An outbreak of food-borne gastroenteritis in two hospitals associated with a Norwalk-like virus

By J. V. S. PETHER

Public Health Laboratory, Taunton & Somerset Hospitals, Musgrove Park Branch, Taunton, Somerset

AND E. O. CAUL

Public Health Laboratory, Myrtle Road, Kingsdown, Bristol

(Received 10 January 1983; accepted 21 March 1983)

SUMMARY

Two connected outbreaks of gastroenteritis in separate hospitals associated with a small round structured virus morphologically indistinguishable from the Norwalk virus are described. The virus was most probably introduced on chicken sandwiches prepared by a member of the kitchen staff who was incubating the disease.

INTRODUCTION

Outbreaks of diarrhoea and vomiting occur frequently in hospitals. It is now well documented that such outbreaks in paediatric units are usually due to rotaviruses (Middleton, Szymanski & Petri, 1977). In adult wards outbreaks are generally less severe, but they are short and apparently highly infectious and often spread to staff. Bacterial agents are only occasionally involved. This paper describes an outbreak in an orthopaedic ward that was unfortunately transferred to a geriatric ward. The agent most probably responsible was a small round structured virus (SRSV) (Caul & Appleton, 1982) morphologically resembling the Norwalk agent (Kapikian *et al.* 1972).

Specimens

MATERIALS AND METHODS

(a) Orthopaedic ward. Stool specimens were obtained from 26 members of staff and patients who were or had been suffering from gastrointestinal symptoms. Specimens were not obtained from the remaining 6 people who were ill. Thirteen of these 26 specimens were obtained within 24 h of the onset of symptoms. The remainder (13) were obtained either before the onset of symptoms or 1-4 days after recovery. Eighteen stool specimens were collected during the outbreak from patients and staff who were not ill and these specimens served as controls.

(b) Geriatric ward. Six stool specimens were obtained from patients and staff suffering from gastrointestinal symptoms. Stool samples from the remaining 15 patients and staff were not obtained. Only three of these six specimens were obtained during the acute stage of the infection, the remainder being obtained 24 h after recovery. Eight stool specimens were obtained from patients and staff who were not ill and these specimens were used as controls.

Bacteriological investigations

All stool samples were examined for the presence of Salmonella, Shigella, Campylobacter and Clostridium species.

Virus isolation

344

Faecal emulsions were inoculated onto secondary baboon kidney cells, MRC5 (human fibroblasts), Hep-2 and Bristol HeLa cells.

Electron microscopy

Faecal extracts were prepared as 10 % suspensions in Earle's saline containing 0.5 % lactalbumen hydrolysate and clarified by low speed centrifugation at 3000 r.p.m. for 30 min. Virus particles were concentrated by precipitation with ammonium sulphate and negatively stained with 1.5 % phosphotungstic acid pH 6.5 as previously described (Caul, Ashley & Egglestone, 1978). All preparations were examined using an AEI 801 electron microscope.

RESULTS

Epidemiological investigations

The orthopaedic ward. The outbreak of gastroenteritis occurred in one of the four wards of an old (1809) voluntary aided hospital in Taunton, Somerset, England. This ward can accommodate up to 33 patients, mostly female. The hospital has recently been extensively modernized but the wards still resemble the Nightingale pattern. They are clean with high ceilings and the standard of nursing and medical care is high. The ages of patients ranged from the early twenties to 94 years.

The geriatric ward. This ward is one of four wards at an older hospital situated about half a mile from the first hospital. In spite of some modernization the washing and toilet facilities remain inadequate and cramped and, to some extent, this frustrates the excellent nursing and medical care. This ward can accommodate up to 26 patients for rehabilitation. There was no staff interchange between the two hospitals and each was served by a separate kitchen.

The outbreak. During the night of 12 September 1979 seven patients, one doctor, a nurse and a member of the evening domestic staff, all associated with the orthopaedic ward, developed diarrhoea and vomiting (Fig. 1). There was a strong association with the consumption of chicken sandwiches which was the only food prepared and eaten on the evening of 11 September. The illness seemed too sudden in onset to have a bacterial cause and faecal specimens collected on 13 September were taken to the Regional PHLS laboratory in Bristol. The ward was closed to further admissions on the 13 September. Between 13 and 16 September eight further patients, four members of the medical staff and eight nurses developed diarrhoea and vomiting. The outbreak ended with a nurse falling ill on 19 September and another doctor on 21 September; he in turn probably transmitted the infection to his wife, as she became ill on 26 September. Unfortunately, before



Fig. 1. The occurrence of cases of gastroenteritis in staff (\square) and patients (\blacksquare) in the orthopaedic ward.



Fig. 2. The occurrence of cases of gastroenteritis in staff (\Box) and patients (\blacksquare) in the geriatric ward.

the seriousness of the outbreak was realized one patient was moved on the 12 September to the geriatric ward where she developed diarrhoea as soon as she arrived. On 16 September, two nurses and two patients in the geriatric ward developed diarrhoea and subsequently a further sixteen cases occurred (Fig. 2). The last case occurred on 26 September. The outbreak in the geriatric ward could not be fully investigated but some information could be gleaned from it. Towards the end of the outbreak in the geriatric ward a visiting female hairdresser became ill on 28 September and transmitted the infection to her husband. He was one of the few cases who had chest pain (see below) before, in turn, developing diarrhoea and vomiting on 30 September.

The source. The baby of the only member of the kitchen staff on duty on the evening of 11 September had had diarrhoea and vomiting on 10 September. His $4\frac{1}{2}$ -year-old brother had a similar illness on 11 September and the father, having prepared no other food but sandwiches on the evening of the same day, was off sick on 12 September. It cannot be definitely determined that he was symptom free on the evening of 11 September and it was unfortunate that we failed to obtain a faecal specimen from any member of this family nor any other member of the

kitchen staff. Three other members of the kitchen staff were away ill with diarrhoea on 8, 11 and 13 September and the original source of the infection is most likely to have been one of these people. Enquiries concerning the food consumed by the first wave of affected patients and staff during the 48 h prior to illness revealed that only the consumption of chicken sandwiches on the evening of 11 September was closely associated with the subsequent occurrence of gastroenteritis. Almost all patients ate the sandwiches as did some of the medical and nursing staff on duty that evening. It was clearly remembered that the doctor, the nurse and the domestic who were ill on 12 September had eaten the sandwiches provided the previous evening. Further enquiries revealed that the first batch of the chicken sandwiches had been delivered to the affected ward on the evening of 11 September, subsequent batches going to the other wards. There must have been a decreasing and variable faecal contamination of sequential batches of sandwiches which would have caused the preponderance of affected persons in the ward that received the first batch.

There were, in fact, two other cases of illness, both nurses, from the other wards in the orthopaedic hospital. The presumption must be that later batches of sandwiches contained individually a lesser dose of virus or the nurses had acquired infection from the community where outbreaks of clinically similar illness were occurring.

The illness. Usually the illness was short starting with nausea followed by projectile vomiting, to be followed after 4 h by abdominal cramp and diarrhoea. It was noticeable, however, that the course of the illness was more protracted in the geriatric ward both in the patients and staff than in the orthopaedic ward. A few persons were feverish but most had recovered after 24–48 h when previously affected patients were continent and seen to be sitting up and eating breakfast with a healthy appetite. Two nurses were off sick for one day, came back to work for an hour and a working day respectively, but then had a recurrence of the diarrhoea that necessitated their absence for a further day. Two members of staff reported that they had chest pain which preceded the diarrhoea and vomiting and, as previously mentioned, the husband of the hairdresser also had chest pain. The incubation period of the illness in the orthopaedic ward was difficult to determine. It is impossible to decide whether those who became ill in the ward between 13 and 16 September had acquired the virus from those ill on the 12 September or whether the dose of the virus determined the incubation period. However, apart from the single transferred person, patients in the geriatric hospital had not eaten chicken sandwiches and the incubation period in these cases seemed to be 48 h or less. Likewise two nurses returned from holiday and fell ill 48 h and 72 h afterwards. Several general practitioners in Taunton (population 50000 persons) subsequently confirmed that there was an outbreak of a short acute gastrointestinal illness in the town.

Microbiological observations

Orthopaedic ward. Small round structured viruses (SRSV) measuring 32–34 nm in diameter (Fig. 2) were detected in the faeces of nine affected patients by electron microscopy. Comparative studies demonstrated a morphological resemblance with Norwalk virus (Caul, Ashley & Pether, 1979). SRSV could be detected in nine of 26



Figure 3. Norwalk like small round structured virus (SRSV) from patients with gastroenteritis (A–C). Small round featureless virus (SRFV) present in symptomatic and asymptomatic patients (D). Bar represents 100 nm.

patients from whom faecal specimens were obtained (36%). Two patients were excreting a small round featureless virus (SRFV, parvovirus-like) measuring approximately 22 nm in diameter (Plate 1).

Geriatric ward. SRSVs indistinguishable from those detected in the orthopaedic ward outbreak were seen in two of six stool specimens examined. SRFV particles were seen in the faeces of one patient.

Controls. Virus particles resembling the Norwalk group of viruses were not detected by electron microscopy in any of the 26 control specimens from patients and staff who were not ill collected during the course of the outbreak. SRFVs were seen in one control specimen.

Neither bacterial nor viral pathogens could be cultivated from any specimen.

DISCUSSION

It seems reasonable to assume that the small round structured virus (Norwalk-like or SRSV) detected by electron microscopy was originally transmitted passively on the chicken sandwiches prepared by a member of the kitchen staff who, probably, was beginning to excrete virus. It was unfortunate that an accurate history of those who had eaten the sandwiches and the few who may not have eaten them was not obtained. It was concluded that all the patients and the majority of the staff on duty on the evening of 11 September in the orthopaedic ward had eaten the sandwiches. Certainly the members of staff who were ill had partaken. It was demonstrated that virus was associated only with those patients and staff who were clinically ill and we failed to demonstrate virus in the examined stools of three patients and staff who were not ill. Patients and staff who did not develop gastroenteritis in the initial wave may have eaten sandwiches that either, by chance, had no contaminating virus, were contaminated with a dose of virus that was insufficient to infect, or were immune from previous exposure to an antigenically related virus. Subsequent cases in members of staff in the orthopaedic ward, where the incubation period appeared to be 24-48 h, were probably due to faecal-oral spread from the patients, although others have demonstrated the presence of virus in vomit (Greenberg, Wyatt & Kapikian, 1979). In the geriatric ward where the source of infection was quite clear, successive crops of cases most probably resulted from cross infection by the faecal-oral route. It is possible that meticulous hand-washing and attention to personal hygiene would have helped to minimize the outbreak but in a crowded geriatric ward with inadequate facilities this is impracticable.

Outbreaks similar to that described in this paper are inevitable with such a highly infectious virus which, on clinical evidence, seems to have been widespread in the community. The clinical presentation of the illness accords with that of previous authors but we did notice that three cases had quite severe chest pain. No electrocardiography was performed nor were the levels of serum enzymes studied but it may be worthwhile to examine such cases in future outbreaks.

Griffin *et al.* (1982) described an outbreak of gastroenteritis due to the Norwalk virus that was epidemiologically associated with the consumption of a green salad at a luncheon banquet. They noted that one of the two employees who had prepared the green salad became ill the next day. There was subsequently serological evidence of infection by the Norwalk virus in this employee but not his colleague who had remained well. Unfortunately, in our outbreak it was not possible to obtain faecal or blood specimens from the person who had prepared the chicken sandwiches. However, no other food was prepared by him and it is most likely that the sandwiches were the passive vehicle that spread the virus. Oshiro *et al.* (1981) described an outbreak of acute non-bacterial gastroenteritis among the elderly patients in a convalescent home in North California which persisted for 14 months. The responsible agent was shown to be serologically unrelated to the Norwalk virus. They did not discover how this virus was introduced into the convalescent home.

Oysters (Murphy et al. 1979), contaminated drinking water supply (Kaplan et al. 1982) and swimming in sewage-contaminated water (Baron et al. 1982) have

been incriminated in the transmission of small round structured viruses (Norwalklike or SRSV) which have been shown to constitute a separate group of viruses (Caul & Appleton, 1982) from the small round featureless viruses (parvovirus-like or SRFV) recently incriminated in an outbreak associated with the consumption of cockles (Appleton & Periera, 1977). It is unusual and usually impossible to identify a specific item of food in the spread of enteric viruses (Gunn et al. 1980). Unfortunately, there appears no way to prevent such outbreaks other than meticulous hand-washing by those handling food. The number of virus particles that constitute an infectious dose is not known and, by analogy with bacteria, it is possible that the old and immunologically compromised patient will become ill from a smaller dose than that which would infect people outside hospitals. It is possible that those patients and staff who were never affected and whom we were thus able to use as controls may either not have had a dose high enough to produce disease, not been a secondary faecal oral contact or had become immune from a previous episode. Epidemiological evidence from the outbreak described in this paper suggests that the chef who prepared the sandwiches was instrumental in the transmission of the agent. If kitchen staff were forbidden to work merely because someone in their family or one of their colleagues was ill, it would soon lead to staff shortages.

In this study we detected two morphologically distinct small round viruses. In three patients a SRFV measuring approximately 22 nm was observed which was indistinguishable from the 'Wollan' agent originally described by Paver *et al.* (1973). This virus was not shown to cause gastroenteritis in the original studies and no evidence was obtained in the present study to support an aetiological role. Dual infections with agents morphologically similar to SRFV and SRSV have been reported previously (Murphy *et al.* 1979; Grohmann *et al.* 1980; Caul & Appleton, 1982). It was notable that in the present study SRFV's were seen in a control specimen but Norwalk-like particles were never seen in controls.

The second virus detected in 11 patients in our study was a SRSV measuring approximately 32-34 nm which has been shown to be morphologically indistinguishable from the Norwalk group of viruses (Caul, Ashley & Pether, 1979). Much evidence exists that this group of viruses cause gastroenteritis (Kapikian *et al.* 1982) and it was considered to be the cause of the outbreak in our study. Thornhill *et al.* 1975 suggest that viruses in this group are shed in profusion while the patient is ill, but for only a short time afterwards. Negative results on further specimens collected at 1, 2 and 4 days respectively from three patients support this view, although more prolonged excretion has been documented by us in other outbreaks (unpublished observations). It is apparent, however, from our studies on this outbreak and our observations with similar outbreaks that electron microscopy is not the ideal means for the detection of this group of viruses. More sensitive methods are needed and in this respect the radio-immune assay techniques described by Kapikian *et al.* (1982) is being developed in our laboratory to study this important group of enteric viruses.

REFERENCES

- APPLETON, H. & PERIERA, M. S. (1977). A possible virus aetiology in outbreaks of food-poisoning from cockles. *Lancet* i, 780–781.
- BARON, R. C., MURPHY, F. D., GREENBERG, H. B., DAVIS, C. E., BREGMAN, D. J., GARY, G. W., HUGHES, J. M. & SCHONBERGER, L. B. (1982). Norwalk gastrointestinal illness. American Journal of Epidemiology 115, 163-172.
- CAUL, E. O. & APPLETON, H. (1982). The electron microscopical and physical characteristics of small round human fecal viruses: an interim scheme for classification. Journal of Medical Virology 9, 257-265.
- CAUL, E. O., ASHLEY, C. R. & EGGLESTONE, S. I. (1978). An improved method for the routine identification of faecal viruses using ammonium sulphate precipitation. *FEMS Microbiology Letters* 4, 1–4.
- CAUL, E. O., ASHLEY, C. & PETHER, J. V. S. (1979). 'Norwalk'-like particles in epidemic gastroenteritis in the U.K. *Lancet* ii, 1292.
- GREENBERG, H. B., WYATT, R. G. & KAPIKIAN, A. Z. (1979). Norwalk virus in vomitus. Lancet i, 55.
- GRIFFIN, M. R., SUROWIEC, J. J., MCCLOSKEY, D. I., CAPUANO, B., PIERZYNSKI, B., QUINN, M., WOJNARSKI, R., PARKIN, W. E., GREENBERG, H. & GARY, G. W. (1982). Foodborne Norwalk virus. American Journal of Epidemiology 115, 178–184.
- GROHMANN, G. S., GREENBERG, H. B., WELCH, B. M. & MURPHY, A. M. (1980). Oysterassociated gastroenteritis in Australia: the detection of Norwalk virus and its antibody by immune electronmicroscopy and radioimmunoassay. *Journal of Medical Virology* 6, 11–19.
- GUNN, R. A., TERRANOVA, W. A., GREENBERG, H. B., YASHUK, J., GARY, G. W., WELLS, J. G., TAYLOR, P. R. & FELDMAN, R. A. (1980). Norwalk virus gastroenteritis aboard a cruise ship: an outbreak on five consecutive cruises. *American Journal of Epidemiology* **112**, 820–827.
- KAPIKIAN, A. Z., GREENBERG, H. B., WYATT, R. G., KALICA, A. R. & CHANOCK, R. M. (1982). The Norwalk group of viruses-agents associated with epidemic viral gastroenteritis. In Virus Infections of the Gastrointestinal Tract (ed Tyrell, D. A. J. and Kapikian, A. Z.), pp. 147–177. New York: Marcell Dekker.
- KAPIKIAN, A. Z., WYATT, R. G., DOLIN, R., THORNHILL, T. S., KALICA, A. R. & CHANOCK, R. M. (1972). Visualisation by immune electron microscopy of a 27 nm particle associated with acute infectious nonbacterial gastroenteritis. *Journal of Virology* 10, 1075–1081.
- KAPLAN, J. E., GOODMAN, R. A., SCHONBERGER, L. B., LIPPY, E. C. & GARY, G. W. (1982). Gastroenteritis due to Norwalk virus: an outbreak associated with a municipal water system. Journal of Infectious Diseases 146, 190–197.
- MIDDLETON, P. J., SZYMANSKI, M. T. & PETRI, M. (1977). Viruses associated with acute gastroenteritis in young children. American Journal of Diseases of Children 131, 733-737.
- MURPHY, A. M., GROHMANN, G. S., CHRISTOPHER, P. J., LOPEZ, W. A., DAVEY, G. R. & MILLSOM, R. H. (1979). An Australia-wide outbreak of gastroenteritis from oysters caused by Norwalk virus. *Medical Journal of Australia* 2, 329–333.
- OSHIRO, L. S., HALEY, C. E., ROBERTO, R. R., RIGGS, J. L., CROUGHAN, M., GREENBERG, H. & KAPIKIAN, A. Z. (1981). A 27 nm virus isolated during an outbreak of acute infectious nonbacterial gastroenteritis in a convalescent hospital: a possible new serotype. Journal of Infectious Diseases 143, 791-795.
- PAVER, W. K., CAUL, E. O., ASHLEY, C. R. & CLARKE, S. K. R. (1973). A small virus in human faeces. Lancet i, 237-240.
- THORNHILL, T. S., KALICA, A. R., WYATT, R. G., KAPIKIAN, A. Z. & CHANOCK, R. M. (1975). Pattern of shedding of the Norwalk particle in stools during experimentally induced gastroenteritis in volunteers as determined by immune electron microscopy. *Journal of Infectious Diseases* 132, 28-34.