumstances that potentially impede good personal hygiene. Such outbreaks involving food handlers should be prioritised for investigation by public health authorities in order to better estimate the burden of the illness due to asymptomatic carriers. This investigation highlights that any single vomiting episode should be immediately reported to the management in order to prevent spread of gastrointestinal illness.

Acknowledgements

We would like to thank Patricia Garvey for her critical scientific feedback, Dr Brian O'Herlihy, former Director of Public Health, and Dr Maire O'Connor, Specialist in Public Health Medicine, Health Service Executive, for their advice and support in the investigation of this outbreak. We would like to thank the environmental health officers from the Communicable Disease Unit, Health Service Executive Dublin North East. We also wish to thank Dr Eleanor McNamara and the Public Health Laboratory, Cherry Orchard Dublin, for advice and technical support. We would like to thank Barbara Hennessy and Bernie Clarke for administrative support.

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Coxsackieviral infections involved in aseptic meningitis: a study in Slovakia from 2005 to 2009

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Citation style for this article:
Sojka M, Wsólová L, Petrovičová A. Coxsackieviral infections involved in aseptic meningitis: a study in Slovakia from 2005 to 2009. Euro Surveill. 2011;16(30):pii=19927. Available online: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=19927

Article published on 28 July 2011

A wide range of diseases is associated with enteroviruses. They are reported to be responsible for viral meningitis, especially in children, but also in adults. This study analysed infection with eight selected coxsackievirus serotypes as the cause of aseptic meningitis in 480 patients in Slovakia from 2005 to 2009, using a quantitative assay for the detection of intrathecal antibodies. Intrathecal production of antibodies against selected coxsackieviruses was proved in 21% of these patients. A significant decrease from 35% in 2005 to 8,5% in 2009 (p=0.004) in the proportion of patients with proven intrathecal production of virus-specific antibodies was observed during the study period. We conclude that coxsackievirus B4 was the endemic serotype in Slovakia and was responsible for most cases of coxsackieviral meningitis in the study period.

Introduction

Human enteroviruses (HEV) are ubiquitous faecal-orally transmitted, small RNA viruses, belonging to the family *Picornaviridae*. HEV are classified into four species: human enterovirus A-D. These viruses cause a wide variety of diseases, and it is known that non-polio enteroviruses are the most common cause of aseptic meningitis in adults as well as children [1-3]. Enteroviruses are responsible for approximately 26% of adult aseptic meningitis cases with identified causative agent [4]. Individual enteroviral serotypes are not clearly associated with particular disease syndromes, but have a propensity to cause particular symptoms [5]. Echovirus serotypes are frequently reported to be responsible for meningitis [5-7]. Among coxsackieviruses, the leading serotypes associated with central nervous system (CNS) diseases are B1 to B6, A7 and A9 [6-10].

Many reports concerning enteroviral meningitis are based on diagnosis of enterovirus infection by the conventional method, i.e. virus isolation from cerebrospinal fluid (CSF), stool or throat wash (swab) in cell culture, followed by identification of virus by virus neutralisation assay using type-specific antisera [6,7]. This method is highly specific, but laborious and time-consuming. Another problem with this procedure is that although positive virus isolation from stool and throat confirms virus infection of the patient, it does not necessarily prove that it is the causative agent of current disease [11], considering the existing high proportion of inapparent enteroviral infections [10]. Evidence of the intrathecal production of virus-specific antibodies has been used to diagnose poliovirus [11,12], echovirus 6, coxsackievirus B6 and A9 [13,14] and other viral infections [15,16], using different approaches. A positive result demonstrates a clear link between the infection and CNS disease [11]. To our knowledge, no epidemiological study has been published to date applying this method in laboratory diagnostics of coxsackieviral meningitis in eastern and central Europe.

In 2004, the live Sabin oral polio vaccine, which had been in use since 1960 in the poliomyelitis vaccination programme, was replaced in Slovakia by the Salk inactivated polio vaccine. It is well known that two or more enteroviruses can propagate simultaneously in the alimentary tract, but multiplication of one of them may interfere with growth of the second. Thus, an active enterovirus infection may block live vaccine poliovirus replication in the gut [17,18]. On the other hand, vaccine poliovirus strain replicating in the gut may block the propagation of other enteroviruses. Shedding of poliovirus typically occurs one to four weeks after vaccination and may last months or years in immunocompromised individuals. The shift from live vaccine to inactivated vaccine means opening the living space in the gut for the other, competing, enteroviruses, and therefore may influence the spectrum of enteroviruses circulating in population. This study was designed to identify the role of selected coxsackievirus serotypes as the causative agents of aseptic meningitis by demonstrating intrathecal production of virus-specific neutralising antibodies. Furthermore, it aimed at defining the prevalence of these serotypes after the change in the polio vaccine in Slovakia.

Materials and methods

Pairs of CSF and serum samples from patients with viral meningitis (ICD-10 code A87) of suspected enteroviral aetiology, hospitalised in neurological and

infectious disease departments of hospitals in all parts of Slovakia, that were sent to the National Reference Centre for the Identification of Enteric Viruses in Bratislava for analysis between January 2005 and December 2009, were included in this study.

Antibody titre determination

Titres of virus-specific neutralising antibodies in CSF and heat-inactivated sera were determined by a standard virus neutralisation test as described previously [11]. Standard coxsackievirus strains used to determine the virus-neutralising antibodies were coxsackievirus B1 (strain Conn), B2 (Ohio), B3 (Nancy), B4 (JVB), B5 (Faulkner), B6 (Schmitt), A7 (Parker), and A9 (Griggs). The standard viruses, obtained from the Institute of Sera and Vaccines (Prague, Czech Republic), were propagated in Vero cells and the virus serotypes were periodically verified using standard LBM Pools (Lim Benyesh-Melnick antiserum pools from Statens Serum Institut, Copenhagen, Denmark). The lowest serum and CSF dilutions that were analysed for the presence of virus-neutralising antibodies were 1:8 for serum and 1:2 for CSF. Titres of virus-neutralising antibodies were expressed as the reciprocals of the highest dilution of serum or CSF that showed neutralisation of virus. Titres of virus-specific antibodies were used to calculate the serum/CSF antibody titre ratio for each individual. Based on our previous experience with laboratory findings and the clinical course of the disease, the CSF and serum sample pair was considered to be positive for the intrathecal antibody production if a ratio of the serum and CSF titres was less than 100. This shows a relative increase in the level of virus-specific antibodies in the CSF compared with the serum and indicates local (intrathecal) antibody production linked to acute infection with a given virus type. Currently, there is no agreed standard cut-off value for serum/CSF titre ratio indicating intrathecal production of specific antibodies; various authors have used different values (32–400) depending on the viruses and methods used [11,14,19,20]. The presence of virus-neutralising antibodies against a given serotype in the serum (without proven intrathecal antibody production) was considered only as a sign of previous infection with this virus type.

Statistical analysis

The trend in the proportion of patients with proven intrathecal antibody production during the five years of the study was determined by means of linear regression. Statistical software SPSS 16.0 was used.

Results

Samples from 480 patients (214 men and 266 women) were analysed during the study, ranging between 43 and 147 patients per year. The median age was 36 years, ranging from 1 to 69 years (Figure 1).

During the study period, intrathecal production of virus-specific neutralising antibodies against the eight selected coxsackieviruses was shown in 100 (21%) patients, ranging between nine and 30 per year. A statistically significant decrease (p=0.004) in the proportion of positive cases was observed over time, from 35% in 2005 to 8.5% in 2009 (Figure 2). The highest titre of antibodies against coxsackieviruses measured in a serum sample was 2,048.

In the 100 patients with proven intrathecal antibody production against the studies viruses, the aetiological agents were identified as follows: 46 coxsackievirus serotype B4, 19 serotype B3, 16 serotype B5, eight serotype A9, five serotype A7, three serotype B1 and three serotype B2. Coxsackievirus B6 was not identified as a causative agent of aseptic meningitis in this study. Coxsackievirus B4 was identified as the dominant serotype in all years of the study, except in 2005, where B3 was the dominant serotype (Figure 3). Coxsackievirus B4 was the dominant serotype in all age groups, except for the group of 10-14 year-olds, in whom coxsackievirus B5 was the dominant serotype, and the 40-49 year-old patients, in whom coxsackievirus B3 was the dominant serotype (Figure 4). The

Figure 1

Age structure of patients with aseptic meningitis, Slovakia, 2005–2009 (n=480)

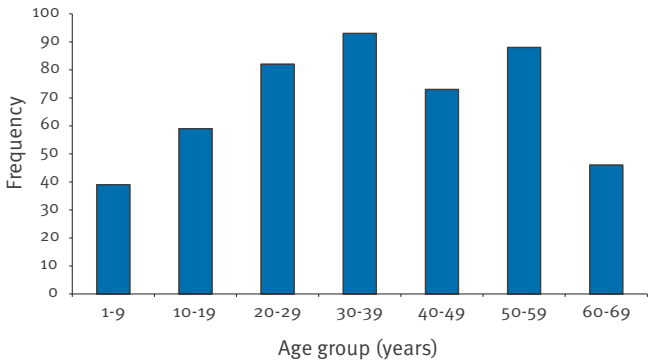
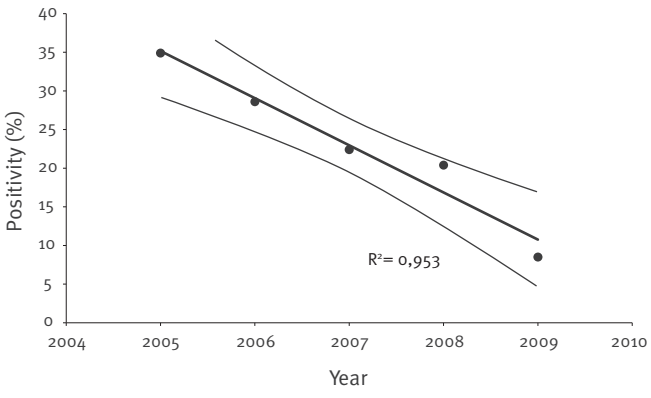


Figure 2

Proportion of aseptic meningitis patients with proven intrathecal antibody production against coxsackieviruses, Slovakia, 2005–2009 (n=480)



Linear trend (thick line) with 95% confidence intervals (thin lines).

proportion of 40-49 year-old patients in 2005 and 2006 was 21% and similar in 2007 (17%), when B4 serotype was dominant.

We compared these results with the general prevalence of these eight coxsackievirus serotypes in the population by looking at the prevalence of virus-specific neutralising antibodies in serum samples as a marker of previous infection (Table). Antibodies against coxsackievirus B4 were the most prevalent in all years of the study, ranging between 73% and 81%. Antibodies against other coxsackieviruses were less prevalent.

Discussion and conclusion

This paper relates the occurrence of infection with eight selected coxsackievirus serotypes to that of aseptic meningnitis in Slovakia. Unlike in other studies [5,6], a large proportion (78%) of our patients were older than 20 years. This may partially be explained by the fact that the majority of samples included in the study were from hospital departments for adults.

Almost all published reports on enteroviruses as the cause of diseases in humans are based on virus isolation from different biological materials such as stool, throat swabs or CSF [5,21]. Our study was based on the evidence of intrathecal production of virus-neutralising antibodies as a consequence of central nervous system infection with a given virus. This approach enables identification of the virus as the causative agent of an ongoing CNS disease without the necessity of virus isolation and identification, which may be difficult. On the other hand, the method allows detection of only a limited number of virus serotypes that are set up in the routine virus neutralisation assay.

Certain enteroviral serotypes can be endemic in a particular geographical area with little or only gradual changes over time [21]. Coxsackieviruses were established as the aetiological agent of CNS inflammatory disease in 21% of patients followed in our study. Coxsackieviruses are rare among enterovirus serotypes causing meningitis. Comparing the serotypes followed up in this study with other reports, different coxsackieviruses are dominant in different regions and times: Serotype B5 was relatively more abundant in Hungary (2000–2008) [7] and Spain (1988–1997) [21], B5 and B3 in Belgium (1980–1994) [8], B5 and B4 in the United States (2002–2005) [22], B3 in Tunisia (1992–2003) [9] and B2 in Cyprus (2000–2002) [6]. Coxsackie B4 virus appeared to be the endemic serotype in Slovakia, responsible for most cases of coxsackieviral meningitis during the period of our study. This finding is supported by the fact that in all years of the study, the most prevalent antibodies in sera of meningitis patients were those against coxsackievirus B4. However, it should be emphasised that we studied only selected eight of the coxsackievirus serotypes that may be responsible for the inflammatory CNS disease in humans, and our results are therefore not fully comparable with other studies aimed to all HEV serotypes. Other enteroviral serotypes were not investigated here because of the limitations of the virus neutralisation test.

Figure 3

Coxsackievirus serotypes causing aseptic meningitis, by year, Slovakia, 2005–2009 (n=100)

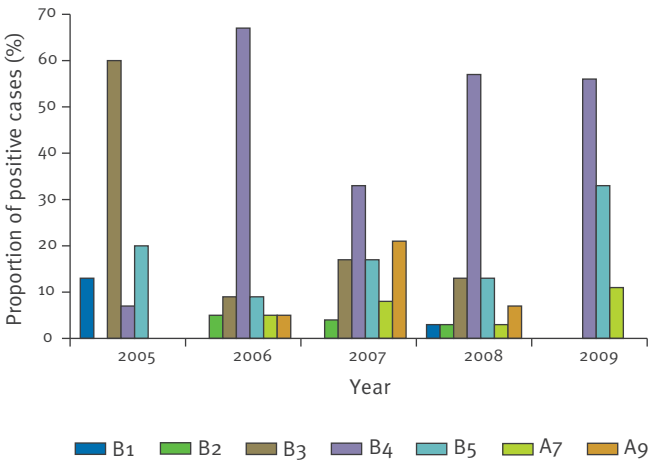
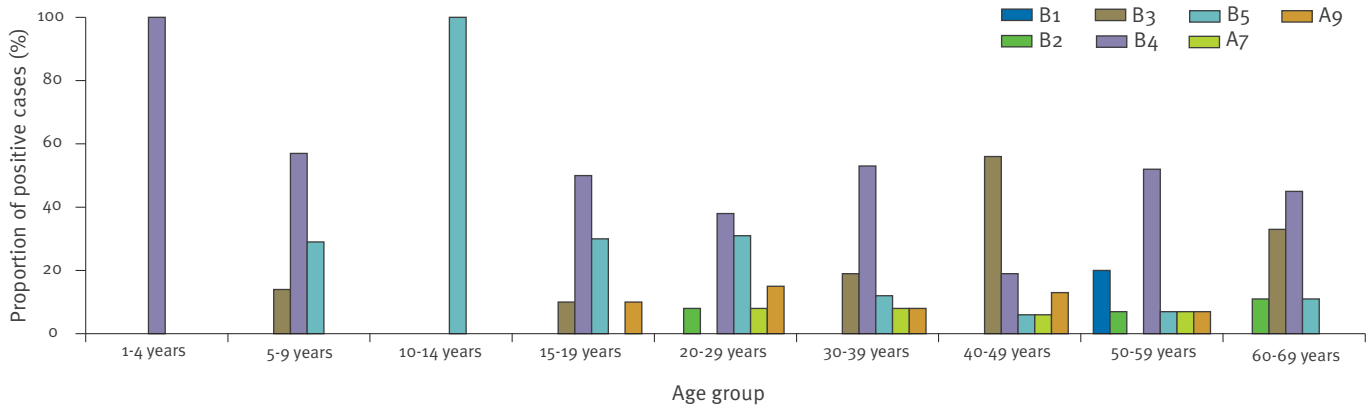


Figure 4

Coxsackievirus serotypes causing of inflammatory central nervous system disease, by age group, Slovakia, 2005–2009 (n=100)



We observed a significant decrease during the study in the proportion of patients with coxsackieviral meningitis as evidenced by intrathecal production of virus-specific antibodies. We may speculate that this decline could be related to changes in the circulation of coxsackieviruses and other enteroviral serotypes in the Slovak population that are related to the modification of the polio vaccination programme when the Sabin live oral polio vaccine was replaced by the Salk inactivated vaccine in 2004 and other enteroviruses may have colonised the free living space, possibly interfering with the coxsackievirus strains we have here identified as declining. It should be taken in account that age, the characteristics of the biological agent and epidemiological conditions modulate the patterns of intrathecal immunoglobulin synthesis [14] and that possible alterations in the virulence of circulating enteroviruses may also lead to a change in the virus-specific intrathecal immune response pattern. In our previous seroprevalence study [23], we observed a consecutive decrease in proportion of patients that had antibodies against studied coxsackieviruses in the years 1985 to 2004. In 2005 this trend stopped and a mild increase in seropositivity to all studied serotypes was observed.

In conclusion this study demonstrates that coxsackieviruses were a significant cause of viral meningitis in Slovakia. These viruses circulate in the population and the prevalence of antibodies against the studied serotypes did not change during the study period. The identified trend in the proportion of diseases caused by the studied viruses indicates that in the future, studies on the other non-polio enteroviruses, e.g. echoviruses, may become of greater importance in the diagnosis of CNS diseases.

Acknowledgements

This study was supported by a grant from the Ministry of Health of the Slovakia (MZSR 2006/12-SZU-07). We thank Shubhada Bopegamage for reading the article and giving valuable suggestions, and Iveta Jandová and Alica Stračinová for their technical assistance.

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Table

Prevalence of antibodies against coxsackieviruses B1 to B6, A7 and A9 in sera of aseptic meningitis patients, Slovakia, 2005–2009 (n=480)

Year	Number of patients	Number of patients with serum antibodies n (%)							
		B1	B2	B3	B4	B5	B6	A7	A9
2005	43	15 (35)	22 (51)	33 (77)	35 (81)	22 (51)	2 (5)	30 (70)	24 (56)
2006	77	24 (31)	43 (56)	44 (57)	59 (77)	36 (47)	3 (4)	51 (66)	46 (60)
2007	107	35 (33)	56 (52)	73 (68)	87 (81)	49 (46)	13 (12)	68 (64)	72 (67)
2008	147	55 (37)	87 (59)	94 (64)	113 (77)	83 (56)	13 (9)	94 (64)	90 (61)
2009	106	36 (34)	67 (63)	59 (56)	77 (73)	59 (56)	10 (9)	55 (52)	51 (48)
Total	480	165 (34)	275 (57)	303 (63)	371 (77)	249 (52)	41 (9)	298 (62)	283 (59)

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World Hepatitis Day 2011

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Citation style for this article:
Eurosurveillance editorial team. World Hepatitis Day 2011.
Euro Surveill. 2011;16(30):pii=19926. Available online: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=19926

Article published on 28 July 2011

The World Heath Organization (WHO) is today calling for attention to the global health problem posed by viral hepatitis. The first official WHO-supported World Hepatitis Day on 28 July 2011, coordinated by the World Hepatitis Alliance, comes under the slogan: *Hepatitis affects everyone, everywhere. Know it. Confront it.*

It is emphasised that although some 350 million people worldwide live with chronic hepatitis B and 170 million people with chronic hepatitis C, awareness of the disease is low. In Europe, there are an estimated 14 million chronic hepatitis B cases, and about 9 million people infected with hepatitis C. The WHO Regional Office for Europe sees the greatest challenge in improving the currently weak surveillance of hepatitis.

More information can be found on the following websites:

- World Hepatitis Day website (www.worldhepatitisday.info)
- World Hepatitis Alliance (www.worldhepatitisalliance.org/WorldHepatitisDay.aspx)
- World Health Organization (www.who.int)
- World Health Organization Regional Office for Europe (www.euro.who.int/en/home)