Methicillin resistant *Staphylococcus aureus* in milk

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SUMMARY Three separate outbreaks of gentamicin and methicillin resistant *Staphylococcus aureus* in a special care baby unit are described. The outbreaks ceased only after a milk bank worker was identified as a carrier of the strain. It is postulated that the infant milk feeds served as a vehicle of spread.

Resistance to methicillin among *Staphylococcus aureus* rapidly followed the introduction of the drug in the 1960s. Recently, problems with gentamicin/methicillin resistant *Staph. aureus* have occurred world wide and they are becoming widespread in the United Kingdom. Outbreaks have only occasionally caused problems in neonatal units. An outbreak of multiresistant methicillin resistant *Staph. aureus* (MRSA) that colonised at least 30 newborn babies in our special care baby unit over a period of 16

months in three separate episodes is reported. The problems encountered in eradicating the strain, until the source was identified, are described.

The outbreak and control measures

The Table shows details of the babies who were possibly infected with the MRSA. Initial nasal and umbilical swabs from babies and nasal swabs from staff revealed six colonised and three infected babies and one staff carrier.

A strict admission policy, barrier nursing, and topical treatment with nasal 1% chlorhexidine cream, 0.33% hexachlorophane talc, and daily chlorhexidine baths produced ineffective control as seven further minor clinical infections (cases 4–10) occurred and a further seven babies became colonised.

Similar treatment failed to eliminate the MRSA

Table Details of babies with infection due to methicillin resistant *Staphylococcus aureus*

Case No	Age (days)	Diagnosis	Birth weight (g)	Gestational age (wks)	Date of isolation of MRSA	Site	Previous antibiotics	Clinical notes
1	4	Ambiguous genitalia	2080	34	21.08.83	Umbilicus	None	Umbilical flare
2	12	Hyaline membrane disease	2100	33	17.01.84	Oral suction	None	Pustules in groin
3	2	Extrauterine pregnancy	2180	37	7.09.83	Groin	None	
4	27	Preterm	1150	28	10.09.83	Tongue	None	Mouth coated, spots on neck, spots on waist
5	15	Congenital rubella syndrome	1520	37	13.09.83	Spots	Amoxycillin	Snuffly nose, watery eye
6	8	Spontaneous pneumothorax	3640	38	21.09.83	Spots		
7	19	Small for dates	1330	37	22.09.83	Nose		
8	5	Hyaline membrane disease	1500	31	3.10.83	Eye		
9	22	Transient tachypnoea	2260	36	22.09.83	Umbilicus	None	Moist umbilicus
10	87*	Hyaline membrane disease, subglottic stenosis	1280	31	2.10.83	Nose	None	Snuffly nose
11	6	Small for dates	1900	36	10.10.83	Pustule	None	Pustule on forehead
12	60	Meningoencephalocele	2840	40	17.10.83	Umbilicus	None	Umbilical flare, pustules behind ears, sticky eyes
13	2	Total aganglionosis	2680	40	28.10.83	Eye		
					24.10.83	Groin	Penicillin+gentamicin	Sore groin
					5.12.83	Oropharynx	Ceftazidime	Possible chest infection, snuffly nose, osteomyelitis
					12.12.83	Oropharynx	Penicillin+gentamicin	
					13.01.84	Nose		
					12.12.83	Pustule	None	Pustule in groin, sticky eyes
					13.12.83	Eye		
					21.07.84	Brain swab (postmortem)	None	Group B <i>Streptococcus</i> +coliforms also isolated from brain at post-mortem examination
					1.11.84	Umbilicus	None	Routine swab post-operative wound
					12.11.84	Wound		

MRSA=Methicillin resistant *Staph. aureus*.

*Admitted to the unit at 30 days.

from the staff carrier. Subsequently, fusidic acid cream given nasally eliminated the strain and recolonisation has not occurred.

Case 4 was subsequently admitted to another hospital with possible staphylococcal pneumonia, and MRSA were isolated from his secretions. This first 'serious' infection prompted us to treat all colonised babies with fusidic acid nasal cream as the MRSA was reported to be resistant to other nasal applications. Three weeks later, however, a ventilated baby developed possible osteomyelitis, and oropharyngeal secretions revealed three strains of *Staph. aureus*, which were resistant to fusidic acid, methicillin, and gentamicin, respectively. Treatment with intravenous gentamicin, flucloxacillin, and fusidic acid produced a good clinical result, but the baby remained colonised and subsequently introduced MRSA to another hospital.

The bacitracin resistance was rechecked and the MRSA was found to be sensitive to bacitracin, which was used for nasal carriage and superficial infections. One month later the last remaining colonised baby left the unit and no further isolates were recorded for six months when a postmortem brain swab from a child with a meningoencephalocoele revealed the MRSA strain. Previously adopted measures were reintroduced. One additional colonised baby was identified and after successful treatment discharged.

Three months later a routine specimen of pooled, pasteurised, expressed breast milk yielded MRSA, and investigation of the milk bank staff revealed one nasal carrier. She was removed from the milk bank and given bacitracin nasal cream and 4% chlorhexidine detergent for bathing. This and a subsequent course of pseudomonic acid failed to eradicate the strain from her nose. Concurrently, an outbreak of loose stools revealed three further colonised babies and subsequently one colonised baby developed a postoperative wound infection with MRSA at another hospital.

MRSA has not been isolated in the 24 months after departure of the last colonised baby and removal of the nurse from the milk bank.

All isolates of MRSA were bacteriophage type 75 and tests performed at the Staphylococcal Reference Laboratory, Colindale, showed the strain to be identical to types found in South Africa and distinguishable from that causing problems recently in the midlands and currently in the south east of England.

Discussion

Methicillin/gentamicin resistant *Staph. aureus* have been isolated with increasing frequency in the last

few years. Less than 1% of isolates sent to the Staphylococcal Reference Laboratory before 1978 were methicillin/gentamicin resistant, but this figure had risen to 28% by 1983.¹ There are only a few reports of outbreaks in neonatal units.²⁻⁵

The source of many outbreaks has remained unclear, but staff carriers have been implicated and prolonged carriage by staff may result despite topical treatment.⁶ Our medical staff carrier probably acquired the organism by handling colonised babies as she did not spread the organism to babies in the other hospital she attended. We believe the source of our strain was a midwife in the milk bank who had returned from South Africa one month before the first isolate was detected. While in South Africa she had been briefly admitted to hospital after an accident. She remained undetected for 18 months as she had no direct work or social contact with the unit.

Milk has been widely reported as a vehicle for the spread of organisms, but we are unaware of other reports implicating it as a vehicle of infection with MRSA. The frequency or degree to which the milk bank nurse contaminated feeds remains unknown. The only contact with the positive milk sample was to remove the sample tube from the pasteuriser and check the tightness of the cap. The sample probably became contaminated from the outside of the tube or cap during transit to or on opening in the laboratory.

Feeding of contaminated milk or contact with contaminated bottles or teats presumably led to either oral or gastrointestinal contamination, or both, with MRSA and subsequent skin colonisation. As previously reported colonisation was more common than infection and most infections were minor.²⁻⁴⁻⁵

Prolonged staphylococcal carriage by staff and patients with possible subsequent re-admission or transfer to another hospital highlights the need for close liaison between medical, nursing, laboratory, and control of infection personnel both within and between hospitals if outbreaks are to be detected early and controlled successfully.

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Treatment of campylobacter gastroenteritis

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SUMMARY Twin boys suffered from recurrent diarrhoea due to *Campylobacter jejuni* after entering a day nursery. Stool sampling of all 17 children at the nursery revealed *C. jejuni* in 12. Simultaneous treatment with antibiotics of all children with positive cultures successfully eradicated the infection.

Campylobacter gastroenteritis has been recognised in recent years as a major cause of diarrhoeal diseases in children.^{1,2} It accounts for considerable morbidity and may result in chronic diarrhoea and failure to gain weight.² This organism has also been responsible for outbreaks in day care centres,³ where reinfection is common and eradication difficult.

We present our experience in the management of an outbreak of campylobacter gastroenteritis in a nursery and suggest a practical approach to eradicate the infection.

Case report

Eleven month old twin boys were referred to us eight weeks after entering a day care centre with a history of recurrent diarrhoea. The diarrhoea had begun two weeks after they had started at the centre. It diminished when the mother removed them for periods of several days, only to return on re-admission. The diarrhoea consisted of between four and nine yellowish-green, soft, bulky stools each day for periods of up to two weeks. Their body weight dropped from the 75th to the 25th centile. *Campylobacter jejuni* was cultured from one infant; no other enteric pathogen was isolated.

Erythromycin estolate 50 mg/kg/day was administered to both children for seven days, with complete

resolution of symptoms. Repeated stool cultures for *C. jejuni* after the treatment yielded negative results. Two weeks later, after re-entering the centre, diarrhoea occurred again in both children and *C. jejuni* was again isolated. Clinical and laboratory recovery was again observed after a second course of treatment.

A further episode of diarrhoea occurred 10 days after stopping treatment, and *C. jejuni* was isolated from the stools of both boys. On this occasion, the father, who was a physician, collected stool samples from the other children at the nursery. *C. jejuni* was isolated in the stools of 12 of the 17 children. All these children had suffered from at least one period of diarrhoea during the past two months. By the time of stool sampling, however, eight of the children were already asymptomatic.

All children with positive stool culture were treated simultaneously with a course of erythromycin. A week later stool cultures from these children yielded negative results. From then until the end of the school year (eight months) no further outbreaks of diarrhoea occurred in the centre.

Discussion

Outbreaks of gastroenteritis among children in day care centres are well known.⁴ Close personal contact and poor hygiene in young children, especially in those who are not yet toilet trained, enable the spread of enteric pathogens. The problem of reinfection by the same agent ('ping pong mechanism') is common among these children and can ruin efforts to eradicate the infection.

Recently, attention has been called to the role of *C. jejuni* in infectious diarrhoea in children, including outbreaks in day care centres.^{1,3} Relapses of this infection are documented in our patients, as reported before.^{5,6} The high rate of relapse usually