also nasal carriers. After these cases the importance of diligent hand-washing with chlorhexidine was emphasised and the problem widely discussed. Despite this six further cases occurred.

Comment

Since the widespread outbreaks of staphylococcal disease in nurseries for newborn babies in the early 1950s and early 1970s outbreaks of bullous impetigo are now rarely seen. Their occurrence depends on the phage type and virulence of the prevalent staphylococci, with bullous impetigo usually caused by organisms of phage group II. Infants are usually colonised from their mothers or by contact with nursing and medical staff, and the incidence of colonisation may be reduced by scrupulous hand-washing before and after handling each infant. Antiseptic baths with hexachlorophane have been withdrawn from routine use in nurseries for the newborn as it is now known to be absorbed through intact skin. Detectable concentrations of hexachlorophane have been recovered from brain tissue in very immature infants who were shown to have a spongiform myelinopathy after repeated bathing.¹ The daily use of dusting powder containing 0.33% hexachlorophane may result in a reduction of staphylococcal colonisation² but it is difficult to provide definite evidence that skin preparations are necessary in the newborn.³

Seven different strains of *Staph aureus* were isolated in the outbreak described, and the cases appeared at variable intervals and on several different wards. Rather than being spread from a common source, this was a cluster of cases appearing over a fairly short period. Our experience suggests that staphylococcal bullous impetigo might again create a problem in nurseries for newborn babies.

¹ Anderson JM, Kilshaw BH, Harkness RA, Kelly RW. Spongiform myelinopathy in premature infants. *Br Med J* 1965;iii:175.

² Gillespie WA, Garner BD, Burman D, Alder VG. Antimicrobial applications for prophylaxis in the newborn. *Chemotherapy* 1976;3:361.

³ Davies PA. Treatment of neonatal bacterial infection. In: Valman HB, ed. *Paediatric therapeutics*. Oxford: Blackwell Scientific Publications, 1979: 185.

(Accepted 7 September 1981)

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Pseudomembranous colitis in a 5-week-old infant

Pseudomembranous colitis has been recognised as a rare complication of giving antibiotics. We report a case that occurred in a 5-week-old infant after amoxicillin.

Case report

A 5-week-old Asian boy weighing 3000 g was admitted with a history of fever and refusing feeds. He had had a normal birth at term, weighed 2340 g, and been bottle-fed. Two days before admission he had been prescribed amoxicillin for an upper respiratory tract infection. On admission he was feverish (39.5°C) and irritable but had no localising signs of infection. White cell count was $17.4 \times 10^9/l$ ($17\,400/mm^3$). Cerebrospinal fluid and blood cultures were sterile; nose and throat swabs grew normal flora; and urine culture showed no appreciable growth.

On the third day of illness he developed bloodstained stools with mucus. He was given electrolyte solution by mouth, intravenous ampicillin, and intravenous 0.18% saline in 4% dextrose solution. Repeated stool cultures failed to grow shigella, salmonella, enteropathogenic *Escherichia coli*, or campylobacter and were negative for rotavirus (Rotazyme, Abbott Laboratories Ltd). On the fourth day he developed a complete ileus with greatly distended abdomen, and abdominal x-ray examination showed dilated bowel loops and multiple fluid levels. There was serious electrolyte disturbance (urea 5.0 mmol/l (30 mg/100 ml), sodium 112 mmol(mEq)/l, and potassium 8.6 mmol(mEq)/l), which was resistant to treatment with sodium polystyrene

sulphonate enemas and 0.45% saline in 5% dextrose drip. Oral fluids were stopped and bile aspirated via a nasogastric tube.

By the fifth day the white cell count had risen to $69.6 \times 10^9/l$ ($69\,000/mm^3$) with 88% polymorphonuclear cells and his general condition had deteriorated. Urine output was poor, and 20 ml plasma/kg body weight was given intravenously followed by physiological saline. Gentamicin and hydrocortisone were added. Laparotomy was performed, which excluded a surgically treatable condition: it showed massive generalised straw-coloured ascites and externally the whole colon was thickened, inflamed, and oedematous. After operation he continued to deteriorate despite active management including metronidazole. Appreciable metabolic acidosis, recurrent episodes of hypoglycaemia, and sclerema developed, and he died eight days after the onset of illness.

Postmortem specimen of opened large bowel. Pseudomembrane has been removed on the left to show underlying necrotic mucosa.

At necropsy the small bowel was normal but the entire large-bowel mucosa was replaced by a thick bright yellow membrane that separated easily from the submucosa (figure). Histologically the membrane was a fibrinopurulent exudate replacing the mucosa and often the submucosa. *Clostridium difficile* was isolated from the large-gut contents and neutralisable toxin detected to a titre of 1/1000 in Hep 2 cells.

Comment

Many antibiotics have been associated with pseudomembranous colitis. Larson *et al*¹ suggested that the colitis is caused by *C difficile* and an associated toxin. They found, however, that five of eight healthy neonates had *C difficile* in their stools and two also had toxin. In a retrospective study Rietra *et al*² found a cytotoxic effect in 14% of stool samples from infants aged up to 5 months. Donta *et al*³ described pseudomembranous colitis in a neonate beginning within three days of a course of ampicillin.

A Scandinavian survey⁴ of 218 children aged 2 weeks to 15 years found toxigenic *C difficile* in 43 (20%) but in only two out of 49 children aged under 1 month. We examined the faeces of 35 children aged under 1 month. *C difficile* was isolated from 11 (only two had received antibiotics) and none produced toxin in culture filtrates or the faeces. Thus the exact importance of *C difficile* and its toxin in the bowel of neonates and young infants is still not fully understood.

Our infant was initially well apart from fever but deteriorated steadily after receiving oral amoxicillin. We then used intravenous ampicillin when blood and mucus appeared in the stool, thinking that the infant had dysentery. This calls into question the generous use of antibiotics previously thought to be relatively harmless. Cases of pseudomembranous colitis not associated with antibiotics have been described, and there may be other factors to take into account.

¹ Larson HE, Price AB, Honour P, Borriello SP. *Clostridium difficile* and the aetiology of pseudomembranous colitis. *Lancet* 1978;ii:1063-6.

² Rietra PJGM, Slaterus KW, Zanen HC, Meuwissen SGM. Clostridial toxin in faeces of healthy infants. *Lancet* 1978;ii:319.

³ Donta ST, Stuppy MS, Myers MG. Neonatal antibiotic-associated colitis. *Am J Dis Child* 1981;135:181-2.

⁴ Holst E, Helin I, Mardh P-A. Recovery of *Clostridium difficile* from children. *Scand J Infect Dis* 1981;13:41-5.

(Accepted 8 September 1981)

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