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An Investigation into the Possible Role of the Family Unit in the Transmission of Rotavirus Infections of Children

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A survey of young children hospitalized with viral gastroenteritis, and of the members of some of their families, was carried out. Results showed rotavirus to be the principal agent associated with the condition though other types were also detected. In addition, rotavirus was found in the stools of some of the members of each of the families investigated, and it is suggested that means of spread is the faecal–oral route within family groups.

Introduction

Human infantile gastroenteritis caused by rotavirus infection is well documented.[1,2] Although the faecal-oral route is a likely candidate for the way in which the virus is transferred from person to person, in terms of virus shedding little notice has been taken of potential contacts or children and other members of the family with gastroenteritis. The object of the study reported here was to perform a survey of children admitted to hospital with gasteroenteritis and where possible to investigate the family as it is probable that young children may be infected by older siblings and/or their parents.

Methods

Faecal samples were obtained during a 9-month period, October to May, from 20 successive cases of gastroenteritis admitted to one ward of the Children's Hospital, Sunderland. The ages of the children ranged from 2 weeks to 5 years. Samples from patients with gastroenteritis were screened for the absence of bacterial pathogens before virological examination, and control samples were obtained from patients hospitalized for reasons other than suspected infection, for example, treatment for hernia or circumcision. Specimens were coded by a third party, transported from hospital to laboratory in ice, and were stored for not more than 48 hr at 4 °C before processing. Where they were obtainable, samples from members of the patient's family were obtained as soon as confirmation was made that the original infection was viral in origin.

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Screening of the samples was carried out by electron microscopy. Twenty per cent (w/v) suspensions were prepared from each specimen in phosphate-buffered saline (PBS) and mixed gently. These suspensions were then centrifuged for 15 min at low speed (800× g) to sediment gross debris and the supernatants were ultracentrifuged at 12,000×g for 45 min at 4°, the resulting pellet then being washed in PBS and the ultracentrifugation repeated. The pellet from this second spin was resuspended in a small volume (0·1–0·2 ml) of distilled water, one drop mixed with an equal volume of 3·0% phosphotungstic acid, pH 6·0 and transferred to a carbon-formvar coated grid, which was dried and examined in a Siemens Elmiskop 1A electron microscope. When virus particles were present they were usually detected quite quickly, within 4–5 minutes of examining the specimen, although in some instances more prolonged examination of the grid was necessary.

Clinical Features

The 30 children with gastroenteritis comprised 18 (60%) boys and 12 (40%) girls, similar to a survey by Carr, McKendrick & Spyridakis.[3] Seventy per cent of the children were under 6 months of age. All the children were admitted to hospital between the second and fourth day of their illness with a history of diarrhoea. The stools were mainly loose, yellow motions. A few children passed explosive watery stools. Nineteen (63%) children had upper respiratory tract symptoms and/or signs such as snuffles, cough, otitis media or pharyngitis.

None of the children was seriously ill. Only one patient was moderately dehydrated and required intravenous fluid therapy; the remaining 29 patients were treated with soya bean (Velactin) feeds for 48 hours and then infants were given their usual dried milk preparation, whilst older children were recommended on their normal diet. The average length of stay in hospital was seven days.

Results

Of the 30 children with gastroenteritis, 23 (77%) had rotavirus in their stools. One child had coronavirus and another a pathogenic *E. coli*. No viruses or pathogenic bacteria were found in the stools of the remaining five patients.

There were 15 control children, none of whom had diarrhoea and no rotavirus or pathogenic organisms were detected in the stools. However, coronavirus was found in the faeces of one control child. For contact studies, samples of faeces were obtained from members of five families.

In the first family, the father and a 5-year-old brother of the patient, and a sister aged 2 were all found to be shedding rotavirus in large amounts, although father and brother were symptom-free.

The second family comprised the patient aged 3 months, and her mother; both had rotavirus in their stools, whilst a 4-year-old brother was excreting adenovirus.

The third family with rotavirus in the stools consisted of a 2-month-old baby and a symptom-free mother. The fathers in the second and third families showed no rotavirus.

In the fourth family, the patient, a 2-year-old girl, and her two older sisters aged 4 and 6, all had rotavirus in their stools. The two older girls had slight diarrhoea but were not admitted to hospital. Mother and father's stool examinations were negative for rotavirus.

In the fifth family, twins aged nine months were both found to be positive for rotavirus. Whether one twin contracted the infection from the other, or whether both acquired it from a parent is not clear. Specimens from both parents were negative.

Discussion

Infection with rotavirus as a cause of non-bacterial gastroenteritis is now well established, particularly in very young children although other viruses are clearly involved as well.[4,5] In the present study, rotavirus was found in 23 of the 30 patients, an incidence of 77%. This is higher than most reports, e.g. Chrystie *et al.*[6] and Davidson *et al.*[7] both reported 48% infection rate in their studies. However, higher infection rates have been reported in the winter period,[2,8] which was the time during which our study was conducted.

The means whereby rotavirus gastroenteritis is contracted is still not conclusive. The most likely explanation seems to be direct contact from infected to uninfected individuals. Murphy, Albrey & Crewe[9] suggested environmental spread from neonate to neonate in the study of babies in newborn humans. As most clinical reports are of young children with gastroenteritis, direct contact would appear to be important. In 1976 Zissis[10] and his colleagues reported transmission of rotavirus gastroenteritis from a 5-year-old boy to his mother.

Although the present study is not large, we have been able to study potential means of spread of rotavirus within families, a subject which has hitherto been largely a matter of conjecture. In each of the five unrelated families studies, one or more members were found to be shedding rotavirus, though not necessarily showing symptoms of infection. It is therefore quite likely that a parent-to-child or sibling-to-sibling-spread is the principal means by which rotavirus is disseminated.

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