Epidemic gastroenteritis in children associated with rotavirus infection

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Rotavirus was demonstrated by electron microscopic examination in all the ten faecal samples tested from an epidemic of childhood diarrhoea in north Kerala. No bacterial cause could be established for the epidemic. It is therefore postulated that the epidemic may have been due to rotavirus infection. This is believed to be the first report of a large community epidemic of rotavirus diarrhoea.

Gastroenteritis constitutes a major cause of morbidity and mortality in children the world over, particularly in the developing countries in the tropics. In India, diarrhoeal diseases excluding cholera, account for 1-4 million deaths in children annually. The aetiology remains unidentified in the large majority of childhood diarrhoeas. Several studies have shown that causative agents could be established only in about 30 per cent of cases. Although the failure to identify bacterial pathogens in the bulk of childhood diarrhoeas suggested that viruses may be responsible for this condition, attempts to incriminate known viruses had been largely unsuccessful.

In 1973, workers in Melbourne observed a virus with a characteristic morphology by electron microscopy of duodenal biopsy specimens from children with acute diarrhoeas. The same virus was identified in Birmingham by electron microscopic examination of negatively stained preparations of faecal samples from children with diarrhoea. This virus, designated variously as duovirus, orbivirus, rotavirus, reovirus-like agent and infantile gastroenteritis virus, has now been reported from various parts of the world in association with childhood diarrhoeas. It is closely related morphologically and antigenically to viruses that cause epidemic diarrhoea in infant mice and diarrhoeal disease in calves and piglets.

There have been annual epidemics of gastroenteritis in infants and children in north Kerala during the last several years. The disease starts in November, reaches epidemic proportions by December-January and subsides by March. Several thousands of children are affected each year. These epidemics had been investigated by the Department of Microbiology, Calicut and by the National Institute of Communicable Diseases, Delhi. The results had been inconclusive. Miscellaneous bacterial pathogens had been isolated from occasional patients, but none of them could account for the epidemic disease.
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We report here evidence indicating that these epidemics might have been due to rotavirus infection.

Material and Methods

As part of a continuing study of diarrhoeal diseases in children in north Kerala, faecal samples from patients have been examined for established and presumptive bacterial pathogens, parasites and Candida albicans. This report relates to children under 5 years of age admitted to the paediatric wards of the Medical College Hospital, Calicut, with acute gastroenteritis during the period between November, 1975 and March, 1976. Faecal samples were collected using rectal catheters, directly in sterile screw-capped bottles and processed within an hour of collection. The gross appearance of the sample was noted and a microscopic examination done. Cultures were put up on the following media: MacConkey, deoxycholate-citrate agar, alkaline bile salt agar and thiosulphate-citrate-bile-salt-sucrose agar for primary plating, and alkaline peptone water, 7 per cent salt broth and selenite medium for enrichment. Bacterial isolates were identified by standard methods.

For electron microscopy, faecal samples were collected by catheter into pre-chilled screw-capped bottles containing Hanks’ balanced salt solution, transported to the laboratory on ice, homogenised, and after centrifugation at 5000 rpm for 30 minutes at 4°C, the supernatants were stored at —20°C till they were transported under refrigeration to the Christian Medical College Hospital, Vellore. On arrival at Vellore, the specimens were centrifuged at 25000 g. for 90 minutes in a refrigerated centrifuge. The pellets were resuspended in a drop of distilled water. The suspension was then transferred to collection carbon coated grids, allowed to dry and stained with ammonium molybdate. The grids were examined in a Philips electron microscope EM 200.

Results

A total of 110 faecal samples were examined by culture during the 5 months. Bacterial pathogens could be isolated only from 7 samples, i.e. 4 strains of enteropathogenic Escherichia coli (belonging to 3 different serotypes), one strain of Vibrio cholerae el tor and two strains of non-cholera (non-agglutinating) vibrios. Diarrhoeagenic parasites or Candida albicans could not be demonstrated in any specimen.

Ten specimens collected in February 1976, in the latter part of the epidemic were examined by electron microscopy. Rotavirus was present in large numbers in all the ten samples. Electron micrograph of one such specimen is given in (Fig. 1, Plate XXXVI) showing a clump of rotaviruses, with the typical morphology of the complete virion having both inner and outer layers of capsids. In some specimens, both complete and incomplete virions were observed (Fig. 2, Plate XXXVI).

After the epidemic subsided, 6 faecal samples collected from children with diarrhoea, in May 1976 were examined. Rotavirus was not seen in any of these, though two specimens contained adenoviruses.
Fig. 1. Electron micrograph of faeces pellet, showing a large clump of complete rotavirus particles with the double-rim capsid structure (Magnification 1 cm.=100 nm).

Fig. 2. Electron micrograph of faeces pellet, showing rotavirus particles without the outer rim, and cylindrical capsid protein (Magnification 1 cm.=100 nm).
Patients in whose faeces rotavirus was found, ranged in age from 7 months to 4 years. Seven of them were males and 3 females. All of them were admitted with diarrhoea and vomiting. On admission, dehydration was severe in two children, moderate in 4, mild in 3 and not apparent in one. All were discharged after having been cured, the duration of hospitalisation ranging from 2 to 6 days. The faeces in all cases was watery, yellow or yellowish green in colour, with white flakes, and having an offensive odour. No blood or mucus was present in any sample. The faecal samples did not yield any other pathogen. The clinical features of these cases are summarised in the Table.

**Table. Clinical features of patients with rotavirus infection.**

<table>
<thead>
<tr>
<th>Serial No.</th>
<th>Age in years</th>
<th>Sex</th>
<th>Degree of dehydration</th>
<th>Duration of stay in hospital</th>
</tr>
</thead>
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<tr>
<td>D. 223</td>
<td>7/12</td>
<td>F</td>
<td>Severe</td>
<td>4</td>
</tr>
<tr>
<td>D. 228</td>
<td>8/12</td>
<td>M</td>
<td>Mild</td>
<td>4</td>
</tr>
<tr>
<td>D. 229</td>
<td>10/12</td>
<td>M</td>
<td>Nil</td>
<td>4</td>
</tr>
<tr>
<td>D. 230</td>
<td>18/12</td>
<td>M</td>
<td>Severe</td>
<td>5</td>
</tr>
<tr>
<td>D. 231</td>
<td>18/12</td>
<td>M</td>
<td>Mild</td>
<td>2</td>
</tr>
<tr>
<td>D. 233</td>
<td>8/12</td>
<td>F</td>
<td>Moderate</td>
<td>4</td>
</tr>
<tr>
<td>D. 235</td>
<td>1</td>
<td>F</td>
<td>Moderate</td>
<td>6</td>
</tr>
<tr>
<td>D. 236</td>
<td>10/12</td>
<td>M</td>
<td>Mild</td>
<td>2</td>
</tr>
<tr>
<td>D. 238</td>
<td>18/12</td>
<td>M</td>
<td>Moderate</td>
<td>6</td>
</tr>
<tr>
<td>D. 241</td>
<td>4</td>
<td>M</td>
<td>Moderate</td>
<td>2</td>
</tr>
</tbody>
</table>

**Discussion**

The failure to establish a bacterial cause during the last 5 years and the presence of rotavirus in all ten samples tested during the epidemic indicate that the annual epidemics of gastroenteritis in infants and children in north Kerala could be attributed to rotavirus infection. Studies in Australia and England have shown that rotavirus infection occurs mainly in the winter months, when as much as 75 per cent of childhood diarrhoeas may be caused by the virus. North Kerala does not have a noticeable winter, but the epidemic season i.e. November to March, represents the coolest part of the year.

Rotavirus infection associated with childhood diarrhoea has now been documented from different parts of the world, including one report from India. Such reports have mostly been of sporadic childhood diarrhoeas. There have also been reports of outbreaks of diarrhoea in closed communities such as hospitals and nurseries. The importance of the virus in epidemic diarrhoea has not been established. The present report is believed to be the first instance of an epidemic of childhood diarrhoea in a large open community which could be related to rotavirus infection.
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The demonstration that epidemic childhood diarrhoea in the tropics may be associated with rotavirus infection has an important bearing on the treatment of the disease. Though antibiotics are not indicated in non-invasive diarrhoeas, it is the usual practice in this area to administer broad spectrum antibiotics orally for these cases. Evidence that many of these cases may be of viral origin further emphasises the futility of antibiotic therapy of childhood diarrhoeas. Indiscriminate antibiotic consumption during diarrhoea epidemics may lead to the emergence and spread of multiple-drug resistant bacteria. The sudden appearance of chloramphenicol-resistant typhoid bacilli in north Kerala in 1972 is believed to be related to the widespread use of an oral chloramphenicol preparation during an epidemic of gastroenteritis\(^2\).

All cases in this series in whom rotavirus infection was demonstrated, presented a reasonably uniform clinical picture, similar to that described in an earlier report\(^3\). With further study, it may be possible to obtain guidelines for the definition and clinical diagnosis of rotavirus diarrhoea.

Acknowledgment

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References

