Eur J Clin Microbiol Infect Dis (2000) 19:542–544

Note

Outbreak of *Campylobacter* Infection in a Subartic Community

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Abstract A presumably waterborne outbreak of *Campylobacter jejuni/coli* infection in a subarctic community is described. Drinking water supplied to residents was delivered unchlorinated during a 4-week period. No *Campylobacter* sp. was recovered from the water supply. Three hundred thirty individuals (15% of the 2200 exposed) became ill. Diarrhoea, abdominal pain, fever, nausea and joint pain occurred in 81%, 30%, 29%, 43% and 21%, respectively. Nine percent reported swelling of joints, and two cases of reactive arthritis occurred. A *Campylobacter* sp. was isolated from 9 of 33 individuals who became ill and from 1 of 33 healthy controls. All culture-positive individuals, 46% of culture-negative ill persons and 27% of healthy controls were seropositive. All strains recovered had an identical DNA profile.

Introduction

Several waterborne outbreaks of campylobacteriosis have been reported from Scandinavia as well as from other countries [1–8]. Sequential sera, together with clinical information and culture results, have been available in very few of these outbreaks. The present report of an outbreak of gastroenteritis in a subarctic community (total population 3085), where clinical information, culture results and serum samples were available from a number of patients, is an extension of a brief note on the clinical data presented previously [9].

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Materials and Methods

The water source of this community is a river on which a dam has been built. Thus, surface water served as drinking water common to 2200 of the inhabitants in the community. During the summer of 1988, the rinsing system (chlorination) was to be renewed and the water was delivered unchlorinated for a period of approximately 4 weeks. During this period, the community health officers were faced with increased morbidity presenting as gastrointestinal illness.

As soon as the outbreak was evident, residents were encouraged to contact the local health care centre. A subset of the individuals who visited the health care centre during this period – whether suffering from gastrointestinal illness or not (i.e. the latter belonging to, in this context, a healthy control group) – were randomly allocated to a patient group or a healthy control group. Faecal specimens and serum samples were obtained from these individuals, and a brief clinical history was taken. Faecal samples for culture were obtained from 66 symptomatic individuals at the first visit, i.e. 1–2 weeks after the onset of disease. Faecal samples were obtained from an equal number of other individuals who visited the health care centre but did not experience the symptoms of the gastrointestinal illness associated with the outbreak. Examination of these latter samples enabled us to assess the presence of possible pathogens in the general population.

Sera were obtained 1–2 weeks after the onset of illness from 91 individuals, after 4 weeks from 86 individuals, after 8 weeks from 68 individuals and after 4–5 months from 72 individuals. When the first serum sample was drawn, the date of the onset of the illness was noted. The outbreak was also assessed by a questionnaire, which was sent to all 3085 residents in the community. Faecal specimens from 33 ill persons (Table 1, groups I and II) and from 33 controls (Table 1, groups III and V) were inoculated onto Skirrow's medium. The plates were incubated in a microaerophilic atmosphere at 42 °C for 48 h. Gram-negative curved rods with typical morphology and motility that were oxidase and catalase positive and resistant to cephalothin were identified as *Campylobacter jejuni/coli*. All strains isolated were susceptible to nalidixic acid.

The serotyping assays were performed using the indirect haemagglutination technique for heat-stable (HS) antigens as described by Penner et al. [10] using 65 polyclonal HS sera. The direct slide

Table 1 Characteristics of 88 patients studied in the acute phase of the *Campylobacter jejuni* epidemic in Skjervøy

Group[a]	No. of patients[b]	Culture result	Illness present	Mean age (years)	Percent seropositive
I	9	positive	yes	44.9	100
II	24	negative	yes	36.9	46
III	1	positive	no	88.0	100
IV	2	ND	yes	46.0	50
V	32	negative	no	44.3	27
VI	20	ND	no	37.8	15

[a] See text for details
[b] First sample
ND, culture not done

agglutination technique for detection of heat-labile (HL) antigens was performed with whole, live bacteria as described by Lior et al. [11] using adsorbed polyclonal HL antisera for 122 reference strains. DNA fingerprinting was done according to Lind et al. [12]. Strains were grown overnight on agar plates containing horse blood. The bacteria were harvested in saline and centrifuged and the pellet resuspended in Tris-EDTA, lysozyme, sodium dodecyl sulfate and pronase as described previously [12]. Phenol and chloroform were used for DNA extraction; DNA was harvested by ethanol precipitation. Different restriction endonucleases were used. *Hin*dIII and BSU 36 I gave the best DNA pattern. The serological study was performed using a diffusion-in-gel (DIG)-enzyme immunoassay (EIA) that employs an ethanol-inactivated multireacting (HS 6, 7) strain from the same region as the target antigen [13]. The sensitivity of the assay ranges from 77 to 86%, depending on whether the patient resides in the northern or southern part of Norway [13]. Cases were defined as positive when zone sizes exceeded the mean ± 2 SD observed in healthy blood donors.

Results and Discussion

The characteristics of the outbreak are presented in Figure 1 and Table 1. Single cases occurred in July 1988, but the majority of cases occurred during the weekend of August 19–21. Questionnaires were returned from 520 (17%) of the 3085 inhabitants to whom they were sent. Seventy-seven (15%) of the 520 respondents, all consumers of the common drinking water supply, became ill. Among those who reported using only boiled tap water during the epidemic, no illness was recorded. All age groups were affected. Diarrhoea, fever, abdominal pain, nausea, joint swelling or joint pain was reported in 81%, 29%, 30%, 43%, 21% and 9%, respectively. We estimate that 330 of 2200 persons became ill during the epidemic. This attack rate is much lower than that observed during an earlier epidemic in the same region of Norway [7] and is also lower than the attack rate observed during a waterborne outbreak in Sweden [2].

Each individual was allocated to one of the groups listed in Table 1. Thus, from 9 of 33 (groups I and II) patients, *Campylobacter jejuni/coli* was isolated from stool samples, compared to 1 of 33 healthy persons (groups III and V). All strains isolated were identified as serotype HS7 (heat-stable antigen) and serotype HL5 (heat-labile antigen). The DNA restriction enzyme cleavage pattern obtained using *Hin*dIII revealed an identical pattern of fragments obtained after digestion of the DNA from the strains isolated during the epidemic. These strains were distinct from strains involved in single cases observed elsewhere [12].

All culture-positive patients ($n=9$) were positive in acute-stage sera (specific IgG, IgM or IgA). Twenty-seven percent of the culture-negative healthy controls ($n=33$) and 46% of the culture-negative patients ($n=24$) were seropositive. Acute sera had comparable antibody levels, i.e. mean zones in millimetres observed

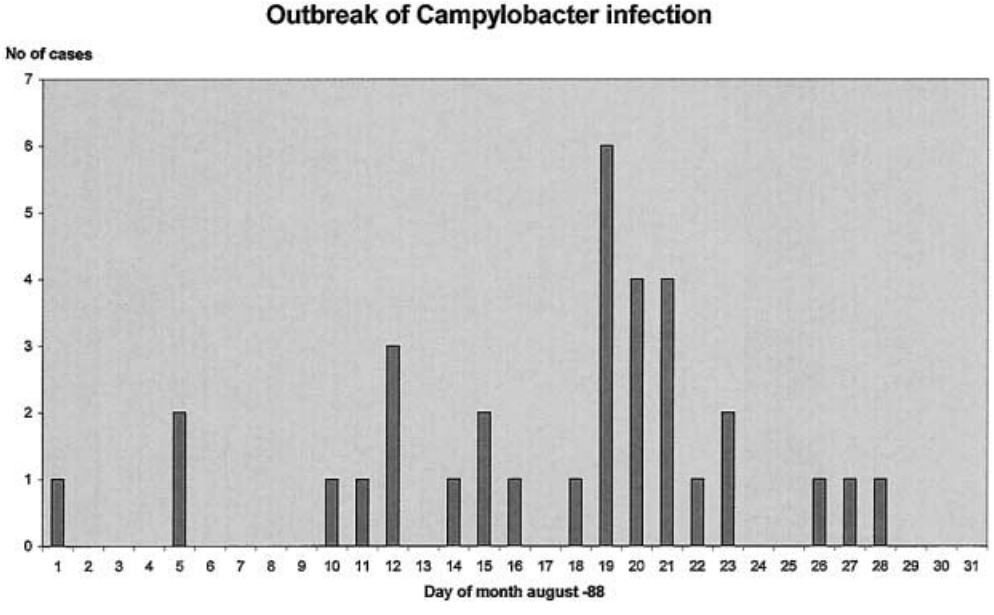

Figure 1 Onset of diarrhoea reported by 33 patients at the first visit during the Skjervøy epidemic

in the DIG-EIA system, against the outbreak strain or the multireacting strain. The immune response was most pronounced in ill patients, in whom specific antibody levels decreased during the observation period (data not shown). Two cases of reactive arthritis occurred. In both cases, the patients were seropositive for either IgA or IgG antibodies. This figure (2/330) is higher than that observed in the previous epidemic [7]. Differences in population and the strain involved are possible explanations. The disease most likely affected more than those who were culture positive, since many culture-negative and antibody-positive cases were observed.

In contrast to the findings in the previous outbreak in the region [7], no significant difference in the level of IgG antibodies in acute sera was observed whether using the local strain (HS7:HL5) or the multireacting strain (serotype HS 6, 7) as target in the DIG-EIA system. The multireacting strain represents a common isolate from the region and shares antigens with the outbreak strain. PEN O:7 and PEN O:6,7 have been shown to be two of the leading causes of outbreaks in the region [14]. The lower attack rate in this outbreak may indicate that some immunity may prevail in the community, which might explain the reduced number of cases in this setting in contrast to the former outbreak [7]. Low numbers of *Campylobacter* in the water also may have contributed to the low attack rate in the present outbreak. The lack of isolation of the microbe from drinking water during the outbreak adds support to this explanation. Culture-positive symptomatic patients had a serological response to the *Campylobacter* antigen. This finding may also account for the asymptomatic seropositive individuals that were observed. Several culture-negative patients had antibodies to *Campylobacter* spp. Faecal samples were obtained several days after the onset of illness. This may explain the low recovery rate of *Campylobacter* spp. from our patients. The finding of an immune response in ill patients who were culture negative lends support to this hypothesis. Furthermore, the low recovery rate may also suggest additional unknown factors that provoke this illness in the community. Guillain-Barré syndrome, reported to be associated with different O serotypes [15], did not occur.

As the illness occurred only among consumers of unboiled tap water, we believe that the temporary lack of water chlorination may explain the occurrence of this presumably waterborne *Campylobacter* epidemic. This epidemic has shown that strict regimens regarding the handling of drinking water are essential in order to prevent this type of illness. When examining such an epidemic, various tools should be applied. For typing the infecting organism, serotyping combined with DNA typing is useful [12]. In order to determine the number of cases involved, both typing and serological testing, used in conjunction with a well-designed questionnaire, are required.

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