
An outbreak of echovirus 11 in a children's home

E. SOMEKH^{1*}, T. SHOHAT², R. HANDSHER³ AND F. SEROUR⁴

¹ *The Pediatric Infectious Diseases Unit, The Edith Wolfson Medical Center, Holon, PO Box 5 Holon 58100, Israel*

² *The Tel Aviv Health District, Ministry of Health, 12 Haarbaha Street, Tel Aviv, Israel*

³ *The Central Viral Laboratory, The Chaim Sheba Medical Center, Tel Hashomer, Ramat Gan, Israel*

⁴ *The Division of Pediatric Surgery, The Edith Wolfson Medical Center, PO Box 5 Holon 58100, Israel (affiliated to the Sackler School of Medicine, Tel Aviv University, Tel Aviv, Israel)*

(Accepted 13 December 2000)

SUMMARY

An outbreak of echovirus 11 infection was observed in a children's home that housed 16 children. Nine children younger than 1 year shared a large room on the first floor, which contained a large basin. Three of them presented with aseptic meningitis with CSF and stool samples positive for echovirus 11. The other six infants who shared the room were asymptomatic but their stools were positive for echovirus 11. Seven infants aged 1–2 years stayed on the second floor and were asymptomatic. One of them had positive stool culture for echovirus 11. No virus was isolated from stool samples taken from the 26 staff members. However, serology was suggestive for recent echovirus 11 infection in seven asymptomatic staff members. All seven worked either exclusively on the first floor or alternately on both floors. Our survey demonstrated that echovirus 11 may spread very efficiently in children's homes. The rate of meningitis in the infected infants was 30% while all the recently infected adults were asymptomatic.

INTRODUCTION

Echovirus 11 is a common serotype among the 30 serotypes of this group of enteroviruses in the Picornaviridae family and causes a wide range of symptoms, including aseptic meningitis [1, 2], and outbreaks of serious systemic illness in neonatal units [1, 3–6]. However, outbreaks in children's homes for older infants have not been recorded.

In August 1999, three infants from the same children's home were admitted with aseptic meningitis. We carried out an epidemiological investigation in order to determine the extent of the epidemic in this children's home including asymptomatic

infection in both children and members of the staff, and the risk factors for infection by the virus.

METHODS

The affected children's home housed infants who were candidates for adoption until completion of all the legal procedures of adoption. Typically, infants were admitted to this home for social reasons after leaving the nursery, mainly because of inadequacy of the biological mother to care for her baby.

Sixteen children were housed at the children's home at time of the outbreak. Their ages ranged from 1·5 month to 2 years. Nine children younger than 1 year

* Author for correspondence.

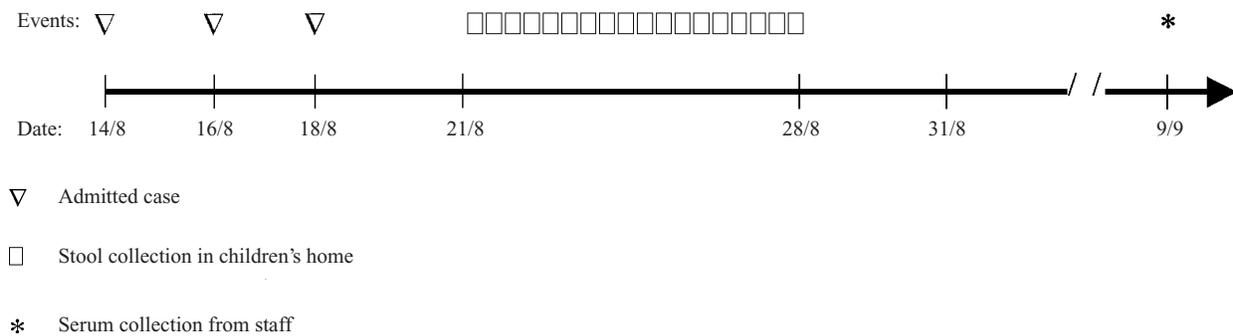


Fig. 1. Temporal data of echovirus 11 outbreak in a children's home.

(5 females and 4 males) shared a large room sized 6×13.5 metres on the first floor. This room, which contained a large basin, was used by the infants for sleeping and bathing. Seven older children (5 males and 2 females, aged 1–2 years) slept in two smaller rooms on the second floor. A third room was used for playing and bathing. The children were examined routinely by a paediatrician at least once a week and all medical details were recorded.

The staff members included 26 women aged 37–60 years (mean 49 years). Ten of them worked exclusively on the first floor, six exclusively on the second floor, and the other ten worked alternately on both floors. Rooms were cleaned at least once daily. The food was prepared in a single kitchen and babies did not share food or bottles. All employees were instructed to wash their hands before and after taking care of infants.

Specimens collected for viral cultures included cerebrospinal fluid (CSF) from the hospitalized patients, and stool specimens from all children regardless of their medical condition. Both stool and serum specimens were collected from all members of staff. Stool specimens from the asymptomatic children and from the staff members were collected in the week that followed the admission of the third infant with aseptic meningitis, and serum samples were obtained 3 weeks later (Fig. 1). For virus isolation, three different cell lines were used: human rhabdomyosarcoma cells (RD), buffalo green monkey cell line (BGM), and a local human kidney cell line (HuKi). All three cell lines were sensitive to the recent prevalent echovirus 11.

The first isolates were identified by performing microneutralization assays, as described by Egberston and Mayo [7] using enterovirus antisera pools [from National Institute of Public Health and the Environment (RIVM), Bilthoven, The Netherlands]. The rest of the isolates were identified by monovalent

specific antisera to echovirus 11 (NIH, Bethesda, Maryland).

Neutralizing antibodies to echovirus 11 were measured in a microneutralization test as described previously [8] using a strain of echovirus 11 isolated from one of the patients. A titre ≥ 64 was considered as suggestive of recent infection. This cut-off point has been based on a serological survey performed by one of us (R. H.) on 250 individuals during a non-epidemic period (1998), in which the highest titre was 16 (Central Viral Laboratory, Israel, unpublished data).

RESULTS

Three infants (2 females and 1 male), aged 1.5 months, 1.5 months, and 6 months were hospitalized with aseptic meningitis. They were admitted on 14, 16 and 18 August 1999, respectively (Fig. 1). CSF cell count of these patients ranged from 300 to $700/\mu\text{l}$ (neutrophil differential count ranged from 70 to 90%, and lymphocyte differential count from 10 to 30% of total white cell count). CSF glucose concentration ranged from 48 to 58 mg/dl and protein concentration ranged from 32 to 50 mg/dl. Bacterial cultures of the CSF and blood of these three patients were all negative. Viral cultures of CSF and stool specimens from the hospitalized patients yielded echovirus 11. All three patients recovered without any neurological sequelae. Stool samples taken from seven other children were positive for echovirus 11; all were asymptomatic. Six of the seven infants resided on the first floor (in addition to the three hospitalized patients that also slept at the first floor), and their ages ranged from 2 to 10 months. The other patient, a 1.5-year-old child, was the only second-floor patient with echovirus 11 in stool culture. The age of the hospitalized patients did not differ significantly from that of non-hospitalized children.

No viruses were isolated from stool samples taken from the staff members. However, serology was suggestive of recent echovirus infection in seven (27%) (neutralizing antibody titres ranged from 64 to 1024). All were asymptomatic. All seven employees worked either exclusively on the first floor ($n = 3$) or alternately on both floors ($n = 4$). None of the six employees who worked exclusively on the second floor had serological evidence of recent echovirus 11 infection.

DISCUSSION

Enterovirus outbreaks in children's homes due to serotypes other than echovirus 11 have been described [9–14]. Serotypes involved in these outbreaks included echovirus 25 that caused a skin rash in 15 of 22 infants, some of them also had mild enteric and respiratory symptoms [9]. Mixed infection with Coxsackie B3 and B4 that affected 74 of 120 children, manifested with moderately severe upper respiratory symptoms [10]. Another outbreak caused by Coxsackie B3, involved 81% of the children accommodated in a welfare home and was manifested with febrile illness and pharyngitis [11]. Coxsackie B2, Coxsackie B5 and echovirus 26 were also identified in outbreaks involving children's homes [12–14]. However, our report is the first description of an outbreak in children's home due to echovirus 11 serotype.

The outbreak described herein has several common features of infection caused by enteroviruses. First, the transmission of the virus was very efficient in a setting of young infants under 1 year of age residing in a single room and sharing the same bath. In this room, the infectivity rate was 100% (9 out of 9 infants). The common modes of transmission of enteroviruses is faecal–oral and respiratory spread. Even though the source of the outbreak had not been identified, the infected infants could have acquired the virus from an infected asymptomatic staff member via hands, or through direct baby-to-baby transmission. However, since some of the infected staff members worked also in the second floor where the infection was much less efficient (1 out of 7 infants was infected), it would appear that the staff were infected from the children. The source of infection of the infants on the first floor could have been the common basin. Indeed, it has been reported that exposure to a common source of water such as a swimming pool, may lead to enterovirus infection [15, 16].

A high infectivity rate of other enteroviruses among infants in children's homes, reaching up to 81%, has been reported [9–14]. The effective viral spread in these circumstances has even triggered the speculation that the virus might have been spread by airborne transmission [1]. The rate of meningitis among the infected infants was high (30%), but the disease was self-limiting and the affected infants recovered completely. In contrast to some reports [1], there was no male preponderance among our patients with aseptic meningitis.

In conclusion, our survey demonstrated that echovirus 11 may spread efficiently in a children's homes especially among infants being bathed in a common basin. The rate of meningitis in the infected infants was 30% while all the adults with serological markers of recent infection were asymptomatic. These results further emphasize that simple hygienic measures such as hand washing and care of common baths and basins may prevent enteroviral outbreaks in institutions where infants are being cared for.

REFERENCES

1. Sawyer MH. Enterovirus infection: diagnosis and treatment. *Pediatr Infect Dis J* 1999; **18**: 1033–40.
2. Berry PJ, Nagington J. Fatal infection with echovirus 11. *Arch Dis Child* 1982; **57**: 22–9.
3. Modlin JF. Fatal echovirus 11 disease in premature neonates. *Pediatrics* 1980; **66**: 775–80.
4. Isaacs D, Dobson SR, Wilkinson AR, Hope PL, Eglin R, Moxon ER. Conservative management of an echovirus 11 outbreak in a neonatal unit. *Lancet* 1989; **i**: 543–5.
5. Kinney JS, McCray E, Kaplan JE, et al. Risk factors associated with echovirus 11 infection in a hospital nursery. *Pediatr Infect Dis* 1986; **5**: 192–7.
6. Nagington J, Gandy G, Walker J, Gray JJ. Use of normal immunoglobulin in an echovirus 11 outbreak in a special-care baby unit. *Lancet* 1983; **ii**: 443–6.
7. Egbertson SH, Mayo DR. A microneutralization test for the identification of enterovirus isolates. *J Virol Methods* 1986; **14**: 305–7.
8. Birenbaum E, Handsher R, Kuint J, et al. Echovirus type 22 outbreak associated with gastro-intestinal disease in a neonatal intensive care unit. *Am J Perinatol* 1997; **14**: 469–73.
9. Guidotti MB. An outbreak of skin rash by echovirus 25 in an infant home. *J InfectM* 1983; **6**: 67–70.
10. Hierholzer JC, Mostow SR, Dowdle WR. Prospective study of a mixed coxsackie virus B3 and B4 outbreak of upper respiratory illness in a children's home. *Pediatrics* 1972; **49**: 744–52.
11. Nakayama T, Urano T, Osano M, et al. Outbreak of herpangina associated with Coxsackie virus B3 infection. *Pediatr Infect Dis J* 1989; **8**: 495–8.

12. Carmichael J, McGuckin R, Gardner PS. Outbreak of Coxsackie type B2 virus in a children's home in Newcastle upon Tyne. *BMJ* 1968; **2**: 532–3.
13. Sanders DY, Powell RV, Smith A. Outbreak of Coxsackie B5 virus in a children's home. *South Med J* 1969; **62**: 474–6.
14. Moritsugu Y, Sawada K, Hinohara M, et al. An outbreak of type 26 echovirus infection with exanthem in an infant home near Tokyo. *Am J Epidemiol* 1968; **87**: 599–608.
15. Keswick BH, Gerba CP, Goyal SM. Occurrence of enterovirus in community swimming pools. *Am J Publ Hlth* 1981; **71**: 1026–30.
16. Melnick JL, Gerba CP. The ecology of enteroviruses in natural waters. *CRC Crit Rev Environ Control* 1980; **10**: 65–93.