Surveillance of congenital cytomegalovirus in the UK and Ireland

Claire L Townsend,1 Catherine S Peckham,1 Pat A Tookey1

ABSTRACT

Objective To explore the presentation and management of congenital cytomegalovirus (CMV) identified through routine clinical investigations, and ascertain outcome in early childhood.

Design Active population-based surveillance.

Setting UK and Ireland.

Methods Infants born in 2001–2002 with confirmed or suspected congenital CMV infection were reported through the British Paediatric Surveillance Unit, and clinicians completed questionnaires on presentation, diagnosis, management and subsequent outcome.

Results 86 confirmed and 70 possible cases of congenital CMV infection were reported. Over a third (27/72) of singleton infants with confirmed and 44% (27/61) with possible congenital infection were preterm (<37 weeks gestation). Among confirmed cases, 75% (64/85) presented with neonatal manifestations compatible with congenital CMV, over half (34/64) of whom had neurological signs; 17 infants were treated with gancyclovir. Among confirmed cases with information on outcome, 31% (24/78) were developing normally, 18% (14/78) had mild, 24% (19/78) moderate and 14% (11/78) severe sequelae, and 13% (10/78) had died. Median age at follow-up among survivors was 18 months (IQR 15–22 months). Children with neonatal CMV manifestations were significantly more likely than those without to have moderate or severe outcomes (including death) (60%, 36/60, vs 22%, 4/18, p=0.001). 27% of survivors (17/63) had bilateral hearing loss.

Conclusions The number of confirmed cases of diagnosed congenital CMV reported in this study was lower than expected, highlighting the need for early and appropriate investigations when congenital infection is suspected. Due to the unexpectedly high proportion of preterm infants, resulting from differential case ascertainment, it was difficult to distinguish prematurity and CMV-related symptoms.

INTRODUCTION

Cytomegalovirus (CMV) is the most common congenital infection worldwide. Seroprevalence of CMV in women of childbearing age in the UK is around 50%, but varies by ethnicity, social class and parity.1 Congenital CMV infection can result from recurrent (reactivation of latent infection or reinfection with a new strain) or primary maternal infection in pregnancy.2–3 The birth prevalence of congenital CMV in resource-rich countries varies according to maternal seroprevalence, and in the UK is estimated to be around 3 per 1000.4–5 About 10–15% of congenitally infected infants are symptomatic at birth,6 and most of these experience serious long-term complications, such as cerebral palsy, cognitive impairment and sensorineural hearing loss.5–8 In contrast, most asymptomatic infants develop normally, although a minority (10–15%) develop long-term sequelae, sensorineural deafness being the most common problem.6–9 There is some evidence that intravenous gancyclovir treatment may improve hearing outcomes in severely affected infants;10–11 however, due to concerns about toxicity, treatment is currently restricted to infants with central nervous system involvement.12

CMV is also commonly acquired in infancy, mainly through breastfeeding: in one UK study, 12% of infants born to CMV seropositive mothers acquired infection by 3 months, and 20% by 1 year.13 Although postnatal acquisition is usually asymptomatic in term infants, symptomatic infection can occur in very preterm infants.14 Congenital infection is conventionally distinguished from postnatal acquisition by virus isolation or DNA from urine or tissue samples collected in the first 2–3 weeks of life.15 For infants tested after this time, it is often not possible to distinguish between congenital and acquired infection, although testing stored neonatal dried blood spots can be helpful, with test sensitivity ranging from 71% to 100%.16

Surveillance of congenital CMV was carried out in the UK and Ireland, between 2001 and 2003, to ascertain the population prevalence of diagnosed
congenital CMV disease, management strategies and clinical disease outcome.  

METHODS
Data collection
The study was carried out through the British Paediatric Surveillance Unit (BPSU) of the Royal College of Paediatrics and Child Health, an active monthly surveillance system for rare conditions of childhood. 16, 19 Paediatricians were asked to report all infants with confirmed or suspected congenital CMV infection born in the UK or Ireland in 2001 or 2002. Confirmed cases were infants with congenital CMV infection determined by PCR or virus isolation from urine, blood, saliva or tissue taken at biopsy within 3 weeks of birth; suspected cases were infants with symptoms compatible with congenital CMV infection aged under 12 months, with CMV isolated after 3 weeks of age. Since there was no national screening programme, it was not anticipated that asymptomatic infants or those with non-specific symptoms at birth would be reported. Paediatricians completed standard questionnaires on diagnosis, demographic and clinical factors, maternal details, treatment and outcome; at least two reminders were sent to non-responders. Follow-up information was requested during the second or third year of life to establish outcome. All diagnoses and investigations were undertaken locally as part of routine care.

Case definition
For this analysis, congenital CMV infection was defined as ‘confirmed’ based on a positive PCR, virus isolation or detection of early antigen fluorescent foci on a urine, blood, saliva or tissue sample taken at <21 days of age. 6 Infants reported with only a positive IgM result during this period were classified as ‘possible’ cases, as were infants with earliest positive CMV results on samples taken at 21 days or later. Infants with laboratory evidence of postnatal CMV acquisition were excluded.

Variables
In this paper we describe infants as symptomatic if they had one or more of the following neonatal manifestations compatible with congenital CMV: hepatomegaly, splenomegaly, petechiae, thrombocytopenia, chorioretinitis, seizures, intracranial calcification or microcephaly (the latter four were also classified as neurological signs). The remaining infants are described as asymptomatic, even if they had other non-specific signs. Microcephaly and small for gestational age were based on age- and sex-specific z-scores derived from British 1990 population standards using the LMS method, 20 21 and defined as an occipital-frontal circumference or birth weight below the third centile; where these measurements were missing, classification was based on clinical report. Subsequent outcome was categorised according to the presence and severity of reported sequelae, including hearing or visual impairment, seizures, motor or mental impairment or developmental delay. A summary measure of final outcome for surviving children was derived following review of all available information provided by clinicians, and classified by the researchers as normal, or mild, moderate or severe impairment (see table 1).

Data management and statistical analysis
Data were managed in Access 2003 (Microsoft, Redmond, Washington, USA) and analysed using Stata v 11 (StataCorp LP, College Station, Texas, USA). Differences in proportions were compared using χ 2 or Fisher’s exact tests. The annual number of live births in England and Wales was obtained from Office for National Statistics data. 22

RESULTS
Altogether, 224 paediatricians made 290 reports; 86 infants (born to 81 women) had confirmed congenital CMV, and 70 were possible cases. Fifty-eight infants were reported in error (most born outside the study period (n=31) or with postnatally acquired CMV infection). The remaining reports were duplicates (n=55) or could not be assessed due to non-response (n=21). Reports of confirmed and possible cases came from all parts of the British Isles, with about a fifth from the London area, and over 60% from elsewhere in England (table 2). Seventy-two confirmed reports were from England and Wales (58 in 2001, 54 in 2002), giving an annual prevalence of 0.06 per 1000 live births (38/594 634 in 2001, 34/596 122 in 2002 22).

Maternal and infant characteristics
Demographic characteristics were similar for confirmed and possible cases (table 2), apart from the proportion of twins: 15% of confirmed cases (including five twin pairs), and 7% of possible cases (no pairs) were twins. Median maternal age was 28 years (IQR 21–32 years for confirmed cases, n=81 mothers; IQR 23–33 years for possible cases, n=64 mothers). Among the confirmed singleton cases, 37% (27/72) were preterm (<37 weeks gestation), including 15% (11/72) born at <32 weeks; all 13 twins were preterm, with six born at <32 weeks (including two twin pairs). Among possible cases, 44% (27/61) of singletons and all five twins were preterm; a third (21/61) of singletons and two twins were born at <32 weeks.

Confirmed cases of congenital CMV
Diagnosis and presentation
Among infants with confirmed congenital CMV, the first available positive test was usually PCR or virus isolation; 88% were performed on urine samples (76/86), and 91% (78/86) at <7 days of age (table 3).

Three quarters of infants had neonatal symptoms, over half with neurological involvement (table 3). Of 21 asymptomatic neonates, four had ultrasound abnormalities reported, and the mothers of seven had symptoms suggestive of CMV in pregnancy, including six with confirmed seroconversion. Other specific indications for neonatal testing were reported for

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Classification of infant outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>No hearing impairments or other impairments reported</td>
</tr>
<tr>
<td>Mild impairment</td>
<td>Unilateral hearing loss and/or other mild sequelae</td>
</tr>
<tr>
<td>Examples</td>
<td>Unilateral SNHL and mild speech delay</td>
</tr>
<tr>
<td></td>
<td>Congenital heart defect repaired, mild delay</td>
</tr>
<tr>
<td></td>
<td>Mild language/motor/developmental delay</td>
</tr>
<tr>
<td>Moderate impairment</td>
<td>Bilateral hearing loss and/or other moderate sequelae</td>
</tr>
<tr>
<td>Examples</td>
<td>Profound hearing loss</td>
</tr>
<tr>
<td></td>
<td>Developmental delay and visual problems</td>
</tr>
<tr>
<td></td>
<td>Poor vision, unilateral SNHL, mild developmental delay</td>
</tr>
<tr>
<td>Severe impairment</td>
<td>Multiple disabilities</td>
</tr>
<tr>
<td>Examples</td>
<td>Cerebral palsy, severe learning difficulties, bilateral SNHL</td>
</tr>
<tr>
<td></td>
<td>Bilateral SNHL, global delay</td>
</tr>
<tr>
<td></td>
<td>Quadriplegia, blindness, SNHL</td>
</tr>
</tbody>
</table>

SNHL, sensorineural hearing loss.
eight additional infants without CMV symptoms or maternal indications (table 3). Ultrasound abnormalities or maternal symptoms were also reported for 27% (17/64) of symptomatic infants. In addition to signs classified as CMV compatible, many infants had other non-specific signs, for example, jaundice or anaemia. There were no statistically significant differences in neonatal presentation between term and preterm infants: 69% (31/45) of term infants were symptomatic, and 54% (12/22) of infants born at 32–36 weeks and 62% (24/39) of those born at <32 weeks had moderate or severe outcomes according to gestational age: 52% (22/42) of preterm infants (33/40, p=0.11); 42% (19/45) and 37% (19/54) of those born at 32–36 weeks and >36 weeks, respectively, had neurological signs (p=0.006). Hearing problems were significantly more common in children with congenital CMV infection born in 2001–2002 (44%) than in those born at >36 weeks (22%) (p=0.001) and the rate was highest (44%) if neurological signs were present neonatally (table 5). Among surviving infants, 79% (11/14) of those who received gancyclovir had moderate or severe impairment, as did 35% (19/54) of those who did not (p=0.006).

Outcome

Follow-up information was provided for 89% (70/78) of children alive at first report. Two additional deaths were reported, both under 12 months of age (table 4); the median age of surviving children when last seen was 17.7 months (IQR 14.9–21.8 months; range 4.2–33.9 months). Among 78 children with known outcome, 31% (24/78) were developing normally, 18% (14/78) had mild, 24% (19/78) moderate and 14% (11/78) severe sequelae, and 15% (10/78) had died. There were no significant differences in the proportion of children with moderate or worse outcomes according to gestational age: 52% (22/42) of term infants, 54% (12/22) of infants born at 32–36 weeks and 37% (6/16) of those born <32 weeks had moderate or severe outcomes or died (p=0.55). Symptomatic infants were significantly more likely than others to have moderate or worse outcomes if neurological signs were present neonatally (table 5). Among surviving infants, 79% (11/14) of those who received gancyclovir had moderate or severe impairment, as did 35% (19/54) of those who did not (p=0.006). Hearing problems were more common in children with neonatal symptoms (table 5), although this was not statistically significant (p=0.09).

Eighty-two children had bilateral sensorineural hearing impairment, 79% (65/82) of those with confirmed congenital CMV infection born in 2001–2002 died within 8 weeks of birth; four had severe complications of prematurity and one a congenital syndrome (table 4). Four surviving children had congenital abnormalities: cleft palate, positional talipes equinovarus, imperforate anus and cleft lip, and atrial septal defect.

Gancyclovir treatment

Seventeen infants with confirmed infection received intravenous gancyclovir, for a median duration of 6 weeks (range 4 days to 3 months); all but one had neonatal symptoms, including 11 with neurological signs (table 5). Treatment was discontinued for three infants due to neutropaenia (after 4 days), thrombocytopenia (after 4 weeks) or abnormal liver function tests (after 5 weeks). Six infants with possible congenital CMV also received gancyclovir; all presented after 1 month of age with either bronchiolitis or pneumonitis.

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**DISCUSSION**

This observational surveillance study highlighted important issues relating to ascertainment of congenital CMV infection. We sought notification of infants with confirmed or suspected congenital CMV identified through routine investigations, and expected reports of infants presenting with manifestations of CMV disease. However, infants without typical CMV symptoms were also reported, and almost half of confirmed and suspected cases were preterm. In a similar Australian surveillance study, only 9% of reported infants were preterm (<36 weeks),23 and congenital CMV is not an established risk factor for preterm birth.24 This excess of preterm infants could be due to differential case ascertainment arising from opportunistic screening policies; indeed, one respondent reported a local CMV screening policy for babies admitted to the special care unit. Case classification was particularly challenging, as in preterm infants it was difficult to distinguish symptoms compatible with CMV from those of prematurity. Furthermore, test results were sometimes inadequate and/or too late to distinguish congenital from postnatal infection. Symptomatic CMV disease following postnatal infection is more likely in low birthweight infants,14 and could have contributed to the high proportion of preterm infants among the possible cases. However, the natural history of early postnatal CMV acquisition remains poorly understood.25

**Table 5** Treatment and clinical outcome according to neonatal presentation

<table>
<thead>
<tr>
<th>Neuronal manifestations compatible with congenital cytomegalovirus</th>
<th>Total</th>
<th>No</th>
<th>Yes, non-neuro</th>
<th>Yes, neuro</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment (n=85)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>68 (80)</td>
<td>20 (95)</td>
<td>25 (83)</td>
<td>23 (68)</td>
</tr>
<tr>
<td>Yes</td>
<td>17 (20)</td>
<td>1 (5)</td>
<td>5 (17)</td>
<td>11 (32)</td>
</tr>
<tr>
<td>Summary outcome (n=78)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>24 (31)</td>
<td>13 (72)</td>
<td>6 (21)</td>
<td>5 (16)</td>
</tr>
<tr>
<td>Mild</td>
<td>14 (18)</td>
<td>1 (6)</td>
<td>8 (29)</td>
<td>5 (16)</td>
</tr>
<tr>
<td>Moderate/severe/died</td>
<td>40 (51)</td>
<td>4 (22)</td>
<td>14 (50)</td>
<td>22 (69)</td>
</tr>
<tr>
<td>Hearing (n=63)*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>34 (54)</td>
<td>13 (76)</td>
<td>10 (40)</td>
<td>11 (52)</td>
</tr>
<tr>
<td>Unilateral/mixed hearing loss</td>
<td>12 (19)</td>
<td>1 (6)</td>
<td>8 (32)</td>
<td>3 (14)</td>
</tr>
<tr>
<td>Bilateral sensorineural hearing loss</td>
<td>17 (27)</td>
<td>31 (18)</td>
<td>7 (28)</td>
<td>7 (33)</td>
</tr>
</tbody>
</table>

*Excludes 10 children who died.
†All term infants, one small for gestational age, one borderline microcephalic (4th centile).
Neuro, neurological symptoms.

Possible congenital CMV

Among 70 infants with possible congenital CMV, three had unconfirmed positive IgM results in the first 3 weeks of life. First available CMV test samples were taken at a median age of 65 days (range 0–264 days; IQR 35–107 days). A third (24/70) of these infants had neonatal symptoms compatible with congenital CMV, over half (13/24) of whom had neurological signs. Eleven infants died, at median age of 2.7 months (range 13 days to 9.5 months); nine were very preterm (<32 weeks, including six <28 weeks), one was reported as a sudden infant death and one died of respiratory and cardiac failure. Among surviving children with outcome information available, over half (32/56) were developing normally and 23% (13/56) had moderate or severe outcomes. Median age at follow-up was 20.4 months (IQR 11.4–28.1 months).
This study was carried out through the BPSU, a well-established active surveillance system, relying on clinical diagnosis through routine investigations. Overall, we identified 86 infants with confirmed congenital CMV born in 2001–2002, a reported prevalence of diagnosed congenital CMV (0.06 per 1000 live births) more than sixfold lower than expected if actual prevalence was 3 per 1000 births, with 10–15% symptomatic at birth. This low case ascertainment is probably due to the non-specific clinical presentation of congenital CMV, and the delay in taking appropriate samples early in life, as well as to under-reporting.

A further 70 infants suspected by clinicians of having congenital infection lacked confirmatory tests in the first 3 weeks of life and were classified as possible cases. This group is likely to have included both infants with congenital infection and those with early-acquired postnatal infection. Although TORCH screening (for toxoplasmosis, rubella, CMV and herpes simplex virus) has been discouraged over the last 20 years, this was specified in some reports with CMV and herpes simplex virus) has been discouraged over the last 20 years, this was specified in some reports with CMV and herpes simplex virus. Although TORCH screening (for toxoplasmosis, rubella, CMV and herpes simplex virus) has been discouraged over the last 20 years, this was specified in some reports with CMV and herpes simplex virus) has been discouraged over the last 20 years, this was specified in some reports with CMV and herpes simplex virus. However, we could not assess the extent to which neonatal dried blood spots can also assist in the retrospective diagnosis of congenital CMV, but sensitivity can vary. In an exercise reported elsewhere, only 75% of blood spots retrieved for confirmed cases in this study were positive.

Almost half of the children in this study had serious disabling conditions or died, and almost a third of survivors had bilateral sensorineural hearing loss at a median follow-up age of 18 months. Despite study differences in definitions and case ascertainment, outcomes were similar to those reported elsewhere. For example, among children with neonatal symptoms, 32% had bilateral hearing loss, consistent with rates of 30–40% reported in the literature. Cases of late-onset hearing loss, which have been reported in other studies, could have been missed in this study due to the limited duration of follow-up. The proportion of symptomatic neonates with moderate or severe outcomes (about half) was comparable to the estimated 40–58% reported in a recent meta-analysis. However, we could not assess the extent to which neonatal manifestations or reported sequelae were affected by complications of prematurity or other underlying conditions. We were also unable to assess the effectiveness of ganciclovir treatment due to the lack of controls and selective use of treatment.

The children reported to this study were a subset of those born with congenital CMV in 2001 and 2002, and did not include those who were asymptomatic in infancy but developed late-onset sequelae. The true burden of disease associated with congenital CMV remains unknown. Nevertheless, the poor outcomes observed highlight the need for safe and effective treatment for congenitally infected infants. In light of recent advances in antiviral therapy for CMV, there is an urgent need to identify those children most likely to benefit from treatment in the future.

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Competing interests None.

Ethics approval Ethics approval for this study was granted by the Great Ormond Street Hospital for Children NHS Trust/Institute of Child Health Ethics Committee.

Provenance and peer review Not commissioned; externally peer reviewed.

Contributors CT carried out the statistical analyses and drafted the paper. All authors contributed to the interpretation of the study, commented on all drafts of the paper and approved the final version.

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