Water-borne outbreak of viral gastroenteritis and Sonne dysentery

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Outbreaks of water-borne gastroenteritis and dysentery are relatively rare in Britain, yet many of the smaller water supplies in which water polluted with sewage is purified for domestic consumption are vulnerable to a breakdown in the purification process. An example of such a breakdown occurred at Montrose, a seaside town (population 10,800) situated 30 miles to the north-east of Dundee (population 185,000) in the County of Angus, Scotland.

THE OUTBREAK

History

On the evening of 18 August 1966 the general practitioners in Montrose received many calls to attend patients suffering from vomiting or diarrhoea or both these symptoms. Adults and children over 1 year of age were affected throughout the town and the cases were confined mainly to the town. A measure of the prevalence of the illness is seen in the fact that half of the night staff of a large canning factory failed to attend for work. On the following morning (19 August) one-quarter of the schoolchildren attending the three main schools were absent. The major wave of new calls to practitioners occurred on 19 and 20 August. There were no deaths in the outbreak. In one practice (population 4000) the number of new patients with diarrhoea or vomiting seen on the 6 days preceding the outbreak (13–18 August) were 0, 0, 3, 0, 1 and 2, and the numbers on the days 19–23 August were 49, 30, 3, 12 and 1.

It is estimated that 40–50% of the population of Montrose was affected. In forty families visited routinely by health visitors on 22 August, 44 (27 adults and 17 children) out of 109 persons had been ill. In September, 553 food handlers were interviewed and 37% gave a history of having had gastro-enteritis in the period 18–21 August and a further 8% reported having been ill between 22 and 27 August.
Clinical features

The symptoms were colicky abdominal pain, nausea, vomiting, headache, general malaise and diarrhoea. Many patients had limb pains and a few were pyrexial. Two or three collapsed, with transient loss of consciousness. In those becoming ill during the first 24 hr. of the outbreak the predominant symptom was projectile vomiting, e.g. five or six episodes in as many hours, and one or two loose stools were passed during or just after the vomiting. In patients becoming ill later in the outbreak, diarrhoea was the principal feature and there was little or no vomiting. Epigastric tenderness was present in all patients seen by one practitioner. Blood and mucus were present in the faeces of only a few patients.

Treatment of most patients was with a sedative kaolin mixture, of which 32 gallons were sold in Montrose between 19 and 26 August. Many were also treated with tetracycline, but without obviously better results. Most patients showed full clinical recovery in 7–10 days and relapses were common.

Epidemiological investigations

In view of the simultaneous onset of the illness in large numbers of unrelated persons in the town, milk, food-stuffs and water were considered as possible vehicles of infection. The investigations of the early cases did not incriminate any of the three milk suppliers or any particular foodstuff, since all the suppliers of milk and foodstuffs distributed also to areas outside Montrose in which no cases occurred.

The widespread distribution of the cases led to the water supply being suspected as the vehicle of infection. Cases of the illness occurred throughout Montrose and in certain premises outside Montrose which were supplied with water from the Montrose main. Gastroenteritis did not occur, except in a few individuals who had visited Montrose, among the inhabitants of the nearby villages of Hillside and Ferryden, which depend on Montrose for food and milk but have a different water supply. No cases occurred in Sunnyside Royal Hospital, 2 miles from Montrose, which receives water drawn from the same supply as Montrose but chlorinated in a separate plant.

The Montrose water supply is filtered and chlorinated. The water is drawn from the River North Esk at Kinnaber and pumped to the water works near Sunnyside Royal Hospital. This river is polluted with sewage effluents from several villages along its course, Edzell Aerodrome and Stracathro Hospital. The water is treated with aluminium sulphate to precipitate organic matter and the pH adjusted with sodium carbonate. It is then subjected to mechanical pressure filtration through a battery of Bell’s filters, chlorinated, normally to 0·4 parts per million, and held in a tank for 3–10 hr. before being distributed. Bacteriological results in the months preceding August had shown this treatment was adequate.

On 19 August, within 24 hr. of the start of the outbreak, the amount of free chlorine in a sample taken from a tap in the Burgh office was less than 0·1 p.p.m. and instructions were issued for the dose of chlorine to be increased to 1 p.p.m.
Samples of water taken at 2 p.m. and 2.30 p.m. on the same day from taps at two other points in the town were graded unsatisfactory on bacteriological examination; their confirmed *Escherichia coli* counts were > 180 and 90 per 100 ml. water. Samples taken on the following days were satisfactory, reflecting the effect of the increased dosage of chlorine.

The chlorination plant was examined some days later and a defect was found in the automatic switching gear which changes the supply of chlorine from an emptied cylinder to a standby full one. This defect allowed some chlorine to pass from the full to the empty cylinder, so that the whole of the calculated dose of chlorine did not pass into the water. No information could be obtained to show exactly when the breakdown took place but the unsatisfactory bacteriological findings on the water samples collected on 19 August demonstrated that the purification process had been inadequate for a period before the time of testing. In addition to the breakdown of chlorination, spates had occurred on 6, 10 and 14 August and the flood waters may have contributed to the failure of the process.

It was necessary to exclude, as a cause of the gastroenteritis, contamination of the water supply with pesticides and herbicides which had been sprayed on the agricultural land on the banks of the River North Esk just before the outbreak. Samples of canned vegetables known to contain water distributed on 16–19 August were tested in the laboratory of the Government Chemist for contamination with pesticides and herbicides but the tests were negative.

**Bacteriological and virological investigations**

Specimens of faeces were cultured on deoxycholate citrate agar plates and in tetrathionate broth and strains of *Shigella sonnei* were identified by standard biochemical and serological techniques. Antibiotic sensitivity tests were done with 'high level' Oxoid sensitivity disks and strains were considered to be sensitive if there was a zone of inhibition that extended more than 2 mm. beyond the edge of the disk. Colicine typing of strains of *Sh. sonnei* was kindly done by Dr R. R. Gillies, Bacteriology Department, University of Edinburgh.

For isolation of viruses the specimens of faeces were treated with penicillin and streptomycin and inoculated into secondary monkey kidney tissue cultures and primary human thyroid tissue culture. The thyroid cultures were prepared and maintained as described by Duncan (1960, 1961). The methods of isolation and identification of the viruses were as described by Weir, Jamieson & Green (1964). Coxackie B antisera were from Burroughs Wellcome and Co., and the echovirus type 30 antiserum was kindly supplied by Prof. N. R. Grist and Dr Eleanor J. Bell, Regional Virus Laboratory, Ruchill Hospital, Glasgow.

**RESULTS**

**Bacteriological results**

Between 19 August and 19 September specimens of faeces were collected from 201 patients in Montrose who had vomiting or diarrhoea, 25 healthy convalescents who had a history of symptoms at the time of the outbreak, and 36 healthy
contacts of patients. *Sh. sonnei* was isolated from 98 of the 201 patients (49%), 4 of the 25 convalescents (16%) and 8 of the 36 contacts (22%). Antibiotic sensitivity tests and colicine typing were done on 56 of the strains, and all 56 showed the same antibiotic sensitivity pattern and colicine type (type 4). The organisms were sensitive to chloramphenicol, nitrofurantoin and tetracycline, and resistant to ampicillin, kanamycin, neomycin, paromomycin, streptomycin and sulphonamide. The only variations were seen in 10 strains which showed borderline zones of inhibition with ampicillin disks, and one strain which was resistant to tetracycline. This antibiotic sensitivity pattern had not been seen previously in any of the 216 strains of *Sh. sonnei* tested from amongst those isolated in Montrose, Dundee or elsewhere in the County of Angus in the 2 years preceding the outbreak. Out of 195 strains isolated in Angus before the outbreak and colicine-typed by Dr R. R. Gillies only 6, isolated from patients in Montrose (1 patient 1964), Brechin (2 patients, 1964, 1964), and Dundee (3 patients, 1963, 1963, 1965), were colicine type 4. These 6 strains had a different antibiotic sensitivity pattern from that of the Montrose strain, being sensitive to ampicillin, kanamycin, neomycin and paromomycin, to which the Montrose strains were resistant. Moreover, these 6 strains did not produce the colicine active only against Abbott and Shannon’s indicator strain 17 (DRL 10033) that is characteristic of the Montrose strains (personal communication from Mrs Janette Whyte.) Thus the strains from the Montrose outbreak formed a homogeneous group, which differed in several characters from all strains previously found in the region. The findings are consistent with the view that the Montrose strains originated from a common source and that the parent strain was a recent importation from another region and did not originate by variation from a resident strain. By courtesy of Dr R. R. Gillies we obtained a collection of *Sh. sonnei* strains of colicine type 4 that had been isolated in Glasgow and Leeds in 1965 and 1966. Some of the Leeds strains had the same antibiotic sensitivity pattern and colicine activity as the Montrose strains, so that at least one source is known from which the Montrose strain could have been derived. Moore swabs (Moore, 1948) put in the river at the inlet of the Montrose water supply on several occasions after the outbreak did not yield *Sh. sonnei*.

In the period 13–29 September, i.e. 3–5 weeks after the outbreak, specimens of faeces were examined from 504 healthy individuals who lived or worked in Montrose and whose occupation involved the handling of food. Only three strains of *Sh. sonnei* were isolated and these strains belonged to colicine type 4 and showed the same pattern of antibiotic sensitivities as the strains from patients in the outbreak. The three carriers had presumably been infected at the time of the outbreak. Apart from this finding no further isolations of *Sh. sonnei* were made in Montrose. In April 1967, thirty specimens of faeces from patients with symptoms of gastroenteritis were examined and neither *Sh. sonnei* nor viruses were isolated from any of them. At the same time, four Moore swabs taken from the main Montrose sewers were negative for *Sh. sonnei*. The strain of *Sh. sonnei* that caused the outbreak apparently failed to establish itself in chronic carriers and disappeared from the town within a few months.
The ‘Montrose’ strain of *Sh. sonnei* did, however, spread from Montrose to other towns in Angus, namely Edzell, Brechin and Dundee, and it established itself for a period of at least several months as an endemic strain in Dundee, a town having a population eighteen times greater than that of Montrose. Out of 139 strains of *Sh. sonnei* isolated from patients in Dundee in the 6 months October 1966–March 1967, twenty-two were colicine type 4 and had the Montrose pattern of antibiotic sensitivities. Nine of the Montrose-type strains were isolated in a day nursery for pre-school children, which experienced a small outbreak of dysentery due to this organism. Throughout the whole period, however, the predominant strain of *Sh. sonnei* in Dundee was a colicine-untypable organism that did not show the Montrose pattern of antibiotic sensitivities. This strain has been the main *Sh. sonnei* strain in Dundee for at least 3 years.

In March 1967, a patient in Dundee yielded a strain of *Sh. sonnei* with the Montrose pattern of antibiotic sensitivities which unexpectedly was found to be colicine-untypable, and in the period March–May 1967 a further forty-five strains with the same characters were obtained in Dundee. Also in this period, seven strains of another new variety of *Sh. sonnei* were isolated from patients in Dundee; these were of colicine type 4 but they differed from the Montrose type-4 strain in being sensitive to kanamycin, neomycin and paromomycin. Type-4 strains with the Montrose pattern of antibiotic sensitivities have not been isolated since March 1967, and the organism seems now to have disappeared from Dundee. The two new varieties of *Sh. sonnei* just mentioned may be importations into the region, but it also seems possible that they have been derived by variation from the Montrose organism, the one by a loss of colicine production, the other by a loss of resistance to kanamycin, neomycin and paromomycin.

**Virological results**

Specimens for virus isolation were obtained from 32 patients in Montrose who first became ill between 10 and 29 August; 3 of these patients became ill before the outbreak (10, 16, 17 August); 18 were involved in the main outbreak (19–21 August) and 11 in the following week.

Sixteen (50%) of the patients were found to be excreting viruses and 7 (21%) to be excreting *Sh. sonnei* (Table 1). Eighteen of the patients were under 20 years of age and the 7 isolations of *Sh. sonnei* were obtained in this group together with 7 of the virus isolations.

The first case was a woman who became ill on 10 August with a febrile illness with diarrhoea, 8 days before the outbreak began. A specimen of faeces taken 3 weeks later at a time when the diarrhoea was still continuing yielded Coxsackie virus type B5. The cases taking ill with a feverish illness on 16 and 17 August were twins aged 21 months; they developed diarrhoea on the following day. The faeces of both contained Coxsackie virus type B6 when they were examined three weeks later. These three cases occurred before 18 August and indicate that Coxsackie viruses types B5 and B6 were already in the community when the outbreak took place.

Faeces from eighteen cases who were taken ill between 18 and 21 August were
examined for viruses and the specimens yielded four echoviruses type 30, four Coxsackie viruses type B6 and three strains of *Sh. sonnei*. Specimens from eleven patients becoming ill on 24–29 August yielded one strain each of Coxsackie viruses type B5 and B6, three strains of echovirus type 30, and four of *Sh. sonnei*. Two patients yielded both *Sh. sonnei* and echovirus type 30 from the same specimen of faeces.

Table 1. *Date of onset of illness and isolations of viruses and Shigella sonnei from thirty-two patients whose faeces were examined for both bacteria and viruses*  

<table>
<thead>
<tr>
<th>Number of patients yielding stated pathogen from faeces, who first became ill on the following days of August</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pathogen isolated</td>
</tr>
<tr>
<td>Echovirus type 30</td>
</tr>
<tr>
<td>Coxsackie virus B5</td>
</tr>
<tr>
<td>Coxsackie virus B6</td>
</tr>
<tr>
<td><em>Sh. sonnei</em></td>
</tr>
<tr>
<td>None of above</td>
</tr>
</tbody>
</table>

Two patients on 27 August had both echovirus type 30 and *Sh. sonnei* in their faeces. The first day of the outbreak was 18 August.

Table 2. *Principal symptoms correlated with isolation of pathogens in thirty-two patients from whom faecal specimens were submitted for examination*  

<table>
<thead>
<tr>
<th>Principal symptoms</th>
<th>Number of cases</th>
<th>Number of isolations of a pathogenic organism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastroenteritis</td>
<td>14</td>
<td>4 <em>Sh. sonnei</em></td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>10</td>
<td>5 Coxsackie virus B6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 Coxsackie virus B5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2 Echovirus type 30</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 <em>Sh. sonnei</em></td>
</tr>
<tr>
<td>General malaise and enteritis</td>
<td>4</td>
<td>2 Echovirus type 30</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 Coxsackie virus B5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2 Coxsackie virus B6</td>
</tr>
<tr>
<td>Blood and mucus</td>
<td>2</td>
<td>2 Echovirus type 30 \double <em>Sh. sonnei</em></td>
</tr>
<tr>
<td>Headache and dizziness</td>
<td>2</td>
<td>1 Echovirus type 30</td>
</tr>
</tbody>
</table>

Table 2 shows the relation between the organisms isolated from the thirty-two cases and the clinical diagnoses. The strains of *Sh. sonnei* were found in the patients with gastroenteritis and those having blood and mucus in the stools. Most of the Coxsackie viruses were associated with diarrhoeal illnesses. The echovirus type 30 strains were associated with enteritis illnesses except for one isolated from a boy with dizziness and fever, suggesting a meningitic type of illness.

Attempts to isolate viruses from Moore swabs taken from the river near the water supply inlet were not successful.
DISCUSSION

This explosive outbreak of gastroenteritis that within two days probably affected about 4000 persons in a town of 10,800 inhabitants was almost certainly due to water-borne infection. The reasons for this conclusion are: (1) the agreement between the distribution of cases and that of the main water supply to the town, (2) the absence of a relationship between the distribution of cases and the distribution of food and milk supplies, (3) the demonstration of a fault in the equipment used to chlorinate the water supply, (4) the demonstration that the water reaching the town at the time of the outbreak contained an inadequate amount of residual chlorine, and (5) the demonstration that the water reaching the town at the time of the outbreak contained large numbers of viable Escherichia coli organisms and, therefore, that there had been a failure in its purification.

The nature of the water-borne pathogens is not fully known but Shigella sonnei was almost certainly the cause of about half of the cases since it was isolated from the faeces of 49% of patients subjected to bacteriological examination. Possibly Sh. sonnei caused the illness in a further proportion of cases, but was not isolated in these cases because of the technical limitation inherent in the single bacterial culture attempted. The outbreak could be attributed wholly to Sh. sonnei if it were assumed that the proportion of missed cases was 51%, but we consider it unlikely that the efficiency of our cultural methods was as low as that.

On the other hand, our isolation of viruses from half the faeces examined virologically suggests that a proportion of the illnesses in the outbreak may have been caused by water-borne viruses. Since the purification process was defective and the water supply known to be heavily polluted with Esch. coli organisms, it is very probable that enteroviruses were also present. Although two Coxsackie B viruses were present in the community before 18 August, the echovirus type 30 infections appear to have started along with the Sh. sonnei infections on the 18th August and echovirus type 30 accounted for seven out of the 16 virus isolations.

Outbreaks of water-borne Sh. sonnei have not occurred often in Great Britain. Green & Macleod (1943) described an explosive epidemic among 400 persons in a town of 10,000 inhabitants. Sh. sonnei was isolated from some patients and also from a laboratory tap. The tap sample was satisfactory in other respects by usual bacteriological tests. The cause of the epidemic was inadequate chlorination of deep well water. The symptoms were similar to those in Montrose, i.e. prostration, sickness and vomiting, colic and diarrhoea.

Ross & Gillespie (1952) reported an outbreak of water-borne gastroenteritis affecting employees in a factory; three specimens of stool were examined from sixty-six patients, Sh. sonnei being isolated from nine patients, Salmonella typhimurium from one patient and Shigella flexneri from one patient. Sh. sonnei was later isolated from river water. The majority of cases were thought to be due to ‘non-specific’ causes as no other pathogens were demonstrated. The cause of the outbreak was the contamination of the town water mains by crude river water through a cross-connexion in the factory between two pipes, one carrying river water at 240 lb. pressure, the other carrying town water at 25–30 lb. pressure.

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Murchison (1966) described an outbreak of *Sh. sonnei* dysentery in a village community in Scotland in which 25–30% of people developed dysentery. Water from a well contaminated by flood-borne excreta played a part in the spread of the disease.

In the United States, Freitag (1960) reported a sudden and extensive outbreak of dysentery due to *Sh. sonnei* in Ravena in Albany County; 45–50% of the population had a clinical illness, there being about 1400 cases in a population of 3200 inhabitants. The outbreak was due to heavy pollution of the water supply with *Sh. sonnei* and a breakdown in the treatment of the water, a period of time from a few hours to a day elapsing during which no chlorine and possibly no alum was added to the raw water.

An outbreak of Sonne dysentery in a town in the Ukraine was reported by Sorvina in 1946; sewage contamination of a piped water supply was probably the cause but *Sh. sonnei* was not demonstrated in the water. Dordević, Sokolovski & Miladinović (1965) described four water-borne epidemics of dysentery that took place in 1962–64 in one unit of a military garrison. The morbidity rates varied from 126-4 to 312-4 per 1000 population. One outbreak was due to *Sh. sonnei* the others to *Sh. flexneri* type Z or *Sh. schmitzii*.

There is evidence that enterovirus infection may be water-borne. Lamb, Chin & Scarce (1964) tested 164 weekly samples of sewage and river water collected from July to November 1962. Eighty-one (49%) of samples were found to contain viruses. Raw sewage yielded the highest frequency of positive samples (80%); 52% of effluent samples were positive. About one-quarter of the river water samples contained viruses, Group-B Coxsackie viruses and polio-viruses being most commonly found. Enteroviruses can survive in river water for at least 1 mile from the outfall. Since enteroviruses apparently can survive in water and in sewage, it is obvious that they can be distributed by an inadequately chlorinated water supply and it is possible that the Coxsackie virus B6 and echovirus type 30 isolated in Montrose had been distributed in this way.

The conditions of filtration and chlorination required to render water free from infective virus are not precisely known. There is evidence that the enteroviruses require a higher dose of chlorine and a longer holding time than is required to kill the bacteria observed as a guide to cleanliness of water. Kelly & Sanderson (1958) showed that complete elimination of enteroviruses was not achieved by the usual conditions for bacterial disinfection of water supplies, i.e. free residual chlorine concentration of 0.2 p.p.m. for 10 min. contact at pH 7. A longer period of contact was necessary. Isherwood (1965) has shown that the inactivation of Coxsackie virus type A13 required a combination of activated sludge treatment using a very high sludge volume index in excess of 700 ml./l. followed by sand filtration and chlorination to a residual level of 0.5 p.p.m. with a contact time of 8 hr. McLean & Brown (1966) have shown that currently used methods of filtration do not remove viruses and that high-level chlorination is required; with about 0.8 p.p.m. residual chlorine it was found that small amounts of polio-virus type 1 were rapidly inactivated in running bath water.

The ability of the types of viruses isolated in Montrose to cause an illness
characterized by acute vomiting and diarrhoea is not fully established. However, in a localized outbreak in Italy, Felici *et al.* (1962) recovered Coxsackie virus B3 from 33% of infants and children with summer diarrhoea and from 12% of healthy contacts. A break in the sewage system appeared to be the primary cause in this outbreak. Presumably the Coxsackie B5 and B6 viruses isolated in Montrose were capable of giving the same type of clinical illness. It has not been suggested previously that water-borne echovirus type 30 might give rise to an intestinal type of illness. Irvine, Irvine & Gardner (1967) have described an outbreak in a general practice in which echovirus type 30 was isolated from a majority of cases in which the principal symptoms included headache, malaise, muscle pain, vomiting and abdominal pain. These symptoms are more of a ‘gastro-intestinal’ type than those usually associated with this virus and are similar to the symptoms found in some of the cases in Montrose.

In this outbreak the infecting agents were *Sh. sonnei* and three enteroviruses. None of the agents isolated produced life-endangering illnesses, but where *Sh. sonnei* and these three enteroviruses can go, so can *Salmonella typhi*, poliovirus type 1, infectious hepatitis virus, *Entamoeba histolytica* and other agents producing severe disease. It is clear, therefore, that more thought and attention must be given to the purification of water supplies drawn from sources liable to pollution with sewage. The points of importance would appear to be adequate filtration, adequate chlorination and adequate holding time after chlorination. Supervision of the purification process should be adequate to guard against the possibility of accidental breakdowns, and this may be difficult to achieve in small water-supply undertakings. The observations in the literature indicate that a higher residual chlorine level than is used for bacteriological disinfection is required to render viruses inactive. If mechanical filters are used in place of the more efficient biological (slow sand) filters, it is particularly important that high levels of chlorine should be used.

**SUMMARY**

1. An explosive outbreak of gastroenteritis that affected 40–50% of people in a town of 10,800 inhabitants (Montrose) is described.

2. There is epidemiological evidence that the outbreak was water-borne. The chlorination of the water supply was faulty at the time of the outbreak.

3. Echovirus type 30 and two types of Coxsackie B viruses were isolated from sixteen out of thirty-two patients examined.

4. *Shigella sonnei* was isolated from the faeces of 110 out of 262 patients and contacts examined. Fifty-six strains tested for colicine activity were all colicine type 4 and had the same antibiotic sensitivity pattern.

5. This particular strain of *Shigella sonnei* (Montrose strain) spread to surrounding areas, although it disappeared quickly from Montrose.

6. The problem of inactivating virus in water supplies contaminated with sewage is discussed briefly.

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