

Short reports

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Severe echo 19 virus infection in a neonatal unit

Frequent isolations of echo 19 virus were reported in the United Kingdom in the years 1967, 1968 (*British Medical Journal*, 1970), and again in 1974 (*Communicable Disease Report*, 1974). Sporadic isolations only were reported from Birmingham in that year but they increased in 1975 and rose sharply in May to assume the proportions of a minor epidemic (*Communicable Disease Report*, 1975).

Experience in neonatal unit

During the week starting 21 July 1975, between 14 and 22 infants were being nursed in the neonatal unit and between 47 and 57 term infants were being cared for in the lying-in wards of Birmingham Maternity Hospital. On 22 July two preterm infants aged 2 and 4 weeks respectively suddenly became very ill with similar symptoms and signs. They refused feeds, became irritable especially on handling, and had several cyanotic attacks, one case needing endotracheal intubation and assisted ventilation. They were febrile, had a tachycardia, and developed abdominal distension with reduced bowel sounds. The fontanelle tension was

raised so cerebrospinal fluid was examined and blood and urine cultured, and then they were treated as suspected bacterial infection with penicillin and gentamicin. They remained very ill for 3 days and on the fourth made a rapid and uneventful recovery. When 3 more babies became similarly affected, viral studies were made in the belief that echo 19 was likely to be the responsible agent. Over the whole period, 21 July-21 August 1975, 12 infants and 5 staff members became infected (see Fig.).

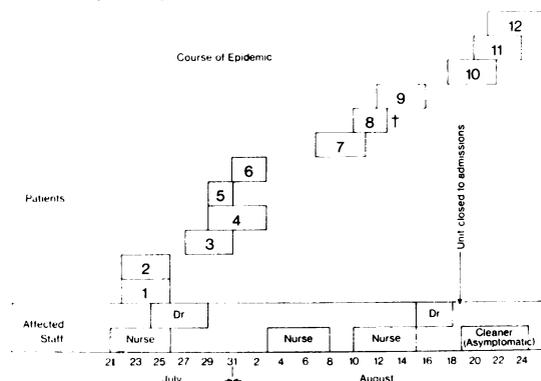


FIG.—Course of epidemic.

TABLE II
Clinical and pathological findings in

Case no.	Sex	Gestation (w)	Birthweight (kg)	Age at onset (d)	Highest recorded	
					Temperature	Pulse
1	M	36	2.23	14	38.2	160
2	M	32	1.55	30	38.0	200
3	F	36	1.98	5	37.0	160
4	M	33	2.24	10	38.4	160
5	M	40	3.28	5	39.0	156
6	M	34	2.00	10	38.0	160
7	M	36	2.04	7	40.0	190
8	M	36	2.4	6	38.5	160
9	F	34	2.1	14	38.0	160
10	F	38	2.21	4	39.0	NR
11	M	39	3.93	2	38.0	140
12	F	36	2.93	7	38.0	NR

NR, not recorded; NT, not tested; T/S, throat swab; +, positive isolation; —, negative isolation.

Of the 12 infants affected, 10 showed cerebral irritability, 7 had marked abdominal distension with reduced bowel sounds, and 5 had apnoeic attacks. 4 had increased fontanelle tension. There were no respiratory symptoms. The other clinical and pathological findings are summarized in the Table. This shows predominance of males and preterm delivery, pyrexial presentation, irrelevance of the peripheral leucocyte count, and despite absence of CSF pleocytosis in all but 3, a 'raised' CSF protein in all those examined. There is, however, a wide range of normal in the newborn period (Bauer, New, and Miller, 1965).

Despite virological screening of all babies and staff in the unit and intensification of barrier nursing techniques and geographical separation with different nursing teams between infected and noninfected babies, further cases occurred and one infant died from whom virus was isolated from brain, lung, and myocardium at necropsy. It was therefore decided to stop admissions to the unit and to direct mothers in preterm labour to neighbouring hospitals.

Virological studies

Virus transport medium was used for washing throat swabs and for preparing homogenates of specimens of faeces; the latter were centrifuged at 5000 rpm for 30 minutes and the supernatant was retained. All specimens were inoculated into HeLa and HEp 2 cell monolayers and incubated as rolling cultures at 35°C. All isolates were identified with a specific antiserum, using microtitre techniques.

The infected staff shown in the Fig. were kept off clinical duties until their throat swabs were negative. The virus was grown from the faeces of the mother of Case 3 but faeces from all other mothers tested proved to be negative. Throat swabs were taken from 176 medical and nursing staff in the rest of the hospital and from none was the virus isolated. 2 term babies in the unit were found on routine screening to have virus growing in their stools but were asymptomatic.

Reopening the unit

The virological findings suggested that the neonatal unit was the source of infection. The remaining 4 infants were discharged, the walls and furniture were cleaned with hypochlorite, and the cubicles fumigated with formaldehyde. After 9 days the unit was reopened and there has been no recurrence. Only staff with negative throat swabs were allowed to return to work. This decision was justified by the detection of echo 19 virus in a prospective member of the nursing staff and the policy was continued until the epidemic in Birmingham declined.

Discussion

Echo 19 virus was not recognized as a fatal illness until Philip and Larson (1973) described 3 neonatal deaths after transplacental infection. This epidemic was postnatally acquired and confined to the neonatal unit except for Case 11. Though it is usually difficult to differentiate between bacterio-

12 infants infected with echo 19 virus

Peripheral white count ($\times 10^9$, polymorphs)	CSF		Echo 19 virus isolation				Clinical illness
	WBC/mm ³	Protein (g/100 ml)	Stool	T/S	CSF	Urine	
9200	No excess	NT	+	-	NT	NT	Severe
P74%	No excess	5.3	+	-	NT	NT	Severe
16 400	No excess	1.55	+	-	NT	NT	Moderate
P54%	34	1.37	+	+	+	+	Severe
7200	No excess	0.92	+	+	+	NT	Severe
NR	No excess	NT	+	+	-	NT	Moderate
15 500	No excess	1.35	+	-	-	NT	Severe
P89%	No excess	1.02	+	+	+	NT	Died on 2nd day
NR	No excess	1.16	+	+	-	+	Moderate
7100	No excess	1.8	+	+	+	+	Mild
P41%	No excess	1.42	+	-	-	NT	Moderate
7200	11	0.84	+	+	+	-	Mild
P36%	No excess						
16 000	No excess						
P56%	69						
NR	No excess						
NR	No excess						
NR	No excess						

logical and virological infection in the newborn, the epidemic form and similarity of clinical features in this outbreak, coupled with the speed and ease of isolation of echo 19 enabled an early recognition of its nature to be established. The predominance of males has previously been noted (*British Medical Journal*, 1970) and in our series preterm delivery resulted in much more serious illness.

In the absence of specific therapy, the decision as to whether or not to close the neonatal unit was finely drawn. At that time 1 of the 10 affected babies had died. This is a similar proportion for the mortality of infants with idiopathic respiratory distress if treated in a suitably staffed and equipped unit (Robertson and Tizard, 1975). To close such a unit would inevitably lead to a rising mortality for small babies requiring intensive care. By temporarily diverting mothers in preterm labour to other hospitals we were not presented with this dilemma for the short period of closure.

Summary

An epidemic of echo 19 virus infection in a neonatal unit affecting 12 babies with one death is described. With one exception it was confined to the neonatal unit and medical and nursing staff were also affected. The unit was closed for 9 days, then was disinfected, and there was no recurrence.

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REFERENCES

- Bauer, C. H., New, M. I., and Miller, J. M. (1965). Cerebrospinal fluid protein values of premature infants. *Journal of Pediatrics*, **66**, 1017.
- British Medical Journal* (1970). *Epidemiology*, **3**, 594.
- Communicable Disease Report* (1974). Public Health Laboratory Service, London.
- Communicable Disease Report* (1975). Public Health Laboratory Service, London.
- Philip, A. G. S., and Larson, E. J. (1973). Overwhelming neonatal infection with echo 19 virus. *Journal of Pediatrics*, **82**, 391.
- Robertson, N. R. C., and Tizard, J. P. M. (1975). Prognosis for infants with idiopathic respiratory distress syndrome. *British Medical Journal*, **3**, 271.

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Organic aciduria

Treatable cause of floppy infant syndrome

The floppy infant syndrome has several causes ranging from disorders of the lower motor neurone and muscle, such as infantile spinal muscular atrophy and congenital myopathy, to various endocrine and metabolic disturbances. These have been fully reviewed by Dubowitz (1969). This report describes a floppy infant in whom hypotonia was due to a rare disorder of organic acid metabolism which responded to treatment with pharmacological doses of the vitamin biotin.

Case report

A girl was the first child of healthy Irish parents, who are second cousins. There have been 3 unexplained childhood deaths in the family. She was born by normal delivery at term after a normal pregnancy, birthweight 3300 g, and was in good condition at birth. There was some initial feeding difficulty and vomiting but by the end of the first week she was feeding well. Development was thought to be normal; by 5 months she was able to sit supported with cushions, roll from supine to prone, make crawling movements, support some weight on her legs and reach out for toys.

When she was 5 months old she developed an upper respiratory tract infection and was treated with ampicillin. She seemed to recover from this infection but the parents noticed that her respirations had become more rapid than before. The mother recalls that at about this time she changed the milk from Cow and Gate 'V' formula to Babymilk 2, representing an increase in protein intake from 1.8 to 3.3 g/100 ml reconstituted milk.

From the age of 8 months there was a gradual onset of floppiness and weakness. She stopped sitting and lost her good head control. She no longer reached out for toys and eventually most of her spontaneous movements ceased so that she lay in her cot, limp and uninterested. She continued to have deep and rapid respirations with inspiratory stridor. Numerous investigations directed mainly toward the respiratory system, including laryngoscopy, bronchoscopy, and tests for avian precipitins, failed to show the cause of her tachypnoea and hypotonia. She was given a trial of steroids for one month on suspicion that she had some form of pulmonary fibrosis, but this had no effect. She was referred to the Hammersmith Hospital at the age of 10 months for further assessment of what was still thought to be primarily a respiratory problem.

Examination showed a profoundly hypotonic, cushingoid female child. She was relatively uninterested in her surroundings, seeming to have difficulty focusing on objects and exhibiting rolling eye movements. She lay in a frog-like posture, the hips being easily abducted to 90° with no resistance. She had a very weak grasp of objects placed in her hand. On arm traction there was gross head lag (Fig. 1a) and on ventral suspen-