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Change of guard in *Eurosurveillance*

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In March 2007, *Eurosurveillance* moved to the European Centre for Disease Prevention and Control (ECDC) from its two previous hosts, the Institut de Veille Sanitaire in Paris, France (*Eurosurveillance Monthly*) and the Health Protection Agency in London (*Eurosurveillance Weekly*) [1]. This marked an important date for the then young ECDC, as this was the first major European Union funded public health project moving to the Centre [2] and I was proud to become the journal's Editor-in-chief.

From the start, my aim was to build on the successful work of my predecessors in Paris and London and to continue to provide and further develop a platform for the exchange of scientific information for all those engaged in the surveillance, prevention and control of communicable diseases [1]. Moreover, I was convinced that the opportunity to have the editorial team for both publications physically in the same place for the first time would open new possibilities such as merging the two formats into one. It would allow *Eurosurveillance* to be strengthened and established as a prime European source of scientific information in its field, in other words to become Europe's journal on infectious disease surveillance, prevention and control.

In fact, relying on a strong network of dedicated experts across Europe and a committed team of editors and with the editorial independence guaranteed by two consecutive ECDC Directors and the ECDC Management Board, *Eurosurveillance* is now firmly established. In 2009, the journal applied for and was accepted to receive an impact factor [3] and during the 2009 influenza A(H1N1) pandemic much attention was paid worldwide to the timely publication of peer-reviewed papers in our journal [4,5]. Timeliness has been and will remain a strength and key distinctive feature of the journal. The rapid sharing of information has on several occasions contributed to linking and detecting similar outbreaks and to controlling them [6-9]. Moreover, a recent reader survey has demonstrated a high level of satisfaction and much support for our journal.

After four exciting years as Editor-in-chief of *Eurosurveillance*, I take over new responsibilities at ECDC where I will build up the new *Public Health Capacity and Communication Unit* [10]. Therefore I am now handing over the full leadership of the journal to Dr Ines Steffens. Ines has since the transfer of the journal to ECDC in 2007 been Managing Editor of *Eurosurveillance*, leading the day-to-day work of the Editorial Office. In this position she has been instrumental in the successful development of the journal, not least broadening the group of authors and readers to a truly global one.

Ines has a firm public health and communicable disease knowledge and passion for quality. Together with an enthusiastic editorial team, her vision is that of a strong journal, which provides knowledge and evidence for decisions that help to prevent and control infectious diseases thus contributes to the many efforts in improving health overall. I am therefore convinced that *Eurosurveillance* will continue to thrive, and although with a sad eye the heart is light when now stepping down from the lead of the journal. I will continue to follow the development of the journal and support it as Associate Editor in the future.

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First detection of *Echinococcus multilocularis* in Sweden, February to March 2011

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Surveillance for the fox tapeworm, *Echinococcus multilocularis*, has been carried out in Sweden since 2000, with about 300 red foxes analysed annually. We report the first finding of *E. multilocularis* in Sweden, in a fox shot in December 2010 in the south-west of the country. A second infected fox shot in the same location was detected in March 2011. This paper describes the national monitoring programme and the ongoing work to estimate the prevalence and spread of the infection.

Detection of *Echinococcus multilocularis* in red foxes in Sweden

In February 2011, *E. multilocularis* was detected for the first time in the south-west of Sweden, in a red fox (*Vulpes vulpes*) shot in December 2010. A second infected fox, shot in the same location, was detected in March 2011.

Background

E. multilocularis is endemic in large parts of Europe and has been increasingly reported in animals from countries near Sweden, such as Latvia, Estonia and Denmark [1-4]. Although a rare disease in humans, it is of considerable public health concern due to its high mortality if untreated as well as high treatment costs [5]. In Sweden, infection with *E. multilocularis* in humans and all animal species are notifiable. Due to detection of the parasite in foxes in Denmark in 2000, a surveillance programme was initiated in Sweden in the same year. The surveillance is designed and implemented by the National Veterinary Institute and financed by the Board of Agriculture. It makes use of hunters submitting foxes for examination on a voluntarily basis, against a small remuneration. From 2000 to 2009, a total of 2,962 red foxes (*Vulpes vulpes*), 68 raccoon dogs (*Nyctereutes procyonoides*) and 35 wolves (*Canis lupus*) were examined for *E. multilocularis*: all were negative [6]. Samples from most foxes (n=2,675) were examined by ELISA for the presence of the *E. multilocularis* coproantigen [7] and the rest, plus those from which samples were ELISA positive, were examined using the sedimentation and counting technique (SCT) (n=726) [8]. The raccoon dogs and wolves were examined by SCT. Since 2000, a total of 6,455 hunted foxes have been submitted for *E. multilocularis* analysis.

Surveillance in 2010

During 2010, 304 foxes were examined for *E. multilocularis*. A total of 103 were tested by SCT and 201 by taeniid egg isolation and real-time PCR. One fox, analysed in February 2011, was found to be positive – a young female, shot in December 2010 in Västra Götaland county, in south-west Sweden (Figure). A faecal sample from the fox was examined by egg flotation [9] followed by detection of egg DNA by real-time PCR, using an in-house protocol. The result was confirmed by conventional PCR [9] followed by sequencing. Furthermore, the intestine of the fox was examined by SCT and the parasites present were identified as *E. multilocularis*, both morphologically and by detection of parasite DNA by real-time PCR and sequencing. Although no formal counting of all worms was done, it was estimated that the animal harboured more than 500 tapeworms. Of the remaining 303 foxes found to be negative, 54 originated from the same county.

Surveillance in 2011

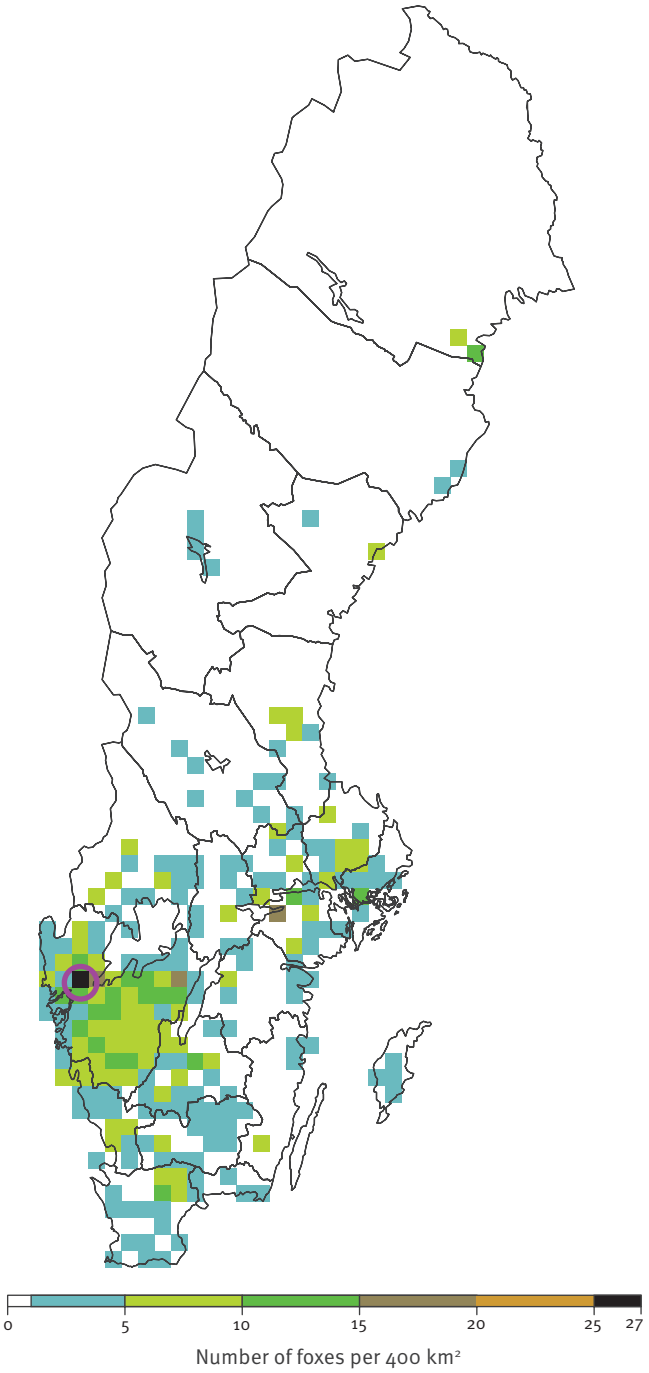
After the positive finding in February 2011, the sampling of foxes was intensified. In the south-western part of Sweden, hunters were requested to submit approximately 10 foxes per municipality in the 93 municipalities in the four counties (Skåne, Blekinge, Halland and Västra Götaland), and four foxes from each of the remaining 197 municipalities in other parts of Sweden. This intensified sampling ceased with the end of the hunting season (i.e. between 28 February and 31 March, ending at the earlier date in the south). By 31 March 2011, a total of 3,189 foxes had been submitted for screening. This sample size is sufficient to detect a prevalence of 0.1% on a country basis, with approximately 95% confidence. The intestines of the foxes were examined by the segmental sedimentation and counting technique (SSCT), which is more cost-effective compared with SCT, but still has a very high

sensitivity [10]. A total of 1,140 foxes had been analysed for *E. multilocularis* by 31 March 2011: one additional fox was found to be positive, an adult female, shot in early March in the same location and by the same hunter as the first infected fox (Figure).

In addition to surveillance of foxes, faecal samples are being collected from 140 hunting dogs in the four municipalities around the parish where the infected foxes were shot, and surveillance in rodents will be initiated once the snow cover has melted.

Figure

Geographical distribution of all georeferenced[a] foxes shot in Sweden and analysed for *Echinococcus multilocularis*, 1 January–31 March 2011 (n=1,025)



The circle indicates the location where the two *E. multilocularis*-positive foxes were shot: one in December 2010 and one in March 2011. The lines indicate the county boundaries.

[a] Coordinate system RT90.

Discussion

It is not yet known how and exactly when *E. multilocularis* was introduced into Sweden. However, considering the frequency of dog movements between Sweden and countries in Europe where the parasite is present, it is regarded as most probable that it was introduced by a dog, despite the legal requirement to deworm dogs before entering the country. Assessments of the risk of introducing *E. multilocularis* into Sweden and the United Kingdom have highlighted dog movement as a risk factor [11,12]. An event that further supports this hypothesis is the introduction in 2003 of another dog-borne parasite (non-zoonotic), the French heartworm (*Angiostrongylus vasorum*) on the coast of Västra Götaland county, where *E. multilocularis* has now been found [13]. This is similar to the situation in Denmark, where *E. multilocularis* was first found in the area where *A. vasorum* had been introduced some decades earlier (unpublished data). This emphasises the need for efficient methods to prevent introduction of the parasite to other *E. multilocularis*-free countries.

After the identification of *E. multilocularis* in Sweden, deworming of dogs and cats has been recommended by the authorities in the four municipalities surrounding the location where the positive fox was shot. These recommendations also apply to dogs and cats entering and leaving this area. Guidance regarding safety precautions has been issued to hunters handling foxes, in line with recommendations given in other countries where *E. multilocularis* occurs [14].

At present, the geographical extent of *E. multilocularis* infection is not known. However, the fact that there were two positive foxes in the same location indicates that this may not be merely a place to which the foxes had wandered and that it may harbour intermediate hosts. The ongoing surveillance is expected to provide more information once up to 3,000 foxes have been examined by early summer this year, and once the dog and rodent screenings have been finalised. However, as the prevalence of *E. multilocularis* infection may be very low, extensive sampling may be needed to define the affected area.

Conditions for the establishment of *E. multilocularis* in Sweden are likely to be favourable: the climate is temperate, allowing worm eggs to survive in the environment for extended periods, and rodents reported to be intermediate hosts of the parasite – such as the water vole (*Arvicola amphibius (terrestris*) and the bank vole (*Myodes glareolus*) – are prevalent. Furthermore, in the northern parts of Sweden, other known intermediate hosts such as muskrats (*Ondatra zibethicus*) and lemmings (*Lemmus lemmus*) are present. The surveillance of rodents is aimed at clarifying which species in Sweden are intermediate hosts. Future actions will depend on the results of the surveillance efforts.

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Gonorrhoea treatment failures to cefixime and azithromycin in England, 2010

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Successful treatment of gonorrhoea is the mainstay of public health control. Cefixime and ceftriaxone, highly active third generation cephalosporins, are today the recommended first-line agents in most countries and azithromycin is a second-line agent. However, there is increasing evidence of decreasing susceptibility and emergence of therapeutic failures. In this report two cases of clinical failure to cefixime are described, one of which additionally shows failure to azithromycin and selection of a less susceptible strain during treatment.

Introduction

Cefixime and ceftriaxone are third generation cephalosporins recommended for first-line therapy for gonorrhoea in the United Kingdom [1]. Cefixime is administered orally in a single dose and is often used in preference to ceftriaxone, which is given intramuscularly (IM). The relationship between dosage given, susceptibility results and treatment failure is still poorly understood but recent reports from Norway [2] and Sweden [3] of treatment failures with cefixime and ceftriaxone, respectively, are beginning to increase our understanding. Azithromycin is an alternative treatment, as a 2-gram dose administered orally, but again the relationship between laboratory findings and treatment failure is unclear. We report here two cases of treatment failure to cefixime, one of which also demonstrated treatment failure to azithromycin, in North East England.

Case 1

In October 2010, a 51 year-old English man, presented at a genitourinary medicine (GUM) clinic in North East England. Prior to this, he had attended his general practitioner (GP) with urethral discharge and dysuria and was treated with amoxicillin and clavulanic acid (co-amoxiclav) for seven days before the tests results were available, which is not first-line treatment for either gonorrhoea or chlamydia, the most common causes of urethral discharge. Tests taken at the visit to the GP returned positive for gonorrhoea and negative for chlamydia and he was referred to the GUM clinic.

At the initial visit (day 1) at the GUM clinic he was symptomatic and reported having had a regular female partner for one year, with whom he last had sex two weeks before. He reported no other sex partners in the last year and no history of sex abroad. On examination, he had a profuse urethral discharge which was diagnosed as presumptive gonorrhoea on microscopy and treated immediately with cefixime 400 mg orally. The laboratory confirmed the diagnosis by isolation of *Neisseria gonorrhoeae* (GC) but reported that the infecting strain of *N. gonorrhoeae* showed decreased susceptibility to cefixime. He was negative for chlamydia, syphilis, and HIV (Table 1).

On recall (day 5), he was still symptomatic and was retreated with azithromycin. The patient returned a further two times, on day 22 and day 30, and remained culture positive for *N. gonorrhoeae* on both occasions (Table 1). He was given a further treatment with azithromycin on day 22 and then on day 40 was treated with ceftriaxone 250 mg IM following the isolation of *N. gonorrhoeae* from urethral sample taken on day 30 (Table 1). His test of cure on day 46 was negative. He reported he had sex with the same contact seven days following his first azithromycin treatment (day 12) but no other sexual contact.

The female sex partner attended another GUM clinic, tested GC culture negative but was treated preventively as a contact of Case 1 with cefixime 400 mg and subsequently azithromycin. She attended GUM Northumberland on day 40 with the index case, declined testing and was treated with ceftriaxone, 250 mg IM. She declared no sex partners other than Case 1.

Case 2

In October 2010, a 23 year-old man attended a different GUM clinic in North East England, as a contact of a chlamydia patient (day 1). He had no symptoms, reported sex with a man two weeks previously, and was treated with azithromycin one gram as a single dose because of his contact with the chlamydia case. He was tested for gonorrhoea (urethra, throat and

rectum) using culture and for gonorrhoea and chlamydia at the same sites using nucleic acid amplification (NAAT) (Aptima Combo 2, Gen-Probe), all of which proved negative. He was also tested for syphilis, HIV, hepatitis B and C markers and was negative (Table 2).

The patient came back to the clinic with symptoms over a month later, reporting having had sex with the same male partner one week prior, with whom he had been having a sexual relationship for the previous eight weeks. He reported no other sex partner in the previous six months. It is, therefore, likely that he acquired his gonococcal infection from this partner since his initial visit, as he tested negative for gonorrhoea on day 1, or from another source although he denied any other partners. A presumptive diagnosis of gonorrhoea was made at this visit (day 37) by microscopy and he was treated with cefixime 400 mg and doxycycline 100 mg twice daily for one week. Gonorrhoea was confirmed by isolation of *N. gonorrhoeae* from the urethra and presence of *N. gonorrhoeae* specific DNA in the urine. Susceptibility to cefixime and ceftriaxone was determined using discs and to penicillin and ciprofloxacin using Etests (Table 2) but the isolate was not available for confirmatory testing at the reference laboratory. Samples taken from the rectum and throat were negative for *N. gonorrhoeae* using culture and for *N. gonorrhoeae* and *Chlamydia trachomatis* using NAATs.

The patient came again to the clinic on day 48 and reported persistent, intermittent dysuria. He reported no sexual contact since day 30 but was again presumptively diagnosed with gonorrhoea by microscopy and treated with ceftriaxone 250 mg IM. *N. gonorrhoeae* was isolated from the urethra and exhibited decreased susceptibility to cefixime (MIC 0.25 mg/L). Six days later (day 54) the test of cure showed the patient was successfully treated.

Table 1

Clinical and microbiological findings and treatment given for gonorrhoea, Case 1, England, 2010

Case 1	Symptoms	Test results	Susceptibility results	Treatment
Day 1	Dysuria Urethral discharge	Gonorrhoea: culture and NAAT Urethra-positive Throat-negative Chlamydia: NAAT Urethra-negative Throat-negative Syphilis serology and HIV - negative	Cefixime: 0.19mg/L Ceftriaxone: 0.064mg/L Azithromycin: 0.25mg/L Ciprofloxacin: 6mg/L Penicillin: 1.5mg/L Spectinomycin: 12mg/L	Cefixime 400 mg orally
Day 5	Recalled for treatment Remained symptomatic	None	NA	Azithromycin 2 grams orally
Day 22	Urethral discharge	Gonorrhoea: urethra Microscopy intracellular GNDC Culture and NAAT- positive	Cefixime: 0.19mg/L Ceftriaxone: 0.047mg/L Azithromycin: 0.25mg/L Ciprofloxacin: 8mg/L Penicillin: 2.0mg/L Spectinomycin: 12mg/L	Azithromycin 2 grams orally
Day 30	Returned for review Asymptomatic	Gonorrhoea: urethra Culture positive	Cefixime: 0.19mg/L Ceftriaxone: 0.047mg/L Azithromycin: 1.0mg//L Ciprofloxacin: 8mg/L Penicillin: 2.0mg/L Spectinomycin: 12mg/L	None given
Day 40	Discharge returned	None	NA	Ceftriaxone 250 mg intramuscularly
Day 46	Returned for test of cure	Gonorrhoea: urethra Culture negative	NA	None given

GNDC: Gram-negative **intracellular** diplococcic; HIV: human immunodeficiency virus; NA: not available; NAAT: nucleic acid amplification tests.

The male sex partner first came to the GUM clinic mentioned above in September 2010 for a check-up, as a chlamydia contact of a female patient and was given azithromycin one gram. He considers himself gay but has some female contacts. No link to this patient was made until eight weeks later when Case 2 was diagnosed with gonorrhoea. When he was recalled, he refused to provide any additional samples although accepted treatment with cefixime.

Microbiology

The three gonococcal isolates from Case 1 and one of the two isolates from Case 2 were available for extended susceptibility tests using Etests. In addition to decreased susceptibility to cefixime, all were sensitive to ceftriaxone and spectinomycin and were resistant to ciprofloxacin and penicillin (Tables 1 and 2). The isolates from Case 1 showed an increase in the minimal inhibitory concentration (MIC) to azithromycin from 0.25 mg/L (day 1 and 22) to 1.0 mg/L (day 30) and Case 2 showed a MIC of 0.5 mg/L. The three isolates from Case 1 were indistinguishable by *Neisseria gonorrhoeae* multi-antigen sequence typing (NG-MAST) [4],

Table 2

Clinical and microbiological findings and treatment given for gonorrhoea, Case 2, England 2010

Case 2	Symptoms	Test results	Susceptibility results	Treatment
Day 1	Asymptomatic Contact of a chlamydia case	Gonorrhoea: culture Urethra-negative Throat-negative Rectum-negative Gonorrhoea and chlamydia: NAAT Urine-negative Throat-negative Rectum-negative Syphilis serology and HIV- negative Hepatitis B and hepatitis C markers-negative	NA	Azithromycin 1 gram orally
Day 37	Dysuria Urethral discharge	Gonorrhoea: microscopy Urethra –intracellular GNDC Gonorrhoea: culture Urethra- positive Rectal-negative Throat-negative Gonorrhoea and chlamydia-NAAT Urine- GC positive/CT negative Throat-negative Rectum-negative	Cefixime: sensitive[a] Ceftriaxone: sensitive[a] Penicillin 0.25mg/L Ciprofloxacin 8mg/L	Cefixime 400 mg orally Doxycycline 100 mg bd / seven days
Day 48	Dysuria persisting intermittently Purulent discharge on examination	Gonorrhoea: microscopy Urethra –GNDC Gonorrhoea: Culture Urethra- positive	Cefixime: 0.25mg/L Ceftriaxone: 0.064mg/L Azithromycin: 0.5mg/L Ciprofloxacin: 8mg/L Penicillin: 2mg/L Spectinomycin: 8mg/L	Ceftriaxone 250 mg intramuscularly
Day 54	Symptoms subsided No discharge	Gonorrhoea: culture Urethra- negative	NA	None given

bd: twice daily; CT: *Chlamydia trachomatis*; GC: *Neisseria gonorrhoeae*; GNDC: Gram-negative intracellular diplococcic; HIV: human immunodeficiency virus; NA: not available; NAAT: nucleic acid amplification tests.

[a] Disc sensitivity testing only available.

belonging to sequence type (ST) 3779 (*por*, 2147; *tbp*B, 110) but distinct from the isolate from Case 2 which belonged to ST 3431 (*por*, 2078; *tbp*B 110). All isolates contained the *pen*A mosaic allele [5]. Susceptibility to cefixime of both isolates from Case 2 had been determined using discs at the local laboratory and appeared initially sensitive and then exhibiting reduced susceptibility. This could indicate acquisition of a different strain but could also reflect difficulties in testing gonococcal susceptibility and remains unresolved as the initial isolate was unavailable for confirmatory testing at the reference laboratory. In the absence of treatment failures, the relationship of zone size to clinical failure is unknown and the second isolate was referred to Sexually Transmitted Bacteria Reference Laboratory (STBRL) by request of the clinician suspecting treatment failure.

Discussion

We report two cases of treatment failures to cefixime in England, one of which fulfils all the criteria for a verified failure [6]. The second case has limited information on the pre-treatment isolate but otherwise is consistent with a treatment failure and similar to a previous report [7]. The MICs to cefixime of 0.19-0.25 mg/L are consistent with the report from Norway [2] and with the European Committee on Antimicrobial Susceptibility Testing (EUCAST) [8] breakpoints of MIC 0.12 mg/L, although these remain putative until the relationship with clinical failure is fully clarified. They are also compatible with Monte Carlo Simulation modeling that suggests peak serum cefixime concentrations are inadequate for successful eradication of infections exhibiting MICs of 0.125 mg/L and above at the current doses used [9].

Case 1 also demonstrated treatment failure to azithromycin on potentially two occasions following treatment, on days 5 and 22. The patient admitted having had sex with the same contact between the two treatments and so he could have been re-infected with the same strain, which subsequently then failed azithromycin treatment again. However, the female contact was never diagnosed with gonorrhoea. The MIC breakpoint that equates with treatment failure for azithromycin is not known and is currently arbitrary, but in this instance the increase in MIC over time suggests selection of resistant strain during therapy, as previously demonstrated in the laboratory [10].

Dissemination of gonococcal isolates with cefixime decreased susceptibility in England and Wales has been largely clonal, belonging to ST 1407 or closely related STs, all sharing the *tbp*B 110 allele, as in these isolates [unpublished data]. Public health control of gonorrhoea is dependent on successful antimicrobial therapy and lessons should be learnt from the extraordinary ability of the gonococcus to be resistant and innovative treatment regimens will need to be used to prevent gonorrhoea becoming an infection difficult to treat. A viable organism is essential to detect emerging resistance as well as for susceptibility testing for individual patient management and therefore maintaining culture will be of paramount importance.

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Outbreak of rotavirus gastroenteritis in a nursing home, Slovenia, December 2010

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A gastroenteritis outbreak affected 45 people (40 residents and five staff) in a nursing home for the elderly in the Celje region, north-east Slovenia, between 17 December and 31 December 2010. Rotavirus group A was laboratory confirmed in the stools of five ill individuals. The outbreak was identified when the number of affected persons was high but was successfully controlled after implementing preventive measures.

Background

On 28 December 2010, the regional epidemiologist of the Institute of Public Health Celje, North East Slovenia, was informed that several residents and staff of a nursing home in the Celje region had symptoms of acute gastroenteritis. Symptoms had first occurred in two residents on 17 December. On 26 December, an 88 year-old resident had been hospitalised for dehydration because of diarrhoea and vomiting. By 28 December, 32 people (four staff and 28 residents) were reporting one or a combination of symptoms including diarrhoea, vomiting, malaise and in four cases elevated body temperature. On 28 December, the Department of Medical Microbiology, Institute of Public Health, Celje confirmed the presence of rotavirus group A antigens in the 88 year-old resident's stool.

Rotavirus infections are well documented in preschool children and present a problem in developed and developing countries alike. Worldwide, 870,000 children under five years old die from rotavirus infections every year [1,2]. In adults, symptomatic rotavirus infections are relatively rare, but can cause health problems and outbreaks in the elderly and in immunocompromised individuals [3,4]. For children under five, there are two licensed vaccines against rotavirus infections.

Rotaviruses are RNA viruses from the Reoviridae family; they are divided into seven serogroups (A to G) on the basis of antigen groups. Infections in humans are caused by serogroups A, B and C, serogroup A being the most common.

Outbreak investigation

On 28 December, an outbreak investigation was initiated. The nursing home for the elderly comprised 121 residents aged from 66 to 95 years, 85 females and 36 males. The residents were cared for by 30 of a total 62 staff which also included 14 kitchen staff and 18 support personnel (cleaners, drivers and janitor). Of the residents, 66 were fully mobile, 26 were wheelchair users and 29 were bed-bound. The rooms for residents are either equipped with one or two beds and are located in the basement, on the ground floor, at the first level, and in two lofts. In addition, there are four small kitchens on each respective floor, a dining hall and a living room. The nursing home does not have a separate unit for bed-bound residents. Mobile residents can go about freely in and around the nursing home.

Enterovirus infection was suspected based on the microbiological confirmation of rotavirus gastroenteritis in the hospitalised resident. Every resident and staff member (epidemiological link) who presented with at least one of the following symptoms and signs from 17 December was classified as a probable case: diarrhoea (›three loose stools/day), vomiting and elevated body temperature (›37°C). A confirmed case was considered as a case with clinical symptoms and laboratory confirmation.

A total of 151 epidemiological questionnaires were distributed to all residents and nursing staff with questions on the date of onset of symptoms if any, gastroenteritis-related health problems and their duration, treatments, and ingestion of food and beverages outside the nursing home. The residents were also asked to identify the room they occupied, and the nursing staff reported which residents they cared for and possible onset of symptoms of gastroenteritis in their family members, if applicable. In parallel, information on measures to prevent the spread of the disease and instructions on how and what samples to collect (vomit, stool) for microbiological analysis were distributed [5].

We received completed questionnaires for all nursing staff and all residents by 4 January 2011. Residents from all building levels of the nursing home felt ill; no level-based clustering was observed. All the staff affected had provided nursing care to symptomatic

residents. According to the probable case criteria, the two residents who became ill on 17 December 2010 (11 days before we were informed of the outbreak) were identified as the first two cases in the outbreak. Between 28 and 30 December, 15 residents became ill, and no further cases were identified after 31 December (Figure). A total of five of 30 nursing staff (16.7%), and 40 of 121 residents (33%) became ill during the outbreak. The overall attack rate was 30%. Only one resident was hospitalised. None of the kitchen staff and support personnel became ill as they were informed about the outbreak and asked to report if they had any symptoms. The staff did not report any symptoms of gastroenteritis in their family members.

Diarrhoea was reported by all 45 affected individuals, 19 experienced vomiting and four had elevated body temperature. Some patients also reported abdominal pain (Table). The median age of the affected staff was 35 years (mean: 35 years, age range: 23 to 44 years), the median age of the affected residents was 78 years (mean: 82.4 years, age range: 66 to 95 years). The average duration of symptoms of gastroenteritis was 2.4 days (from one to four days) in staff, and three days in residents (one to nine days). The outbreak affected 26 women and 19 men. The highest proportion of resident cases was among fully mobile residents (29 of 40 cases), followed by bed-bound residents (seven of 40 cases) and residents on wheelchairs (four of 40 cases).

Laboratory investigation

One stool sample was collected from the 88 year-old hospitalised resident on 26 December and was sent to the Department of Medical Microbiology, Institute of Public Health Celje. On 28 December, results of enzyme-linked immunosorbent assay (ELISA) testing for antigens of adenoviruses, astroviruses and group A rotaviruses were available. Routine diagnostic procedures for rotavirus infections usually include spectrophotometric enzyme immunoassay (EIA), which is highly sensitive and detects group A rotaviruses only. Qualitative EIA was used to confirm antigens of group A rotaviruses (ProSpectTM Rotavirus Microplate Assay, OXOID). Up to 28 December, cultures for *Salmonella* spp., *Shigella* spp., *Campylobacter* spp. *Yersinia* spp., *Clostridium difficile* toxin A and B, and *C. difficile* did not point to infection with these bacteria and were confirmed to be negative on 30 December.

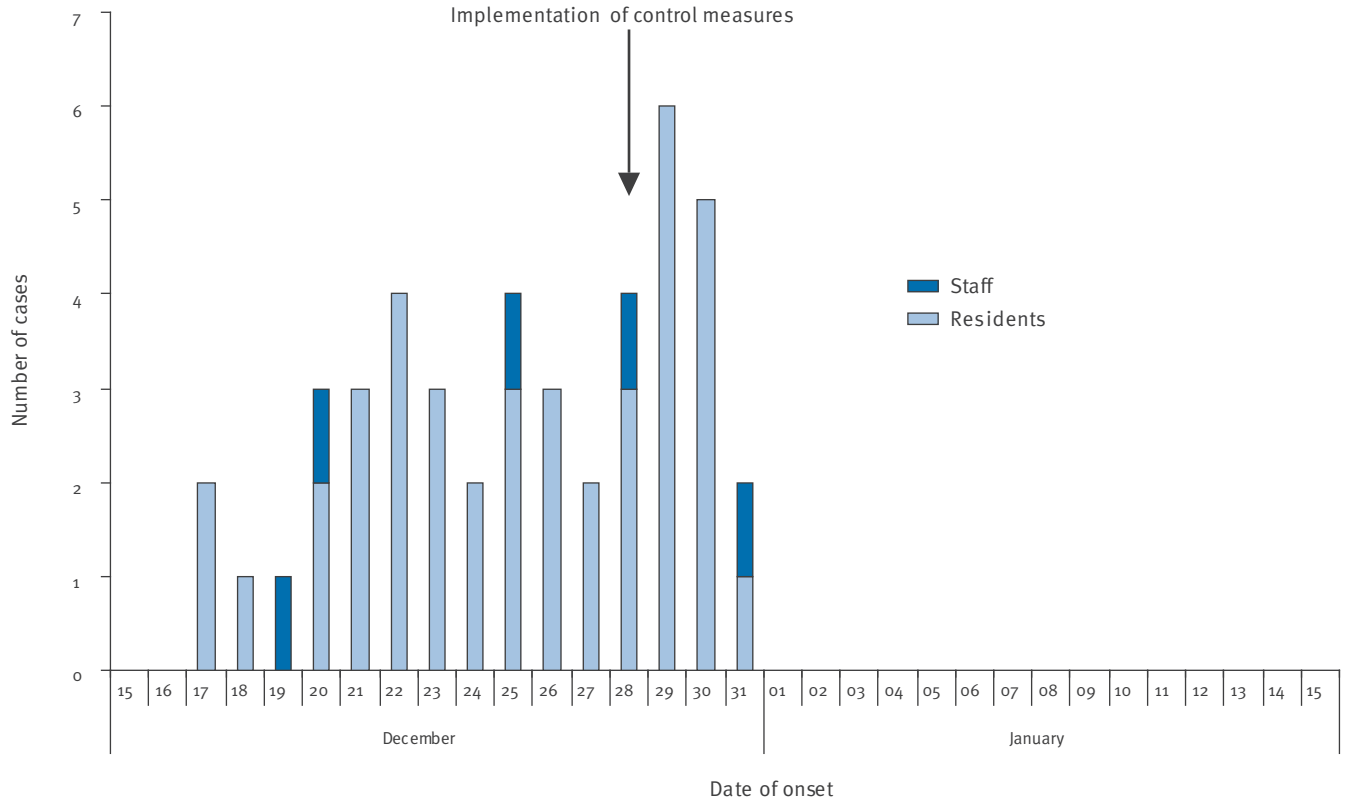
On 28 and 29 December, taking in consideration the result of the hospitalised patient, five additional stool samples from four symptomatic residents and one staff member were tested only for the presence of antigens of astroviruses, adenoviruses and group A rotaviruses. EIA was used to confirm rotavirus group A antigens in four samples, including three from the residents and one from the staff; one sample was negative. All individuals tested were negative for noroviruses.

Control measures

On 30 December, following the confirmation of a rotavirus outbreak, a special sanitary inspection of the

Figure

Epidemic curve for cases of rotavirus gastroenteritis in a nursing home for the elderly, Slovenia, December 2010 (n=45)



nursing home was performed. Measures to prevent the spread of viral diarrhoea were put in place; strict hand hygiene and cleaning with an appropriate disinfectant for viruses, cleaning and disinfection of equipment, surfaces and rooms. Regular airing of premises was recommended. Sanitary inspection of proper disposal of incontinence pads with excrements from residents was conducted. As a temporary measure, contacts between the affected and non-affected residents were limited; cohort isolation of the affected was not implemented. The affected staff were removed from work for a period of one to four days until they did not present any more symptoms [6,7].

Discussion

We describe an outbreak of rotavirus gastroenteritis in a nursing home for the elderly. On 17 December, two residents became ill at the same time; the first resident was bed-bound and the second was mobile and visiting the first one. The first member of the staff fell ill on 19 December (Figure). The outbreak, affected 40 of 121 residents and five of 30 nursing staff. All five affected members of the staff had provided nursing care to bed-bound residents. The most frequent symptoms were diarrhoea, vomiting and elevated body temperature. The average duration of illness was different for staff and residents, 2.4 and three days, respectively. All affected persons made full recovery; only one resident was hospitalised.

Rotavirus gastroenteritis symptoms usually accompany primary infection in childhood, which is followed by protection against subsequent symptomatic infection. For this reason, the ratio of symptomatic to asymptomatic infection decreases with age. In prospective studies, symptomatic infection rates were highest in children under two years, and lowest in those of 45 years of age [3]. Rotavirus infection in immunocompromised adults can have a variable course from asymptomatic to severe and sustained infection [4]. Vaccination for infants from six to 26 weeks of age, which has already been included in some national vaccination programmes, will serve to decrease the burden of rotavirus infections in the future [8,9]. In Slovenia, rotavirus vaccination for infants is available against payment [10].

Table

Clinical manifestation in ill individuals, rotavirus gastroenteritis outbreak, Slovenia, December 2010 (n=45)

Clinical manifestation	Number of individuals[a]
Diarrhoea	45
Vomiting	19
Elevated temperature	4
Stomach pains	1
Feeling unwell	3
Malaise	12

[a] Each individual could record up to six symptoms listed.

Before 2008, rotavirus gastroenteritis outbreaks in Slovenia were reported mostly in preschool and school environments [11]. In 2008, however, rotavirus gastroenteritis outbreaks in nursing homes for the elderly in Slovenia were first recorded in addition to norovirus infections [12]

Our investigation shows another outbreak of rotavirus gastroenteritis in an elderly nursing home, highlighting the potential of rotavirus outbreaks in such a setting. Our results are in agreement with other studies reporting that long-term residence in a closed community is a risk for rotavirus illness [13]. Noteworthy in our investigation, is that five of 30 younger nursing staff (ranging from 23 to 44 years) were affected. This indicates that rotavirus infections can occur in all age groups and affect caretakers of an elderly home, who in turn can contribute to the spread of the disease. This is not entirely unexpected as faeces and vomit from infected individuals can contain more than 10^{13} infectious infectious particles* per gram and only 10 to 100 of these are required to transmit infection [5]. Future epidemiological studies are needed to assess the impact of rotavirus infections in the elderly.

To this effect, outbreaks need to be not only registered, but also reported as close as possible to onset, so that microbiological diagnostic and complete monitoring can be implemented as fast as possible. In the present outbreak, public health authorities were only notified once the number of affected persons was high. This situation is likely to occur frequently because of the speed at which rotavirus gastroenteritis outbreaks can spread, so our investigation highlights the importance of a tight collaboration and dialogue between nursing home staff and public health authorities. More efforts need to be focused on increasing vigilance among caretakers for elderly or vulnerable groups and training caretakers to communicate outbreaks in a timely manner. This will prevent delays in putting in place containment measures and will allow for better care of vulnerable groups such as the elderly or immunocompromised patients.

*Erratum: The number 10^{13} was corrected on 09 April 2011.

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Outbreak of Shigella sonnei infections in the Orthodox Jewish community of Antwerp, Belgium, April to August 2008

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In the beginning of April 2008 three cases of *Shigella sonnei* infection were identified among the Orthodox Jewish community of Antwerp, Belgium. We conducted a descriptive study and a household cohort study to identify potential risk factors. Stool samples were cultured and antibiotic susceptibility of the isolates was determined. Between April and August 2008, 42 cases were registered. All characterised isolates (n=20) shared an identical pulsed-field gel electrophoresis profile and were indistinguishable from one of the twelve main strains detected in Israel in 2008, where the index case's father had stayed before the outbreak. The secondary attack rate in households was 8.5% (95% confidence interval (CI): 4.3–12.7). Multivariate analysis identified the following risk factors for secondary spread: households with more than three children (adjusted relative risk (RR): 9.17; 95% CI: 1.21–69.13), children younger than five years (adjusted RR: 5.45; 95% CI: 2.44–12.62), and children younger than 12 years assisting in washing younger siblings (adjusted RR: 5.45; 95% CI: 2.44–12.17). Rigorous hand washing, use of disposable towels, information for parents and caregivers, and exclusion of symptomatic children from day care, preschool and school for a minimum of 48 hours were implemented.

Introduction

Infection with *Shigella sonnei* is a major cause of bacterial gastroenteritis and a leading cause of bacillary dysentery in Belgium [1,2]. Shigellosis is a highly communicable disease and requires a low dose for infection [1,2]. In industrialised countries person-to-person transmission accounts for most cases of *S. sonnei* infections, which occur commonly in children aged between six months and 10 years [2,3].

Shigellosis has been a statutorily notifiable disease for clinicians and microbiologists in Belgium since 1971 [4]. Between 2000 and 2009, the number of laboratory isolates of shigellae registered annually by the Belgian reference laboratory of salmonellae and shigellae varied from 316 to 500. *S. sonnei* has been the predominant agent causing 65% to 75% of all registered Shigella infections [4,5].

In the beginning of April 2008, a microbiologist of one of the town hospitals informed the Department of Communicable Disease Control of Antwerp that *S. sonnei* had been isolated from three children. The patients belonged to the Orthodox Jewish community of the town. The standardised post-notification interview with their general practitioners and parents showed that the patients had not been out of the country in the month before onset of symptoms. The father of the first case had just returned from a stay in Israel where he felt sick during three days before his return.

Antwerp has an Orthodox Jewish, highly insular community of approximately 10,000 persons living in one quarter of town. The community is characterised by relative social isolation and frequent international contacts especially with New York, London, and Israel [6]. Although sporadic cases of shigellosis have been identified among members of the Orthodox Jewish community in Antwerp before, a well documented outbreak in Belgium has never been described [4].

The first aim of the study was to describe the extent of the outbreak and to identify risk factors for secondary transmission. In addition, we tried to compare the strains from identified cases to confirm that they were genetically indistinguishable and to compare them to the circulating strains in Israel. Using the information

obtained from these objectives, we wanted to implement appropriate public health control and prevention measures in order to stop the propagation of the disease. An outbreak control team was established to oversee the coordination of this study.

Methods

Case definition

A confirmed case was defined as a person living in the town of Antwerp, who had a positive stool culture for *S. sonnei* in the period between 1 April and 31 August 2008. A probable case was defined as a person who had diarrhoea (three or more loose stools within 24 h), fever (≥38°C) and nausea, and who lived in a household where a confirmed case had been detected. An index case in a household or school was the first laboratory-confirmed shigellosis case in each household or school class. A secondary case was a confirmed or a probable case occurring within seven days after the detection of an index case in a household or in a classroom.

Case finding

Cases of shigellosis were reported by peripheral microbiological laboratories and clinicians in accordance to statutory notification of infectious diseases. Active case finding among members of affected households and school classes was performed by the local health authorities. General practitioners and paediatricians were asked to report cases to the outbreak control team.

Data collection

For each identified case, information on demographics, and clinical and microbiological characteristics was collected using a standardised questionnaire. The questionnaire also collected information on possible exposures including any recent travel, attending family gatherings, contacts with other cases, names of household contacts, and attendance at schools or day care centres. It was administered by telephone or by face-to-face interviews at home. Household contacts were followed prospectively for clinical symptoms during one week after contact with the index case. Social Service of the Antwerp Jewish community assisted in contacting people in order to avoid language and cultural barriers. Demographic data collected on household members were compared to data collected from the municipal registry office.

Secondary attack rate study

To identify specific risk factors for secondary transmission, a retrospective cohort study was conducted among the household contacts of the index cases. A household contact was defined as a person living in the same house as the household index patient. A secondary attack rate was calculated by identifying secondary cases in proportion to the number of household contacts after exclusion of the index case. Potential risk factors for transmission in the household were assessed as follows: the number of children in the household, the age of the children, the presence of children with nappies, the practice of hand washing after washing children, the number of toilets, whether children younger than 12 years (primary school children) were assisting their parents in washing siblings with gastrointestinal symptoms or assisting them at going to the toilet, whether the index case received antimicrobial treatment or whether the index case was admitted to hospital.

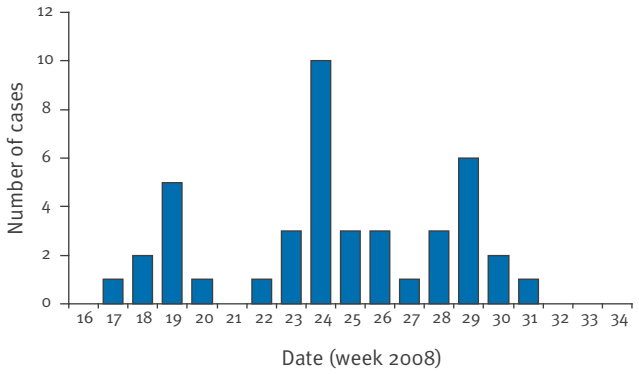
Laboratory investigations

Shigella strains isolated from patients in peripheral clinical laboratories were sent on a voluntary basis to the National Reference Centre for Salmonella and *Shigella* for serotyping by slide agglutination with commercial antisera (Denka Seiken Co). To evaluate antimicrobial susceptibility, *S. sonnei* specimens were tested by disk diffusion (Kirby-Bauer) following recommendations of the National Committee for Clinical and Laboratory Standards Institute (CLSI), formerly the National Committee for Clinical Laboratory Standards (NCCLS) [7]. Antibiotics tested (BioRad disks) were: ampicillin, amoxicillin/clavulanic acid, cefotaxime, chloramphenicol, tetracycline, naladixic acid, ciprofloxacin, streptomycin, kanamycin, gentamicin, sulfonamides, trimethoprim, and trimethoprim/sulfamethoxazole. Interpretation of inhibition zones was performed according to the CLSI criteria, and quality control was performed using the *Escherichia coli* ATCC 25922 reference strain [7].

S. sonnei strains were analysed by pulsed-field gel electrophoresis (PFGE) according to the PulseNet method and digested with the restriction endonuclease *Xba*I (New England Biolabs) [8]. *Salmonella enterica* serovar Braenderup H9812 was used as size marker. Fingerprinting II Informatix software (Bionumerics, BioRad) was used to compare the PFGE profiles. *Salmonella enterica* serovar Braenderup H9812 was used as size marker. FingerprintingII Informatix software (Bionumerics, BioRad) was used to compare the PFGE profiles. In addition, we included as internal reference five unrelated *Shigella* strains from national collections that had been isolated from Belgian patients

Figure 1

Onset of illness among *Shigella sonnei* cases, by week, Jewish community Antwerp, 17 April–31 August 2008 (n=42)



in 2008. The PFGE profiles of the outbreak strains in Antwerp were also compared to patterns, obtained with the same PulseNet method, of *S. sonnei* isolated from different orthodox Jewish community outbreaks in Israel between 2000 and 2008. The PFGE gel for them was provided by the Central Laboratories Ministry of Health of Israel. The bands had been analysed using the Dice coefficient and the unweighed-pair group method using average linkage with a tolerance of 1%.

Statistics

Univariate analysis was performed on data collected in the retrospective household cohort study and crude relative risk (RR) along with 95% confidence intervals (CI) were calculated to determine associations between potential risk factors and infection. Adjusted RRs were calculated using a binomial regression model. All statistical analyses were performed using Stata software version 11 (StataCorp).

Results

Outbreak description

Between 17 April and 31 August 2008, 42 cases of shigellosis were identified in Antwerp and all of them belonged to the Orthodox Jewish community, with the highest number of cases in week 24 (Figure 1). Thirty-two of them were confirmed cases and 10 were probable cases. Cases occurred in 19 Jewish families and in four confessional schools. Two additional reported cases of *S. sonnei* identified outside the Jewish community of Antwerp were excluded from the study because the disease started during a stay in Egypt. They were classified as travel-associated cases.

Of the 42 cases, 20 were male and 22 female. The arithmetic mean of age of cases was 4.4 years with a range from three months to 61 years. Four patients were younger than two years, 19 were between two and five years-old, seven were between six and 10 years-old, six cases were between 11 and 15 years-old, and six cases were older than 20. The affected families had an average of 4.6 children (range: 1–12). All patients had their residence in an enclosed area in the town centre.

Eighteen cases reported fever ≥38°C and bloody or mucopurulent diarrhoea and abdominal cramps, and 32 cases were hospitalised. The average duration of illness was eight days with a range from six to 11 days. The average stay at the hospital was 3.4 days.

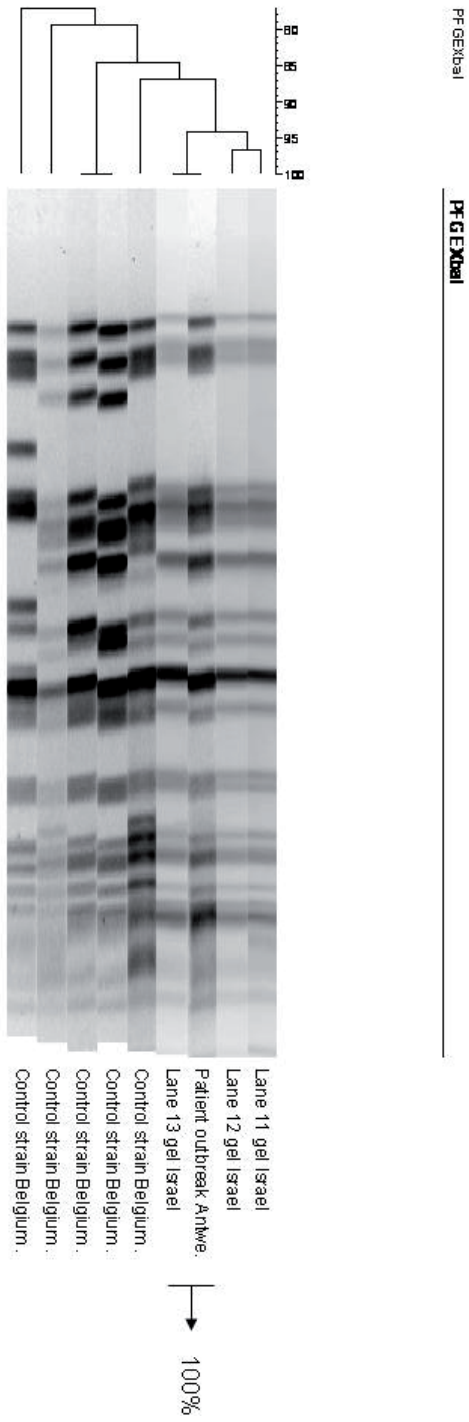
Of the 42 cases, 15 cases met the criteria for a secondary case. The generation interval was 3.5 days (range from one to four). Three children developed illness within two to five days of detection of a case in a classmate.

Laboratory data

We received antibiotic susceptibility results from 28 of the 32 confirmed *S. sonnei* isolates. All of them were resistant to amoxicillin and trimethoprim-sulfamethoxazole, but were susceptible to levofloxacin and

Figure 2

Cluster analysis of PFGE fingerprinting of *Shigella sonnei* isolated in Antwerp and Israel in 2008



'Lane 11, 12 and 13 gel Israel': *S. sonnei* from different outbreaks in Orthodox Jewish communities in Israel in 2008.

'Patient outbreak Antwerp': *S. sonnei* strain from the outbreak in Antwerp in 2008.

'Control strains': unrelated *S. sonnei* strains from the national collection of the Belgian national reference laboratory in 2008.

cefotaxim. PFGE was performed on 20 of the 32 isolates and showed that all strains isolated during this outbreak displayed the same restriction-fragment patterns, confirming the relatedness of these isolates. The outbreak strain in Antwerp was compared to 12 different outbreak strains detected in *Shigella sonnei* shigellosis outbreaks in Orthodox Jewish communities in Israel between 2000 and 2008. Figure 2 presents the results of a cluster analysis on the basis of PFGE fingerprinting of isolates from Antwerp and Israel. The isolate called 'Lane 13 gel Israel', *S. sonnei* isolated in 2008 in Israel, was indistinguishable from the Belgian outbreak strain. The isolates shown as 'Lane 11 and 12 gel Israel', also isolated in Israel in 2008, had a closely related profile with the Belgian outbreak strain. Five unrelated *S. sonnei* strains originating from national Belgian collections ('Control strain Belgium' from 2008) were used as internal reference.

Secondary attack rate study

For the 29 affected households with confirmed cases, we identified 175 household contacts, of whom 15 developed shigellosis. A secondary attack rate of 8.5% (95% CI: 4.3–12.7) was calculated. Information on hand washing, the number of toilets in the home and the use of disposable towels was only provided by four of the 25 interviewed households. These questions were excluded in the analysis. The calculated crude and adjusted RRs for the other risk factors are shown in the Table. In the uni- and multivariate analysis, having more than three children in the family, having children younger than 12 years who assisted their parents washing siblings and helping them go to the toilet, and having children younger than five years, were significantly associated with a higher risk of secondary transmission. Having more than three children in the household was associated with the highest risk, with an adjusted RR of 9.17 (95% CI: 1.21–69.13). Hospitalisation and treatment with antibiotics of the household index cases were not significantly associated with a lower risk of secondary infection, with a respectively adjusted RR of 0.88 (95% CI: 0.61-3.1) and an adjusted RR of 1.8 (95% CI: 0.80-4.34).

Control measures

To prevent further spread of the disease, parents of the affected families were advised of the importance of hand washing with running water and liquid soap after using the toilet or washing the children and also on the importance using disposable towels and cleaning the toilets with chlorine. The need to decontaminate toys was highlighted. In June 2008 educational presentations for parents, caregivers and teachers were organised. Information was also published in the local media. Physicians were informed via articles in the local medical infectious disease journal. Schools were informed on the hygiene of hand washing facilities. We insisted on excluding symptomatic children for a minimum of 48 hours after clinical recovery from day care centres, preschool and school attendance [9].

Discussion

We identified a cluster of 42 cases of shigellosis in the Orthodox Jewish community of Antwerp with 32 isolates laboratory-confirmed as *S. sonnei* with the same genetic profile. Temporal and spatial clustering in one area of town affecting one specific community supported the hypothesis of a single ongoing outbreak, maintained through person-to-person transmission. Statutory laboratory-based surveillance of shigellosis failed to identify concurrent cases outside this community. Two additional *S. sonnei* cases notified in the study period in the province of Antwerp in people who were not Jewish were most probably not linked to the outbreak. The disease started during a stay in Egypt and they were classified as travel-associated cases.

The index case was most probably infected by their father, who had suffered from gastrointestinal problems during a stay in Tel-Aviv, Israel until two days before symptom onset in the index case but did not seek medical care. No exceptional family gatherings could be identified except for synagogue attendance. The father also reported having been in contact with relatives coming from London.

To investigate a possible link between the outbreak in Antwerp and an ongoing outbreak in Israel [10], the circulating strains in both outbreaks were compared. Such a link was supported by the microbiological analysis in which the main strain circulating in Israel at the time and the outbreak strain in Antwerp were indistinguishable. The father of the index case also reported having been in contact with relatives coming from

Table

Risk factors of illness among household contacts of an index case with shigellosis, Jewish community Antwerp, 17 April–31 August 2008 (n=42)

	Univariate analysis		Multivariate analysis	
Exposure	Crude relative risk	95% confidence interval	Adjusted relative risk	95% confidence interval
>3 children in household	8.47	1.14-62.98	9.17	1.21-69.13
Children with nappies	2.41	0.90-6.48	1.59	0.84-3.01
Children <5 years in household	6.0	1.39-25.80	5.24	1.17-23.62
Children <12 years assisting parents washing siblings	6.54	2.59-16.51	5.45	2.44-12.17
Index case in household hospitalised	1.02	0.38-2.75	0.88	0.61-3.10
Index case in household treated with antibiotics	1.42	0.50-3.99	1.87	0.80-4.34

London. Addiman et al. reported on an outbreak of shigellosis in London starting a month before the onset of our outbreak in Antwerp in 2008 [11]. A strain from the outbreak of London 2008 could not be obtained for comparison.

Outbreaks of shigellosis with *S. sonnei* and recurrent increases in the number of cases in Orthodox Jewish populations have already been notified in 2008 and before in different countries. Calderon-Margalit et al. showed that between 1998 and 2006, outbreaks of shigellosis followed a biennial pattern in Israel with annual rates that ranged from 18 to 353 cases per 100,000 population [12]. Also in 2009 outbreaks of *S. sonnei* in Israel were still continuing [10]. Close contacts, day care attendance and having many young children in the families were considered risk factors. The characteristics of the outbreak in Antwerp are comparable with prolonged outbreaks of *S. sonnei* reported by Sobel et al. in North America in traditionally observant Jewish communities between 1994 and 1996 [13] , with outbreaks reported by Garret et al. in New York in 2005 [14] and with the outbreak in London in 2008 [11].

The secondary attack rate of 8.5% found in this study is comparable to those noticed in other studies [2,15]. In larger studies, secondary attack rate differed according to age and to the species of bacterium [2]. Due to the limited number of cases in our study, age-specific attack rates could not be calculated. Dupont et al. showed that for one to four year-olds, the secondary attack rate can reach 40% [2]. The combination of high communicability due to the low infective dose, crowding, and frequent contacts are known explanations for the high secondary attack rate for shigellosis [2,14,15]. In our study we analysed specific risk factors which might explain the noted secondary attack rate. Having more than three children in the household and having children younger than five years of age was significantly associated with the occurrence of secondary cases, which is consistent with data from other authors [14]. Contrary to what we expected, having children with nappies in the household was not a significant independent risk factor in our study. This could be due to good hygienic habits of the adults when providing care for their babies. That the index case of the family was hospitalised was hypothesised to be a protective factor, but the adjusted RR was 0.88 (95% CI: 0.61–3.10). The low number of cases and the different intervals between onset of the disease and moment of hospitalisation of the cases might have interfered with the association. Being treated with antibiotics was not significantly associated with a lower risk of secondary transmission either. Different delays in the start of therapy, the broad spectrum of used antibiotics and the small number of cases might explain the calculated RR of 1.8 (95% CI: 0.80–4.34).

However, it was remarkable that children younger than 12 years, helping their parents take care of babies, was associated with a higher risk of secondary cases (adjusted RR: 5.45; 95% CI: 2.44–12.17). In families with a high number of children, older children were asked to help. There is a risk that these young children are less sensitive or less knowledgeable than their parents on the risk and the practice of hand hygiene.

Visiting friends and relatives in areas with higher risk of shigellosis might be the seeding event leading to shigellosis outbreaks in especially susceptible communities. This is the case for Jewish communities in Antwerp that are more susceptible due to the high number of children in the families, the many social contacts, living in a relatively small community, and the frequent contact with relatives who live in areas of higher endemic prevalence of shigellosis, like certain neighbourhoods in London or Israel [11,12].

The high hospital admission rate in our study (32 of 42 cases) suggests that we have probably detected only the most severe cases, whereas milder cases could also have been expected. Presenting bloody or mucopurulent diarrhoea was noted in 18 of the cases. This is unusual compared to the expected picture of a *S. sonnei* infection which is normally associated with a milder disease [2,3,5]. We have therefore reason to consider under-diagnosis and under-reporting in this outbreak, which is also mentioned in similar outbreaks of shigellosis elsewhere [2].

Several limitations of the study especially for the secondary attack rate study should be noted. Firstly, the number of cases was limited. This raises concerns about the interpretation of the calculated relative risks. Secondly, we assumed that secondary cases acquired their infection at home. Alternatively they might have been infected during pre-school and school attendance or by visiting friends and relatives. Thirdly, personal questions like ‘did you wash your hands after toilet use?’ and ‘how many toilets do you use at home’ were often not answered, most probably due to their sensitive and private nature [16]. It is likely that not all possible risk factors could be explored in the study of this outbreak.

Early notification of shigellosis enabled prompt reaction, and implementation of the advice most probably put a stop to further propagation. We presume that especially the intensive hand washing campaign in families and schools, the educational presentations and specific information to physicians contributed to stopping the outbreak.

PFGE in studies of clusters has been shown to be a highly effective method of characterising *S. sonnei* and an important tool for outbreak investigations [17]. Provided that the same protocol is used, it allows comparison of strains detected in different outbreak. The genetic relatedness of the strains in this study provides strong evidence that this cluster was a single outbreak and associated with recurrent endemic shigellosis in Israel [10,12].

In conclusion, this is the first well-documented outbreak of *S. sonnei* in the Orthodox Jewish community of Antwerp and Belgium for which a direct link to an ongoing outbreak of endemic shigellosis in Israel could be identified. The combination of case finding, source tracing, and comparing different strains with PFGE was essential for confirming the hypothesis of import of an outbreak strain from Israel into the local community, and implementation of hand washing was important to stop the propagation of the epidemic.

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