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Outbreak of Coxsackie B4 arthritis among newborns and staff of a neonatal unit

SUMMARY We investigated an outbreak of Coxsackie B4 arthritis in a neonatal unit involving 20 neonates and 12 staff members, over an eight-month period. Laboratory investigations, serology tests, indicate that the outbreak was caused by Coxsackie B4 virus. Contamination of one of the overhead water reservoirs, supplying the nursery, was responsible. After the water tanks were cleaned out, no new cases were reported over five years.

Staphylococcus aureus[1], candida[2] and klebsella[3] arthritis in neonates have been reported. Septic arthritis with methicillin-resistant *S. aureus* has been reported from India.[4] Coxsackie arthritis has been reported in adults.[5–7] Outbreak of arthritis due to Coxsackie B4, involving neonates and staff of the neonatal unit, is rare.

Case report

Neonates

The first neonate to be affected was a 32-week preterm weighing 1.32 kg at birth. He was 30 days old when he started crying excessively, had fever, refused to feed, and was not moving his right lower limb. A day later, swelling and redness was noticed over the right hip joint, and there was tenderness on palpation. Septic arthritis was diagnosed. Blood culture was negative. Arthrotomy was suggestive of infection, but the synovial-fluid culture was sterile. The baby received antibiotics including Cloxacillin for 20 days. He became asymptomatic in a few days.

Twenty cases were diagnosed as having arthritis over the next seven months (Table 1). The hip, knee and ankle joints were involved, and more than one joint was involved in the majority. Amphoteracin B and vancomycin was administered to most. Parents of one neonate declined antibiotics. This child also recovered without residue. None died from the disease.

Adults

The first staff member was affected three months later (when 11 neonates had already been affected). He initially had painful movement, especially when climbing stairs and walking briskly. This was followed by a mild swelling of the right knee joint. There was no redness of the overlying skin. He had no systemic symptoms like fever, rash, sore throat or malaise. Over the next four months, 12 staff (nine doctors and three nurses) developed arthritis without systemic complaints. All took anti-inflammatory medication. None received antibiotics. One doctor had a meniscus tear that was repaired arthoscopically. This may not have been directly related to the outbreak.

Control measures

An independent investigation into the outbreak was carried out from 24 February 1998 with the help of an epidemiologist and microbiologist from the National Institute of Communicable disease (NICD). The regular nursery was closed for three weeks from 10 November 1997. All the infants admitted at that time were transferred to a makeshift nursery. The regular nursery was again operational on 2 December 1997 after thorough cleaning with detergents and fumigation with 40% formaldehyde.

Bacterial culture of nine blood samples from staff members and two from affected babies, tested at the NICD, were negative. Of the nine stool samples cultured from affected babies, one grew poliovirus. (Oral polio vaccine is administered in term babies soon after birth.)

Sera were tested in 12 affected adults and nine contacts. Cytomegalovirus and Rubella virus were negative in adult cases. Coxsackie B4 virus antibody was raised in all the affected but none of the contacts. Repeat samples were taken from three, and one of these had three samples collected. The person was not affected with arthritis when the first sample was drawn. She developed arthritis a few days afterwards. Acute sera and post-convalescent samples were collected from her. The first sample showed low titres. There was increase in titres in the acute and post-convalescent samples. Of the two others who had post-convalescent samples taken, one had an eight-fold

Table 1 Month-wise and birth weight-wise attack rates of arthritis among infants admitted to the nursery, September 1997–February 1998

		Nursery admissions			*Cases*			*Attack rate(%)*		
Series no.	*Month*	*LBW/PT*	*NBW/Term*	*Total*	*LBW/PT*	*NBW/Term*	*Total*	*LBW/PT*	*NBW/PT*	*Total*
(1)	August 1997	60	98	158	1	0	1	1.6	0	0.6
(2)	September 1997	51	140	191	2	0	2	3.9	0	1.04
(3)	October 1997	44	123	167	3	1	4	6.8	0.8	2.39
(4)	November 1997	34	91	125	4	0	4	11.76	0	3.2
(5)	December 1997	32	48	80	2	1	3	6.25	2.0	3.75
(6)	January 1998	52	91	143	1	0	1	1.92	0	0.7
(7)	February 1998	29	87	116	3	2	5	10.3	2.2	4.3
	Total	302	678	980	16	4	20	5.2	0.6	2.0

NA, not available; LBW, low birth weight; PT, preterm; NBW, normal birth weight

decline in levels. The person who developed arthritis after the first sample was collected had convalescent serum tested for Parvo-virus serology in another reference laboratory, and this was negative.

Water samples were collected from the nursery on three occasions spread over 20 days. The sample collected from the pantry of the nursery was unsatisfactory showing gross contamination with Pseudomonas and Coliform organisms. Sample from two other areas of the hospital were found to be excellent.

Discussion

This investigation was set up after a number of cases had already developed arthritis. By then, many of the neonates had been discharged. This investigation was heavily dependent on the adult staff for serology tests.

Antibiotics and antifungal medication was given to the neonates. However, one infant whose parents declined treatment recovered without antibiotics. None of the adults received antibiotics. This militates against a bacterial or fungal aetiology.

The acute and convalescent serum sample, in one case, gave a clue to the aetiology of the outbreak. The first sample from this case was collected as a control on 24 February 1998. She developed arthritis on 12 March 1998 and the second sample was collected from her on 19 March 1998. Micro-neutralization test showed a fourfold rise in antibody titre for Coxsackie B4 virus in the second sample, suggesting recent exposure to this virus.[8] The presence of antibodies to Coxsackie B4 virus in high titres in all the staff affected and the absence of antibodies in the controls, the fourfold rise in paired sera samples in the one case who subsequently developed arthritis, and the declining titre in the post-convalescent phase of another, strongly suggest that the aetiology was probably Coxsackie B4 virus. It is a matter of conjectural that the aetiological agent in the neonates affected concurrently was the same.

Coxsackie B4 is an entero-virus. The presence of coliforms in one tap sample suggests that this may have been the source of the problem. Water from the contaminated tap was used to clean the cup and spoon used to feed the neonates, and was also used by the staff for drinking purposes. After the contaminated tank was cleaned out and a protocol for maintenance cleaning was set up, no further cases of arthritis were reported over five years of surveillance.

There are several reports of newborn nursery outbreaks of non-polio enterovirus infection. Most outbreaks have been due to echovirus 11 or to group B Coxsackie virus serotypes 1–5. The source of infection to infants has sometimes been vertical transmission from their mothers.[9] Adenovirus has been previously reported to have affected both neonates and staff in a nursery.[10]

It is suggested that to the prevent the occurrence of such outbreaks in future, the quality of water supplied to the nursery is required to be monitored regularly.

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Penetrating abdominal injuries in a developing country

Introduction

In industrialized countries, injury is the most common cause of deaths and permanent disability in those aged 40 years and below.[1]

This is becoming common in developing countries such as Nigeria.[1] The high incidence of infective pathology in tropical nations seems to overshadow the importance of trauma.[2] The incidence of accidental injury is progressively increasing in our environment due to increasing density and speed of road traffic.[2,3]

Violence and acts of civil strife are becoming a problem in many Nigerian cites.[4,5]

Materials and methods

Between January 1988 and December 2002, the records of all patients aged 15 years and above, managed for