A new *E. coli* O group O158 associated with an outbreak of infantile enteritis

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SYNOPSIS An outbreak of acute diarrhoea occurred amongst babies in a special care baby unit. Thirteen babies were at risk; six suffered diarrhoea and three of these died. An *Escherichia coli* with an unidentifiable O antigen and flagellar antigen H23 was isolated from all six cases. This O antigen has been accepted into the international scheme as O158.

At the reference laboratory it is not uncommon to receive *Escherichia coli* strains which possess O antigens which are not included in the international serotyping scheme. If they are shown to be prevalent or appear to have a causal relationship to human or animal disease they may be established as test strains for new *E. coli* antigens (Ørskov, Ørskov, and Furowicz, 1972).

The Outbreak

The outbreak took place in a special care baby unit consisting of three small wards, but with no cubicles for isolation. The unit admits approximately 200 babies each year from neighbouring maternity units and occasionally admits babies born at home.

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Over the previous two years, mild outbreaks of diarrhoea caused the closure of the unit on several occasions. Some of these outbreaks were due to enteropathogenic *E. coli* belonging to O groups 26, 55, and 126; at the time of the present incident specimens of stool from all babies were routinely examined on the third day after birth and at weekly intervals thereafter.

At the onset of the outbreak the unit housed 13 babies. The first case of diarrhoea occurred on 7 May and was followed by two cases on 8 May and a further three on 9 May (table). Seven babies remained healthy. The six babies with diarrhoea were all born in the hospital. The clinical and epidemiological features of the outbreak suggested that the diarrhoea was infective and that cross infection was occurring within the unit. The unit was closed to

Case No.	Clinical Complications	Date of Birth	Date of Onset of Diarrhoea	First Date <i>E. coli</i> O158 Isolated	Severity of Diarrhoea	Treatment	Result
1	Prematurity (34 weeks) Temporary lactose intolerance	2.5.72	7.5.72	9.5.72	Severe	Intravenous fluids; gentamicin	Recovered
2	Prematurity (34 weeks) Exomphalos repair. Peritonitis with terminal ileus	2.5.72	9.5.72	10.5.72	Severe	Intravenous fluids; gentamicin	Died 18.5.72
3	Prematurity (35 weeks) Caesarian section following failed induction of labour; respiratory distress syndrome	3.5.72	9.5.72	10.5.72	Severe	Intravenous fluids; gentamicin	Initial recovery; relapsed, died 25.5.72
4	Mild respiratory distress syndrome	5.5.72	9.5.72	10.5.72	Mild	Gentamicin	Recovered
5	Prematurity (33 weeks) Caesarian section for toxæmia	5.5.72	8.5.72	9.5.72	Severe	Intravenous fluids; gentamicin	Initial recovery; relapsed, died 20.5.72
6	Caesarian section following failed forceps delivery; second of twins	5.5.72	8.5.72	11.5.72	Mild	Gentamicin	Recovered

Table Clinical features in six cases of enteritis

further admissions and healthy babies were discharged as soon as their clinical condition allowed. All babies in the unit were given gentamicin 3 mg per kg each day for five days.

Clinical Features

The diarrhoea was mild in two patients and severe in four. The four severely affected patients required intravenous fluids and three died. In two of these there was an initial response to treatment followed by relapse (table).

Bacteriology

The investigation of the outbreak included the examination of faecal specimens from all babies in the unit. *Salmonellae* and *Shigellae* were not found and *E. coli* were examined using the limited range of *E. coli* antisera available in the hospital laboratory. This range covered the common enteropathogenic O groups: O26, 55, 86, 111, 112, 114, 124, 125, 126, 127, 128, and 142. Because these O groups were not found representative cultures of *E. coli* from all babies were sent to the Salmonella and Shigella Reference Laboratory, Colindale, for serotyping using the complete serological scheme which covered *E. coli* O1 to O157. An *E. coli* with an unidentifiable O antigen but with flagellar antigen H23 was isolated from the faeces of all six cases and also from throat swabs of two of the fatal cases. A similar *E. coli* was isolated from faeces in five of the seven healthy babies. All strains gave the typical biochemical reactions of *E. coli*. The presence of the H23 antigen suggested that these strains might belong to the same serotype.

Using one of these strains as a vaccine, an O antiserum was produced in rabbits. By agglutination-absorption techniques all 13 isolates were shown to possess the same O antigen. The results suggested that this was a new O antigen and a strain was sent to the International Escherichia Centre, Copenhagen, where the findings were confirmed and the strain designated as *E. coli* O158.H23.

Discussion

The antigenic scheme for serotyping *E. coli* contains over 150 O groups and the scheme remains incomplete with new groups O being added from time to time. Evidence exists that infantile enteritis may be caused by *E. coli* serotypes from about 20 O groups (Taylor, 1961) and some of these have been found to be responsible for outbreaks of infantile enteritis in many countries. Hospital laboratories usually restrict their investigations to a limited range of O groups and in the United Kingdom this range

remained for many years as O26, 55, 86, 111, 119, 125, 126, 127, and 128. As a result of serious outbreaks of infantile enteritis in Manchester in 1968 (Jacobs, Holzel, Wolman, Keen, Miller, Taylor, and Gross, 1970) and in Scotland in 1970 (Rowe and Gross, 1971) this range was extended to include *E. coli* O114 and O142. Recently many laboratories have included O18ac, 44, 112ac, and 124 and this final range forms a good practical compromise.

E. coli O114 and O142 were the causative agents for the Manchester and Scottish outbreaks and these were O groups which already existed in the antigenic scheme. A similar situation existed in the recognition of *E. coli* O91 as the causative agent in an outbreak in Winchester (Hughes, Greaves, and Bettelheim, 1968). The present outbreak differed in that the *E. coli* isolates from 11 babies could not be serotyped using the accepted scheme. However, the recognition by the reference laboratory that they possessed the H23 antigen suggested that a single epidemic strain was involved. These events illustrate the need for hospital laboratories to be supported by a national reference centre capable of undertaking the complete serotyping of *E. coli*.

The inclusion of *E. coli* O158 into the serotyping scheme will allow further surveillance by reference laboratories and an assessment of its prevalence and relationship to disease. In this outbreak the evidence is suggestive, but not conclusive, that *E. coli* O158 may be enteropathogenic; the fact that it was isolated from healthy babies does not preclude this. Until there is a reliable laboratory test, the association of particular serotypes with outbreaks remains an important indication of enteropathogenicity.

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