Increased stool sampling during a waterborne outbreak of cryptosporidiosis does not increase the detection of other faecal pathogens

A O Qamruddin, M G L Keaney, R McCann, P R Chadwick

Aims: To ascertain the effect of enhanced surveillance following an outbreak of waterborne cryptosporidiosis on the number of faecal specimens submitted to the local microbiology laboratory and the number positive for common enteric pathogens. The outbreak provided an opportunity to estimate the extent of routine under ascertainment of common enteric pathogens.

Method: Retrospective search of the computerised microbiology system database for details of faecal examination requests for the period 26 April to 6 June 1998 and 1999 (period of outbreak).

Results: Specimens were received from 378 community patients during the six week period 26 April to 6 June 1999. This was double that for the same period in 1998 [a non-outbreak year]. Oocysts of Cryptosporidium parvum were detected in 59 patients, an eightfold increase compared with the same period in 1998. Despite the greater number of patients tested, the detection of other pathogens in patients with gastroenteritis was not altered when compared with the same period in the previous year.

Conclusion: This study found no evidence of under ascertainment of gastrointestinal infection (common bacterial pathogens and rotavirus) by local general practitioners.

Cryptosporidiosis is caused by the protozoan Cryptosporidium parvum of which there are human and animal associated genotypes. Infection in the immunocompetent individual is characteristically a self limiting diarrhoea, which may take up to a few weeks to resolve. However, in immunocompromised individuals, infection may not be cleared and can result in severe intractable diarrhoea, with associated morbidity and mortality. Transmission occurs by animal to human (zoonotic infection), person to person, and waterborne routes.

An outbreak of cryptosporidiosis occurred in the north west of England in 1999. This followed the detection on the 23 April of 30 cryptosporidium oocysts (normal, < 2) in a routine weekly 10 litre grab sample taken on the 20 April from the raw inlet water for the Dunmail Raise water treatment works. This plant supplies water to many areas in the north west of England, including Salford (which receives almost 100% Thirlmere aqueduct water). The population of Salford is approximately 240 000.

“...in immunocompromised individuals, infection may not be cleared and can result in severe intractable diarrhoea, with associated morbidity and mortality.”

Following an outbreak meeting on 26 April it was decided not to issue a general “boil water” notice because any contaminated water would have already passed through the water distribution system and daily water samples for cryptosporidium oocysts from the 23 April onwards had been within normal limits. A press statement was released and a letter was sent to all general practitioners in Salford, requesting them to send faecal specimens from patients with suspected infective diarrhoea.

A sharp increase in laboratory confirmed cases of cryptosporidiosis was noted in subsequent weeks and affected mainly four health authority areas: Salford and Trafford, Wigan and Bolton, South Lancashire, and Northwest Lancashire. The outbreak subsided by the first week of June 1999.
for details of faecal examination requests from Salford residents for the period 26 April to 6 June in 1998 and in 1999. Data were analysed on a patient basis rather than on a specimen basis (that is, multiple samples from the same patients, of which there were 21 and three during the period 26 April to 6 June in 1999 and in 1998, respectively, were treated as a single request). Statistical analysis included calculation of the 95% confidence intervals for the percentage difference in positive isolates between the two time periods. Illness onset dates for cryptosporidium positive individuals were obtained from the local consultant in communicable disease control (CCDC; RM).

RESULTS

Figure 1 shows illness onset dates for cryptosporidium positive individuals between 26 April and 6 June 1999. The graph shows two peaks, one around the 24 April and the other around the 13 May. Table 1 provides a summary of the number of community patients positive for enteric pathogens from 1998 to 1999 for the period 26 April to 6 June.

Some general practitioners serving Salford patients use microbiology laboratory services other than the Hope Hospital microbiology laboratory. Of all Salford residents notified to the CCDC with laboratory confirmed cryptosporidiosis between 26 April and 6 June 1999, 77% had been tested at Hope Hospital. Our data therefore refer to most, but not all, of the Salford population.
DISCUSSION
This outbreak of cryptosporidiosis was confirmed by both the absolute and proportionate (relative to the total number of patients tested for enteric pathogens) rises in the numbers of cryptosporidium positive patients, compared with the same period the previous year. Despite a twofold increase in the total number of patients tested during the outbreak period, there was no proportionate increase in the numbers of patients positive for other routinely tested enteric pathogens. The rise in the number of patients tested during the outbreak period is more than can be accounted for by all the patients testing positive for the routinely tested pathogens. These excess patients may represent patients with cryptosporidiosis (or other recognised pathogens) testing laboratory negative, patients with non-infective causes of diarrhoea, or patients with viral gastroenteritis (for example, Norwalk-like virus, adenovirus, calicivirus). However, following the enhanced surveillance request and press publicity, the rise was probably the result of a lower threshold for individuals with diarrhoea (regardless of cause) seeking medical advice and a lower threshold for requesting faeces examination by general practitioners.

"The rise in the number of patients tested during the outbreak period is more than can be accounted for by all the patients testing positive for the routinely tested pathogens"

General practitioners were not given explicit advice for obtaining a faecal specimen but were asked to sample patients with suspected infective diarrhoea. It is accepted that there is wide variation in faecal sampling between general practitioners. It was not possible to determine the number of patients presenting with symptoms of gastroenteritis to all general practitioners during the two periods. This would have required a separate study and manual retrieval of data in many practices. One possible explanation for the absence of an increase in the isolation of other pathogens is that local general practitioners are correctly sampling patients who have gastrointestinal infection (common bacterial pathogens and rotavirus) by local general practitioners. The rise in the number of patients tested in 1999 compared with the same period in 1998 (a non-outbreak year) and there was an eightfold increase in the number of patients positive for Cryptosporidium parvum

• Double the number of specimens were received in 1999

• Therefore, there was no evidence of under ascertainment of gastrointestinal infection (common bacterial pathogens and rotavirus) by local general practitioners

Table 2 Microbiology results, with age breakdown, on general practice patients from whom faeces specimens were received during the period 26 April to 6 June, 1998 and 1999

<table>
<thead>
<tr>
<th>Age (in years)</th>
<th>Population of Salford as estimated at 1/1/99</th>
<th>Patients positive for cryptosporidium 26-6-6/6</th>
<th>Patients positive for other tested pathogens 26/4-6/6</th>
<th>Patients negative for all tested faecal pathogens 26/4-6/6</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-4</td>
<td>14224</td>
<td>1</td>
<td>12</td>
<td>35</td>
</tr>
<tr>
<td>5-9</td>
<td>16285</td>
<td>2</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>10-14</td>
<td>15541</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>15-19</td>
<td>16008</td>
<td>2</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>20-24</td>
<td>17601</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>25-29</td>
<td>19190</td>
<td>0</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>30-34</td>
<td>189680</td>
<td>0</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>35-39</td>
<td>17929</td>
<td>0</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>40-44</td>
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<td>0</td>
<td>0</td>
<td>4</td>
</tr>
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<td>45-49</td>
<td>13754</td>
<td>0</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>50-54</td>
<td>14426</td>
<td>0</td>
<td>1</td>
<td>12</td>
</tr>
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<td>11998</td>
<td>0</td>
<td>2</td>
<td>12</td>
</tr>
<tr>
<td>60-64</td>
<td>11238</td>
<td>1</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>65-69</td>
<td>10130</td>
<td>0</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>70-74</td>
<td>9162</td>
<td>0</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>75+</td>
<td>17225</td>
<td>1</td>
<td>0</td>
<td>13</td>
</tr>
<tr>
<td>Not known</td>
<td>--</td>
<td>0</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>Totals</td>
<td>239274</td>
<td>7</td>
<td>59</td>
<td>292</td>
</tr>
</tbody>
</table>
reported to local and national surveillance networks than individuals with viral gastroenteritis.\textsuperscript{4,5} In one of these studies the ratio of cases in the community to cases reported to national surveillance was calculated.\textsuperscript{4} This ratio was lower for bacteria (salmonella, 3.2 : 1) than for viruses (rotavirus, 35 : 1).

However, our study is not directly comparable with these because our objective was to determine whether enhanced surveillance (as a result of an outbreak) picked up additional cases of gastrointestinal infection and not to determine the background incidence of gastrointestinal infection in the community. We acknowledge, however, that our study has limitations because of the small sample size.

A coincidental community wide outbreak of diarrhoea caused by pathogens other than those routinely tested (such as Norwalk-like viruses) cannot be ruled out, but seems unlikely.

**ACKNOWLEDGEMENT**
The authors gratefully acknowledge the help of R Philip and C Bryan in gathering data for this paper.

**REFERENCES**


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