Comment

Callaghan² first reported the restless legs syndrome in uraemia and noted its exacerbation with dialysis. Ekbom had earlier noted the association with iron deficiency,³ and it has also been reported in folate deficiency of pregnancy.⁴ Treatment other than oral iron or folate where indicated was, until Matthews's report,¹ unrewarding. The patients reported here were not deficient in iron, folate, or other vitamins. The aetiology of this curious syndrome is not known, though interestingly uraemic myoclonus, thought to be due to disturbed function of the brain-stem reticular formation, also responds to clonazepam.⁵ The promptness with which clonazepam alone of the benzodiazepines tried suppressed the symptoms was remarkable. We believe that the preliminary results are encouraging enough to warrant a long-term controlled trial.

¹ Matthews WB. Treatment of the restless legs syndrome with clonazepam. *Br Med J* 1979;i:751.

² Callaghan N. Restless legs syndrome in uraemic neuropathy. *Neurology* (Minneapolis) 1966;16:359-61.

³ Ekbom KA. Restless legs syndrome. *Neurology* (Minneapolis) 1960;10:868-73.

⁴ Botez M, Lambert B. Folate deficiency and restless legs in pregnancy. *N Engl J Med* 1977;297:670.

⁵ Chadwick D, French AT. Uraemic myoclonus: an example of reticular reflex myoclonus? *J Neurol Neurosurg Psychiatry* 1979;42:52-5.

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Outbreak of rotavirus gastroenteritis among premature infants

Though rotavirus causes severe gastroenteritis predominantly in infants and young children, it may infect any age group. In neonates rotavirus infection is usually subclinical,^{1 2} and even in premature babies the infection is reportedly³ mostly mild. We report an outbreak of rotavirus infection among premature and low-birthweight babies in a special-care ward. In most cases virus excretion was associated with gastroenteritis. Dehydration, weight loss, and growth retardation occurred in several cases.

Patients, methods, and results

From January to May 1980, 513 faecal specimens were collected from 77 premature and low-birthweight babies admitted to a special-care ward of the Bambino Gesù paediatric hospital in Rome. Babies were placed in isolation and kept in incubators during the first three to 10 days of admission. Visitors were not admitted to the ward, and special attention was paid to gowning and handwashing by nursery staff. The duration of admission varied from 12 days to three months (mean 37 days). Faecal specimens for rotavirus were sought on admission and at intervals thereafter. Initially babies were sampled weekly, but when rotavirus was found in the nursery in February the frequency of sampling was increased to twice weekly throughout March, with a return to weekly sampling in April and May.

Faeces were suspended in Hanks's balanced salt solution without antibiotics to give a 20% w/v suspension. This was homogenised for 10 minutes and then centrifuged at 4500 g for one hour. The aqueous layer was then centrifuged at 180 000 g for 90 minutes. The resulting pellet was resuspended in 0.05 ml distilled water. A small amount of the suspension was placed on a carbon-coated electron microscope grid. The dried specimen was negatively stained with 1% phosphotungstic acid, pH 7.4, and examined at a magnification of 40 000 with a Siemens 102 electron microscope.

In February and March rotavirus was found in 18 newborn babies (figure). It was apparent within 10 days of admission in all affected infants, seven of whom were virus-positive within four days of admission. In seven cases several stool samples gave positive results; in four cases virus excretion persisted for 14-24 days after first being detected. Few viruses other than rotavirus were found: adenovirus and parvovirus-like particles were detected in four and two samples respectively.

From January to May diarrhoea lasting three to 16 days occurred in 28 babies. Most cases occurred in February and March, when all the

rotavirus-positive stool samples were detected. At that time 14 out of 22 babies with diarrhoea (64%) and four out of 23 asymptomatic babies (17%) were rotavirus positive ($p < 0.002$). In a few cases rotavirus excretion lasted for several days after diarrhoea stopped (figure). Diarrhoea persisted for three to five days in six cases and six to 16 days in eight. Vomiting occurred

Premature and low-birthweight infants examined for rotavirus excretion by clinical state and duration of admission to hospital.

in most cases and lasted one to three days. Loss of weight was observed in 10 cases. Growth retardation occurred in 12 babies, five failing to show an increase in body weight for five to nine days and seven for 10 to 18 days after rotavirus gastroenteritis. Management was based on rehydration with oral glucose-electrolyte mixture except in two babies, both of whom required intravenous administration of fluids. Milk intolerance after enteritis did not occur.

Comment

Despite the special attention paid to preventing an outbreak of infection, spread of rotavirus occurred in the ward. The prolonged persistence of the virus suggests a mechanism of transmission from baby to baby within the ward rather than repeated introduction of the virus from outside. The clinical manifestation of rotavirus infection varied from mild to severe gastroenteritis in 14 out of 18 infected babies, and in four cases virus excretion persisted for two to four weeks.

We have no data on the serotype(s) concerned in this outbreak and cannot hypothesise on how prematurity and low birth weight influenced the severity of clinical manifestations in babies who had received less passive immunity than full-term neonates. Our findings, however, suggest that rotavirus infection may constitute a considerable problem in premature babies.

¹ Murphy AM, Albrey MB, Crewe EB. Rotavirus infections of neonates. *Lancet* 1977;ii:1149.

² Blacklaw NR, Cukor G. Viral gastroenteritis. *N Engl J Med* 1981;304:397-406.

³ Van Renterghem L, Borre P, Tilleman J. Rotavirus and other viruses in the stool of premature babies. *J Med Virol* 1980;5:137-42.

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